Pocket Guide to Diagnostic Tests

- Includes over 350 tests
- Answers questions on-the-spot

Diana Nicoll
Stephen J. McPhee
Michael Pignone
Tony M. Chou
William M. Detmer
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Definition</th>
<th>mo</th>
<th>Month</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ab</td>
<td>Antibody</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abn</td>
<td>Abnormal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AFB</td>
<td>Acid-fast bacillus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ag</td>
<td>Antigen</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AIDS</td>
<td>Acquired immuno-deficiency syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ALT</td>
<td>Alanine aminotransferase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ANA</td>
<td>Antinuclear antibody</td>
<td></td>
<td></td>
</tr>
<tr>
<td>AST</td>
<td>Aspartate aminotransferase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CF</td>
<td>Complement fixation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CHF</td>
<td>Congestive heart failure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CIE</td>
<td>Counterimmuno-electrophoresis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CK</td>
<td>Creatine kinase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CNS</td>
<td>Central nervous system</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CSF</td>
<td>Cerebrospinal fluid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CXR</td>
<td>Chest x-ray</td>
<td></td>
<td></td>
</tr>
<tr>
<td>d</td>
<td>Day</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Diff</td>
<td>Differential cell count</td>
<td></td>
<td></td>
</tr>
<tr>
<td>EDTA</td>
<td>Ethylenediaminetetra-acetic acid (edetate)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ELISA</td>
<td>Enzyme-linked immunosorbent assay</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FT4I</td>
<td>Free thyroxine index</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GI</td>
<td>Gastrointestinal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GNR</td>
<td>Gram-negative rod</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GNCB</td>
<td>Gram-negative coccobacillus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GPC</td>
<td>Gram-positive coccus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>GVCB</td>
<td>Gram-variable coccobacillus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>h</td>
<td>Hour</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ig</td>
<td>Immunoglobulin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IM</td>
<td>Intramuscular(ly)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>Intravenous(ly)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>min</td>
<td>Minute</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MN</td>
<td>Mononuclear cell</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MRI</td>
<td>Magnetic resonance imaging</td>
<td></td>
<td></td>
</tr>
<tr>
<td>N</td>
<td>Normal</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neg</td>
<td>Negative</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCR</td>
<td>Polymerase chain reaction</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NPO</td>
<td>Nothing by mouth (nil per os)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PO</td>
<td>Orally (per os)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pos</td>
<td>Positive</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PMN</td>
<td>Polymorphonuclear neutrophil (leukocyte)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PTH</td>
<td>Parathyroid hormone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RBC</td>
<td>Red blood cell</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RPR</td>
<td>Rapid plasma reagin (syphilis test)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>s</td>
<td>Second</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SIADH</td>
<td>Syndrome of inappropriate antidiuretic hormone (secretion)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SLE</td>
<td>Systemic lupus erythematosus</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T3</td>
<td>Triiodothyronine</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T4</td>
<td>Tetraiodothyronine (thyroxine)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TSH</td>
<td>Thyroid-stimulating hormone</td>
<td></td>
<td></td>
</tr>
<tr>
<td>V</td>
<td>Variable</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VDRL</td>
<td>Venereal Disease Research Laboratory (syphilis test)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>WBC</td>
<td>White blood cell</td>
<td></td>
<td></td>
</tr>
<tr>
<td>wk</td>
<td>Week</td>
<td></td>
<td></td>
</tr>
<tr>
<td>yr</td>
<td>Year</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↑</td>
<td>Increased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↓</td>
<td>Decreased</td>
<td></td>
<td></td>
</tr>
<tr>
<td>↔</td>
<td>No change</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
# Contents

Abbreviations ........................................... Inside Front Cover

Preface .................................................. v

1. Basic Principles of Diagnostic Test Use and Interpretation ................................. 1
   Diana Nicoll, MD, PhD, MPA, and Michael Pignone, MD, MPH

2. Laboratory Procedures in the Clinical Setting ................................................. 23
   Stephen J. McPhee, MD

3. Common Laboratory Tests: Selection and Interpretation ........................................ 41
   Diana Nicoll, MD, PhD, MPA, Stephen J. McPhee, MD, and Michael Pignone, MD, MPH

4. Therapeutic Drug Monitoring: Principles and Test Interpretation ............................... 187
   Diana Nicoll, MD, PhD, MPA

5. Microbiology: Test Selection ................................................................. 195
   Mary K. York, PhD

6. Diagnostic Imaging: Test Selection and Interpretation ........................................... 243
   Sean Perini, MD, and Susan D. Wall, MD

7. Basic Electrocardiography ................................................................. 283
   G. Thomas Evans, Jr., MD

   Stephen J. McPhee, MD, Diana Nicoll, MD, PhD, MPA, and Michael Pignone, MD, MPH

Index .................................................................. 403

Quick Reference Guide ........................................ Back Cover
Associate Authors

G. Thomas Evans, Jr., MD
Associate Clinical Professor of Medicine
University of California, San Francisco
Director of Electrocardiography
Moffit-Long Hospitals, San Francisco
*Basic Electrocardiography*

Sean Perini, MD
Clinical Fellow
Section of Interventional Radiology
Department of Radiology
University of California, San Francisco
*Diagnostic Testing: Algorithms, Nomograms, and Tables*

Susan D. Wall, MD
Professor of Radiology and Assistant Chief
Department of Radiology
Veterans Affairs Medical Center, San Francisco
Associate Dean, Graduate Medical Education
University of California, San Francisco
*Diagnostic Imaging: Test Selection and Interpretation*

Mary K. York, PhD
Clinical Professor of Laboratory Medicine
University of California, San Francisco
*Microbiology: Test Selection*
Preface

Purpose

*Pocket Guide to Diagnostic Tests* is intended to serve as a pocket reference manual for medical and other health professional students, house officers, and practicing physicians. It is a quick reference guide to the selection and interpretation of commonly used diagnostic tests, including laboratory procedures in the clinical setting, laboratory tests (chemistry, hematology, and immunology), microbiology tests (bacteriology, virology, and serology), diagnostic imaging tests (plain radiography, CT, MRI, and ultrasonography), and electrocardiography.

This book will enable readers to understand commonly used diagnostic tests and diagnostic approaches to common disease states.

Outstanding Features

- Over 350 tests are presented in a concise, consistent, and readable format.
- Fields covered include internal medicine, pediatrics, general surgery, neurology, and gynecology.
- Costs and risks of various procedures and tests are emphasized.
- Literature references are included for most diagnostic tests.
- An index for quick reference is included on the back cover.

Organization

This pocket reference manual is not intended to include all diagnostic tests or disease states. Rather, the authors have selected those tests and diseases that are most common and relevant to the general practice of medicine.

The *Guide* is divided into eight sections:
1. Basic Principles of Diagnostic Test Use and Interpretation
2. Laboratory Procedures in the Clinical Setting
3. Common Laboratory Tests: Selection and Interpretation
4. Therapeutic Drug Monitoring: Principles and Test Interpretation
5. Microbiology: Test Selection
6. Diagnostic Imaging: Test Selection and Interpretation
7. Basic Electrocardiography

Intended Audience

In this era of rapidly changing medical technology, many new diagnostic tests are being introduced every year and are replacing older tests as they are shown to be more sensitive, specific, or cost-effective.
In this environment, students, house officers, and practicing physicians are looking for a pocket reference on diagnostic tests.

Medical students will find the concise summary of diagnostic laboratory, microbiologic, and imaging studies, and of electrocardiography in this pocket-sized book of great help during clinical ward rotations.

Busy house officers will find the clear organization and citations to the current literature useful in devising proper patient management.

Practitioners (internists, family physicians, pediatricians, surgeons, and other specialists who provide generalist care) may use the Guide as a refresher manual to update their understanding of laboratory tests and diagnostic approaches.

Nurses and other health practitioners will find the format and scope of the Guide valuable for understanding the use of laboratory tests in patient management.

In 1998, the contents of this book were integrated with the contents of Pocket Guide to Commonly Prescribed Drugs, 2nd ed., by Glenn N. Levine, MD, in a new CD-ROM, Current Medical Diagnosis & Treatment 1998 on CD-ROM. An updated version of the CD-ROM, including this book, will be published in 2000.

Acknowledgments

We wish to thank our associate authors for their contributions to this book. In addition, we are grateful to the many physicians, residents, and students who contributed useful suggestions and to Jim Ransom for his careful editing of the manuscript.

We welcome comments and recommendations from our readers for future editions.

Diana Nicoll, MD, PhD, MPA
Stephen J. McPhee, MD
Michael Pignone, MD, MPH
William M. Detmer, MD, MS
Tony M. Chou, MD

San Francisco
September 2000
The clinician’s main task is to make reasoned decisions about patient care despite incomplete clinical information and uncertainty about clinical outcomes. While data elicited from the history and physical examination are often sufficient for making a diagnosis or for guiding therapy, more information may be required. In these situations, clinicians often turn to diagnostic tests for help.

**BENEFITS; COSTS, AND RISKS**

When used appropriately, diagnostic tests can be of great assistance to the clinician. Tests can be helpful for **screening**, ie, to identify risk factors for disease and to detect occult disease in asymptomatic persons. Identification of risk factors may allow early intervention to prevent disease occurrence, and early detection of occult disease may reduce
disease morbidity and mortality through early treatment. Optimal screening tests meet the criteria listed in Table 1–1.

Tests can also be helpful for diagnosis, ie, to help establish or exclude the presence of disease in symptomatic persons. Some tests assist in early diagnosis after onset of symptoms and signs; others assist in differential diagnosis of various possible diseases; others help determine the stage or activity of disease.

Finally, tests can be helpful in patient management. Tests can help (1) evaluate the severity of disease, (2) estimate prognosis, (3) monitor the course of disease (progression, stability, or resolution), (4) detect disease recurrence, and (5) select drugs and adjust therapy.

When ordering diagnostic tests, clinicians should weigh the potential benefits against the potential costs and disadvantages:

1. Some tests carry a risk of morbidity or mortality—eg, cerebral angiogram leads to stroke in 1% of cases.
2. The discomfort associated with tests such as sigmoidoscopy or barium enema will deter some patients from completing a diagnostic work-up.
3. The result of a diagnostic test often has implications for further care in that a test result may mandate further testing or frequent follow-up. This means that a patient with a positive fecal occult blood test may incur significant cost, risk, and discomfort during follow-up sigmoidoscopy, barium enema, or colonoscopy.
4. A false-positive test may lead to further unnecessary testing. Classifying a healthy patient as diseased based on a falsely positive diagnostic test can cause psychologic distress and may lead to risks from unnecessary therapy.

### TABLE 1–1. CRITERIA FOR USE OF SCREENING PROCEDURES.

<table>
<thead>
<tr>
<th>Characteristics of population</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Sufficiently high prevalence of disease.</td>
</tr>
<tr>
<td>2. Likely to be compliant with subsequent tests and treatments.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Characteristics of disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Significant morbidity and mortality.</td>
</tr>
<tr>
<td>2. Effective and acceptable treatment available.</td>
</tr>
<tr>
<td>3. Presymptomatic period detectable.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Characteristics of test</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Good sensitivity and specificity.</td>
</tr>
<tr>
<td>2. Low cost and risk.</td>
</tr>
<tr>
<td>3. Confirmatory test available and practical.</td>
</tr>
</tbody>
</table>
(5) A diagnostic or screening test may identify cases of disease that would not otherwise have been recognized and that would not have affected the patient. For example, early-stage, low-grade prostate cancer detected by PSA screening in an 84-year-old man with known severe congestive heart failure will probably not become symptomatic or require treatment during his lifetime.

(6) An individual test such as MRI of the head can cost more than $1400, and diagnostic tests as a whole account for approximately one-fifth of health care expenditures in the USA.

PERFORMANCE OF DIAGNOSTIC TESTS

Factors affecting both the patient and the specimen are important. The most crucial element in a properly conducted laboratory test is an appropriate specimen.

Patient Preparation

Preparation of the patient is important for certain tests—eg, a fasting state is needed for optimal glucose and triglyceride measurements; posture and sodium intake must be strictly controlled when measuring renin and aldosterone levels; and strenuous exercise should be avoided before taking samples for creatine kinase determinations, since vigorous muscle activity can lead to falsely abnormal results.

Specimen Collection

Careful attention must be paid to patient identification and specimen labeling. Knowing when the specimen was collected may be important. For instance, aminoglycoside levels cannot be interpreted appropriately without knowing whether the specimen was drawn just before (“trough” level) or after (“peak” level) drug administration. Drug levels cannot be interpreted if they are drawn during the drug’s distribution phase (eg, digoxin levels drawn during the first 6 hours after an oral dose). Substances that have a circadian variation (eg, cortisol) can be interpreted only in the context of the time of day the sample was drawn.

During specimen collection, other principles should be remembered. Specimens should not be drawn above an intravenous line, as this may contaminate the sample with intravenous fluid. Excessive tourniquet time will lead to hemoconcentration and an increased concentration of protein-bound substances such as calcium. Lysis of cells during collection of a blood specimen will result in spuriously increased serum
levels of substances concentrated in cells (e.g., lactate dehydrogenase and potassium). Certain test specimens may require special handling or storage (e.g., blood gas specimens). Delay in delivery of specimens to the laboratory can result in ongoing cellular metabolism and therefore spurious results for some studies (e.g., low blood glucose).

**TEST CHARACTERISTICS**

Table 1–2 lists the general characteristics of useful diagnostic tests. Most of the principles detailed below can be applied not only to laboratory and radiologic tests but also to elements of the history and physical examination.

**Accuracy**

The accuracy of a laboratory test is its correspondence with the true value. An inaccurate test is one that differs from the true value even though the results may be reproducible (Figures 1–1A and 1–1B). In the clinical laboratory, accuracy of tests is maximized by calibrating laboratory equipment with reference material and by participation in external quality control programs.

**Precision**

Test precision is a measure of a test’s reproducibility when repeated on the same sample. An imprecise test is one that yields widely varying results on repeated measurements (Figure 1–1B). The precision of diagnostic tests, which is monitored in clinical laboratories by using control material, must be good enough to distinguish clinically relevant changes in a patient’s status from the analytic variability of the test. For instance, the manual white blood cell differential count is not precise

<table>
<thead>
<tr>
<th>TABLE 1–2. PROPERTIES OF USEFUL DIAGNOSTIC TESTS.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Test methodology has been described in detail so that it can be accurately and reliably reproduced.</td>
</tr>
<tr>
<td>2. Test accuracy and precision have been determined.</td>
</tr>
<tr>
<td>3. The reference range has been established appropriately.</td>
</tr>
<tr>
<td>4. Sensitivity and specificity have been reliably established by comparison with a gold standard. The evaluation has used a range of patients, including those who have different but commonly confused disorders and those with a spectrum of mild and severe, treated and untreated disease. The patient selection process has been adequately described so that results will not be generalized inappropriately.</td>
</tr>
<tr>
<td>5. Independent contribution to overall performance of a test panel has been confirmed if a test is advocated as part of a panel of tests.</td>
</tr>
</tbody>
</table>
enough to detect important changes in the distribution of cell types, because it is calculated by subjective evaluation of a small sample (100 cells). Repeated measurements by different technicians on the same sample result in widely different results. Automated differential counts are more precise because they are obtained from machines that use objective physical characteristics to classify a much larger sample (10,000 cells).

**Reference Range**

Reference ranges are method- and laboratory-specific. In practice, they often represent test results found in 95% of a small population presumed to be healthy; by definition, then, 5% of healthy patients will have a positive (abnormal) test (Figure 1–2). As a result, slightly abnormal results should be interpreted critically—they may be either truly abnormal or falsely abnormal. The practitioner should be aware also that the more tests ordered, the greater the chance of obtaining a falsely abnormal result. For a healthy person subjected to 20 independent tests, there is a 64% chance that one test result will lie outside the reference range (Table 1–3). Conversely, values within the reference range may not rule out the actual presence of disease since the reference range does not establish the distribution of results in patients with disease.

It is important to consider also whether published reference ranges are appropriate for the patient being evaluated, since some ranges depend on age, sex, weight, diet, time of day, activity status, or posture. For instance, the reference ranges for hemoglobin concentration are age-
and sex-dependent. Chapter 3 contains the reference ranges for commonly used chemistry and hematology tests. Test performance characteristics such as sensitivity and specificity are needed to interpret results and are discussed below.

Interfering Factors

The results of diagnostic tests can be altered by external factors, such as ingestion of drugs; and internal factors, such as abnormal physiologic states.

External interferences can affect test results in vivo or in vitro. In vivo, alcohol increases $\gamma$-glutamyl transpeptidase, and diuretics can affect

![Figure 1–2. The reference range is usually defined as within 2 standard deviations of the mean test result (shown as -2 and 2) in a small population of healthy volunteers. Note that in this example, test results are normally distributed; however, many biologic substances will have distributions that are skewed.](image)

<table>
<thead>
<tr>
<th>Number of Tests</th>
<th>Probability That One or More Results Will Be Abnormal</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5%</td>
</tr>
<tr>
<td>6</td>
<td>26%</td>
</tr>
<tr>
<td>12</td>
<td>46%</td>
</tr>
<tr>
<td>20</td>
<td>64%</td>
</tr>
</tbody>
</table>
sodium and potassium concentrations. Cigarette smoking can induce hepatic enzymes and thus reduce levels of substances such as theophylline that are metabolized by the liver. In vitro, cephalosporins may produce spurious serum creatinine levels due to interference with a common laboratory method.

Internal interferences result from abnormal physiologic states interfering with the test measurement. As an example, patients with gross lipemia may have spuriously low serum sodium levels if the test methodology used includes a step in which serum is diluted before sodium is measured. Because of the potential for test interference, clinicians should be wary of unexpected test results and should investigate reasons other than disease that may explain abnormal results, including laboratory error.

**Sensitivity and Specificity**

Clinicians should use measures of test performance such as sensitivity and specificity to judge the quality of a diagnostic test for a particular disease. Test **sensitivity** is the likelihood that a diseased patient has a positive test. If all patients with a given disease have a positive test (ie, no diseased patients have negative tests), the test sensitivity is 100%. A test with high sensitivity is useful to exclude a diagnosis because a highly sensitive test will render few results that are falsely negative. To exclude infection with the AIDS virus, for instance, a clinician might choose a highly sensitive test such as the HIV antibody test.

A test’s **specificity** is the likelihood that a healthy patient has a negative test. If all patients who do not have a given disease have negative tests (ie, no healthy patients have positive tests), the test specificity is 100%. A test with high specificity is useful to confirm a diagnosis, because a highly specific test will have few results that are falsely positive. For instance, to make the diagnosis of gouty arthritis, a clinician might choose a highly specific test, such as the presence of negatively birefringent needle-shaped crystals within leukocytes on microscopic evaluation of joint fluid.

To determine test sensitivity and specificity for a particular disease, the test must be compared against a “gold standard,” a procedure that defines the true disease state of the patient. For instance, the sensitivity and specificity of the ventilation/perfusion scan for pulmonary embolus are obtained by comparing the results of scans with the gold standard, pulmonary arteriography. Application of the gold standard examination to patients with positive scans establishes specificity. Failure to apply the gold standard examination following negative scans
may result in an overestimation of sensitivity, since false negatives will not be identified. However, for many disease states (eg, pancreatitis), such a gold standard either does not exist or is very difficult or expensive to apply. Therefore, reliable estimates of test sensitivity and specificity are sometimes difficult to obtain.

Sensitivity and specificity can also be affected by the population from which these values are derived. For instance, many diagnostic tests are evaluated first using patients who have severe disease and control groups who are young and well. Compared with the general population, this study group will have more results that are truly positive (because patients have more advanced disease) and more results that are truly negative (because the control group is healthy). Thus, test sensitivity and specificity will be higher than would be expected in the general population, where more of a spectrum of health and disease are found. Clinicians should be aware of this spectrum bias when generalizing published test results to their own practice.

Test sensitivity and specificity depend on the threshold above which a test is interpreted to be abnormal (Figure 1–3). If the threshold is lowered, sensitivity is increased at the expense of lowered specificity, or vice versa.

Figure 1–4 shows how test sensitivity and specificity can be calculated using test results from patients previously classified by the gold standard as diseased or nondiseased.

![Figure 1–3. Hypothetical distribution of test results for healthy and diseased individuals. The position of the “cutoff point” between “normal” and “abnormal” (or “negative” and “positive”) test results determines the test’s sensitivity and specificity. If point “A” is the cutoff point, the test would have 100% sensitivity but low specificity. If point “C” is the cutoff point, the test would have 100% specificity but low sensitivity. For most tests, the cutoff point is determined by the reference range, ie, the range of test results that are within 2 standard deviations of the mean (point “B”). In some situations, the cutoff is altered to enhance either sensitivity or specificity.](image-url)
The performance of two different tests can be compared by plotting the sensitivity and (1 minus the specificity) of each test at various reference range cutoff values. The resulting receiver operator characteristic (ROC) curve will often show which test is better; a clearly superior test will have an ROC curve that always lies above and to the left of the inferior test curve, and, in general, the better test will have a larger area under the ROC curve. For instance, Figure 1–5 shows the ROC curves for prostate-specific antigen (PSA) and prostatic acid phosphatase (PAP) in the diagnosis of prostate cancer. PSA is a superior test because it has higher sensitivity and specificity for all cutoff values.

### Figure 1–4. Calculation of sensitivity, specificity, and probability of disease after a positive test (posttest probability). (TP, true positive; FP, false positive; FN, false negative; TN, true negative.)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Present</th>
<th>Absent</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Positive Test</strong></td>
<td>TP</td>
<td>FP</td>
</tr>
<tr>
<td><strong>Negative Test</strong></td>
<td>FN</td>
<td>TN</td>
</tr>
</tbody>
</table>

Sensitivity = \( \frac{\text{Number of diseased patients with positive test}}{\text{Number of diseased patients}} = \frac{TP}{TP + FN} \)

Specificity = \( \frac{\text{Number of nondiseased patients with negative test}}{\text{Number of nondiseased patients}} = \frac{TN}{TN + FP} \)

Posttest probability after positive test = \( \frac{\text{Probability of disease if test positive}}{\text{Probability of disease if test positive}} = \frac{TP}{TP + FP} \)

\( = \frac{(\text{Sensitivity})(\text{Pretest probability})}{(\text{Sensitivity})(\text{Pretest probability}) + (1-\text{Specificity})(1-\text{Pretest probability})} \)
The value of a test in a particular clinical situation depends not only on the test’s sensitivity and specificity but also on the probability that the patient has the disease before the test result is known (pretest probability). The results of a valuable test will substantially change the probability that the patient has the disease (posttest probability). Figure 1–4 shows how posttest probability can be calculated from the known sensitivity and specificity of the test and the estimated pretest probability of disease (or disease prevalence).

The pretest probability of disease has a profound effect on the posttest probability of disease. As demonstrated in Table 1–4, when a test with 90% sensitivity and specificity is used, the posttest probability can vary from 1% to 99% depending on the pretest probability of disease. Furthermore, as the pretest probability of disease decreases, it becomes less likely that someone with a positive test actually has the disease and more likely that the result represents a false positive.
As an example, suppose the clinician wishes to calculate the posttest probability of prostate cancer using the PSA test and a cut-off value of 4 ng/mL. Using the data shown in Figure 1–5, sensitivity is 90% and specificity is 60%. The clinician estimates the pretest probability of disease given all the evidence and then calculates the posttest probability using the approach shown in Figure 1–5. The pretest probability that an otherwise healthy 50-year-old man has prostate cancer is equal to the prevalence of prostate cancer in that age group (probability = 10%) and the posttest probability is only 20%—ie, even though the test is positive, there is still an 80% chance that the patient does not have prostate cancer (Figure 1–6A). If the clinician finds a prostate nodule on rectal examination, the pretest probability of prostate cancer rises to 50% and the posttest probability using the same test is 69% (Figure 1–6B). Finally, if the clinician estimates the pretest probability to be 98% based on a prostate nodule, bone pain, and lytic lesions on spine x-rays, the posttest probability using PSA is 99% (Figure 1–6C). This example illustrates that pretest probability has a profound effect on posttest probability and that tests provide more information when the diagnosis is truly uncertain (pretest probability about 50%) than when the diagnosis is either unlikely or nearly certain.

**ODDS-LIKELIHOOD RATIOS**

An easier way to calculate the posttest probability of disease is to use the odds-likelihood approach. Sensitivity and specificity are combined into one entity called the likelihood ratio (LR).

\[
LR = \frac{\text{Probability of result in diseased persons}}{\text{Probability of result in nondiseased persons}}
\]

Every test has two likelihood ratios, one corresponding to a positive test \(LR^+\) and one corresponding to a negative test \(LR^-\):
Figure 1–6. Effect of pretest probability and test sensitivity and specificity on the posttest probability of disease. (See text for explanation.)

\[ LR^+ = \frac{\text{Probability that test is positive in diseased persons}}{\text{Probability that test is positive in nondiseased persons}} \]

\[ = \frac{\text{Sensitivity}}{1 - \text{Specificity}} \]

\[ LR^- = \frac{\text{Probability that test is negative in diseased persons}}{\text{Probability that test is negative in nondiseased persons}} \]

\[ = \frac{1 - \text{Sensitivity}}{\text{Specificity}} \]
Lists of likelihood ratios can be found in some textbooks, journal articles, and computer programs (see Table 1–5 for sample values). Likelihood ratios can be used to make quick estimates of the usefulness of a contemplated diagnostic test in a particular situation. The simplest method for calculating posttest probability from pretest probability and likelihood ratios is to use a nomogram (Figure 1–7). The clinician places a straightedge through the points that represent the pretest probability and the likelihood ratio and then reads the posttest probability where the straightedge crosses the posttest probability line.

### TABLE 1–5. LIKELIHOOD RATIOS (LR) FOR DIAGNOSTIC TESTS.

<table>
<thead>
<tr>
<th>Test</th>
<th>Disease</th>
<th>LR⁺</th>
<th>LR⁻</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amylase (↑)</td>
<td>Pancreatitis</td>
<td>9.1</td>
<td>0.2</td>
</tr>
<tr>
<td>Anti-dsDNA (↑)</td>
<td>SLE</td>
<td>37</td>
<td>0.28</td>
</tr>
<tr>
<td>Antinuclear antibody</td>
<td>SLE</td>
<td>4.5</td>
<td>0.13</td>
</tr>
<tr>
<td>Carcinoembryonic antigen</td>
<td>Dukes A colon cancer</td>
<td>1.6</td>
<td>0.87</td>
</tr>
<tr>
<td>Creatine kinase MB</td>
<td>Myocardial infarction</td>
<td>32</td>
<td>0.05</td>
</tr>
<tr>
<td>Esophagogastroduodenoscopy (+)</td>
<td>Upper GI bleeding</td>
<td>18</td>
<td>0.11</td>
</tr>
<tr>
<td>ESR &gt; 30 mm/h</td>
<td>Temporal arteritis</td>
<td>3.3</td>
<td>0.01</td>
</tr>
<tr>
<td>Exercise echocardiography (new wall motion abnormalities)</td>
<td>Coronary artery disease</td>
<td>6.2</td>
<td>0.23</td>
</tr>
<tr>
<td>Exercise ECG (ST depression &gt; 1 mm)</td>
<td>Coronary artery disease</td>
<td>5.9</td>
<td>0.39</td>
</tr>
<tr>
<td>Ferritin</td>
<td>Iron deficiency anemia</td>
<td>85</td>
<td>0.15</td>
</tr>
<tr>
<td>Free T₄ (↑)</td>
<td>Hyperthyroidism</td>
<td>19</td>
<td>0.05</td>
</tr>
<tr>
<td>Free thyroxine index</td>
<td>Hyperthyroidism</td>
<td>6.8</td>
<td>0.06</td>
</tr>
<tr>
<td>Hepatitis A IgM antibody</td>
<td>Hepatitis A</td>
<td>99</td>
<td>0.01</td>
</tr>
<tr>
<td>Heterophil (+)</td>
<td>Infectious mononucleosis</td>
<td>97</td>
<td>0.03</td>
</tr>
<tr>
<td>Metanephrines (↑)</td>
<td>Pheochromocytoma</td>
<td>11</td>
<td>0.23</td>
</tr>
<tr>
<td>Pleural fluid protein &gt; 3 g/dL</td>
<td>Exudative pleural effusion</td>
<td>10</td>
<td>0.12</td>
</tr>
<tr>
<td>Technetium Tc 99m pyrophosphate scan (highly focal uptake)</td>
<td>Myocardial infarction</td>
<td>&gt; 360</td>
<td>0.64</td>
</tr>
<tr>
<td>Testosterone (↓)</td>
<td>Erectile dysfunction</td>
<td>32</td>
<td>0.03</td>
</tr>
<tr>
<td>TSH (↑)</td>
<td>Hypothyroidism</td>
<td>99</td>
<td>0.01</td>
</tr>
<tr>
<td>24-Hour urinary free cortisol (↑)</td>
<td>Hypercortisolism</td>
<td>10</td>
<td>0.07</td>
</tr>
</tbody>
</table>
Figure 1–7. Nomogram for determining posttest probability from pretest probability and likelihood ratios. To figure the posttest probability, place a straightedge between the pretest probability and the likelihood ratio for the particular test. The posttest probability will be where the straightedge crosses the posttest probability line. (Adapted and reproduced, with permission, from Fagan TJ: Nomogram for Bayes’s theorem. N Engl J Med 1975;293:257.)
A more formal way of calculating posttest probabilities uses the likelihood ratio as follows:

\[
\text{Pretest odds} \times \text{Likelihood ratio} = \text{Posttest odds}
\]

To use this formulation, probabilities must be converted to odds, where the odds of having a disease are expressed as the chance of having the disease divided by the chance of not having the disease. For instance, a probability of 0.75 is the same as 3:1 odds (Figure 1–8).

To estimate the potential benefit of a diagnostic test, the clinician first estimates the pretest odds of disease given all available clinical information and then multiplies the pretest odds by the positive and negative likelihood ratios. The results are the posttest odds, or the odds that the patient has the disease if the test is positive or negative. To obtain the posttest probability, the odds are converted to a probability (Figure 1–8).

For example, if the clinician believes that the patient has a 60% chance of having a myocardial infarction (pretest odds of 3:2) and the creatine kinase MB test is positive (LR+ = 32), then the posttest odds of having a myocardial infarction are

\[
\frac{3}{2} \times 32 = 48
\]

The posttest probability can then be calculated as

\[
\frac{48}{48 + 2} = 0.96
\]

**Odds** = \( \frac{\text{Probability}}{1 - \text{Probability}} \)

**Probability** = \( \frac{\text{Odds}}{\text{Odds} + 1} \)

**Example:** If probability = 0.75, then

\[
\text{Odds} = \frac{0.75}{1 - 0.75} = \frac{0.75}{0.25} = \frac{3}{1} = 3:1
\]

\[
\text{Probability} = \frac{3/1}{3/1 + 1} = \frac{3}{3 + 1} = 0.75
\]

**Figure 1–8.** Formulas for converting between probability and odds.
If the CKMB test is negative (LR⁻ = 0.05), then the posttest odds of having a myocardial infarction are

\[
\frac{3}{2} \times 0.05 = \frac{0.15}{2} \text{ odds} \left( \frac{0.15/2}{0.15/2 + 1} = \frac{0.15}{0.15 + 2} = 7\% \text{ probability} \right)
\]

Sequential Testing

To this point, the impact of only one test on the probability of disease has been discussed, whereas during most diagnostic workups, clinicians obtain clinical information in a sequential fashion. To calculate the posttest odds after three tests, for example, the clinician might estimate the pretest odds and use the appropriate likelihood ratio for each test:

\[
\text{Posttest odds} = \text{Pretest odds} \times LR_1 \times LR_2 \times LR_3
\]

When using this approach, however, the clinician should be aware of a major assumption: the chosen tests or findings must be conditionally independent. For instance, with liver cell damage, the aspartate aminotransferase (AST) and alanine aminotransferase (ALT) enzymes may be released by the same process and are thus not conditionally independent. If conditionally dependent tests are used in this sequential approach, an overestimation of posttest probability will result.

Threshold Approach to Decision Making

A key aspect of medical decision making is the selection of a treatment threshold, ie, the probability of disease at which treatment is indicated. Figure 1–9 shows a possible way of identifying a treatment threshold by considering the value (utility) of the four possible outcomes of the treat/don’t treat decision.

A diagnostic test is useful only if it shifts the disease probability across the treatment threshold. For example, a clinician might decide to treat with antibiotics if the probability of streptococcal pharyngitis in a patient with a sore throat is greater than 25% (Figure 1–10A). If, after reviewing evidence from the history and physical examination, the clinician estimates the pretest probability of strep throat to be 15%, then a diagnostic test such as throat culture (LR⁺ = 7) would be useful only if a positive test would shift the posttest probability above 25%. Use of the nomogram shown in Figure 1–7 indicates that the posttest
probability would be 55% (Figure 1–10B); thus, ordering the test would be justified as it affects patient management. On the other hand, if the history and physical examination had suggested that the pretest probability of strep throat was 60%, the throat culture (LR− = 0.33) would be indicated only if a negative test would lower the posttest probability below 25%. Using the same nomogram, the posttest probability after a negative test would be 33% (Figure 1–10C). Therefore, ordering the throat culture would not be justified.

This approach to decision making is now being applied in the clinical literature.

**Decision Analysis**

Up to this point, the discussion of diagnostic testing has focused on test characteristics and methods for using these characteristics to calculate the probability of disease in different clinical situations. Although useful, these methods are limited because they do not incorporate the many outcomes that may occur in clinical medicine or the values that patients and clinicians place on those outcomes. To incorporate outcomes and values with characteristics of tests, decision analysis can be used.

The basic idea of decision analysis is to model the options in a medical decision, assign probabilities to the alternative actions, assign values (utilities) to the various outcomes, and then calculate which decision gives the greatest value. To complete a decision analysis, the clinician would proceed as follows:
Figure 1–10. Threshold approach applied to test ordering. If the contemplated test will not change patient management, the test should not be ordered. (See text for explanation.)
(1) Draw a decision tree showing the elements of the medical decision.
(2) Assign probabilities to the various branches.
(3) Assign values (utilities) to the outcomes.
(4) Determine the expected utility (the product of probability and utility) of each branch.
(5) Select the decision with the highest expected utility.

Figure 1–11 shows a decision tree where the decision to be made is whether to treat without testing, perform a test and then treat based on the test result, or perform no tests and give no treatment. The clinician

![Decision Tree Diagram]

Figure 1–11. Generic tree for a clinical decision where the choices are (1) to treat the patient empirically, (2) to test and then treat if the test is positive, or (3) to withhold therapy. The square node is called a decision node, and the round nodes are called chance nodes. (p, pretest probability of disease; Sens, sensitivity; Spec, specificity.)
begins the analysis by building a decision tree showing the important elements of the decision. Once the tree is built, the clinician assigns probabilities to all the branches. In this case, all the branch probabilities can be calculated from (1) the probability of disease before the test (pretest probability), (2) the chance of a positive test if the disease is present (sensitivity), and (3) the chance of a negative test if the disease is absent (specificity). Next, the clinician assigns utility values to each of the outcomes.

After the expected utility is calculated, the clinician may identify which alternative has the highest value by this analysis.

Although time-consuming, decision analysis can help to structure complex clinical problems and to make difficult clinical decisions.

**Evidence-Based Medicine**

The focus over the past decade on evidence-based medicine stresses the examination of evidence from clinical research—rather than intuition and pathophysiologic reasoning—as a basis for clinical decision making. Evidence-based medicine relies on systematic reviews of the medical literature to inform clinical practice. Meta-analysis uses statistical techniques to combine evidence from different studies.

Clinical practice guidelines are systematically developed statements intended to assist practitioners and patients in making decisions about health care. Clinical algorithms and practice guidelines are now ubiquitous in medicine. Their utility and validity depend on the quality of the evidence that shaped the recommendations, on their being kept current, and on their acceptance and appropriate application by clinicians. While clinicians are concerned about the effect of guidelines on professional autonomy, many organizations are trying to use compliance with practice guidelines as a measure of quality of care.

**Computer Access to Medical Information**

The development of medical information science and computer technology now offer a vast amount of clinical information on CD-ROM or over the World Wide Web.

**REFERENCES**


This page intentionally left blank.
This chapter presents information on how to perform common bedside laboratory procedures. Information on interpretation of results of body fluid analysis is included in some of the sections. Test results can be used for patient care only if the tests have been performed according to strict federal guidelines.

Contents

1. Obtaining and processing body fluids .................................24
   A. Safety considerations .......................................................24
   B. Specimen handling .........................................................24

2. Basic staining methods .........................................................25
   A. Gram stain ......................................................................25
   B. Wright stain of peripheral blood smear ..........................27

3. Other bedside laboratory procedures ..................................28
   A. Urinalysis .......................................................................28
   B. Vaginal fluid wet preparation .........................................33
   C. Skin or vaginal fluid KOH preparation .........................33
   D. Synovial fluid examination for crystals .......................35
   E. Pulse oximetry ................................................................36

Stephen J. McPhee, MD
1. OBTAINING AND PROCESSING BODY FLUIDS

A. Safety Considerations

General Safety Considerations
Because all patient specimens are potentially infectious, the following precautions should be observed:

a. Universal body fluid and needle stick precautions must be observed at all times.
b. Disposable gloves and sometimes gown, mask, and goggles should be worn when collecting specimens.
c. Gloves should be changed and hands washed after contact with each patient. Dispose of gloves in an appropriate biohazard waste container.
d. Any spills should be cleaned up with 10% bleach solution.

Handling and Disposing of Needles and Gloves
a. Do not resheath needles.
b. Discard needles and gloves only into designated containers.
c. Do not remove a used needle from a syringe by hand. The needle may be removed using a specially designed waste collection system, or the entire assembly may (if disposable) be discarded as a unit into a designated container.
d. When obtaining blood cultures, it is hazardous and unnecessary to change needles.
e. Do not place phlebotomy or other equipment on the patient’s bed.

B. Specimen Handling

Identification of Specimens
a. Identify the patient before obtaining the specimen. (If the patient is not known to you, ask for the name and check the wristband.)
b. Label each specimen container with the patient’s name and identification number.

Specimen Tubes: Standard specimen tubes are now widely available and are easily identified by the color of the stopper (see also p 37):

a. Red-top tubes contain no anticoagulants or preservatives and are used for chemistry tests.
b. Marbled-top tubes contain material that allows ready separation of serum and clot by centrifugation.
c. Lavender-top tubes contain EDTA and are used for hematology tests (eg, blood or cell counts, differentials).
d. Green-top tubes contain heparin and are used for tests that require plasma or anticoagulation.

e. Blue-top tubes contain citrate and are used for coagulation tests.

f. Gray-top tubes contain fluoride and are used for some chemistry tests (eg, glucose) if the specimen cannot be analyzed immediately.

**Procedure**

a. When collecting multiple specimens, fill sterile tubes used for bacteriologic tests, then tubes without additives (ie, red-top tubes) before filling those with additives to avoid the potential for bacterial contamination, transfer of anticoagulants, etc. However, be certain to fill tubes containing anticoagulants before the blood specimen clots.

b. The recommended order of filling tubes is (by type and color): (1) blood culture, (2) red top, (3) blue top, (4) green top, (5) lavender top.

c. Fill each stoppered tube completely. Tilt each tube containing anticoagulant or preservative to mix thoroughly. Place any specimens on ice as required (eg, arterial blood). Deliver specimens to the laboratory promptly.

d. For each of the major body fluids, Table 2–1 summarizes commonly requested tests and requirements for specimen handling and provides cross-references to tables and figures elsewhere in this book for help in interpretation of the results.

---

2. BASIC STAINING METHODS

A. Gram Stain

**Preparation of Smear**

a. Obtain a fresh specimen of the material to be stained (eg, sputum) and smear a small amount on a glass slide. Thin smears give the best results (eg, press a sputum sample between two glass slides).

b. Let the smear air-dry before heat-fixing, because heating a wet smear will usually distort cells and organisms.

c. Heat-fix the smear by passing the clean side of the slide quickly through a Bunsen burner or other flame source (no more than three or four times). The slide should be warm, not hot.

d. Let the slide cool before staining.
# TABLE 2-1. BODY FLUID TESTS, HANDLING, AND INTERPRETATION.

<table>
<thead>
<tr>
<th>Body Fluid</th>
<th>Commonly Requested Tests</th>
<th>Specimen Tube and Handling</th>
<th>Interpretation Guide</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial blood</td>
<td>pH, P_{O_2}, P_{CO_2}</td>
<td>Glass syringe. Evacuate air bubbles; remove needle; position rubber cap; place sample on ice; deliver immediately.</td>
<td>See acid-base nomogram p 337.</td>
</tr>
<tr>
<td>Ascitic fluid</td>
<td>Cell count, differential</td>
<td>Lavender top</td>
<td>See ascitic fluid profiles, p 365.</td>
</tr>
<tr>
<td></td>
<td>Protein, amylase</td>
<td>Red top</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gram stain, culture</td>
<td>Sterile</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cytology (if neoplasm suspected)</td>
<td>Cytology</td>
<td></td>
</tr>
<tr>
<td>Cerebrospinal fluid</td>
<td>Cell count, differential</td>
<td>Tube #1</td>
<td>See cerebrospinal fluid profiles, p 369.</td>
</tr>
<tr>
<td></td>
<td>Protein, glucose</td>
<td>Tube #2</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gram stain, culture</td>
<td>Tube #3</td>
<td></td>
</tr>
<tr>
<td></td>
<td>VDRL or other studies (oligoclonal bands)</td>
<td>Tube #4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cytology (if neoplasm suspected)</td>
<td>Cytology</td>
<td></td>
</tr>
<tr>
<td>Pleural fluid</td>
<td>Cell count, differential</td>
<td>Lavender top</td>
<td>See pleural fluid profiles, p 382.</td>
</tr>
<tr>
<td></td>
<td>Protein, glucose, amylase</td>
<td>Red top</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gram stain, culture</td>
<td>Sterile</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cytology (if neoplasm suspected)</td>
<td>Cytology</td>
<td></td>
</tr>
<tr>
<td>Synovial fluid</td>
<td>Cell count, differential</td>
<td>Lavender top</td>
<td>See synovial fluid profiles, p 389, and Figure 2–6.</td>
</tr>
<tr>
<td></td>
<td>Protein, glucose</td>
<td>Red top</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Gram stain, culture</td>
<td>Sterile</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Microscopic examination for crystals</td>
<td>Green top</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cytology (if neoplasm [villonodular synovitis, metastatic disease] suspected)</td>
<td>Cytology</td>
<td></td>
</tr>
<tr>
<td>Urine</td>
<td>Urinalysis</td>
<td>Clean tube</td>
<td>See Table 8–24, p 395.</td>
</tr>
<tr>
<td></td>
<td>Dipstick</td>
<td>Centrifuge tube</td>
<td>See Table 2–2, p 31.</td>
</tr>
<tr>
<td></td>
<td>Microscopic examination</td>
<td>Sterile</td>
<td>See Figure 2–3, p 34.</td>
</tr>
<tr>
<td></td>
<td>Gram stain, culture</td>
<td>Cytology</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Cytology (if neoplasm suspected)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Staining Technique**

a. Put on gloves.
b. Stain with crystal violet (10 seconds).
c. Rinse with gently running water (5 seconds).
d. Flood with Gram iodine solution (10–30 seconds).
e. Rinse with gently running water (5 seconds).
f. Decolorize with acetone-alcohol solution until no more blue color leaches from the slide (5 seconds).

g. Rinse immediately with water (5 seconds).

h. Counterstain with safranin O (10 seconds).

i. Rinse with water (5 seconds).

j. Let the slide air-dry (or carefully blot with filter paper), then examine it under the microscope.

**Microscopic Examination**

a. Examine the smear first using the low-power lens for leukocytes and fungi. Screen for the number and color of polymorphonuclear cells (cell nuclei should be pink, not blue).

b. Examine using the high-power oil-immersion lens for microbial forms. Screen for intracellular organisms. Review the slide systematically for (1) fungi (mycelia, then yeast), (2) small gram-negative rods (bacteroides, haemophilus, etc) (3) gram-negative cocci (neisseria, etc), (4) gram-positive rods (listeria, etc), and (5) gram-positive cocci (streptococcus, staphylococcus, etc).

c. Label positive slides with the patient’s name and identification number and save them for later review.

d. Figure 2–1 illustrates typical findings on a Gram-stained smear of sputum.

**B. Wright Stain of Peripheral Blood Smear**

**Preparation of Smear**

a. Obtain a fresh specimen of blood by pricking the patient’s finger with a lancet. If alcohol is used to clean the fingertip, wipe it off first with a gauze pad.

b. Place a single drop of blood on a glass slide. Lay a second glass slide over the first one and rapidly pull it away lengthwise to leave a thin smear.

c. Let the smear air-dry. Do not heat-fix.

**Staining Technique**

a. Stain with fresh Wright stain (1 minute).

b. Gently add an equal amount of water and gently blow on the smear to mix the stain and water. Repeat by adding more water and blowing to mix. Look for formation of a shiny surface scum. Then allow the stain to set (3–4 minutes).

c. Rinse with gently running water (5 seconds).

d. Clean the back of the slide with an alcohol pad if necessary.

**Microscopic Examination**

a. Examine the smear first using the low-power lens to select a good area for study (red and white cells separated from one another).
b. Then move to the high-power oil-immersion lens. Review the slide systematically for (1) platelet morphology, (2) white cells (differential types, morphology, toxic granulations and vacuoles, etc), and (3) red cells (size, shape, color, stippling, nucleation, etc).

c. Label slides with the patient’s name and identification number and save them for later review.

d. See Figure 2–2 for examples of common peripheral blood smear abnormalities.

3. OTHER BEDSIDE LABORATORY PROCEDURES

A. Urinalysis

Collection and Preparation of Specimen

a. Obtain a midstream urine specimen from the patient. The sample must be free of skin epithelium or bacteria, secretions, hair, lint, etc.
| **RED BLOOD CELLS** | \[\text{Normal red cells} \] \[\text{Target cells} \] \[\text{Teardrop cells} \] \[\text{Elliptocytes (ovalocytes)} \] \[\text{Acanthocytes} \] \[\text{Macrocytes} \] \[\text{Spherocytes} \] \[\text{Stomatocytes} \] \[\text{Hypochromic microcytic red cells} \] \[\text{Schistocytes (schizocytes)} \] \[\text{Howell-Jolly bodies} \] \[\text{Basophilic stippling} \] \[\text{Echinocytes} \] \[\text{Sickle cells} \] \[\text{Bite cells} \] |
| **WHITE BLOOD CELLS** | \[\text{Monocyte} \] \[\text{Eosinophil} \] \[\text{Lymphocyte} \] \[\text{Basophil} \] \[\text{Band neutrophil} \] \[\text{Neutrophil (polymorphonuclear leukocyte)} \] \[\text{Neutrophil with toxic granulations} \] \[\text{Hypersegmented neutrophil} \] |
| **PLATELETS** | \[\text{}\] |
b. Examine the specimen while fresh (still warm). Otherwise, bacteria may proliferate, casts and crystals may dissolve, and particulate matter may settle out. (Occasionally, amorphous crystals precipitate out, obscuring formed elements. In cold urine, they are amorphous urate crystals; these may be dissolved by gently rewarming the urine. In alkaline urine, they are amorphous phosphate crystals; these may be dissolved by adding 1 mL of acetic acid.)

c. Place 10 mL in a tube and centrifuge at 2000–3000 rpm for 3–5 minutes.

d. Discard the supernatant. Resuspend the sediment in the few drops that remain by gently tilting the tube.

e. Place a drop on a glass slide, cover it with a coverslip, and examine under the microscope; no stain is needed. If bacterial infection is present, a single drop of methylene blue applied to the edge of the coverslip, or a Gram-stained smear of an air-dried, heat-fixed specimen, can assist in distinguishing gram-negative rods (eg, *E coli*, *proteus*, *klebsiella*) from gram-positive cocci (eg, *enterococcus*, *Staphylococcus saprophyticus*).

**Procedural Technique**

a. While the urine is being centrifuged, examine the remainder of the specimen by inspection and reagent strip (“dipstick”) testing.

b. Inspect the specimen for color and clarity. Normally, urine is yellow or light orange. Dark orange urine is caused by ingestion of the urinary tract analgesic phenazopyridine (Pyridium, others); red urine, by hemoglobinuria, myoglobinuria, beets, senna, or rifampin therapy; green urine, by *Pseudomonas* infection or iodochlorhydroxyquin or amitriptyline therapy; brown urine, by bilirubinuria or fecal contamination; black urine, by intravascular hemolysis, alkaptonuria, melanoma, or methyldopa therapy; purplish urine, by porphyria; and milky white urine, by pus, chyluria, or amorphous crystals (urates or phosphates). Turbidity of urine is caused by pus, red blood cells, or crystals.

c. Reagent strips provide information about specific gravity, pH, protein, glucose, ketones, bilirubin, heme, nitrite, and esterase (Table 2–2). Dip a reagent strip in the urine and compare it with the chart on the bottle. Follow the timing instructions carefully. **Note:** Reagent strips cannot be relied on to detect some proteins (eg, globulins, light chains) or sugars (other than glucose).

d. Record the results.
### TABLE 2–2. COMPONENTS OF THE URINE DIPSTICK.¹

<table>
<thead>
<tr>
<th>Test</th>
<th>Values</th>
<th>Lowest Detectable Range</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific gravity</td>
<td>1.001–1.035</td>
<td>1.000–1.030</td>
<td>Highly buffered alkaline urine may yield low specific gravity readings. Moderate proteinuria (100–750 mg/dL) may yield high readings. Loss of concentrating or diluting capacity indicates renal dysfunction.</td>
</tr>
<tr>
<td>pH</td>
<td>5–9 units</td>
<td>5–8.5 units</td>
<td>Excessive urine on strip may cause protein reagent to run over onto pH area, yielding falsely low pH reading.</td>
</tr>
<tr>
<td>Protein</td>
<td>0 15–30 mg/dL albumin</td>
<td>False-positive readings can be caused by highly buffered alkaline urine. Reagent more sensitive to albumin than other proteins. A negative result does not rule out the presence of globulins, hemoglobin, Bence Jones proteins, or mucoprotein. 1+ = 30 mg/dL 3+ = 300 mg/dL 2+ = 100 mg/dL 4+ = ≥ 2000 mg/dL</td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>0 75–125 mg/dL</td>
<td>Test is specific for glucose. False-negative results occur with urinary ascorbic acid concentrations ≥ 50 mg/dL and with ketone body levels ≥ 50 mg/dL Test reagent reactivity also varies with specific gravity and temperature. Trace = 100 mg/dL 1 = 1000 mg/dL 1/4 = 250 mg/dL 2 = ≥ 2000 mg/dL 1/2 = 500 mg/dL</td>
<td></td>
</tr>
<tr>
<td>Ketone</td>
<td>0 5–10 mg/dL acetoacetate</td>
<td>Test does not react with acetone or β-hydroxybutyric acid. (Trace) false-positive results may occur with highly pigmented urines or those containing levodopa metabolites or sulfhydryl-containing compounds (eg, mesna). Trace = 5 mg/dL Moderate = 40 mg/dL Small = 15 mg/dL Large = 80–160 mg/dL</td>
<td></td>
</tr>
<tr>
<td>Bilirubin</td>
<td>0 0.4–0.8 mg/dL</td>
<td>Indicates hepatitis (conjugated bilirubin). False-negative readings can be caused by ascorbic acid concentrations ≥ 25 mg/dL. False-positive readings can be caused by etodolac metabolites. Test is less sensitive than Ictotest Reagent tablets.</td>
<td></td>
</tr>
</tbody>
</table>

(continued)
Microscopic Examination

a. Examine the area under the coverslip under the low-power and high-dry lenses for cells, casts, crystals, and bacteria. (If a Gram stain is done, examine under the oil immersion lens.)

b. Cells may be red cells, white cells, squamous cells, transitional (bladder) epithelial cells, or atypical (tumor) cells. Red cells suggest upper or lower urinary tract infections (cystitis, prostatitis, pyelonephritis), glomerulonephritis, collagen vascular disease, trauma, renal calculi, tumors, drug reactions, and structural abnormalities (polycystic kidneys). White cells suggest inflammatory processes such as urinary tract infection (most common), collagen vascular disease, or interstitial nephritis. Red cell casts are considered pathognomonic of glomerulonephritis; white cell casts, of pyelonephritis; and fatty (lipid) casts, of nephrotic syndrome.

---

### TABLE 2–2 (CONT’D). COMPONENTS OF THE URINE DIPSTICK.¹

<table>
<thead>
<tr>
<th>Test</th>
<th>Values</th>
<th>Lowest Detectable Range</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood</td>
<td>0²</td>
<td>0.015–0.062 mg/dL hemoglobin</td>
<td>Test equally sensitive to myoglobin and hemoglobin (including both intact erythrocytes and free hemoglobin). False-positive results can be caused by oxidizing contaminants (hypochlorite) and microbial peroxidase (urinary tract infection). Test sensitivity is reduced in urines with high specific gravity, captopril, or heavy proteinuria.</td>
</tr>
<tr>
<td>Nitrite</td>
<td>0</td>
<td>0.06–0.1 mg/dL nitrite ion</td>
<td>Test depends on the conversion of nitrate (derived from the diet) to nitrite by gram-negative bacteria in urine. Test specific for nitrite. False-negative readings can be caused by ascorbic acid. Test sensitivity is reduced in urines with high specific gravity.</td>
</tr>
<tr>
<td>Leukocytes (esterase)</td>
<td>0³</td>
<td>6–15 WBCs/hpf</td>
<td>Indicator of urinary tract infection. Test detects esterases contained in granulocytic leukocytes. Test sensitivity is reduced in urines with high specific gravity, elevated glucose concentrations (≥ 4 g/dL), or presence of cephalixin, cephalothin, tetracycline, or high concentrations of oxalate.</td>
</tr>
</tbody>
</table>

¹ Package insert, revised 9/95. Bayer Diagnostics Reagent Strips for Urinalysis, Bayer Corporation.
² Except in menstruating females.
³ Except in females with vaginitis.
c. The finding on a Gram-stained smear of unspun, clean, fresh urine of even one bacterium per field under the oil-immersion lens correlates fairly well with bacterial culture colony counts of greater than 100,000 organisms per µL.

d. See Table 8–24, p 395, for a guide to interpretation of urinalysis; and Figure 2–3 for a guide to microscopic findings in urine.

B. Vaginal Fluid Wet Preparation

**Preparation of Smear and Staining Technique**

a. Place a small amount of vaginal discharge on a glass slide.

b. Add 2 drops of sterile saline solution.

c. Place a coverslip over the area to be examined.

**Microscopic Examination**

a. Examine under the microscope, using the high-dry lens and a low light source.

b. Look for motile trichomonads (undulating protozoa propelled by four flagella). Look for clue cells (vaginal epithelial cells with large numbers of organisms attached to them, obscuring cell borders), pathognomonic of *Gardnerella vaginalis*-associated vaginosis.

c. See Figure 2–4 for an example of a positive wet prep (trichomonads, clue cells) and Table 8–25, p 397 for the differential diagnosis of vaginal discharge.

C. Skin or Vaginal Fluid KOH Preparation

**Preparation of Smear and Staining Technique**

a. Obtain a skin specimen by using a No. 15 scalpel blade to scrape scales from the skin lesion onto a glass slide or to remove the top of a vesicle onto the slide. Or place a single drop of vaginal discharge on the slide.

b. Place 1 or 2 drops of potassium hydroxide (10–20%) on top of the specimen on the slide. Lay a coverslip over the area to be examined.

c. Heat the slide from beneath with a match or Bunsen burner flame until the slide contents begin to bubble.

d. Clean carbon off the back side of the slide with an alcohol pad if necessary.

*Note:* A fishy amine odor upon addition of KOH to a vaginal discharge is typical of bacterial vaginosis caused by *Gardnerella vaginalis*.

**Microscopic Examination**

a. Examine the smear under the high-dry lens for mycelial forms. Branched, septate hyphae are typical of dermatophytosis (eg, trichophyton, epidermophyton, microspo-
Figure 2–3. Microscopic findings on examination of the urine. (Modified and reproduced, with permission from Krupp MA et al: Physician’s Handbook, 21st ed. Originally published by Lange Medical Publications. Copyright © 1985 by The McGraw-Hill Companies, Inc.)
rum species); branched, septate pseudohyphae with or without budding yeast forms are seen with candidiasis (candida species); and short, curved hyphae plus clumps of spores ("spaghetti and meatballs") are seen with tinea versicolor (Malassezia furfur).

b. See Figure 2–5 for an example of a positive KOH prep.

D. Synovial Fluid Examination for Crystals

Preparation of Smear
a. No stain is necessary.
b. Place a small amount of synovial fluid on a glass slide.
c. Place a coverslip over the area to be examined.

Microscopic Examination
a. Examine under a polarized light microscope with a red compensator, using the high-dry lens and a moderately bright light source.
b. Look for needle-shaped, negatively birefringent urate crystals (crystals parallel to the axis of the compensator appear yellow) in gout or rhomboidal, positively birefringent calcium pyrophosphate crystals (crystals parallel to the axis of the compensator appear blue) in pseudogout.

c. See Figure 2–6 for examples of positive synovial fluid examinations for these two types of crystals.

E. Pulse Oximetry  
Indications  
To measure oxygen saturation in a noninvasive and often continuous fashion.
Contraindications

a. Hypotension, hypothermia, low perfusion states, severe or rapid desaturation, and severe anemia (hemoglobin < 5 g/dL) cause inaccurate readings.

b. Hyperbilirubinemia, methemoglobinemia, fetal hemoglobinemia, and carboxyhemoglobinemia can falsely elevate oxygen saturation measurements.

c. Excessive ambient light, simultaneous use of a blood pressure cuff, the presence of intravascular dyes (eg, methylene blue), and electrical interference (eg, MRI scanners, electrosurgery) can also cause erroneous readings.

Approach to the Patient

The patient should be positioned close to the pulse oximeter and should hold the probe site still. The sampling area should have good circulation and be free of skin irritation.

Procedural Technique

a. Plug the pulse oximeter into a grounded AC power outlet or make sure that sufficient battery power is available. Turn the oximeter on and wait until self-calibration is complete.
b. Select the probe to be used and connect it to the pulse oximeter. The probe consists of a light source (a red light-emitting device [LED] in most cases) and a photodetector. Probes are available for the ear, finger, and, in neonates, the foot, ankle, palm, calf, and forearm.

c. Attach the probe to the patient after cleansing the surrounding skin with an alcohol swab. Some probes come with double-sided adhesive disks that improve probe signal.

d. Watch the waveform and pulse indicators to assess the quality of the signal. Readjust if a poor signal is present.

e. Set alarm warnings on the device.

f. Check the probe site at least every 4 hours. Care should be taken not to apply tension to the probe cables.

Possible Complications
Allergic reaction to adhesives.

Comments
Because of the curvilinear nature of the oxygen-hemoglobin dissociation curve, oxygen saturation (SaO₂) is not directly proportionate to oxygen partial pressure (PaO₂). Therefore, a relatively small change in oxygen saturation (eg, from 94% to 83%) can represent a large change in PaO₂ (eg, from 80 mm Hg to 50 mm Hg). In addition, the dissociation curve varies markedly from patient to patient and with pH, temperature, and altitude. To ensure accurate assessment of oxygenation, one should correlate pulse oximetry with arterial blood gas analysis.

REFERENCES
Gram Stain

Urinalysis

**Vaginal Wet Prep**

**Synovial Fluid Examination**

**Pulse Oximetry**
This page intentionally left blank.
Common Laboratory Tests: Selection and Interpretation

Diana Nicoll, MD, PhD, MPA, Stephen J. McPhee, MD, and Michael Pignone, MD, MPH

HOW TO USE THIS SECTION

This section contains information about commonly used laboratory tests. It includes most of the blood, urine, and cerebrospinal fluid tests found in this book, with the exception of drug levels. Entries are in outline format and are arranged alphabetically.

Test/Reference Range/Collection

This first outline listing begins with the common test name, the specimen analyzed, and any test name abbreviation (in parentheses).

Below this in the first outline listing is the reference range for each test. The first entry is in conventional units, and the second entry (in [brackets]) is in SI units (Système International d’Unités). Any panic values for a particular test are placed here after the word “Panic.” The reference ranges provided are from several large medical centers; consult your own clinical laboratory for those used in your institution.
This outline listing also shows which tube to use for collecting blood and other body fluids, how much the test costs (in relative symbolism; see below), and how to collect the specimen. Listed below are the common collection tubes and their contents:

<table>
<thead>
<tr>
<th>Tube Top Color</th>
<th>Tube Contents</th>
<th>Typically Used In</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lavender</td>
<td>EDTA</td>
<td>Complete blood count</td>
</tr>
<tr>
<td>Marbled</td>
<td>Serum separator</td>
<td>Serum chemistry tests</td>
</tr>
<tr>
<td>Red</td>
<td>None</td>
<td>Blood banking (serum)</td>
</tr>
<tr>
<td>Blue</td>
<td>Citrate</td>
<td>Coagulation studies</td>
</tr>
<tr>
<td>Green</td>
<td>Heparin</td>
<td>Plasma studies</td>
</tr>
<tr>
<td>Yellow</td>
<td>Acid citrate</td>
<td>HLA typing</td>
</tr>
<tr>
<td>Navy</td>
<td>Trace metal free</td>
<td>Trace metals (eg, lead)</td>
</tr>
<tr>
<td>Gray</td>
<td>Inhibitor of glycolysis (sodium fluoride)</td>
<td>Lactic acid</td>
</tr>
</tbody>
</table>

The scale used for the cost of each test is:

<table>
<thead>
<tr>
<th>Approximate Cost</th>
<th>Symbol Used in Tables</th>
</tr>
</thead>
<tbody>
<tr>
<td>$1–20</td>
<td>$</td>
</tr>
<tr>
<td>$21–50</td>
<td>$$</td>
</tr>
<tr>
<td>$51–100</td>
<td>$$$</td>
</tr>
<tr>
<td>&gt; $100</td>
<td>$$$$$</td>
</tr>
</tbody>
</table>

**Physiologic Basis**

This outline listing contains physiologic information about the substance being tested. Information on classification and biologic importance, as well as interactions with other biologic substances and processes, is included.

**Interpretation**

This outline lists clinical conditions that affect the substance being tested. Generally, conditions with higher prevalence will be listed first. When the sensitivity of the test for a particular disease is known, that
information will follow the disease name in parentheses, eg, “rheumatoid arthritis (83%).” Some of the common drugs that can affect the test substance in vivo will also be included in this outline listing.

**Comments**

This outline listing sets forth general information pertinent to the use and interpretation of the test and important in vitro interferences with the test procedure. Appropriate general references are also listed.

**Test Name**

The test name is placed as a header to the rest of the outline list to allow for quick referencing.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>ABO grouping</strong>, serum and red cells (ABO)**</td>
<td>The four blood groups A, B, O, and AB are determined by the presence of antigens A and B or their absence (O) on a patient’s red blood cells. Antibodies are present in serum for which red cells lack antigen.</td>
<td>In the US white population, 45% are type O, 40% A, 11% B, 4% AB. In the African-American population, 49% are type O, 27% A, 20% B, 4% AB. In the US Asian population, 40% are type O, 28% A, 27% B, 5% AB. In the Native American population, 79% are type O, 16% A, 4% B, &lt;1% AB.</td>
<td>For both blood donors and recipients, routine ABO grouping includes both red cell and serum testing, as checks on each other. Tube testing is as follows: patient’s red cells are tested with anti-A and anti-B for the presence or absence of agglutination (forward or cell grouping), and patient’s serum is tested against known A and B cells (reverse or serum grouping). <em>Technical Manual of the American Association of Blood Banks</em>, 11th ed. American Association of Blood Banks, 1993.</td>
</tr>
<tr>
<td><strong>Acetaminophen</strong>, serum (Tylenol; others)**</td>
<td>In overdose, liver and renal toxicity are produced by the hydroxylated metabolite if it is not conjugated with glutathione in the liver.</td>
<td><strong>Increased in:</strong> Acetaminophen overdose. Interpretation of serum acetaminophen level depends on time since ingestion. Levels drawn &lt;4 hours after ingestion cannot be interpreted since the drug is still in the absorption and distribution phase. Use nomogram (Figure 8-1, p 336) to evaluate possible toxicity. Levels &gt;150 mg/dL at 4 hours or &gt;50 mg/dL at 12 hours after ingestion suggest toxicity. Nomogram inaccurate for chronic ingestions.</td>
<td>Do not delay acetylcysteine (Mucomyst) treatment (140 mg/kg orally) if stat levels are unavailable. Lancet 1971;1:519. Pediatrics 1975;55:871. Lancet 1976;2:109.</td>
</tr>
<tr>
<td><strong>Acetaminophen</strong></td>
<td>10–20 mg/L [66–132 µmol/L] <strong>Panic:</strong> &gt;50 mg/L</td>
<td><strong>Marbled</strong></td>
<td><strong>Marbled</strong></td>
</tr>
<tr>
<td><strong>Acetaminophen</strong></td>
<td>For suspected overdose, draw two samples at least 4 hours apart, at least 4 hours after ingestion. Note time of ingestion, if known. Order test stat.</td>
<td><strong>For suspected overdose, draw two samples at least 4 hours apart, at least 4 hours after ingestion. Note time of ingestion, if known. Order test stat.</strong></td>
<td><strong>For suspected overdose, draw two samples at least 4 hours apart, at least 4 hours after ingestion. Note time of ingestion, if known. Order test stat.</strong></td>
</tr>
<tr>
<td><strong>Acetoacetate</strong>, serum or urine</td>
<td>Acetoacetate, acetone, and β-hydroxybutyrate contribute to ketoacidosis when oxidative hepatic metabolism of fatty acids is impaired. Proportions in serum vary but are generally 20% acetoacetate, 78% β-hydroxybutyrate, and 2% acetone.</td>
<td><strong>Present in</strong>: Diabetic ketoacidosis, alcoholic ketoacidosis, prolonged fasting, severe carbohydrate restriction with normal fat intake.</td>
<td>Nitroprusside test is semiquantitative; it detects acetoacetate and is sensitive down to 5–10 mg/dL. Trace = 5 mg/dL, small = 15 mg/dL, moderate = 40 mg/dL, large = 80 mg/dL [1 mg/dL = 100 µmol/L]. β-Hydroxybutyrate is not a ketone and is not detected by the nitroprusside test. Acetone is also not reliably detected by this method. Failure of test to detect β-hydroxybutyrate in ketoacidosis may produce a seemingly paradoxical increase in ketones with clinical improvement as nondetectable β-hydroxybutyrate is replaced by detectable acetoacetate. Br Med J 1972;2:565.</td>
</tr>
<tr>
<td>Acetoacetate, acetone, and β-hydroxybutyrate contribute to ketoacidosis when oxidative hepatic metabolism of fatty acids is impaired. Proportions in serum vary but are generally 20% acetoacetate, 78% β-hydroxybutyrate, and 2% acetone.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled or urine container $</td>
<td>Urine sample should be fresh.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Acetylcholine receptor antibody</strong>, serum</td>
<td>Acetylcholine receptor antibodies are involved in the pathogenesis of myasthenia gravis. Sensitive radio-assay or ELISA is available based on inhibition of binding of $^{125}$I alpha-bungarotox in to the acetylcholine receptor.</td>
<td><strong>Positive in</strong>: Myasthenia gravis. Sensitivity = 73%. Single fiber EMG may have best sensitivity.</td>
<td>Titer has been found to correlate with clinical severity. J Neurol Neurosurg Psychiatry 1993;56:496. Clin Chem 1993;39:2053. Muscle Nerve 1992;15:720.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>---------------------------------------</td>
<td>-----------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Adrenocorticotropic hormone, plasma (ACTH)</strong></td>
<td>Pituitary ACTH (release stimulated by hypothalamic corticotropin-releasing factor) stimulates cortisol release from the adrenal gland. There is feedback regulation of the system by cortisol. ACTH is secreted episodically and shows circadian variation, with highest levels at 6:00–8:00 AM; lowest levels at 9:00–10:00 PM.</td>
<td><strong>Increased in:</strong> Pituitary (40–200 pg/mL) and ectopic (200–71,000 pg/mL) Cushing’s syndrome, primary adrenal insufficiency (&gt;250 pg/mL), adrenogenital syndrome with impaired cortisol production. <strong>Decreased in:</strong> Adrenal Cushing’s syndrome (&lt;20 pg/mL), pituitary ACTH (secondary adrenal) insufficiency (&lt;50 pg/mL).</td>
<td>ACTH levels (RIA) can only be interpreted when measured with cortisol after standardized stimulation or suppression tests. Postgrad Med 1998;104:61.</td>
</tr>
<tr>
<td>20–100 pg/mL [4–22 pmol/L]</td>
<td>Heparinized plastic container $$$ Send promptly to laboratory on ice. ACTH is unstable in plasma, is inactivated at room temperature, and adheres strongly to glass. Avoid all contact with glass.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Alanine aminotransferase, serum (ALT, SGPT, GPT)</strong></td>
<td>Intracellular enzyme involved in amino acid metabolism. Present in large concentrations in liver, kidney; in smaller amounts, in skeletal muscle and heart. Released with tissue damage, particularly liver injury.</td>
<td><strong>Increased in:</strong> Acute viral hepatitis (ALT &gt; AST), biliary tract obstruction (cholangitis, choledocholithiasis), alcoholic hepatitis and cirrhosis (AST &gt; ALT), liver abscess, metastatic or primary liver cancer; right heart failure, ischemia or hypoxia, injury to liver (“shock liver”), extensive trauma. Drugs that cause cholestasis or hepatotoxicity. <strong>Decreased in:</strong> Pyridoxine (vitamin B6) deficiency.</td>
<td>ALT is the preferred enzyme for evaluation of liver injury. Screening ALT in low-risk populations has a low (12%) positive predictive value. Compr Ther 1994;20:50. Hosp Pract (Off Ed) Nov 1994;29:32. Dig Dis Sci 1993;38:2145.</td>
</tr>
<tr>
<td>0–35 U/L [0–0.58 µkat/L] (laboratory-specific)</td>
<td>Marbled $</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Albumin, serum</strong></td>
<td><strong>Marbled</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------</td>
<td>------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.4–4.7 g/dL</td>
<td>$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>[34–47 g/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Major component of plasma proteins; influenced by nutritional state, hepatic function, renal function, and various diseases. Major binding protein. While there are more than 50 different genetic variants (allo-albumins), only occasionally does a mutation cause abnormal binding (eg, in familial dysalbuminemic hyperthyroxinemia).**

**Increased in:** Dehydration, shock, hemococoncentration.

**Decreased in:** Decreased hepatic synthesis (chronic liver disease, malnutrition, malabsorption, malignancy, congenital analbuminemia [rare]). Increased losses (nephrotic syndrome, burns, trauma, hemorrhage with fluid replacement, fistulas, enteropathy, acute or chronic glomerulonephritis). Hemodilution (pregnancy, CHF). Drugs: estrogens.

**Serum albumin gives an indication of severity in chronic liver disease. Useful in nutritional assessment if there is no impairment in production or increased loss of albumin and is an independent risk factor for all-cause mortality in the elderly (age >70). There is a 10% reduction in serum albumin level in late pregnancy (related to hemodilution).**

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Aldosterone, plasma</strong></td>
<td>Aldosterone is the major mineralocorticoid hormone and is a major regulator of extracellular volume and serum potassium concentration. For evaluation of hyperaldosteronism (associated with hypertension and hypokalemia), patients should be salt-loaded and recumbent when specimen is drawn. For evaluation of hypoaldosteronism (associated with hyperkalemia), patients should be salt-depleted and upright when specimen is drawn.</td>
<td><strong>Increased in:</strong> Primary hyperaldosteronism (72%). <strong>Decreased in:</strong> Primary or secondary hypoaldosteronism.</td>
<td>Testing for hyperaldosteronism and hypoaldosteronism must be done using specific protocols, and results must be interpreted based on reference values from the laboratory performing the test. 24-hour urinary excretion of aldosterone is the most sensitive test for hyperaldosteronism. (See Aldosterone, urine, below.) The significance of an elevated plasma aldosterone level is difficult to interpret without simultaneous determination of plasma renin activity (PRA). In primary aldosteronism, plasma aldosterone is usually elevated while PRA is low; in secondary hyperaldosteronism, both plasma aldosterone and PRA are usually elevated. Am J Med 1983;74:641. Med Clin North Am 1988;72:1117. Mayo Clin Proc 1990;65:96.</td>
</tr>
<tr>
<td><strong>Salt-loaded</strong> (120 meq Na⁺/d): Supine: 3–10 Upright: 5–30 ng/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Salt-depleted</strong> (10 meq Na⁺/d): Supine: 12–36 Upright: 17–137 ng/dL [1 ng/dL = 27.7 pmol/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lavender or green $$$$$ Early AM fasting specimen. Separate immediately and freeze.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Aldosterone, urine**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Range (µg/24 h)</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Salt-loaded</strong> (120 meq Na+/d for 3–4 days):</td>
<td>1.5–12.5</td>
<td>-</td>
</tr>
<tr>
<td><strong>Salt-depleted</strong> (20 meq Na+/d for 3–4 days):</td>
<td>18–85</td>
<td>-</td>
</tr>
</tbody>
</table>

[1 µg/24 h = 2.77 nmol/d]

Bottle containing boric acid

* To evaluate hyperaldosteronism, patient is salt-loaded and recumbent. Obtain 24-hour urine for aldosterone (and sodium to check that sodium excretion is >250 meq/day). To evaluate hypoaldosteronism, patient is salt-depleted and upright; check patient for hypotension before 24-hour urine collected.

Secretion of aldosterone is controlled by the renin-angiotensin system. Renin (synthesized and stored in juxtaglomerular cells of kidney) is released in response to both decreased perfusion pressure at the juxtaglomerular apparatus and negative sodium balance. Renin then hydrolyses angiotensinogen to angiotensin I, which is converted to angiotensin II, which then stimulates the adrenal gland to produce aldosterone.

**Increased in:** Primary and secondary hyperaldosteronism, some patients with essential hypertension.

**Decreased in:** Primary hypoaldosteronism (eg, 18-hydroxylase deficiency), secondary hypoaldosteronism (hyporeninemic hypoaldosteronism).

Urinary aldosterone is the most sensitive test for primary hyperaldosteronism. Levels >14 µg/24h after 3 days of salt-loading have a 96% sensitivity and 93% specificity for primary hyperaldosteronism. Only 7% of patients with essential hypertension have urinary aldosterone levels >14 µg/24h after salt-loading. Neither serum potassium nor plasma renin activity (PRA) is a satisfactory screening test for hyperaldosteronism. Hypokalemia is present in only 73% of patients with hyperaldosteronism on a normal sodium diet, and in 86% after salt loading. Suppressed PRA has only a 64% sensitivity and 83% specificity for hyperaldosteronism.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amebic serology, serum</strong>&lt;br&gt;41–133 IU/L [0.7–2.2 µkat/L] (method- and age-dependent)</td>
<td>Test for presence of <em>Entamoeba histolytica</em> by detection of antibodies which develop 2–4 weeks after infection. Tissue invasion by the organism may be necessary for antibody production.</td>
<td>Increased in: Current or past infection with <em>E histolytica</em>. Amebic abscess (91%), amebic dysentery (84%), asymptomatic cyst carriers (9%), patients with other diseases and healthy people (2%).</td>
<td>In some endemic areas, as many as 44% of those tested have positive serologies. Precipitin or indirect hemagglutination (IHA) and recombinant antigen-based ELISA tests are available. N Engl J Med 1978;298:262. Ann Trop Parasitol 1993;87:31.</td>
</tr>
<tr>
<td><strong>Alkaline phosphatase, serum</strong></td>
<td>Alkaline phosphatases are found in liver, bone, intestine, and placenta.</td>
<td>Increased in: Obstructive hepatobiliary disease, bone disease (physiologic bone growth, Paget’s disease, osteomalacia, osteogenic sarcoma, bone metastases), hyperparathyroidism, rickets, benign familial hyperphosphatasemia, pregnancy (third trimester), GI disease (perforated ulcer or bowel infarct), hepatotoxic drugs. Decreased in: Hypophosphatasia.</td>
<td>Alkaline phosphatase performs well in measuring the extent of bone metastases in prostate cancer. Normal in osteoporosis. Alkaline phosphatase isoenzyme separation by electrophoresis or differential heat inactivation is unreliable. Use γ-glutamyl transpeptidase (GGT), which increases in hepatobiliary disease but not in bone disease, to infer origin of increased alkaline phosphatase (ie, liver or bone). Endocrinol Metab Clin North Am 1990;19:1. Int J Urol 1997;4:572.</td>
</tr>
<tr>
<td>Marbled $</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| **Ammonia, plasma** (NH₃)  
18–60 µg/dL  
[11–35 µmol/L] | **Ammonia** is liberated by bacteria in the large intestine or by protein metabolism and is rapidly converted to urea in liver.  
In liver disease or portal-systemic shunting, the blood ammonia concentration increases.  
In acute liver failure, elevation of blood ammonia may cause brain edema; in chronic liver failure, it may be responsible for hepatic encephalopathy. | **Increased in:** Liver failure, hepatic encephalopathy (especially if protein consumption is high or if there is GI bleeding), fulminant hepatic failure, Reye’s syndrome, portacaval shunting, cirrhosis, urea cycle metabolic defects, urea-splitting urinary tract infection with urinary diversion, and organic acidemias. Drugs: diuretics, acetazolamide, asparaginase, fluorouracil (5-FU) (transient), others.  
Spuriously increased by any ammonia-containing detergent on laboratory glassware.  
**Decreased in:** Decreased production by gut bacteria (kanamycin, neomycin). Decreased gut absorption (lactulose). | Correlates poorly with degree of hepatic encephalopathy. Test not useful in adults with known liver disease. Test is not as useful as CSF glutamine (see p 97).  
Green $$  
Separate plasma from cells immediately.  
Avoid hemolysis.  
Analyze immediately.  
Place on ice. |
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Amylase, serum</strong></td>
<td>Amylase hydrolyzes complex carbohydrates. Serum amylase is derived primarily from pancreas and salivary glands and is increased with inflammation or obstruction of these glands. Other tissues have some amylase activity, including ovaries, small and large intestine, and skeletal muscle.</td>
<td><strong>Increased in:</strong> Acute pancreatitis (70–95%), pancreatic pseudocyst, pancreatic duct obstruction (cholecystitis, cholelithiasis, pancreatic cancer, stone, stricture, duct spasm), bowel obstruction and infarction, mumps, parotitis, diabetic ketoacidosis, penetrating peptic ulcer, peritonitis, ruptured ectopic pregnancy, macroamylasemia. Drugs: azathioprine, hydrochlorothiazide. <strong>Decreased in:</strong> Pancreatic insufficiency, cystic fibrosis. Usually normal or low in chronic pancreatitis.</td>
<td>Macroamylasemia is indicated by high serum but low urine amylase. Serum lipase is an alternative test for acute pancreatitis. Amylase isoenzymes are not of practical use because of technical problems. Gastroenterol Clin North Am 1990;19:793. J Gastroenterol 1994;29:189. Gastroenterologist 1994;2:119. Pancreas 1998;16:45.</td>
</tr>
<tr>
<td><strong>Angiotensin-converting enzyme, serum (ACE)</strong></td>
<td>ACE is a dipeptidyl carboxypeptidase that converts angiotensin I to the vasopressor, angiotensin II. ACE is normally present in the kidneys and other peripheral tissues. In granulomatous disease, ACE levels increase, derived from epithelioid cells within granulomas.</td>
<td><strong>Increased in:</strong> Sarcoidosis (sensitivity = 63%, specificity = 93%, LRT = 9.0) (when upper limit of normal is 50), hyperthyroidism, acute hepatitis, primary biliary cirrhosis, diabetes mellitus, multiple myeloma, osteoarthritis, amyloidosis, Gaucher’s disease, pneumoconiosis, histoplasmosis, miliary tuberculosis. Drugs: dexamethasone. <strong>Decreased in:</strong> Renal disease, obstructive pulmonary disease, hypothyroidism.</td>
<td>Test is not useful as a screening test for sarcoidosis (low sensitivity). Specificity is compromised by positive tests in diseases more common than sarcoidosis. Some advocate measurement of ACE to follow disease activity in sarcoidosis. J Clin Pathol 1983;36:938.</td>
</tr>
<tr>
<td>Antibody screen, serum</td>
<td>Detects antibodies to non-ABO red blood cell antigens in recipient’s serum, using reagent red cells selected to possess antigens against which common antibodies can be produced. Further identification of the specificity of any antibody detected (using panels of red cells of known antigenicity) makes it possible to test donor blood for the absence of the corresponding antigen.</td>
<td>Positive in: Presence of alloantibody, autoantibody.</td>
<td>In practice, a type and screen (ABO and Rh grouping and antibody screen) is adequate workup for patients undergoing operative procedures unlikely to require transfusion. A negative antibody screen implies that a recipient can receive type-specific (ABO-Rh identical) blood with minimal risk. Technical Manual of the American Association of Blood Banks, 11th ed. American Association of Blood Banks, 1993.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Antidiuretic hormone, plasma (ADH)</strong></td>
<td>Antidiuretic hormone (vasopressin) is a hormone secreted from the posterior pituitary that acts on the distal nephron to conserve water and regulate the tonicity of body fluids. Water deprivation provides both an osmotic and a volume stimulus for ADH release by increasing plasma osmolality and decreasing plasma volume. Water administration lowers plasma osmolality and expands blood volume, inhibiting the release of ADH by the osmoreceptor and the atrial volume receptor mechanisms.</td>
<td>Increased in: Nephrogenic diabetes insipidus, syndrome of inappropriate antidiuretic hormone (SIADH). Drugs: nicotine, morphine, chlorpropamide, clofibrate, cyclophosphamide. Normal relative to plasma osmolality in: Primary polydipsia. Decreased in: Central (neurogenic) diabetes insipidus. Drugs: ethanol, phenytoin.</td>
<td>Test very rarely indicated. Measurement of serum and urine osmolality usually suffices. Test not indicated in diagnosis of SIADH. Patients with SIADH show decreased plasma sodium and decreased plasma osmolality, usually with high urine osmolality relative to plasma. These findings in a normovolemic patient with normal thyroid and adrenal function are sufficient to make the diagnosis of SIADH without measuring ADH itself. Semin Nephrol 1994;14:368.</td>
</tr>
</tbody>
</table>

**Antibody screen**

Red $Properly identified and labeled blood specimens are critical.

Lavender $$$$ Draw in two chilled tubes and deliver to lab on ice. Specimen for serum osmolality must be drawn at same time.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antiglobulin test,</strong> <strong>direct,</strong> red cells <em>(Direct Coombs, DAT)</em></td>
<td>Direct antiglobulin test demonstrates in vivo coating of washed red cells with globulins, in particular IgG and C3d. Washed red cells are tested directly with antihuman globulin reagent. DAT is positive (shows agglutination) immediately when IgG coats red cells. Complement or IgA coating may only be demonstrated after incubation at room temperature.</td>
<td>Positive in: Autoimmune hemolytic anemia, hemolytic disease of the newborn, alloimmune reactions to recently transfused cells, and drug-induced hemolysis. Drugs: cephalosporins, levodopa, methadone, methyldopa, penicillin, phenacetin, quinidine.</td>
<td>A positive DAT implies in vivo red cell coating by immunoglobulins or complement. Such red cell coating may or may not be associated with immune hemolytic anemia. Poly-specific and anti-IgG reagents detect approximately 500 molecules of IgG per red cell, but autoimmune hemolytic anemia has been reported with IgG coating below this level. 10% of hospital patients have a positive DAT without clinical manifestations of immune-mediated hemolysis. A false-positive DAT is often seen in patients with hypergammaglobulinemia, eg, in some HIV-positive patients. Technical Manual of the American Association of Blood Banks, 11th ed. American Association of Blood Banks, 1993. The technique is used in antibody detection and identification and in the major cross-match prior to transfusion (see Type and Cross-Match, p 175). Technical Manual of the American Association of Blood Banks, 11th ed. American Association of Blood Banks, 1993.</td>
</tr>
<tr>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lavender or red $</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blood anticoagulated with EDTA is used to prevent in vitro uptake of complement components. A red top tube may be used, if necessary.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>α₁-Antiprotease (α₁-antitrypsin), serum</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>110–270 mg/dL</strong> [1.1–2.7 g/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Antistreptolysin O titer, serum (ASO)**

- **Children <5 years:** <85
- **5–19 years:** <170
- **Adults:** <85 Todd units (laboratory-specific)

Marbled $$

**α₁-Antiprotease** is an α₁ globulin glycoprotein serine protease inhibitor (Pi) whose deficiency leads to excessive protease activity and panacinar emphysema in adults or liver disease in children (seen as ZZ and SZ phenotypes). Cirrhosis of the liver and liver cancer in adults are also associated with the Pi Z phenotype.

**Increased in:** Inflammation, infection, rheumatic disease, malignancy, and pregnancy because it is an acute phase reactant.

**Decreased in:** Congenital α₁-antiprotease deficiency, nephrotic syndrome.

Smoking is a much more common cause of chronic obstructive pulmonary disease in adults than is α₁-antiprotease deficiency.


**Antistreptolysin O titer**

Detected the presence of antibody to the antigen streptolysin O produced by group A streptococci. Streptococcal antibodies appear about 2 weeks after infection. Titer rises to a peak at 4–6 weeks and may remain elevated for 6 months to 1 year. Test is based on the neutralization of hemolytic activity of streptolysin O toxin by antistreptolysin O antibodies in serum.

**Increased in:** Recent infection with group A beta-hemolytic streptococci: scarlet fever, erysipelas, streptococcal pharyngitis/tonsillitis (40–50%), rheumatic fever (80–85%), poststreptococcal glomerulonephritis. Some collagen-vascular diseases. Certain serum lipoproteins, bacterial growth products, or oxidized streptolysin O may result in inhibition of hemolysis and thus cause false-positive results.

**Increased in:** Influenza, infection, rheumatic disease, malignancy, and pregnancy because it is an acute phase reactant.

**Decreased in:** Congenital α₁-antiprotease deficiency, nephrotic syndrome.

Standardization of (Todd) units may vary significantly from laboratory to laboratory.

ASO titers are not useful in management of acute streptococcal pharyngitis.

In patients with rheumatic fever, test may be a more reliable indicator of recent streptococcal infection than throat culture.

An increasing titer is more suggestive of acute streptococcal infection than a single elevated level. Even with severe infection, ASO titers will rise in only 70–80% of patients.


<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antithrombin III (AT III), plasma 84–123% (qualitative) 22–39 mg/dL (quantitative) Blue $$</td>
<td>Antithrombin III is a serine protease inhibitor that protects against thrombus formation by inhibiting thrombin and factors IXa, Xa, XIa, XIIa, plasmin, and kallikrein. It accounts for 70–90% of the anticoagulant activity of human plasma. Its activity is enhanced 100-fold by heparin. There are two types of assay: functional (qualitative) and immunologic (quantitative). Since the immunologic assay cannot rule out functional AT III deficiency, a functional assay should be ordered first. Functional assays test AT III activity in inhibiting thrombin or factor Xa. Given an abnormal functional assay, the quantitative immunologic test indicates whether there is decreased synthesis of AT III or intact synthesis of a dysfunctional protein.</td>
<td>Increased by: Oral anticoagulants. Decreased in: Congenital and acquired AT III deficiency (renal disease, chronic liver disease), oral contraceptive use, chronic disseminated intravascular coagulation, acute venous thrombosis (consumption), and heparin therapy.</td>
<td>Congenital and acquired AT III deficiency results in a hypercoagulable state, venous thromboembolism, and heparin resistance. Congenital AT III deficiency is present in 1:2000–1:5000 people and is autosomal codominant. Heterozygotes have AT III levels 20–60% of normal. Semin Thromb Hemost 1982;8:276. Thromb Haemost 1993;69:231.</td>
</tr>
<tr>
<td>Aspartate aminotransferase, serum (AST, SGOT, GOT) 0–35 IU/L [0–0.58 μkat/L] (laboratory-specific) Marbled $</td>
<td>Intracellular enzyme involved in amino acid metabolism. Present in large concentrations in liver, skeletal muscle, brain, red cells, and heart. Released into the bloodstream when tissue is damaged, especially in liver injury.</td>
<td>Increased in: Acute viral hepatitis (ALT &gt; AST), biliary tract obstruction (cholangitis, choledocholithiasis), alcoholic hepatitis and cirrhosis (AST &gt; ALT), liver abscess, metastatic or primary liver cancer; right heart failure, ischemia or hypoxia, injury to liver (“shock liver”), extensive trauma. Drugs that cause cholestasis or hepatotoxicity. Decreased in: Pyridoxine (vitamin B6) deficiency.</td>
<td>Test is not indicated for diagnosis of myocardial infarction. AST/ALT ratio &gt; 1 suggests cirrhosis in patients with hepatitis C. Compr Ther 1994;20:50. Hosp Pract (Off Ed) Nov 1994;29:32. Am J Gastroenterol 1998;93:44.</td>
</tr>
<tr>
<td><strong>B cell immunoglobulin heavy chain gene rearrangement</strong></td>
<td>In general, the percentage of B lymphocytes with identical immunoglobulin heavy chain gene rearrangements is very low; in malignancies, however, the clonal expansion of one population leads to a large number of cells with identical B cell immunoglobulin heavy chain gene rearrangements. Southern blot is used to identify a monoclonal population.</td>
<td><strong>Positive in:</strong> B cell neoplasms such as lymphoma.</td>
<td>Samples with &gt; 10% of cells showing a given B cell rearrangement are considered positive. However, a large monoclonal population is consistent with—but not diagnostic of—malignancy. Arch Path Lab Med 1988;112:117.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Whole blood, bone marrow, or frozen tissue</td>
<td>Lavender</td>
<td><strong>bcr/abl translocation</strong></td>
<td>Blood</td>
</tr>
<tr>
<td><strong>bcr/abl translocation</strong></td>
<td>Approximately 95% of chronic myelogenous leukemia (CML) is associated with the “Philadelphia chromosome,” a translocation that moves the c-abl proto-oncogene from chromosome 9 to the break-point cluster (bcr) region of chromosome 22. Southern blot is used to identify the translocation.</td>
<td><strong>Positive in:</strong> Chronic myelogenous leukemia (sensitivity 95%) and acute lymphocytic leukemia (sensitivity 10–15%).</td>
<td>This assay will detect the 9;22 translocation if it has taken place in &gt;10% of the cells. CML patients with bone marrow transplants can be monitored for recurrence of disease with this test. N Engl J Med 1988;319:990.</td>
</tr>
<tr>
<td>Lavender</td>
<td>Lavender</td>
<td>$$$</td>
<td>$$</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Bilirubin, serum</strong></td>
<td>Bilirubin, a product of hemoglobin metabolism, is conjugated in the liver to mono- and diglucuronides and excreted in bile. Some conjugated bilirubin is bound to serum albumin, so-called D (delta) bilirubin. Elevated serum bilirubin occurs in liver disease, biliary obstruction, or hemolysis.</td>
<td><strong>Increased in:</strong> Acute or chronic hepatitis, cirrhosis, biliary tract obstruction, toxic hepatitis, neonatal jaundice, congenital liver enzyme abnormalities (Dubin-Johnson, Rotor’s, Gilbert’s, Crigler-Najjar syndromes), fasting, hemolytic disorders. Hepatotoxic drugs.</td>
<td>Assay of total bilirubin includes conjugated (direct) and unconjugated (indirect) bilirubin plus delta bilirubin (conjugated bilirubin bound to albumin). It is usually clinically unnecessary to fractionate total bilirubin. The fractionation is unreliable by the diazo reaction and may underestimate unconjugated bilirubin. Only conjugated bilirubin appears in the urine, and it is indicative of liver disease; hemolysis is associated with increased unconjugated bilirubin. Persistence of delta bilirubin in serum in resolving liver disease means that total bilirubin does not effectively indicate the time course of resolution. Pediatrics 1992;89:80. Br J Hosp Med 1994;51:181. Pediatr Rev 1994;15:233.</td>
</tr>
<tr>
<td><strong>0.1–1.2 mg/dL</strong></td>
<td>0.1–1.2 mg/dL [2–21 \mu\text{mol/L}]</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Direct (conjugated to glucuronide) bilirubin:</strong></td>
<td>0.1–0.4 mg/dL [&lt;7 \mu\text{mol/L}]; Indirect (unconjugated) bilirubin: 0.2–0.7 mg/dL [&lt;12 \mu\text{mol/L}]</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Marbled $$$</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Bleeding time</strong></td>
<td><strong>Increased in:</strong> Platelet disorders, thrombocytopenia, Bernard-Soulier syndrome, thrombasthenia. Also elevated in some forms of von Willebrand’s disease, which is a disorder of factor VIII coagulant activity and not primarily a platelet disorder. Drugs: aspirin and other preparations containing aspirin.</td>
<td>Test is useful as a screening test (with aspirin challenge) for diagnosis of von Willebrand’s disease and platelet disorders. Test adds no clinically useful information to the prediction of clinically significant bleeding beyond that obtained from the history, physical examination, and other laboratory tests—platelet count, blood urea nitrogen (BUN), prothrombin time (PT), and partial thromboplastin time (PTT). In patients with no history of bleeding and no intake of nonsteroidal anti-inflammatory drugs, an increased bleeding time does not correlate with actual surgical bleeding. Semin Thromb Hemost 1990;16:1. Blood 1994;84:3363. Med Clin North Am 1994;78:577.</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>2–10 minutes</td>
<td>This is a test of platelet function, not a test of coagulation factors.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$$ Test done by laboratory personnel. Simplate (presterilized device with spring-loaded blade) is used to make single cut 1 mm deep and 6 mm long on dorsal aspect of forearm after inflation of sphygmomanometer to 40 mm Hg. Filter paper is used to absorb blood from wound margins every 30 seconds, and time to cessation of bleeding is noted.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>--------------------------</td>
<td>-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Blood urea nitrogen, serum</strong> (BUN)</td>
<td>Urea, an end product of protein metabolism, is excreted by the kidney. BUN is directly related to protein intake and nitrogen metabolism and inversely related to the rate of excretion of urea. Urea concentration in glomerular filtrate is the same as in plasma, but its tubular reabsorption is inversely related to the rate of urine formation. Thus, the BUN is a less useful measure of glomerular filtration rate than the serum creatinine (Cr).</td>
<td><strong>Increased in:</strong> Renal failure (acute or chronic), urinary tract obstruction, dehydration, shock, burns, CHF, GI bleeding. Nephrotoxic drugs (eg, gentamicin). <strong>Decreased in:</strong> Hepatic failure, nephrotic syndrome, cachexia (low-protein and high-carbohydrate diets).</td>
<td>Urease assay method commonly used. BUN/Cr ratio (normally 12:1–20:1) is decreased in acute tubular necrosis, advanced liver disease, low protein intake, and following hemodialysis. BUN/Cr ratio is increased in dehydration, GI bleeding, and increased catabolism. Nursing 1994;24:88. Ann Emerg Med 1992;21:713.</td>
</tr>
<tr>
<td><strong>Brucella antibody, serum</strong></td>
<td>Patients with acute brucellosis generally develop an agglutinating antibody titer of $\geq 1:160$ within 3 weeks. The titer may rise during the acute infection, with relapses, brucellergin skin testing, or use of certain vaccines (see Interpretation). The agglutinin titer usually declines after 3 months or after successful therapy. Low titers may persist for years.</td>
<td><strong>Increased in:</strong> <em>Brucella</em> infection (except <em>B canis</em>) (97% within 3 weeks of illness); recent brucellergin skin test; infections with <em>Francisella tularensis, Yersinia enterocolitica</em>, salmonella, Rocky mountain spotted fever; vaccinations for cholera and tularemia. <strong>Normal in:</strong> <em>B canis</em> infection.</td>
<td>This test will detect antibodies against all of the <em>Brucella</em> species except <em>B canis</em>. A fourfold or greater rise in titer in separate specimens drawn 1–4 weeks apart is indicative of recent exposure. Final diagnosis depends on isolation of organism by culture. J Clin Microbiol 1980;11:691. J Infect Dis 1989;159:219. Rev Infect Dis 1991;13:359.</td>
</tr>
<tr>
<td><strong>C-reactive protein, serum</strong></td>
<td>Marker of inflammation.</td>
<td><strong>Increased in:</strong> Inflammatory states.</td>
<td>Elevated C-reactive protein level appears to be an independent risk factor for coronary heart disease events. Ann Intern Med 1999;130:933.</td>
</tr>
<tr>
<td><strong>C1 esterase inhibitor (C1 INH), serum</strong></td>
<td>C1 esterase inhibitor (C1 INH) is an alpha-globulin, which controls the first stage of the classic complement pathway and inhibits thrombin, plasmin, and kallikrein. Deficiency results in spontaneous activation of C1, leading to consumption of C2 and C4. The functional assay involves the measurement of C1 INH as it inhibits the hydrolysis of a substrate ester by C1 esterase. Immunoassay of C1 INH is also available.</td>
<td><strong>Decreased in:</strong> Hereditary angioedema (HAE) (85%) (15% of patients with HAE will have normal levels by immunoassay, but the protein is non-functional and levels determined by the functional assay will be low).</td>
<td>C1 esterase inhibitor deficiency is an uncommon cause of angioedema. There are two subtypes of hereditary angioedema. In one, the protein is absent; in the other, it is non-functional. Acquired angioedema has been attributed to massive consumption of C1 INH (presumably by tumor or lymphoma-related immune complexes) or to anti-C1 INH autoantibody. When clinical suspicion exists, a serum C4 level screens for HAE. Low levels of C4 are present in all cases during an attack. C1 esterase inhibitor levels are not indicated unless either the C4 level is low or there is a very high clinical suspicion of HAE in a patient with normal C4 during an asymptomatic phase between attacks. In acquired C1 INH deficiency, the C1 level is also significantly decreased (often 10% of normal), whereas in HAE the C1 level is normal or only slightly decreased. Am J Med 1990;88:656. Ann Allergy 1991;67(2 Part 1):107. Med Clin North Am 1992;76:805. South Med J 1992;85:1084.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>C-peptide, serum</strong></td>
<td>C-peptide is an inactive by-product of the cleavage of proinsulin to active insulin. Its presence indicates endogenous release of insulin. C-peptide is largely excreted by the kidney.</td>
<td><strong>Increased in:</strong> Renal failure, ingestion of oral hypoglycemic drugs, insulinomas, B cell transplants. <strong>Decreased in:</strong> Factitious hypoglycemia due to insulin administration, pancreatectomy, type I diabetes mellitus (decreased or undetectable).</td>
<td>Test is most useful to detect factitious insulin injection (increased insulin, decreased C-peptide) or to detect endogenous insulin production in diabetic patients receiving insulin (C-peptide present). A molar ratio of insulin to C-peptide in peripheral venous blood &gt;1.0 in a hypoglycemic patient is consistent with surreptitious or inadvertent insulin administration but not insulinoma. Arch Intern Med 1977;137:625. Am J Med 1989;86:335. Arch Intern Med 1993;153:650.</td>
</tr>
<tr>
<td>0.8–4.0 ng/mL [µg/L] Marbled $$$ Fasting sample preferred.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Calcitonin, plasma</strong></td>
<td>Calcitonin is a 32-amino-acid poly-peptide hormone secreted by the parafollicular C cells of the thyroid. It decreases osteoclastic bone resorption and lowers serum calcium levels.</td>
<td><strong>Increased in:</strong> Medullary thyroid carcinoma (&gt;500 pg/mL on two occasions), Zollinger-Ellison syndrome, pernicious anemia, pregnancy (at term), newborns, carcinoma (breast, lung, pancreas), chronic renal failure.</td>
<td>Test is useful to diagnose and monitor medullary thyroid carcinoma, although stimulation tests may be necessary (eg, pentagastrin test). Genetic testing is now available for the diagnosis of multiple endocrine neoplasia type II. (MEN II is the most common familial form of medullary thyroid carcinoma.) Mayo Clin Proc 1975;50:53. Ann Intern Med 1995;122:118.</td>
</tr>
<tr>
<td>Male: &lt;90 pg/mL [ng/L] Female: &lt;70 pg/mL [ng/L] Green $$$ Fasting sample required. Place on ice.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Calcium, serum (Ca^{2+})**

8.5–10.5 mg/dL
[2.1–2.6 mmol/L]

**Panic:** <6.5 or >13.5 mg/dL

Marbled

$\text{Prolonged venous stasis during collection causes false increase in serum calcium.}$

Serum calcium is the sum of ionized calcium plus complexed calcium and calcium bound to proteins (mostly albumin).

Level of ionized calcium is regulated by parathyroid hormone and vitamin D.

**Increased in:** Hyperparathyroidism, malignancies secreting parathyroid hormone–related protein (PTHrP) (especially squamous cell carcinoma of lung and renal cell carcinoma), vitamin D excess, milk-alkali syndrome, multiple myeloma, Paget’s disease of bone with immobilization, sarcoidosis, other granulomatous disorders, familial hypocalciuria, vitamin A intoxication, thyrotoxicosis, Addison’s disease.

Drugs: antacids (some), calcium salts, chronic diuretic use (eg, thiazides), lithium, others.

**Decreased in:** Hypoparathyroidism, vitamin D deficiency, renal insufficiency, pseudohypoparathyroidism, magnesium deficiency, hyperphosphatemia, massive transfusion, hypoalbuminemia.

Need to know serum albumin to interpret calcium level. For every decrease in albumin by 1 mg/dL, calcium should be corrected upward by 0.8 mg/dL. In 10% of patients with malignancies, hypercalcemia is attributable to coexistent hyperparathyroidism, suggesting that serum PTH levels should be measured at initial presentation of all hypercalcemic patients (see pp 134 and 354).

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium, ionized, serum</td>
<td>Calcium circulates in three forms: as free Ca$^{2+}$ (47%), protein-bound to albumin and globulins (43%), and as calcium-ligand complexes (10%) (with citrate, bicarbonate, lactate, phosphate, and sulfate). Protein binding is highly pH-dependent, and acidosis results in an increased free calcium fraction. Ionized Ca$^{2+}$ is the form that is physiologically active. Ionized calcium is a more accurate reflection of physiologic status than total calcium in patients with altered serum proteins (renal failure, nephrotic syndrome, multiple myeloma, etc), altered concentrations of calcium-binding ligands, and acid-base disturbances. Measurement of ionized calcium is by ion-selective electrodes.</td>
<td>Increased in: ↓ blood pH. Decreased in: ↑ blood pH, citrate, heparin, EDTA.</td>
<td>Ionized calcium measurements are not needed except in special circumstances, eg, massive blood transfusion, liver transplantation, neonatal hypocalcemia, and cardiac surgery. Validity of test depends on sample integrity. Ann Clin Lab Sci 1991;21:297.</td>
</tr>
</tbody>
</table>
**Calcium, urine** (U<sub>Ca</sub>)

- **100–300 mg/24 h** (2.5–7.5 mmol/24 h or 2.3–3.3 mmol/12 h)
- Urine bottle containing hydrochloric acid

### Collect 24-hour urine or 12-hour overnight urine.

**Ordinarily there is moderate urinary calcium excretion, the amount depending on dietary calcium, parathyroid hormone (PTH) level, and protein intake.**

Renal calculi occur much more often in hyperparathyroidism than in other hypercalcemic states.

**Increased in:** Hyperparathyroidism, osteolytic bone metastases, myeloma, osteoporosis, vitamin D intoxication, distal RTA, idiopathic hypercalciuria, thyrotoxicosis, Paget’s disease, Fanconi’s syndrome, hepatolenticular degeneration, schistosomiasis, sarcoidosis, malignancy (breast, bladder), osteitis deformans, immobilization.

Drugs: acetazolamide, calcium salts, cholestyramine, corticosteroids, dihydrotachysterol, initial diuretic use (eg, furosemide), others.

**Decreased in:** Hypoparathyroidism, pseudohypoparathyroidism, rickets, osteomalacia, nephrotic syndrome, acute glomerulonephritis, osteoblastic bone metastases, hypothyroidism, celiac disease, steatorrhea, hypocalciuric hypercalcemia, other causes of hypocalcemia.

Drugs: aspirin, bicarbonate, chronic diuretic use (eg, thiazides, chlorothalidone), estrogens, indomethacin, lithium, neomycin, oral contraceptives.

**Approximately one-third of patients with hyperparathyroidism have normal urine calcium excretion.**

The extent of calcium excretion can be expressed as a urine calcium (U<sub>Ca</sub>)/urine creatinine (U<sub>Cr</sub>) ratio.

Normally,

\[
\frac{U_{Ca}(mg/dL)}{U_{Cr}(mg/dL)} < 0.14
\]

and

\[
\frac{U_{Ca}(mmol/L)}{U_{Cr}(mmol/L)} < 0.40
\]

Hypercalciuria is defined as a ratio >0.20 or >0.57, respectively.

Test is useful in the evaluation of renal stones but is not usually needed for the diagnosis of hyperparathyroidism, which can be made using serum calcium (see above) and PTH measurements (see pp 134 and 354). It may be useful in hypercalcemic patients to rule out familial hypocalciuric hypercalcemia.

In the diagnosis of hypercalciuria, U<sub>Ca</sub>/U<sub>Cr</sub> ratios in random single-voided urine specimens correlate well with 24-hour calcium excretions.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Carbon dioxide</strong> (CO(_2), total, serum (bicarbonate))</td>
<td>Bicarbonate-carbonic acid buffer is one of the most important buffer systems in maintaining normal body fluid pH. Total CO(_2) is measured as the sum of bicarbonate concentration plus carbonic acid concentration plus dissolved CO(_2). Since bicarbonate makes up 90–95% of the total CO(_2) content, total CO(_2) is a useful surrogate for bicarbonate concentration.</td>
<td><strong>Increased in:</strong> Primary metabolic alkalosis, compensated respiratory acidosis, volume contraction, mineralocorticoid excess, congenital chloride diarrhea. Drugs: diuretics (eg, thiazide, furosemide). <strong>Decreased in:</strong> Metabolic acidosis, compensated respiratory alkalosis. Fanconi’s syndrome, volume overload. Drugs: acetazolamide, outdated tetracycline.</td>
<td>Total CO(_2) determination is indicated for all seriously ill patients on admission. If arterial blood gas studies are done, total CO(_2) test is redundant. Simultaneous measurement of pH and Pco(_2) is required to fully characterize a patient’s acid-base status.</td>
</tr>
<tr>
<td><strong>Carboxyhemoglobin, whole blood (HbCO)</strong></td>
<td>Carbon monoxide (CO) combines irreversibly with hemoglobin at the sites that normally bind oxygen. This produces a decrease in oxygen saturation and a shift in the oxyhemoglobin dissociation curve, resulting in decreased release of oxygen to the tissues.</td>
<td><strong>Increased in:</strong> Carbon monoxide poisoning. Exposure to automobile exhaust or smoke from fires. Cigarette smokers can have up to 9% carboxyhemoglobin, nonsmokers have &lt;2%.</td>
<td>Test (if available within minutes, together with O(_2) saturation by oximeter) is useful in evaluation of CO poisoning. PO(_2) is usually normal in CO poisoning. Test measures carboxyhemoglobin spectrophotometrically. N Engl J Med 1989;321:1474.</td>
</tr>
<tr>
<td>Carcinoembryonic antigen, serum (CEA)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0–2.5 ng/mL [µg/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CEA is an oncofetal antigen, a glycoprotein associated with certain malignancies, particularly epithelial tumors.

**Increased in:** Colon cancer (72%), lung cancer (76%), pancreatic cancer (91%), stomach cancer (61%), cigarette smokers, benign liver disease (acute 50% and chronic 90%), benign GI disease (peptic ulcer, pancreatitis, colitis). Elevations >20 ng/mL are generally associated with malignancy. For breast cancer recurrence (using 5 ng/mL cut-off), sensitivity = 44.4% and specificity = 95.5%.

**Screening:** Test is not sensitive or specific enough to be useful in cancer screening.

**Monitoring after surgery:** Test is used to follow progression of colon cancer after surgery (elevated CEA levels suggest recurrence 3–6 months before other clinical indicators), although such monitoring has not yet been shown to improve survival rates. If monitoring is done, the same assay method must be used consistently in order to eliminate any method-dependent variability.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CD4/CD8 ratio, whole blood</strong></td>
<td>Lymphocyte identification depends on specific cell surface antigens (clusters of differentiation, CD), which can be detected with monoclonal antibodies using flow cytometry. CD4 cells are predominantly helper-inducer cells of the immunologic system. They react with peptide class II major histocompatibility complex antigens and augment B cell responses and T cell lymphokine secretion. CD4 cells are the major target of HIV-1. CD8 cells can be divided into suppressor cells, which decrease B cell responses, and cytotoxic T cells.</td>
<td><strong>Increased in:</strong> Rheumatoid arthritis, type I diabetes mellitus, SLE without renal disease, primary biliary cirrhosis, atopic dermatitis, Sézary syndrome, psoriasis, chronic autoimmune hepatitis. <strong>Decreased in:</strong> AIDS/HIV infection, SLE with renal disease, acute CMV infection, burns, graft-versus-host disease, sunburn, myelodysplasia syndromes, acute lymphocytic leukemia in remission, recovery from bone marrow transplantation, herpes infection, infectious mononucleosis, measles, ataxia-telangiectasia, vigorous exercise.</td>
<td>Progressive decline in the number and function of CD4 lymphocytes seems to be the most characteristic immunologic defect in AIDS. Absolute CD4 measurement is particularly useful (more useful than the CD4/CD8 ratio) in determining eligibility for therapy (usually when CD4 &lt; 500 cells/µL) and in monitoring the progress of the disease. Most AIDS-defining infections occur when the CD4 count drops below 200 cells/µL. Absolute CD4 count depends, analytically, on the reliability of the white blood cell differential count, as well as on the percentage of CD4 cells identified using the appropriate monoclonal antibody. Hematol Oncol Clin North Am 1991;5:215. Arch Intern Med 1994;154:1561.</td>
</tr>
</tbody>
</table>

Lavender $$$
If an absolute CD4 count is required, also request a CBC and differential.
**Centromere antibody, serum (ACA)**

Negative

Marbled $$

**Ceruloplasmin, serum**

20–35 mg/dL [200–350 mg/L]

Marbled $$

**Anticentromere antibodies** are antibodies to nuclear proteins of the kinetochore plate.

**Positive in:** CREST (70–90%), scleroderma (10–15%), Raynaud’s disease (10–30%).

In patients with connective tissue disease, the predictive value of a positive test is >95% for scleroderma or related disease (CREST, Raynaud’s disease). Diagnosis of CREST is made clinically (calcinosis, Raynaud’s disease, esophageal dysmotility, sclerodactyly, and telangiectasia).

In the absence of clinical findings, the test has low predictive value. (See also Autoantibodies table, p 367.)


In patients with connective tissue disease, the predictive value of a positive test is >95% for scleroderma or related disease (CREST, Raynaud’s disease). Diagnosis of CREST is made clinically (calcinosis, Raynaud’s disease, esophageal dysmotility, sclerodactyly, and telangiectasia).

**Ceruloplasmin, serum**

Ceruloplasmin, a 120,000–160,000 MW α₂-glycoprotein synthesized by the liver, is the main (95%) copper-carrying protein in human serum.

**Increased in:** Acute and chronic inflammation, pregnancy. Drugs: oral contraceptives, phenytoin.

**Decreased in:** Wilson’s disease (hepato-lenticular degeneration) (95%), CNS disease other than Wilson’s (15%), liver disease other than Wilson’s (23%), malabsorption, malnutrition, primary biliary cirrhosis, nephrotic syndrome, severe copper deficiency, Menkes’ disease (X-linked inherited copper deficiency).

Slitlamp examination for Kayser-Fleischer rings and serum ceruloplasmin level recommended for diagnosis of Wilson’s disease. Serum copper level is very rarely indicated. Screening all patients with liver disease is ineffective. 5% of patients with Wilson’s disease have low-normal levels of ceruloplasmin.


J Hepatol 1997;27:358.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Chloride, serum (Cl⁻) 98–107 meq/L [mmol/L] | Chloride, the principal inorganic anion of extracellular fluid, is important in maintaining normal acid-base balance and normal osmolality. If chloride is lost (as HCl or NH₄Cl), alkalosis ensues; if chloride is ingested or retained, acidosis ensues. | **Increased in:** Renal failure, nephrotic syndrome, renal tubular acidosis, dehydration, overtreatment with saline, hyperparathyroidism, diabetes insipidus, metabolic acidosis from diarrhea (loss of HCO₃⁻), respiratory alkalosis, hyperadrenocorticism. Drugs: acetazolamide (hyperchloremic acidosis), androgens, hydrochlorothiazide, salicylates (intoxication).  
### Cholesterol, serum

Desirable <200
Borderline 200–239
High risk >240 mg/dL
[Desirable <5.2
Borderline 5.2–6.1
High risk >6.2 mmol/L]

Marbled $
Fasting preferred for LDL cholesterol.
HDL and total cholesterol can be measured nonfasting.

Cholesterol level is determined by lipid metabolism, which is in turn influenced by heredity, diet, and liver, kidney, thyroid, and other endocrine organ functions.

Total cholesterol (TC) = low density lipoprotein (LDL) cholesterol + high density lipoprotein (HDL) cholesterol + (triglycerides [TG] / 5) (valid only if TG < 400).

Since LDL cholesterol is the clinically important entity, it is calculated as:

$$\text{LDL} = \text{TC} - \text{HDL} - \frac{\text{TG}}{5}$$

This calculation is valid only if specimen is obtained fasting (in order to obtain relevant triglyceride level).

**Increased in:** Primary disorders: polygenic hypercholesterolemia, familial hypercholesterolemia (deficiency of LDL receptors), familial combined hyperlipidemia, familial dysbetalipoproteinemia. Secondary disorders: hypothyroidism, uncontrolled diabetes mellitus, nephrotic syndrome, biliary obstruction, anorexia nervosa, hepatoma, Cushing’s syndrome, acute intermittent porphyria. Drugs: corticosteroids.

**Decreased in:** Severe liver disease (acute hepatitis, cirrhosis, malignancy), hyperthyroidism, severe acute or chronic illness, malnutrition, malabsorption (eg, HIV), extensive burns, familial (Gaucher’s disease, Tangier disease), abetalipoproteinemia, intestinal lymphangiectasia.

It is important to treat the cause of secondary hypercholesterolemia (eg, hypothyroidism). Need to check total cholesterol and HDL cholesterol because cardiovascular risk may be increased with relatively modest total cholesterol elevation if HDL cholesterol is low.

National Cholesterol Education Program Expert Panel has published clinical recommendations for cholesterol management (see JAMA 1993 reference).

JAMA 1993;260:3015.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Chorionic gonadotropin, β-subunit, quantitative, serum (β-hCG)</strong></td>
<td>Human chorionic gonadotropin is a glycoprotein made up of two sub-units (α and β). Human glycoproteins such as LH, FSH, and TSH share the α subunit of hCG, but the β subunit is specific for hCG. hCG is produced by trophoblastic tissue, and its detection in serum or urine is the basis for pregnancy testing. Serum hCG can be detected as early as 24 hours after implantation at a concentration of 5 mIU/mL. During normal pregnancy, serum levels double every 2–3 days and are 50–100 mIU/mL at the time of the first missed menstrual period. Peak levels are reached 60–80 days after the last menstrual period (LMP) (30,000–100,000 mIU/mL), and levels then decrease to a plateau of 5,000–10,000 mIU/mL at about 120 days after LMP and persist until delivery.</td>
<td><strong>Increased in:</strong> Pregnancy (including ectopic pregnancy), hyperemesis gravidarum, trophoblastic tumors (hydatidiform mole, choriocarcinoma of uterus), some germ cell tumors (teratomas of ovary or testicle, seminoma), ectopic hCG production by other malignancies (stomach, pancreas, lung, colon, liver). Failure of elevated serum levels to decrease after surgical resection of trophoblastic tumor indicates metastatic tumor; levels rising from normal indicate tumor recurrence. <strong>Decreasing over time:</strong> Threatened abortion.</td>
<td>Routine pregnancy testing is done by qualitative serum or urine hCG test. Test will be positive (&gt;50 mIU/mL) in most pregnant women at the time of or shortly after the first missed menstrual period. <strong>Quantitative</strong> hCG testing is indicated for (1) the evaluation of suspected ectopic pregnancy (where levels are lower than in normal pregnancy at the same gestational age) if the routine pregnancy test is negative; (2) the evaluation of threatened abortion. In both situations, hCG levels fail to demonstrate the normal early pregnancy increase. Test is also indicated for following the course of trophoblastic and germ cell tumors. Hum Reprod 1992;7:701. West J Med 1993;159:195. Urology 1994;44:392.</td>
</tr>
</tbody>
</table>

Marbled $$
**Clostridium difficile enterotoxin**, stool  
Negative (≤1:10 titer)  
Urine or stool container $$$  
Must be tested within 12 hours of collection as toxin (B) is labile.

**Clostridium difficile**, a motile, gram-positive rod, is the major recognized agent of antibiotic-associated diarrhea, which is toxigenic in origin (see Antibiotic-associated colitis, p 224). There are two toxins (A and B) produced by *C difficile*. Cell culture is used to detect the cytopathic effect of the toxins, whose identity is confirmed by neutralization with specific antitoxins. Toxin A (more weakly cytopathic in cell culture) is enterotoxic and produces enteric disease. Toxin B (more easily detected in standard cell culture assays) fails to produce intestinal disease.

**Positive in:** Antibiotic-associated diarrhea (15–25%), antibiotic-associated colitis (50–75%), and pseudomembranous colitis (90–100%). About 3% of healthy adults and 10–20% of hospitalized patients have *C difficile* in their colonic flora. There is also a high carrier rate of *C difficile* and its toxin in healthy neonates.

**Prolonged in:** Heparin therapy, severe deficiency of clotting factors (except factors VII and XIII), functional platelet disorders, afibrinogenemia, circulating anticoagulants.  
**Normal in:** Thrombocytopenia, factor VII deficiency, von Willebrand’s disease.

**Clotting time, activated**, whole blood (ACT)  
114–186 seconds  
Special black tube $$  
Performed at patient bedside. Avoid traumatic venipuncture, which may cause contamination with tissue juices and decrease clotting time.

A bedside or operating room test that assesses heparinization by measuring time taken for whole blood to clot.

**Positive in:** Antibiotic-associated diarrhea (15–25%), antibiotic-associated colitis (50–75%), and pseudomembranous colitis (90–100%). About 3% of healthy adults and 10–20% of hospitalized patients have *C difficile* in their colonic flora. There is also a high carrier rate of *C difficile* and its toxin in healthy neonates.


Many consider this test unreliable. Reproducibility of prolonged ACTs is poor. Increasingly, the ACT has been used in the operating room, dialysis units, critical care centers and during interventional cardiology/radiology procedures to monitor anticoagulation and titrate heparin dosages. At centers without experience, should not be used to regulate therapeutic heparin dosage adjustments; use partial thromboplastin time (PTT) instead.  
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Coccidioides antibodies, serum or CSF</strong></td>
<td>Screens for presence of antibodies to <em>Coccidioides immitis</em>. Some centers use the mycelial-phase antigen, coccidioidin, to detect antibody. IgM antibodies appear early in disease in 75% of patients, begin to decrease after week 3, and are rarely seen after 5 months. They may persist in disseminated cases, usually in the immunocompromised. IgG antibodies appear later in the course of the disease. Meningeal disease may have negative serum IgG and require CSF IgG antibody titers.</td>
<td><strong>Positive in:</strong> Infection by coccidioides (90%). <strong>Negative in:</strong> Coccidioidin skin testing, many patients with chronic cavitary coccidioides; 5% of meningeal coccidioides is negative by CSF complement fixation (CF) test.</td>
<td>Diagnosis is based upon culture and serologic testing. Precipitin and CF tests detect 90% of primary symptomatic cases. Precipitin test is most effective in detecting early primary infection or an exacerbation of existing disease. Test is diagnostic but not prognostic. CF test becomes positive later than precipitin test, and titers can be used to assess severity of infection. Titers rise as the disease progresses and decline as the patient improves. Enzyme immunoassay now available; data suggest good performance. N Engl J Med 1995;332:1077. Am J Clin Pathol 1997;107:148.</td>
</tr>
<tr>
<td><strong>Cold agglutinins, plasma</strong></td>
<td>Detects antibodies that agglutinate red blood cells in the cold (strongly at 4°C, weakly at 24°C, and weakly or not at all at 37°C). These antibodies are present in primary atypical pneumonias due to <em>Mycoplasma pneumoniae</em>, in certain autoimmune hemolytic anemias, and in normal persons (not clinically significant).</td>
<td><strong>Increased in:</strong> Chronic cold agglutinin disease, lymphoproliferative disorders (eg, Waldenström’s macroglobulinemia), autoimmune hemolytic anemia, collagen-vascular diseases, <em>M pneumoniae</em> pneumonia, infectious mononucleosis, mumps orchitis, cytomegalovirus, tropical diseases (eg, trypanosomiasis).</td>
<td>In <em>Mycoplasma</em> pneumonia, titers rise early, are maximal at 3–4 weeks after onset, and then disappear rapidly. These antibodies are usually IgM anti-I antibodies distinct from antibodies to <em>M pneumoniae</em>. A rise in cold agglutinin antibody titer is suggestive of recent mycoplasma infection but is found in other diseases. N Engl J Med 1977;297:583.</td>
</tr>
<tr>
<td><strong>Complement C3</strong>&lt;br&gt;<strong>serum</strong>&lt;br&gt;64–166 mg/dL [640–1660 mg/L]&lt;br&gt;Marbled $$</td>
<td>The classic and alternative complement pathways converge at the C3 step in the complement cascade. Low levels indicate activation by one or both pathways. Most diseases with immune complexes will show decreased C3 levels. Test is usually performed as an immunoassay (by radial immunodiffusion or nephelometry).</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Increased in:</strong> Many inflammatory conditions as an acute phase reactant, active phase of rheumatic diseases (e.g., rheumatoid arthritis, SLE), acute viral hepatitis, myocardial infarction, cancer, diabetes mellitus, pregnancy, sarcoidosis, amyloidosis, thyroiditis.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Decreased by:</strong> Decreased synthesis (protein malnutrition, congenital deficiency, severe liver disease), increased catabolism (immune complex disease, membranoproliferative glomerulonephritis [75%], SLE, Sjögren’s syndrome, rheumatoid arthritis, disseminated intravascular coagulation, paroxysmal nocturnal hemoglobinuria, autoimmune hemolytic anemia, gram-negative bacteremia), increased loss (burns, gastrointestinal enteropathies).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Complement C3 levels may be useful in following the activity of immune complex diseases. The best test to detect inherited deficiencies is CH50. N Engl J Med 1987;316:1525.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Complement C4</strong>&lt;br&gt;<strong>serum</strong>&lt;br&gt;15–45 mg/dL [150–450 mg/L]&lt;br&gt;Marbled $$</td>
<td>C4 is a component of the classic complement pathway. Depressed levels usually indicate classic pathway activation. Test is usually performed as an immunoassay and not a functional assay.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Increased in:</strong> Various malignancies (not clinically useful).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Decreased by:</strong> Decreased synthesis (congenital deficiency), increased catabolism (SLE, rheumatoid arthritis, proliferative glomerulonephritis, hereditary angioedema), and increased loss (burns, protein-losing enteropathies).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Complement CH50,</strong> plasma or serum (CH50)</td>
<td>The quantitative assay of hemolytic complement activity depends on the ability of the classic complement pathway to induce hemolysis of red cells sensitized with optimal amounts of anti-red cell antibodies. For precise titrations of hemolytic complement, the dilution of serum that will lyse 50% of the indicator red cells is determined as the CH50. This arbitrary unit depends on the conditions of the assay and is therefore laboratory-specific.</td>
<td><strong>Decreased with:</strong> &gt;50–80% deficiency of classic pathway complement components (congenital or acquired deficiencies). <strong>Normal in:</strong> Deficiencies of the alternative pathway complement components.</td>
<td>This is a functional assay of biologic activity. Sensitivity to decreased levels of complement components depends on exactly how the test is performed. It is used to detect congenital and acquired severe deficiency disorders of the classic complement pathway. N Engl J Med 1987;316:1525.</td>
</tr>
<tr>
<td>22–40 U/mL (laboratory-specific)</td>
<td>Marbled $$$</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cortisol,</strong> plasma or serum</td>
<td>Release of corticotropin-releasing factor (CRF) from the hypothalamus stimulates release of ACTH from the pituitary, which in turn stimulates release of cortisol from the adrenal. Cortisol provides negative feedback to this system. Test measures both free cortisol and cortisol bound to cortisol-binding globulin (CBG). Morning levels are higher than evening levels.</td>
<td><strong>Increased in:</strong> Cushing’s syndrome, acute illness, surgery, trauma, septic shock, depression, anxiety, alcoholism, starvation, chronic renal failure, increased CBG (congenital, pregnancy, estrogen therapy). <strong>Decreased in:</strong> Addison’s disease; decreased CBG (congenital, liver disease, nephrotic syndrome).</td>
<td>Cortisol levels are useful only in the context of standardized suppression or stimulation tests. (See Cosyntropin stimulation test, p 77, and Dexamethasone suppression tests, p 83). Circadian fluctuations in cortisol levels limit usefulness of single measurements. Analysis of diurnal variation of cortisol is not useful diagnostically. Crit Care Clin 1991;7:23. Endocrinol Metab Clin North Am 1994;23:511.</td>
</tr>
</tbody>
</table>
### Cosyntropin stimulation test, serum or plasma

<table>
<thead>
<tr>
<th>Description</th>
<th>Details</th>
</tr>
</thead>
</table>
| Urine bottle containing boric acid | $$$
| Collect 24-hour urine. |
| Cosyntropin (synthetic ACTH preparation) stimulates the adrenal to release cortisol. | A normal response is a doubling of basal levels or an increment of 7 µg/dL (200 nmol/L) to a level above 18 µg/dL (>504 nmol/L). A poor cortisol response to cosyntropin indicates adrenal insufficiency (see Adrenocortical insufficiency algorithm, Fig. 8-3, p 338). |

<table>
<thead>
<tr>
<th>Increased in:</th>
<th>Cushing’s syndrome, acute illness, stress.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Not Increased in:</strong> obesity.</td>
<td></td>
</tr>
</tbody>
</table>

| Decreased in: | Adrenal insufficiency, pituitary insufficiency, AIDS. |

| Test does not distinguish primary from secondary (pituitary) adrenal insufficiency, since in secondary adrenal insufficiency the atrophic adrenal may be unresponsive to cosyntropin. Test may not reliably detect pituitary insufficiency. Metyrapone test (p 127) may be useful to assess the pituitary-adrenal axis. |

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Creatine kinase, serum (CK)</strong> 32–267 IU/L [0.53–4.45 µkat/L] (method-dependent) Marbled $</td>
<td>Creatine kinase splits creatine phosphate in the presence of ADP to yield creatine and ATP. Skeletal muscle, myocardium, and brain are rich in the enzyme. CK is released by tissue damage.</td>
<td><strong>Increased in:</strong> Myocardial infarction, myocarditis, muscle trauma, rhabdomyolysis, muscular dystrophy, polymyositis, severe muscular exertion, malignant hyperthermia, hypothyroidism, cerebral infarction, surgery, Reye’s syndrome, tetanus, generalized convulsions, alcoholism, IM injections, DC countershock. Drugs: clofibrate, HMG-Co A reductase inhibitors.</td>
<td>CK is as sensitive a test as aldolase for muscle damage, so aldolase is not needed. During a myocardial infarction (MI), serum CK level rises rapidly (within 3–5 hours); elevation persists for 2–3 days post-myocardial infarction. Total CK is not specific enough for use in diagnosis of MI, but a normal total CK has a high negative predictive value. A more specific test is needed for diagnosis of MI (eg, CK-MB or cardiac troponin I). Cardiac troponin I and CK-MB or CK-MB mass concentration are better markers for myocardial infarction. Br Heart J 1994;72:112.</td>
</tr>
</tbody>
</table>
**Creatine kinase MB, serum (CKMB), enzyme activity**

- $<16$ IU/L
- $<0.27$ µkat/L
- $<4\%$ of total CK
- $<7$ µg/L mass units (laboratory-specific)

**Marbled $\$$**

<table>
<thead>
<tr>
<th><strong>Creatine kinase MB</strong></th>
<th><strong>Increased in:</strong> Myocardial infarction, cardiac trauma, certain muscular dystrophies, and polymyositis. Slight persistent elevation reported in a few patients on hemodialysis.</th>
<th><strong>CKMB is a relatively specific test for MI. It appears in serum approximately 4 hours after infarction, peaks at 12–24 hours, and declines over 48–72 hours. CKMB mass concentration is a more sensitive marker of MI than CKMB isoenzymes or total CK within 4–12 hours after infarction. Cardiac troponin I levels are useful in the late (after 48 hours) diagnosis of MI since, unlike CKMB, levels remain elevated for 5–7 days. Within 48 hours, sensitivity and specificity of troponin I are similar to CKMB. Specificity of troponin I is higher than CKMB in patients with skeletal muscle injury or renal failure, or post-operatively. Cardiac troponin I is therefore the preferred test. Estimation of CKMM and CKBB is not clinically useful. Use total CK.</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
</tr>
<tr>
<td>-----------------------</td>
<td>------------------</td>
<td>----------------</td>
</tr>
<tr>
<td><strong>Creatinine, serum (Cr)</strong></td>
<td>Endogenous creatinine is excreted by filtration through the glomerulus and by tubular secretion. Creatinine clearance is an acceptable clinical measure of glomerular filtration rate (GFR), though it sometimes overestimates GFR (eg, in cirrhosis). For each 50% reduction in GFR, serum creatinine approximately doubles.</td>
<td><strong>Increased in:</strong> Acute or chronic renal failure, urinary tract obstruction, nephrotoxic drugs, hypothyroidism. <strong>Decreased in:</strong> Reduced muscle mass.</td>
</tr>
<tr>
<td>0.6–1.2 mg/dL [50–100 µmol/L] Marbled $</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Creatinine**
Common Laboratory Tests: Selection and Interpretation

Creatinine clearance, (C\textsubscript{1Cr})

Adults: 90–130 mL/min/1.73 m\textsuperscript{2} BSA

Collect carefully timed 24-hour urine and simultaneous serum/plasma creatinine sample. Record patient’s weight and height.

Widely used test of glomerular filtration rate (GFR). Theoretically reliable, but often compromised by incomplete urine collection. Creatinine clearance is calculated from measurement of urine creatinine (U\textsubscript{Cr} [mg/dL]), plasma/serum creatinine (P\textsubscript{Cr} [mg/dL]), and urine flow rate (V [mL/min]) according to the formula:

\[
C_{\text{1Cr}} (\text{mL/min}) = \frac{U_{\text{Cr}} \times V}{P_{\text{Cr}}}
\]

where

\[
V (\text{mL/min}) = \frac{24\text{-hour urine volume(mL)}}{1440}
\]

Creatinine clearance is often “corrected” for body surface area (BSA [m\textsuperscript{2}]) according to the formula:

\[
C_{\text{1Cr}} \text{(corrected)} = \frac{C_{\text{1Cr}} \times 1.73}{\text{BSA}}
\]

Increased in: High cardiac output, exercise, acromegaly, diabetes mellitus (early stage), infections, hypothyroidism.

Decreased in: Acute or chronic renal failure, decreased renal blood flow (shock, hemorrhage, dehydration, CHF). Drugs: nephrotoxic drugs.

Serum Cr may, in practice, be a more reliable indicator of renal function than 24-hour C\textsubscript{1Cr} unless urine collection is carefully monitored. An 8-hour collection provides results similar to those obtained by a 24-hour collection. C\textsubscript{1Cr} will overestimate GFR to the extent that Cr is secreted by the renal tubules (eg, in cirrhosis). C\textsubscript{1Cr} can be estimated from the serum creatinine using the following formula:

\[
C_{\text{1Cr}} (\text{mL/min}) = \frac{(140 - \text{Age}) \times Wt(kg)}{72 \times P_{\text{Cr}}}
\]

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>globulins are immunoglobulins (IgG, IgM, IgA, or light chains) which precipitate on exposure to the cold. Type I cryoglobulins (25%) are monoclonal proteins, most commonly IgM, occasionally IgG, and rarely IgA or Bence Jones protein, seen in multiple myeloma and Waldenström’s macroglobulinemia. Type II (25%) are mixed cryoglobulins with a monoclonal component (usually IgM but occasionally IgG or IgA) that complexes with autologous normal IgG in the cryoprecipitate. Type III (50%) are mixed polyclonal cryoglobulins (IgM and IgG).</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cytomegalovirus antibody, serum (CMV)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Negative</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Marbled</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Dexamethasone suppression test (single low-dose, overnight), serum</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>8:00 AM serum cortisol level: &lt;5 µg/dL</strong></td>
</tr>
<tr>
<td><strong>[&lt;140 nmol/L]</strong></td>
</tr>
<tr>
<td><strong>$$$</strong></td>
</tr>
<tr>
<td><strong>Give 1 mg dexamethasone at 11:00 PM. At 8:00 AM, draw serum cortisol level.</strong></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Cytomegalovirus antibody</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Increased in:</strong> Previous or active CMV infection. False-positive CMV IgM tests occur when rheumatoid factor or infectious mononucleosis is present.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Dexamethasone suppression test (low-dose)</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>In normal patients, dexamethasone suppresses the 8:00 AM serum cortisol level to below 5 µg/dL. Patients with Cushing’s syndrome have 8:00 AM levels &gt;10 µg/dL (&gt;276 nmol/L).</strong></td>
</tr>
<tr>
<td><strong>Positive in:</strong> Cushing’s syndrome (98% sensitivity, 98% specificity in lean outpatients), obese patients (13%), hospitalized or chronically ill patients (23%).</td>
</tr>
</tbody>
</table>

| **Serial specimens exhibiting a greater than fourfold titer rise suggest a recent infection. Active CMV infection must be documented by viral isolation. Useful for screening of potential organ donors and recipients. Detection of CMV IgM antibody in the serum of a newborn usually indicates congenital infection. Detection of CMV IgG antibody is not diagnostic, since maternal CMV IgG antibody passed via the placenta can persist in newborn’s serum for 6 months. Rev Infect Dis 1988;10:S468.** |

<p>| <strong>Patients taking phenytoin may fail to suppress because of enhanced dexamethasone metabolism. Depressed patients may also fail to suppress morning cortisol level. Ann Clin Biochem 1997; 34(Part 3):222.</strong> |</p>
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dexamethasone suppression test (high-dose, overnight), serum 8:00 AM serum cortisol level: &lt;5 µg/dL [&lt;140 nmol/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dexamethasone suppression test (high-dose, overnight), serum 8:00 AM serum cortisol level: &lt;5 µg/dL [&lt;140 nmol/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dexamethasone suppression test (high-dose, overnight), serum 8:00 AM serum cortisol level: &lt;5 µg/dL [&lt;140 nmol/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Double-stranded-DNA antibody (ds-DNA Ab), serum &lt;1 : 10 titer Marbled $$</td>
<td>IgG or IgM antibodies directed against host double-stranded DNA.</td>
<td><strong>Increased in:</strong> Systemic lupus erythematosus (60–70% sensitivity, 95% specificity) based on &gt;1 : 10 titer. <strong>Not increased in:</strong> Drug-induced lupus.</td>
<td>High titers are seen only in SLE. Titers of ds-DNA antibody correlate well with disease activity and with occurrence of glomerulonephritis. (See also Autoantibodies table, p 367.) West J Med 1987;147:210. Clin Immunol Immunopathol 1988;47:121.</td>
</tr>
<tr>
<td>Epstein-Barr virus antibodies, serum (EBV Ab)</td>
<td>Antiviral capsid antibodies (anti-VCA) (IgM) often reach their peak at clinical presentation and last up to 3 months; anti-VCA IgG antibodies last for life. Early antigen antibodies (anti-EA) are next to develop, are most often positive at 1 month after presentation, typically last for 2–3 months, and may last up to 6 months in low titers. Anti-EA may also be found in some patients with Hodgkin’s disease, chronic lymphocytic leukemia, and some other malignancies. Anti-EB nuclear antigen (anti-EBNA) antibody begins to appear in a minority of patients in the third or fourth week but is uniformly present by 6 months.</td>
<td><strong>Increased in:</strong> EB virus infection, infectious mononucleosis. Antibodies to the diffuse (D) form of antigen (detected in the cytoplasm and nucleus of infected cells) are greatly elevated in nasopharyngeal carcinoma. Antibodies to the restricted (R) form of antigen (detected only in the cytoplasm of infected cells) are greatly elevated in Burkitt’s lymphoma.</td>
<td>Most useful in diagnosing infectious mononucleosis in patients who have the clinical and hematologic criteria for the disease but who fail to develop the heterophile agglutinins (10%) (see Heterophile agglutination, p 107). EBV antibodies cannot be used to diagnose “chronic” mononucleosis. Chronic fatigue syndrome is not caused by EBV. The best indicator of primary infection is a positive anti-VCA IgM (check for false-positives caused by rheumatoid factor). Rose NR et al (editors): Manual of Clinical Laboratory Immunology, 4th ed. American Society for Microbiology, 1992. J Clin Microbiol 1996;34:3240. Lab Med 1983;14:509.</td>
</tr>
</tbody>
</table>

<p>| Erythrocyte count, whole blood (RBC count) | Erythrocytes are counted by automated instruments using electrical impedance or light scattering. | <strong>Increased in:</strong> Secondary polycythemia (hemoconcentration), polycythemia vera. Spurious increase with increased white blood cells. <strong>Decreased in:</strong> Anemia. Spurious decrease with autoagglutination. |  |</p>
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Erythrocyte sedimentation rate</strong>, whole blood (ESR)</td>
<td>In plasma, erythrocytes (red blood cells [RBCs]) usually settle slowly. However, if they aggregate for any reason (usually because of plasma proteins called acute phase reactants, eg, fibrinogen) they settle rapidly. Sedimentation of RBCs occurs because their density is greater than plasma. ESR measures the distance in mm that erythrocytes fall during 1 hour.</td>
<td><strong>Increased in:</strong> Infections (osteomyelitis, pelvic inflammatory disease [75%]), inflammatory disease (temporal arteritis, polymyalgia rheumatica, rheumatic fever), malignant neoplasms, paraproteinemia, anemia, pregnancy, chronic renal failure, GI disease (ulcerative colitis, regional ileitis). For endocarditis, sensitivity = 93%. <strong>Decreased in:</strong> Polycythemia, sickle cell anemia, spherocytosis, anisocytosis, hypofibrinogenemia, hypogammaglobulinemia, congestive heart failure, microcytosis. Drugs: high-dose corticosteroids.</td>
<td>There is a good correlation between ESR and C-reactive protein, but ESR is less expensive. Test is useful and indicated only for diagnosis and monitoring of temporal arteritis and polymyalgia rheumatica. The test is not sensitive or specific for other conditions. ESR is higher in women, blacks, and older persons. Low value is of no diagnostic significance. Am J Med 1985;78:1001. Ann Intern Med 1986;104:515.</td>
</tr>
<tr>
<td>Male: &lt;10 Female: &lt;15 mm/h (laboratory-specific) Lavender $ Test must be run within 2 hours after sample collection.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Erythropoietin</strong>, serum (EPO)</td>
<td>Erythropoietin is a glycoprotein hormone produced in the kidney that induces red blood cell production by stimulating proliferation, differentiation, and maturation of erythroid precursors. Hypoxia is the usual stimulus for production of EPO. In conditions of bone marrow hyporesponsiveness, EPO levels are elevated. In chronic renal failure, EPO production is decreased.</td>
<td><strong>Increased in:</strong> Anemias associated with bone marrow hyporesponsiveness (aplastic anemia, iron deficiency anemia), secondary polycythemia (high-altitude hypoxia, COPD, pulmonary fibrosis), erythropoietin-producing tumors (cerebellar hemangioblastomas, pheochromocytomas, renal tumors), pregnancy, polycystic kidney disease. <strong>Decreased in:</strong> Anemia of chronic disease, renal failure, inflammatory states, primary polycythemia (polycythemia vera) (39%).</td>
<td>Test is not very useful in differentiating polycythemia vera from secondary polycythemia. Since virtually all patients with severe anemia due to chronic renal failure respond to EPO therapy, pretherapy EPO levels are not indicated. Patient receiving EPO as chronic therapy should have iron deficiency screening routinely. Curr Opin Nephrol Hypertens 1994;3:620. Haematologica 1997;82:406.</td>
</tr>
<tr>
<td>5–20 mIU/mL [4–26 IU/L] Marbled $$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ethanol, serum (EtOH)</strong></td>
<td>Measures serum level of ethyl alcohol (ethanol).</td>
<td><strong>Present in:</strong> Ethanol ingestion.</td>
<td>Whole blood alcohol concentrations are about 15% lower than serum concentrations. Each 0.1 mg/dL of ethanol contributes about 22 mosm/kg to serum osmolality. Legal intoxication in many states is defined as &gt;80 mg/dL (&gt;17 mmol/L). N Engl J Med 1976;294:757.</td>
</tr>
<tr>
<td>-------------------------</td>
<td>-------------------------------------------------</td>
<td>----------------------------------</td>
<td>----------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>0 mg/dL [mmol/L]</td>
<td>Marbled $$</td>
<td>Do not use alcohol swab. Do not remove stopper.</td>
<td></td>
</tr>
<tr>
<td><strong>Factor V (Leiden) mutation</strong></td>
<td><strong>Blood</strong> The Leiden mutation is a single nucleotide base substitution leading to an amino acid substitution (glutamine replaces arginine) at one of the sites where coagulation factor V is cleaved by activated protein C. The mutation causes factor V to be partially resistant to protein C, which is involved in inhibiting coagulation. Factor V mutations may be present in up to half of the cases of unexplained venous thrombosis and are seen in 95% of patients with activated protein C resistance.</td>
<td><strong>Positive in:</strong> Hypercoagulability secondary to factor V mutation (specificity approaches 100%).</td>
<td>The presence of mutation is only a risk factor for thrombosis, not an absolute marker for disease. Homozygotes have a 50- to 100-fold increase in risk of thrombosis (relative to the general population) and heterozygotes have a 7-fold increase in risk. The current PCR and reverse dot blot assay only detects the Leiden mutation of factor V; other mutations may yet be discovered. N Engl J Med 1995;332:912. Nature 1994;369:64. Ann Intern Med 1999;130:643.</td>
</tr>
<tr>
<td><strong>Factor V (Leiden) mutation</strong></td>
<td><strong>Blood Lavender or blue</strong> $$ $$ $$ $$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Factor VIII assay, plasma</strong>&lt;br&gt;40–150% of normal, (varies with age) Blue $$$&lt;br&gt;Deliver immediately to laboratory on ice. Stable for 2 hours.</td>
<td>Measures activity of factor VIII (anti-hemophilic factor), a key factor of the intrinsic clotting cascade.</td>
<td><strong>Increased in:</strong> Inflammatory states (acute phase reactant), last trimester of pregnancy, oral contraceptives.&lt;br&gt;<strong>Decreased in:</strong> Hemophilia A, von Willebrand’s disease, disseminated intravascular coagulation, acquired factor VIII antibodies.</td>
<td>Normal hemostasis requires at least 25% of factor VIII activity. Symptomatic hemophiliacs usually have levels ≤5%. Disease levels are defined as severe (&lt;1%), moderate (1–5%), and mild (&gt;5%). Factor VIII assays are used to guide replacement therapy in patients with hemophilia. Semin Hematol 1967;4:93.</td>
</tr>
<tr>
<td><strong>Fecal fat, stool</strong>&lt;br&gt;Random: &lt;60 droplets of fat/ high power field&lt;br&gt;72 hour: &lt;7 g/d $$$&lt;br&gt;Qualitative: random stool sample is adequate. Quantitative: dietary fat should be at least 50–150 g/d for 2 days before collection. Then all stools should be collected for 72 hours and refrigerated.</td>
<td>In healthy people, most dietary fat is completely absorbed in the small intestine. Normal small intestinal lining, bile acids, and pancreatic enzymes are required for normal fat absorption.</td>
<td><strong>Increased in:</strong> Malabsorption from small bowel disease (regional enteritis, celiac disease, tropical sprue), pancreatic insufficiency, diarrhea with or without fat malabsorption.</td>
<td>A random, qualitative fecal fat (so-called Sudan stain) is only useful if positive. Furthermore, it does not correlate well with quantitative measurements. Sudan stain appears to detect triglycerides and lipolytic by-products, whereas 72-hour fecal fat measures fatty acids from a variety of sources, including phospholipids, cholesteryl esters, and triglycerides. The quantitative method can be used to measure the degree of fat malabsorption initially and then after a therapeutic intervention. A normal quantitative stool fat reliably rules out pancreatic insufficiency and most forms of generalized small intestine disease. Gastroenterol Clin North Am 1989; 18:467. Gastroenterology 1992;102:1936.</td>
</tr>
<tr>
<td><strong>Fecal occult blood, stool</strong></td>
<td>Measures blood in the stool using gum guaiac as an indicator reagent. In the Hemoccult test, gum guaiac is impregnated in a test paper that is smeared with stool using an applicator. Hydrogen peroxide is used as a developer solution. The resultant phenolic oxidation of guaiac in the presence of blood in the stool yields a blue color.</td>
<td><strong>Positive in:</strong> Upper GI disease (peptic ulcer, gastritis, variceal bleeding, esophageal and gastric cancer), lower GI disease (diverticulosis, colonic polyps, colon carcinoma, inflammatory bowel disease, vascular ectasias, hemorrhoids).</td>
<td>Although fecal occult blood testing is an accepted screening test for colon carcinoma, the sensitivity and specificity of an individual test are low. The utility of fecal occult blood testing after digital rectal examination has not been well studied. Three randomized controlled trials have shown reductions in colon cancer mortality with yearly (33% reduction) or biennial (15–21% reduction) testing. About 1000 fifty-year-olds must be screened for 10 years to save one life. BMJ 1998;317:559. Ann Intern Med 1997;126:811.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Negative</strong></td>
<td>Patient should be on a special diet free of exogenous peroxidase activity (meat, fish, turnips, horseradish), GI irritants (aspirin, non-steroidal anti-inflammatory drugs), and iron. To avoid false-negatives, patients should avoid taking vitamin C. Patient collects two specimens from three consecutive bowel movements.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td>Ferritin, serum</td>
<td>Ferritin is the body’s major iron storage protein. The serum ferritin level correlates with total body iron stores. The test is used to detect iron deficiency, to monitor response to iron therapy, and, in iron overload states, to monitor iron removal therapy. It is also used to predict homozygosity for hemochromatosis in relatives of affected patients. In the absence of liver disease, it is a more sensitive test for iron deficiency than serum iron and iron-binding capacity (transferrin saturation).</td>
<td><strong>Increased in:</strong> Iron overload (hemochromatosis [sensitivity 85%, specificity 95%], hemosiderosis), acute or chronic liver disease, alcoholism, various malignancies (eg, leukemia, Hodgkin’s disease), chronic inflammatory disorders (eg, rheumatoid arthritis, adult Still’s disease), thalassemia minor, hyperthyroidism, HIV infection, non-insulin-dependent diabetes mellitus, and postpartum state. <strong>Decreased in:</strong> Iron deficiency (60–75%).</td>
<td>Serum ferritin is clinically useful in distinguishing between iron deficiency anemia (serum ferritin levels diminished) and anemia of chronic disease or thalassemia (levels usually normal or elevated). Test of choice for diagnosis of iron deficiency anemia. Liver disease will increase serum ferritin levels and mask the diagnosis of iron deficiency. Am J Hematol 1993;42:177. Br J Haematol 1993;85:787. J Intern Med 1994;236:315. J Gen Intern Med 1992;7:145</td>
</tr>
<tr>
<td>Males 16–300 ng/mL [µg/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Females 4–161 ng/mL [µg/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test</td>
<td>Description</td>
<td>Increased in:</td>
<td>Negative in:</td>
</tr>
<tr>
<td>----------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>α-Fetoprotein, serum (AFP)</strong></td>
<td>α-Fetoprotein is a glycoprotein produced both early in fetal life and by some tumors.</td>
<td>Hepatocellular carcinoma (72%), massive hepatic necrosis (74%), viral hepatitis (34%), chronic active hepatitis (29%), cirrhosis (11%), regional enteritis (5%), benign gynecologic diseases (22%), testicular carcinoma (embryonal) (70%), teratocarcinoma (64%), teratoma (37%), ovarian carcinoma (57%), endometrial cancer (50%), cervical cancer (53%), pancreatic cancer (23%), gastric cancer (18%), colon cancer (5%).</td>
<td>Seminoma.</td>
</tr>
<tr>
<td><strong>Fibrin D-dimers, plasma</strong></td>
<td>Plasmin acts on fibrin to form various fibrin degradation products. The D-dimer level can be used as a measure of activation of the fibrinolytic system.</td>
<td>Disseminated intravascular coagulation (DIC), other thrombotic disorders, pulmonary embolism, venous or arterial thrombosis.</td>
<td>Fibrin D-dimer assay has replaced the Fibrinogen Split Products test as a screen for DIC, because the D-dimer assay can distinguish fibrin degradation products (in DIC) from fibrinogen degradation products (in primary fibrinogenolysis). Since the presence of fibrin D-dimer is not specific for DIC, the definitive diagnosis of DIC must depend on other tests, including the platelet count and serum fibrinogen level.</td>
</tr>
</tbody>
</table>

**Fibrin D-dimers**

Avoid hemolysis.

Fibrin(ogen) Split Products test is a screen for DIC, because the D-dimer assay can distinguish fibrin degradation products (in DIC) from fibrinogen degradation products (in primary fibrinogenolysis). Since the presence of fibrin D-dimer is not specific for DIC, the definitive diagnosis of DIC must depend on other tests, including the platelet count and serum fibrinogen level.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fibrinogen</strong>&lt;br&gt; (functional), plasma 175–433 mg/dL [1.75–4.3 g/L] <strong>Panic:</strong> &lt;75 mg/dL Blue $$</td>
<td>Fibrinogen is synthesized in the liver and has a half-life of about 4 days. Thrombin cleaves fibrinogen to form insoluble fibrin monomers, which polymerize to form a clot.</td>
<td><strong>Increased in:</strong> Inflammatory states (acute phase reactant), use of oral contraceptives, pregnancy. <strong>Decreased in:</strong> Decreased hepatic synthesis, increased consumption (disseminated intravascular coagulation [DIC], thrombolysis). Hereditary: Afibrinogenemia (rare), hypofibrinogenemia, dysfibrinogenemia.</td>
<td>Hypofibrinogenemia is an important diagnostic laboratory feature of DIC. Diagnosis of dysfibrinogenemia depends upon the discrepancy between measurable antigenic and low functional (clottable) fibrinogen levels. Blood 1982;60:284. Ann Intern Med 1993;118:956.</td>
</tr>
<tr>
<td>Folic acid (RBC), whole blood</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>165–760 ng/mL [370–1720 nmol/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lavender $$$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Follicle-stimulating hormone, serum (FSH)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male: 1–10 mIU/mL</td>
</tr>
<tr>
<td>Female: (mIU/mL)</td>
</tr>
<tr>
<td>Follicular 4–13</td>
</tr>
<tr>
<td>Luteal 2–13</td>
</tr>
<tr>
<td>Midcycle 5–22</td>
</tr>
<tr>
<td>Postmenopausal 20–138 (laboratory-specific)</td>
</tr>
<tr>
<td>Marbled $$</td>
</tr>
</tbody>
</table>

**Folate** is a vitamin necessary for methyl group transfer in thymidine formation, and hence DNA synthesis. Deficiency can result in megaloblastic anemia.

**Decreased in:** Tissue folate deficiency (from dietary folate deficiency), $B_{12}$ deficiency (50–60%, since cellular uptake of folate depends on $B_{12}$).

- Red cell folate level correlates better than serum folate level with tissue folate deficiency.
- A low red cell folate level may indicate either folate or $B_{12}$ deficiency.
- A therapeutic trial of folate (and not red cell or serum folate testing) is indicated when the clinical and dietary history is strongly suggestive of folate deficiency and the peripheral smear shows hypersegmented polymorphonuclear leukocytes. However, the possibility of vitamin $B_{12}$ deficiency must always be considered in the setting of megaloblastic anemia, since folate therapy will treat the hematologic, but not the neurologic, sequelae of vitamin $B_{12}$ deficiency. Blood 1983;61:624.

**FSH** is stimulated by the hypothalamic hormone GnRH and is then secreted from the anterior pituitary in a pulsatile fashion. Levels rise during the preovulatory phase of the menstrual cycle and then decline. Elevation of FSH is the most sensitive indicator of onset of menopause.

**Increased in:** Primary (ovarian) gonadal failure, ovarian or testicular agenesis, castration, postmenopause, Klinefelter’s syndrome, drugs.

**Decreased in:** Hypothalamic disorders, pituitary disorders, pregnancy, anorexia nervosa. Drugs: corticosteroids, oral contraceptives.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Free erythrocyte protoporphyrin, whole blood (FEP)</td>
<td>Protoporphyrin is produced in the next to last step of heme synthesis. In the last step, iron is incorporated into protoporphyrin to produce heme. Enzyme deficiencies, lack of iron, or presence of interfering substances (lead) can disrupt this process and cause elevated FEP.</td>
<td>Increased in: Decreased iron incorporation into heme (iron deficiency, infection, and lead poisoning), erythropoietic protoporphyria.</td>
<td>FEP can be used to screen for lead poisoning in children provided that iron deficiency has been ruled out. Test does not discriminate between uroporphyrin, coproporphyrin, and protoporphyrin, but protoporphyrin is the predominant porphyrin measured. Clin Pediatr 1991;30:74. Am J Dis Child 1993;147:66.</td>
</tr>
<tr>
<td>Fructosamine, serum 1.6–2.6 mmol/L</td>
<td>Glycation of albumin produces fructosamine, a less expensive marker of glycemic control than HbA\textsubscript{1c}.</td>
<td>Increased in: diabetes mellitus.</td>
<td>Fructosamine correlates well with fasting plasma glucose (r = 0.74) but cannot be used to predict precisely the HbA\textsubscript{1c}. Acta Diabetologica 1998;35:48.</td>
</tr>
<tr>
<td>Gamma-glutamyl transpeptidase, serum (GGT) 9–85 U/L [0.15–1.42 µkat/L] (laboratory-specific)</td>
<td>GGT is an enzyme present in liver, kidney, and pancreas. It is induced by alcohol intake and is an extremely sensitive indicator of liver disease, particularly alcoholic liver disease.</td>
<td>Increased in: Liver disease: acute viral or toxic hepatitis, chronic or subacute hepatitis, alcoholic hepatitis, cirrhosis, biliary tract obstruction (intrahepatic or extrahepatic), primary or metastatic liver neoplasm, mononucleosis. Drugs (by enzyme induction): phenytoin, carbamazepine, barbiturates, alcohol.</td>
<td>GGT is useful in follow-up of alcoholics undergoing treatment since the test is sensitive to modest alcohol intake. GGT is elevated in 90% of patients with liver disease. GGT is used to confirm hepatic origin of elevated serum alkaline phosphatase. Alcohol Clin Exp Res 1990;14:250. Am J Gastroenterol 1992;87:991.</td>
</tr>
<tr>
<td><strong>Glucose</strong>, serum</td>
<td>Normally, the glucose concentration in extracellular fluid is closely regulated so that a source of energy is readily available to tissues and so that no glucose is excreted in the urine.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------</td>
<td>-----------------------------------------------------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Increased in:</strong></td>
<td>Increased in: Diabetes mellitus, Cushing's syndrome (10–15%), chronic pancreatitis (30%). Drugs: corticosteroids, phenytoin, estrogen, thiazides.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Decreased in:</strong></td>
<td>Decreased in: Pancreatic islet cell disease with increased insulin, insulinoma, adrenocortical insufficiency, hypopituitarism, diffuse liver disease, malignancy (adrenocortical, stomach, fibrosarcoma), infant of a diabetic mother, enzyme deficiency diseases (eg, galactosemia). Drugs: insulin, ethanol, propranolol; sulfonpyrazone, tolbutamide, and other oral hypoglycemic agents.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Gastrin, serum**

<300 pg/mL [ng/L]

Marbled

$\text{Overnight fasting required.}$

Gastrin is secreted from G cells in the stomach antrum and stimulates acid secretion from the gastric parietal cells.

Values fluctuate throughout the day but are lowest in the early morning.

**Increased in:** Gastrinoma (Zollinger-Ellison syndrome) (80–93% sensitivity), antral G cell hyperplasia, hypochlorhydria, achlorhydria, chronic atrophic gastritis, pernicious anemia.

Drugs: antacids, cimetidine, and other H₂ blockers; omeprazole and other proton pump inhibitors.

**Decreased in:** Antrectomy with vagotomy.

Gastrin is the first-line test for determining whether a patient with active ulcer disease has a gastrinoma. Gastric analysis is not indicated.

Before interpreting an elevated level, be sure that the patient is not taking antacids, H₂ blockers, or proton pump inhibitors.

Both fasting and post-secretin infusion levels may be required for diagnosis.


Lancet 1996;347:270.

**Glucose**, serum

60–110 mg/dL [3.3–6.1 mmol/L]

**Panic:** <40 or >500 mg/dL

Marbled

$\text{Overnight fasting usually required.}$

Diagnosis of diabetes mellitus requires a fasting plasma glucose of >126 mg/dL on more than one occasion.

Hypoglycemia is defined as a glucose of <50 mg/dL in men and <40 mg/dL in women.

While random serum glucose levels correlate with home glucose monitoring results (weekly mean capillary glucose values), there is wide fluctuation within individuals. Thus, glycated hemoglobin levels are favored to monitor glycemic control.

JAMA 1999;281:1203.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose tolerance test, serum</td>
<td>The test determines the ability of a patient to respond appropriately to a glucose load.</td>
<td>Increased glucose rise (decreased glucose tolerance) in: Diabetes mellitus, impaired glucose tolerance, gestational diabetes, severe liver disease, hyperthyroidism, stress (infection), increased absorption of glucose from GI tract (hyperthyroidism, gastrectomy, gastroenterostomy, vagotomy, excess glucose intake), Cushing’s syndrome, pheochromocytoma. Drugs: diuretics, oral contraceptives, glucocorticoids, nicotinic acid, phenytoin. Decreased glucose rise (flat glucose curve) in: Intestinal disease (celiac sprue, Whipple’s disease), adrenal insufficiency (Addison’s disease, hypopituitarism), pancreatic islet cell tumors or hyperplasia.</td>
<td>Test is not generally required for diagnosis of diabetes mellitus. In screening for gestational diabetes, the glucose tolerance test is performed between 24 and 28 weeks of gestation. After a 50-g oral glucose load, a 2-hour postprandial blood glucose is measured as a screen. If the result is &gt; 140 mg/dL, then the full test with 100-g glucose load is done using the following reference ranges: Fasting: &lt;105 1-hour: &lt;190 2-hour: &lt;165 3-hour: &lt;145 mg/dL Routine screening for gestational diabetes has not been found to be cost-effective, and is not recommended by the Canadian Task Force on the Periodic Health Examination. J Fam Pract 1993;37:27. Diabetes Care 1999;22(Suppl 1):55.</td>
</tr>
<tr>
<td>Glucose tolerance test, serum</td>
<td>Fasting: &lt;110 1-hour: &lt;200 2-hour: &lt;140 mg/dL [Fasting: &lt;6.4 1-hour: &lt;11.0 2-hour: &lt;7.7 mmol/L] Marbled $$</td>
<td></td>
<td>Subjects should receive a 150- to 200-g/d carbohydrate diet for at least 3 days prior to test. A 75-g glucose dose is dissolved in 300 mL of water for adults (1.75 g/kg for children) and given after an overnight fast. Serial determinations of plasma or serum venous blood glucose are obtained at baseline, at 1 hour, and at 2 hours.</td>
</tr>
<tr>
<td>Glucose tolerance test, serum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose-6-phosphate dehydrogenase screen, whole blood (G6PD)</td>
<td>G6PD is an enzyme in the hexose monophosphate shunt that is essential in generating reduced glutathione and NADPH, which protect hemoglobin from oxidative denaturation. Numerous G6PD isoenzymes have been identified. Most African-Americans have G6PD-A(+) isoenzyme. 10–15% have G6PD-A(−), which has only 15% of normal enzyme activity. It is transmitted in an X-linked recessive manner. Some Mediterranean people have the B− variant that has extremely low enzyme activity (1% of normal).</td>
<td><strong>Increased in:</strong> Young erythrocytes (reticulocytosis). <strong>Decreased in:</strong> G6PD deficiency.</td>
<td>In deficient patients, hemolytic anemia can be triggered by oxidant agents: antimalarial drugs (eg, chloroquine), nalidixic acid, nitrofurantoin, dapsone, phenacetin, vitamin C, and some sulfonamides. Any African-American about to be given an oxidant drug should be screened for G6PD deficiency. (Also screen people from certain Mediterranean areas: Greece, Italy, etc.) Hemolytic episodes can also occur in deficient patients who eat fava beans, in patients with diabetic acidosis, and in infections. G6PD deficiency may be the cause of hemolytic disease of newborns in Asians and Mediterraneans. Ann Intern Med 1985;103:245.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Glutamine, CSF</td>
<td>Glutamine is synthesized in the brain from ammonia and glutamic acid. Elevated CSF glutamine is associated with hepatic encephalopathy.</td>
<td><strong>Increased in:</strong> Hepatic encephalopathy.</td>
<td>Test is not indicated if albumin, alanine aminotransferase (ALT), bilirubin, and alkaline phosphatase are normal or if there is no clinical evidence of liver disease. Hepatic encephalopathy is essentially ruled out if the CSF glutamine is normal. Arch Intern Med 1971;127:1033. Science 1974;183:81.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td>Glycohemoglobin; glycated (glycosylated) hemoglobin, serum (HbA1c)</td>
<td>During the life span of each red blood cell, glucose combines with hemoglobin to produce a stable glycated hemoglobin. The level of glycated hemoglobin is related to the mean plasma glucose level during the prior 1–3 months. There are three glycated A hemoglobins, HbA1a, HbA1b, and HbA1c. Some assays quantitate HbA1c; some quantitate total HbA1; and some quantitate all glycated hemoglobins, not just A.</td>
<td>Increased in: Diabetes mellitus, splenectomy. Falsely high results can occur depending on the method used and may be due to presence of hemoglobin F or uremia. Decreased in: Any condition that shortens red cell life span (hemolytic anemias, congenital spherocytosis, acute or chronic blood loss, sickle cell disease, hemoglobinopathies).</td>
<td>Test is not currently recommended for diagnosis of diabetes mellitus, though it performs well. It is used to monitor long-term control of blood glucose level. Reference ranges are method-specific. Development and progression of chronic complications of diabetes are related to the degree of altered glycemia. Measurement of HbA1c can improve metabolic control by leading to changes in diabetes treatment. Diabetes Care 1994;17:938. JAMA 1996;246:1246.</td>
</tr>
<tr>
<td>3.9–6.9% (method-dependent)</td>
<td>Lavender $$</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Growth hormone, serum (GH)</strong></td>
<td>Growth hormone is a single-chain polypeptide of 191 amino acids that induces the generation of somatomedins, which directly stimulate collagen and protein synthesis. GH levels are subject to wide fluctuations during the day.</td>
<td><strong>Increased in:</strong> Acromegaly (90% have GH levels &gt;10 ng/mL), Laron dwarfism (defective GH receptor), starvation. Drugs: dopamine, levodopa. <strong>Decreased in:</strong> Pituitary dwarfism, hypopituitarism.</td>
<td>Nonsuppressibility of GH levels to &lt;2 ng/mL after 100 g oral glucose and elevation of IGF-1 levels are the two most sensitive tests for acromegaly. Random determinations of GH are rarely useful in the diagnosis of acromegaly. For the diagnosis of hypopituitarism or growth hormone deficiency in children, an insulin hypoglycemia test has been used. Failure to increase GH levels to &gt; 5 ng/mL after insulin (0.1 unit/kg) is consistent with GH deficiency. Endocrinol Metab Clin North Am 1992;21:649. Clin Endocrinol 1997;46:531. Lancet 1998;352:1455.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Haptoglobin, serum</strong></td>
<td>Haptoglobin is a glycoprotein synthesized in the liver that binds free hemoglobin.</td>
<td><strong>Increased in:</strong> Acute and chronic infection (acute phase reactant), malignancy, biliary obstruction, ulcerative colitis, myocardial infarction, and diabetes mellitus. <strong>Decreased in:</strong> Newborns and children, posttransfusion intravascular hemolysis, autoimmune hemolytic anemia, liver disease (10%). May be decreased following uneventful transfusion (10%) for unknown reasons.</td>
<td>Low haptoglobin is considered an indicator of hemolysis, but it is of uncertain clinical predictive value because of the greater prevalence of other conditions associated with low levels and because of occasional normal individuals who have very low levels. It thus has low specificity. High-normal levels probably rule out significant intravascular hemolysis. JAMA 1980;243:1909. Clin Chem 1987;33:1265.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><em>Helicobacter pylori</em> antibody, serum Negative Marbled $$</td>
<td><em>Helicobacter pylori</em> is a gram-negative spiral bacterium that is found on gastric mucosa. It induces acute and chronic inflammation in the gastric mucosa and a positive serologic antibody response. Serologic testing for <em>H pylori</em> antibody (IgG) is by ELISA.</td>
<td><strong>Increased (positive) in:</strong> Histologic (chronic or chronic active) gastritis due to <em>H pylori</em> infection (with or without peptic ulcer disease). Sensitivity 98%, specificity 48%. Asymptomatic adults: 15–50%.</td>
<td>95% of patients with duodenal ulcers and &gt;70% of patients with gastric ulcers have chronic infection with <em>H pylori</em> along with associated histologic gastritis. All patients with peptic ulcer disease and positive <em>H pylori</em> serology should be treated to eradicate <em>H pylori</em> infection. The prevalence of <em>H pylori</em>-positive serologic tests in asymptomatic adults is approximately 35% overall but is &gt;50% in patients over age 60. Fewer than one in six adults with <em>H pylori</em> antibody develop peptic ulcer disease. Treatment of asymptomatic adults is not currently recommended. The role of <em>H pylori</em> in patients with chronic dyspepsia is controversial. There is currently no role for treatment of such patients except in clinical trials. After successful eradication, serologic titers fall over a 3- to 6-month period but remain positive in up to 50% of patients at 1 year. Gastroenterol Clin North Am 1993;22:105. Gut 1994;35:19. Ann Intern Med 1994;120:977. JAMA 1994;272:65. Can J Infect Dis 1998;9:277.</td>
</tr>
<tr>
<td><strong>Hematocrit, whole blood</strong> (Hct)</td>
<td>The hematocrit represents the percentage of whole blood volume composed of erythrocytes. Laboratory instruments calculate the Hct from the erythrocyte count (RBC) and the mean corpuscular volume (MCV) by the formula: ( \text{Hct} = \text{RBC} \times \text{MCV} )</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| Male: 39–49%  
Female: 35–45% (age-dependent) | **Increased in:** Hemoconcentration (as in dehydration, burns, vomiting), polycythemia, extreme physical exercise.  
**Decreased in:** Macrocytic anemia (liver disease, hypothyroidism, vitamin B\(_12\) deficiency, folate deficiency), normocytic anemia (early iron deficiency, anemia of chronic disease, hemolytic anemia, acute hemorrhage) and microcytic anemia (iron deficiency, thalassemia).  
Conversion from hemoglobin (Hb) to hematocrit is roughly \( \text{Hb} \times 3 = \text{Hct} \). Hematocrit reported by clinical laboratories is not a spun hematocrit. The spun hematocrit may be spuriously high if the centrifuge is not calibrated, if the specimen is not spun to constant volume, or if there is “trapped plasma.”  
In determining transfusion need, the clinical picture must be considered in addition to the hematocrit. Point-of-care instruments may not measure hematocrit accurately in all patients.  
| Lavender $ |  |

| **Hemoglobin A\(_2\), whole blood** (HbA\(_2\)) | HbA\(_2\) is a minor component of normal adult hemoglobin (< 3.5% of total Hb).  
**Increased in:** \( \beta \)-Thalassemia major (HbA\(_2\) levels 4–10% of total Hb), \( \beta \)-thalassemia minor (HbA\(_2\) levels 4–8% of total Hb).  
**Decreased in:** Untreated iron deficiency, hemoglobin H disease.  
Test is useful in the diagnosis of \( \beta \)-thalassemia minor (in absence of iron deficiency, which decreases HbA\(_2\) and can mask the diagnosis). Quantitated by column chromatographic or automated HPLC techniques. Normal HbA\(_2\) levels are seen in delta \( \beta \)-thalassemia or very mild \( \beta \)-thalassemias.  
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.5–3.5% of total hemoglobin (Hb)</td>
<td>Lavender $$</td>
</tr>
<tr>
<td>Lavender $</td>
<td>** Hemoglobin A(_2)**</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td><strong>Hemoglobin electrophoresis, whole blood</strong></td>
<td>Hemoglobin electrophoresis is used as a screening test. It is used to detect and differentiate hemoglobin variants. Separation of hemoglobins by electrophoresis is based on different rates of migration of charged hemoglobin molecules in an electric field.</td>
</tr>
<tr>
<td>HbA: &gt; 95</td>
<td></td>
</tr>
<tr>
<td>HbA₂: 1.5–3.5%</td>
<td></td>
</tr>
<tr>
<td>Lavender, blue, or green</td>
<td></td>
</tr>
<tr>
<td><strong>Hemoglobin, fetal, whole blood (HbF)</strong></td>
<td>Fetal hemoglobin constitutes about 75% of total hemoglobin at birth and declines to 50% at 6 weeks, 5% at 6 months, and &lt;1.5% by 1 year. During the first year, adult hemoglobin (HbA) becomes the predominant hemoglobin.</td>
</tr>
<tr>
<td><strong>Hemoglobin, total, whole blood (Hb)</strong></td>
<td>Hemoglobin is the major protein of erythrocytes and transports oxygen from the lungs to peripheral tissues. It is measured by spectrophotometry on automated instruments after hemolysis of red cells and conversion of all hemoglobin to cyanmethemoglobin.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td><strong>Hemosiderin,</strong> urine</td>
<td>Hemosiderin is a protein produced by the digestion of hemoglobin. Its presence in the urine indicates acute or chronic release of free hemoglobin into the circulation with accompanying depletion of the scavenging proteins, hemopexin and haptoglobin. Presence of hemosiderin usually indicates intravascular hemolysis or recent transfusion.</td>
</tr>
<tr>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Urine container $$$</td>
<td>Fresh, random sample.</td>
</tr>
<tr>
<td><strong>Hepatitis A antibody,</strong> serum (Anti-HAV)</td>
<td>Hepatitis A is caused by a non-enveloped 27 nm RNA virus of the enterovirus-picornavirus group and is usually acquired by the fecal-oral route. IgM antibody is detectable within a week after symptoms develop and persists for 6 months. IgG appears 4 weeks later than IgM and persists for years (see Figure 8–7, p 343, for time course of serologic changes).</td>
</tr>
<tr>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Marbled $$$</td>
<td></td>
</tr>
<tr>
<td><strong>Hepatitis B surface antigen</strong>, serum (HBsAg)</td>
<td>In hepatitis B virus infection, surface antigen is detectable 2–5 weeks before onset of symptoms, rises in titer, and peaks at about the time of onset of clinical illness. Generally it persists for 1–5 months, declining in titer and disappearing with resolution of clinical symptoms (see Figure 8–8, p 344, for time course of serologic changes).</td>
</tr>
<tr>
<td><strong>Hepatitis B surface antibody</strong>, serum (HBsAb, anti-HBs)</td>
<td>Test detects antibodies to hepatitis B virus (HBV) which are thought to confer immunity to hepatitis B. Since several subtypes of hepatitis B exist, there is a possibility of subsequent infection with a second subtype.</td>
</tr>
<tr>
<td><strong>Hepatitis B core antibody, total</strong>, serum (HBcAb, anti-HBc)</td>
<td>HBcAB (IgG and IgM) will be positive (as IgM) about 2 months after exposure to hepatitis B. Its persistent positivity may reflect chronic hepatitis (IgM) or recovery (IgG). (See Figure 8–8, p 344, for time course of serologic changes.)</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td><strong>Hepatitis B antigen/antibody (HBeAg/Ab), serum</strong></td>
<td>HBeAg is a soluble protein secreted by hepatitis B virus, related to HBcAg, indicating viral replication and infectivity. Two distinct serologic types of hepatitis B have been described, one with a positive HBeAg and the other with a negative HBeAg and a positive anti-HBe antibody.</td>
</tr>
<tr>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
</tr>
<tr>
<td><strong>Hepatitis C antibody, serum (HCAb)</strong></td>
<td>Detects antibody to hepatitis C virus. Current screening test (ELISA) detects antibodies to proteins expressed by putative structural (HC34) and nonstructural (HC31, C100-3) regions of the HCV genome. The presence of these antibodies indicates that the patient has been infected with HCV, may harbor infectious HCV, and may be capable of transmitting HCV. A recombinant immunoblot assay (RIBA) is available as a confirmatory test.</td>
</tr>
<tr>
<td>Negative</td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
</tr>
<tr>
<td><strong>Hepatitis D antibody</strong></td>
<td>This antibody is a marker for acute or persisting infection with the delta agent, a defective RNA virus that can only infect HBsAg-positive patients. Hepatitis B virus (HBV) plus hepatitis D virus (HDV) infection may be more severe than HBV infection alone. Antibody to HDV ordinarily persists for about 6 months following acute infection. Further persistence indicates carrier status.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Heterophile agglutination</strong>, serum (Monospot, Paul-Bunnell test)</td>
<td>Infectious mononucleosis is an acute saliva-transmitted infectious disease due to the Epstein-Barr virus (EBV). Heterophile (Paul-Bunnell) antibodies (IgM) appear in 60% of mononucleosis patients within 1–2 weeks and in 80–90% within the first month. They are not specific for EBV but are found only rarely in other disorders. Titers are substantially diminished by 3 months after primary infection and are not detectable by 6 months.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td><strong>Negative</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Marbled (serum)</strong></td>
<td></td>
</tr>
</tbody>
</table>

Deliver urine, CSF in a clean plastic or glass container tube.
<table>
<thead>
<tr>
<th><strong>Histoplasma capsulatum precipitins, serum</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
</tr>
<tr>
<td>Marbled $$</td>
</tr>
</tbody>
</table>

Histoplasmosis is the most common systemic fungal infection and typically starts as a pulmonary infection with influenza-like symptoms. This may heal, progress, or lie dormant with reinfection occurring at a later time. This test screens for presence of histoplasma antibody by detecting precipitin “H” and “M” bands. Positive H band indicates active infection, M band indicates acute or chronic infection or prior skin testing. Presence of both suggests active histoplasmosis.

<table>
<thead>
<tr>
<th><strong>Histoplasma capsulatum complement fixation (CF) antibody, serum</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;1:4 titer</td>
</tr>
<tr>
<td>Marbled $$</td>
</tr>
<tr>
<td>Submit paired sera, one specimen collected within 1 week after onset of illness and another 2 weeks later.</td>
</tr>
</tbody>
</table>

Quantitates level of histoplasma antibody. Antibodies in primary pulmonary infections are generally found within 4 weeks after exposure and frequently are present at the time symptoms appear. Two types of CF test are available based on mycelial antigen and yeast phase antigen. The yeast phase test is considerably more sensitive. Latex agglutination (LA) and ELISA tests are also available but are less reliable.

<table>
<thead>
<tr>
<th><strong>Positive in:</strong> Previous, chronic, or acute histoplasma infection, recent histoplasmin skin testing. Cross-reactions at low levels in patients with blastomycosis and coccidioidomycosis.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Increased in:</strong> Previous, chronic, or acute histoplasma infection (75–80%), recent histoplasmin skin testing (20%), other fungal disease, leishmaniasis. Cross-reactions in patients with blastomycosis and coccidioidomycosis.</td>
</tr>
</tbody>
</table>

Histoplasmosis is usually seen in the Mississippi and Ohio River Valleys but may appear elsewhere. Test is useful as a screening test or as an adjunct to complement fixation test (see below) in diagnosis of systemic histoplasmosis. Rose NR et al (editors): Manual of Clinical Laboratory Immunology, 4th ed. American Society for Microbiology, 1992.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HIV antibody</strong>, serum</td>
<td>This test detects antibody against the human immunodeficiency virus-1 (HIV-1), the etiologic agent of AIDS. HIV antibody test is considered positive only when a repeatedly reactive enzyme immunoassay (EIA) is confirmed by a Western blot analysis or immunofluorescent antibody test (IFA).</td>
<td><strong>Positive in:</strong> HIV infection: EIA sensitivity &gt;99% after first 2–4 months of infection, specificity 99%. When combined with confirmatory test, specificity is 99.995%.</td>
<td>A positive p24 antigen test in an HIV antibody-negative individual must be confirmed by a viral neutralization assay. While Western blot test is currently the most sensitive and specific assay for HIV serodiagnosis, it is highly dependent on the proficiency of the laboratory performing the test and on the standardization of the procedure. Ann Intern Med 1987;106:671. Arch Pathol Lab Med 1989;113:975. JAMA 1991;266:2861. Infect Dis Clin North Am 1993;7:203.</td>
</tr>
<tr>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>HLA typing</strong>, serum and blood (HLA)</td>
<td>The human leukocyte antigen (HLA) system consists of four closely linked loci (HLA-A, -B, -C, and -DR) located on the short arm of chromosome 6. The most widely used technique for HLA typing is the microlymphocyte toxicity test. This is a complement-mediated serologic assay in which antiserum containing specific anti-HLA antibodies is added to peripheral blood lymphocytes. Cell death indicates that the lymphocytes carried the specific targeted antigen.</td>
<td><strong>Useful in:</strong> Evaluation of transplant candidates and potential donors and for paternity and forensic testing.</td>
<td>While diseases associated with particular HLA antigens have been identified, HLA typing for the diagnosis of these diseases is not generally indicated. Cell 1984;36:1.</td>
</tr>
<tr>
<td><strong>HLA-B27 typing</strong>, whole blood</td>
<td><strong>5-Hydroxyindoleacetic acid</strong>, urine (5-HIAA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------------------------</td>
<td>--------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td>2–8 mg/24 h [10–40 µmol/d]</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yellow</td>
<td>Urine bottle containing hydrochloric acid</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Specimens must be &lt;24 hours old.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The HLA-B27 allele is found in approximately 8% of the US white population. It occurs less frequently in the African-American population.

There is an increased incidence of spondyloarthritis among patients who are HLA-B27-positive. HLA-B27 is present in 88% of patients with ankylosing spondylitis. It is also associated with the development of Reiter’s syndrome (80%) following infection with *Shigella* or *Salmonella*.

The best diagnostic test for ankylosing spondylitis is a lumbar spine film and not HLA-B27 typing. HLA-B27 testing is not usually clinically indicated.


Serotonin (5-hydroxytryptamine) is a neurotransmitter that is metabolized by monoamine oxidase (MAO) to 5-HIAA and then excreted into the urine. Serotonin is secreted by most carcinoid tumors, which arise from neuroendocrine cells in locations derived from the embryonic gut.

**Increased in:** Metastatic carcinoid tumor (foregut, midgut, and bronchial). Nontropical sprue (slight increase). Diet of bananas, walnuts, avocado, eggplant, pineapple, plums. Drugs: reserpine.

**Negative in:** Rectal carcinoids (usually), renal insufficiency. Drugs: MAO inhibitors, phenothiazines.

Test is often falsely positive because pretest probability is low. Using 5-HIAA/Cr ratio may improve performance.

Since most carcinoid tumors drain into the portal vein and serotonin is rapidly cleared by the liver, the carcinoid syndrome (flushing, bronchial constriction, diarrhea, hypotension, and cardiac valvular lesions) is a late manifestation of carcinoid tumors, appearing only after hepatic metastasis has occurred.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
</table>
| **IgG index, serum and CSF**<br>0.29–0.59 ratio<br>Marbled (for serum) and glass/plastic tube (for CSF)<br>$$$
| **Immunoelectrophoresis, serum (IEP)**<br>Negative<br>Marbled $$$ | Immunoelectrophoresis is used to identify specific immunoglobulin (Ig) classes. Serum is separated electrophoretically and reacted with antisera of known specificity. Newer technique (immunofixation) is available and easier to interpret. | **Positive in:** Presence of identifiable monoclonal paraprotein: multiple myeloma, Waldenström’s macroglobulinemia, Franklin’s disease (heavy chain disease), lymphoma, leukemia, monoclonal gammopathy of undetermined significance. The most common form of myeloma is the IgG type. | Test is indicated to identify an Ig spike seen on serum protein electrophoresis, to differentiate a polyclonal from a monoclonal increase, and to identify the nature of a monoclonal increase. Test is not quantitative and is not sensitive enough to use for the evaluation of immunodeficiency. Order quantitative immunoglobulins for this purpose (see below). Hematol Oncol Clin North Am 1997;11:71. Arch Pathol Lab Med 1999;123:114. Arch Pathol Lab Med 1999;123:126. |
**Immunoglobulins**, serum (Ig)

<table>
<thead>
<tr>
<th>IgA: 78–367 mg/dL</th>
<th>IgG: 583–1761 mg/dL</th>
<th>IgM: 52–335 mg/dL</th>
</tr>
</thead>
<tbody>
<tr>
<td>[IgA: 0.78–3.67 g/L]</td>
<td>[IgG: 5.83–17.6 g/L]</td>
<td>[IgM: 0.52–3.35 g/L]</td>
</tr>
</tbody>
</table>

Marbled $$$

- IgG makes up about 85% of total serum immunoglobulins and predominates late in immune responses. It is the only immunoglobulin to cross the placenta.
- IgM antibody predominates early in immune responses.
- Secretory IgA plays an important role in host defense mechanisms by blocking transport of microbes across mucosal surfaces.

- **IgG**: *Polyclonal*: Autoimmune diseases (eg, SLE, rheumatoid arthritis), sarcoidosis, chronic liver diseases, some parasitic diseases, chronic or recurrent infections.
  *Monoclonal*: Multiple myeloma (IgG type), lymphomas, or other malignancies.

- **IgM**: *Polyclonal*: Isolated infections such as viral hepatitis, infectious mononucleosis, early response to bacterial or parasitic infection.
  *Monoclonal*: Waldenström’s macroglobulinemia, lymphoma.

- **IgA**: *Polyclonal*: Chronic liver disease, chronic infections (especially of the GI and respiratory tracts).
  *Monoclonal*: Multiple myeloma (IgA).

- **↓ IgG**: Immunosuppressive therapy, genetic (SCID, Wiskott-Aldrich syndrome, common variable immunodeficiency).
- **↓ IgM**: Immunosuppressive therapy.
- **↓ IgA**: Inherited IgA deficiency (ataxia-telangiectasia, combined immunodeficiency disorders).

Quantitative immunoglobulin levels are indicated in the evaluation of immunodeficiency or the quantitation of a paraprotein.

IgG deficiency is associated with recurrent and occasionally severe pyogenic infections.

The most common form of multiple myeloma is the IgG type.

Science 1986;231:1241.


<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Inhibitor screen, plasma</strong></td>
<td>Test is useful for evaluating a prolonged partial thromboplastin time (PTT), prothrombin time (PT), or thrombin time. (Presence of heparin should first be excluded.) Patient’s plasma is mixed with normal plasma and a PTT is performed. If the patient has a factor deficiency, the postmixing PTT will be normal. If an inhibitor is present, it will be prolonged.</td>
<td><strong>Positive in:</strong> Presence of inhibitor: Anti-phospholipid antibodies (lupus anticoagulant (LAC) or anticardiolipin antibodies), factor-specific antibodies. <strong>Negative in:</strong> Factor deficiencies.</td>
<td>LAC prolongs a PTT immediately and is the most common inhibitor. Poor sensitivity for lupus anticoagulant owing to relatively high phospholipid levels in this assay system. 1–4 hour incubation period may be needed to detect factor-specific antibodies with low in vitro affinities. About 15% of hemophilia A patients develop inhibitor against factor VIII. Semin Thromb Hemost 1994;20:79. Thromb Haemost 1996;16:146.</td>
</tr>
<tr>
<td><strong>Insulin antibody, serum</strong></td>
<td>Insulin antibodies develop in nearly all diabetics treated with insulin. Most antibodies are IgG and do not cause clinical problems. Occasionally, high-affinity antibodies can bind to exogenous insulin and cause insulin resistance.</td>
<td><strong>Increased in:</strong> Insulin therapy, type I diabetics before treatment (secondary to autoimmune pancreatic B cell destruction).</td>
<td>Insulin antibodies interfere with most assays for insulin. Insulin antibody test is not sensitive or specific for the detection of surreptitious insulin use; use C-peptide level (see p 62). Anti-insulin and islet cell antibodies are poor predictors of IDDM and only roughly correlate with insulin requirements in patients with diabetes. Diabetes 1996;45:1720. Diabetes Care 1996;19:146.</td>
</tr>
<tr>
<td><strong>Insulin, immunoreactive</strong>, serum</td>
<td>Measures levels of insulin, either endogenous or exogenous.</td>
<td><strong>Increased in:</strong> Insulin-resistant states (e.g., obesity, type II diabetes mellitus, uremia, glucocorticoids, acromegaly), liver disease, surreptitious use of insulin or oral hypoglycemic agents, insulinoma (pancreatic islet cell tumor). <strong>Decreased in:</strong> Type I diabetes mellitus, hypopituitarism.</td>
<td>Measurement of serum insulin level has little clinical value except in the diagnosis of fasting hypoglycemia. An insulin-to-glucose ratio of &gt;0.3 is presumptive evidence of insulinoma. C-peptide should be used as well as serum insulin to distinguish insulinoma from surreptitious insulin use, since C-peptide will be absent with exogenous insulin use (see C-peptide, p 62). Eur J Endocrinol 1998;138:86.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>6–35 µU/mL [42–243 pmol/L]</td>
<td>Marbled</td>
<td><strong>Increased in:</strong> Hemosiderosis (e.g., multiple transfusions, excess iron administration), hemolytic anemia, pernicious anemia, aplastic or hypoplastic anemia, viral hepatitis, lead poisoning, thalassemia, hemochromatosis. Drugs: estrogens, ethanol, oral contraceptives. <strong>Decreased in:</strong> Iron deficiency, nephrotic syndrome, chronic renal failure, many infections, active hematopoiesis, remission of pernicious anemia, hypothyroidism, malignancy (carcinoma), postoperative state, kwashiorkor.</td>
<td>Absence of stainable iron on bone marrow aspirate differentiates iron deficiency from other causes of microcytic anemia (e.g., thalassemia, sideroblastic anemia, some chronic disease anemias), but the procedure is invasive and expensive. Serum iron, iron-binding capacity, and transferrin saturation—or serum ferritin—may obviate the need for bone marrow examination. Serum iron, iron-binding capacity, and transferrin saturation are useful (see p 90) in screening family members for hereditary hemochromatosis. Recent transfusion will confound the test results. JAMA 1997;277:973. Ann Intern Med 1998;129:905. Ann Intern Med 1998;129:923.</td>
</tr>
<tr>
<td>Marbled</td>
<td>Fasting sample required. Measure glucose concurrently.</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Iron</strong>, serum (Fe$^{2+}$)</td>
<td>Plasma iron concentration is determined by absorption from the intestine; storage in the intestine, liver, spleen, bone marrow; rate of breakdown or loss of hemoglobin; and rate of synthesis of new hemoglobin.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>50–175 µg/dL [9–31 µmol/L]</td>
<td>Marbled</td>
<td><strong>Increased in:</strong> Hemosiderosis (e.g., multiple transfusions, excess iron administration), hemolytic anemia, pernicious anemia, aplastic or hypoplastic anemia, viral hepatitis, lead poisoning, thalassemia, hemochromatosis. Drugs: estrogens, ethanol, oral contraceptives. <strong>Decreased in:</strong> Iron deficiency, nephrotic syndrome, chronic renal failure, many infections, active hematopoiesis, remission of pernicious anemia, hypothyroidism, malignancy (carcinoma), postoperative state, kwashiorkor.</td>
<td>Absence of stainable iron on bone marrow aspirate differentiates iron deficiency from other causes of microcytic anemia (e.g., thalassemia, sideroblastic anemia, some chronic disease anemias), but the procedure is invasive and expensive. Serum iron, iron-binding capacity, and transferrin saturation—or serum ferritin—may obviate the need for bone marrow examination. Serum iron, iron-binding capacity, and transferrin saturation are useful (see p 90) in screening family members for hereditary hemochromatosis. Recent transfusion will confound the test results. JAMA 1997;277:973. Ann Intern Med 1998;129:905. Ann Intern Med 1998;129:923.</td>
</tr>
<tr>
<td>$</td>
<td>Avoid hemolysis.</td>
<td>$</td>
<td>$</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td>Iron-binding capacity, total, serum (TIBC) 250–460 µg/dL [45–82 µmol/L] Marbled $$</td>
<td>Iron is transported in plasma complexed to transferrin, which is synthesized in the liver. Total iron-binding capacity is calculated from transferrin levels measured immunologically. Each molecule of transferrin has two iron-binding sites, so its iron-binding capacity is 1.47 mg/g. Normally, transferrin carries an amount of iron representing about 16–60% of its capacity to bind iron (ie, % saturation of iron-binding capacity is 16–60%).</td>
<td>Increased in: Iron deficiency anemia, late pregnancy, infancy, hepatitis. Drugs: oral contraceptives. Decreased in: Hypoproteinemic states (eg, nephrotic syndrome, starvation, malnutrition, cancer), hyperthyroidism, chronic inflammatory disorders, chronic liver disease, other chronic disease.</td>
<td>Increased % transferrin saturation with iron is seen in iron overload (iron poisoning, hemolytic anemia, sideroblastic anemia, thalassemia, hemochromatosis, pyridoxine deficiency, aplastic anemia). Decreased % transferrin saturation with iron is seen in iron deficiency (usually saturation &lt;16%). Transferrin levels can also be used to assess nutritional status. Recent transfusion will confound the test results. Clin Chem 1997;43:2408. Ann Intern Med 1998;129:925. Ann Intern Med 1998;129:962.</td>
</tr>
</tbody>
</table>
**Lactate dehydrogenase, serum (LDH)**

- **88–230 U/L**
  
  [1.46–3.82 µkat/L] (laboratory-specific)

- Marbled

- $Hemolyzed specimens are unacceptable.

LDH is an enzyme that catalyzes the interconversion of lactate and pyruvate in the presence of NAD/NADH. It is widely distributed in body cells and fluids. Because LDH is highly concentrated in red blood cells (RBCs), spuriously elevated serum levels will occur if RBCs are hemolyzed during specimen collection.

**Increased in:** Tissue necrosis, especially in acute injury of cardiac muscle, RBCs, kidney, skeletal muscle, liver, lung, or skin. Commonly elevated in various carcinomas and in *Pneumocystis carinii* pneumonia (78–94%) and lymphoma in AIDS. Marked elevations occur in hemolytic anemias, vitamin B₁₂ deficiency anemia, folate deficiency anemia, polycythemia vera, thrombotic thrombocytopenic purpura (TTP), hepatitis, cirrhosis, obstructive jaundice, renal disease, musculoskeletal disease, CHF. Drugs causing hepatotoxicity (eg, acetaminophen) or hemolysis.

**Decreased in:** Drugs: clofibrate, fluoride (low dose).

LDH is elevated after myocardial infarction (for 2–7 days), in liver congestion (eg, in CHF), and in *P carinii* pneumonia.

LDH is not a useful liver function test, and it is not specific enough for the diagnosis of hemolytic or megaloblastic anemias. Its main diagnostic use has been in myocardial infarction, when the creatine kinase-MB elevation has passed (see CK-MB, p 79, and Figure 8–17, p 353). LDH isoenzymes are preferred over total serum LDH in late diagnosis of MI, but both tests are now being replaced by cardiac troponin I levels. Arch Intern Med 1997;157:1441. Chest 1997;111:1187.

**Lactate dehydrogenase isoenzymes, serum (LDH isoenzymes)**

- **LDH₁/LDH₂** < 0.85

- Marbled

- $$Hemolyzed specimens are unacceptable.

LDH consists of five isoenzymes separable by electrophoresis. The fraction with the greatest electrophoretic mobility is called LDH₁; the one with the least, LDH₅. LDH₁ is found in high concentrations in heart muscle, RBCs, and kidney cortex; LDH₅ in skeletal muscle and liver.

**Increased in:** LDH₁/LDH₂ >0.85 in myocardial infarction, hemolysis (hemolytic or megaloblastic anemia) or acute renal infarction. LDH₂ is increased in liver disease, congestive heart failure, skeletal muscle injury, and essential thrombocytopenia.

**The only clinical indication for LDH isoenzyme measurement has been to rule out myocardial infarction in patients presenting more than 24 hours after onset of symptoms (LDH₁/LDH₂ >0.85 is usually present within 12–48 hours). It may also be helpful if CK-MB results cannot be easily interpreted. The test is being replaced by measurement of cardiac troponin I (see CK-MB, p 79). Arch Intern Med 1997;157:1441.**
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Lactate, venous blood</strong> 0.5–2.0 meq/L [mmol/L] Gray $$ Collect on ice in gray-top tube containing fluoride to inhibit in vitro glycolysis and lactic acid production.</td>
<td>Severe tissue anoxia leads to anaerobic glucose metabolism with production of lactic acid.</td>
<td><strong>Increased in:</strong> Lactic acidosis, ethanol ingestion, sepsis, shock, liver disease, diabetic ketoacidosis, muscular exercise, hypoxia; regional hypoperfusion (bowel ischemia); prolonged use of a tourniquet (spurious elevation); type I glycogen storage disease, fructose 1,6-diphosphatase deficiency (rare), pyruvate dehydrogenase deficiency. Drugs: phenformin, metformin, isoniazid toxicity.</td>
<td>Lactic acidosis should be suspected when there is a markedly increased anion gap (&gt;18 meq/L) in the absence of other causes (eg, renal failure, ketosis, ethanol, methanol, or salicylate). Lactic acidosis is characterized by lactate levels &gt;5 mmol/L in association with metabolic acidosis. Tissue hypoperfusion is the most common cause. Blood lactate levels may indicate whether perfusion is being restored by therapy. Am J Med 1996;101:109. Ann Intern Med 1997;127:170. Medicine 1998;77:73. Semin Nephrol 1998;18:83.</td>
</tr>
<tr>
<td><strong>Lead, whole blood (Pb)</strong></td>
<td>Lead salts are absorbed through ingestion, inhalation, or the skin. About 5–10% of ingested lead is found in blood and 95% of this is in erythrocytes. 80–90% is taken up by bone, where it is relatively inactive. Lead poisons enzymes by binding to protein disulfide groups, leading to cell death. Lead levels fluctuate. Several specimens may be needed to rule out lead poisoning.</td>
<td><strong>Increased in:</strong> Lead poisoning, including abnormal ingestion (especially lead-containing paint, moonshine whiskey), occupational exposures (metal smelters, miners, welders, storage battery workers, auto manufacturers, ship builders, paint manufacturers, printing workers, pottery workers, gasoline refinery workers), retained bullets.</td>
<td>Subtle neurologic impairment may be detectable in children with lead levels of 15 µg/dL and in adults at 30 µg/dL; full-blown symptoms appear at &gt;60 µg/dL. Most chronic lead poisoning leads to a moderate anemia with basophilic stippling of erythrocytes on peripheral blood smear. Acute poisoning is rare and associated with abdominal pain and constipation. Blood lead levels are useful in the diagnosis. Industrial workers’ limit: &lt;50 µg/dL. Pediatrics 1994;93:201. Pediatrics 1996;97:79. Ann Intern Med 1999;130:7.</td>
</tr>
<tr>
<td><strong>Child (&lt;6 yrs):</strong></td>
<td>&lt;10 mg/dL</td>
<td>Navy $$ Use trace metal-free navy blue top tube with heparin.</td>
<td></td>
</tr>
<tr>
<td><strong>Child (&gt;6 yrs):</strong></td>
<td>&lt;25 mg/dL</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adult:</strong></td>
<td>&lt;40 µg/dL</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>[Child (&lt;6):</strong></td>
<td>&lt;0.48 mmol/L</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Child (&gt;6):</strong></td>
<td>&lt;1.21 mol/L</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Adult:</strong></td>
<td>&lt;1.93 µmol/L</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Navy $$$ Collect in a plastic tube.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<p>| <strong>Lecithin/sphingomyelin ratio, amniotic fluid (L/S ratio)</strong> | This test is used to estimate lung maturity in fetuses at risk for hyaline membrane disease. As fetal pulmonary surfactant matures, there is a rapid rise in amniotic fluid lecithin content. To circumvent the dependency of lecithin concentrations on amniotic fluid volume and analytic recovery of lecithin, the assay examines the lecithin/sphingomyelin ratio. | <strong>Increased in:</strong> Contamination of amniotic fluid by blood, meconium, or vaginal secretions that contain lecithin (false-positives). <strong>Decreased in:</strong> Fetal lung immaturity; 95% of normal fetuses. | Test identifies fetal lung maturity effectively only 60% of the time: ie, 40% of fetuses with an L/S ratio of &lt;2.0 will not develop hyaline membrane disease. Precision of L/S ratio test is poor: results on a single sample may vary by ±25%. Test is not reliable to assess fetal lung maturity in offspring of diabetic mothers. Med Decis Making 1990;10:201. Clin Chem 1994;40:541. Am J Obstet Gynecol 1998;179;1640. |
| <strong>&gt;2.0 (method-dependent)</strong> | | |
| $$$ Collect in a plastic tube. |</p>
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Legionella antibody</strong>, serum</td>
<td><em>Legionella pneumophila</em> is a weakly staining gram-negative bacillus that causes Pontiac fever (acute influenza-like illness) and Legionnaire’s disease (a pneumonia that may progress to a severe multi-system illness). It does not grow on routine bacteriologic culture media. Antibodies are detected by indirect immunofluorescent tests to serogroup 1 of <em>L pneumophila</em>. There are at least six serogroups of <em>L pneumophila</em> and at least 22 species of <em>Legionella</em>.</td>
<td><strong>Increased in:</strong> Legionella infection (80% of patients with pneumonia have a fourfold rise in titer); cross-reactions with other infectious agents (<em>Yersinia pestis</em> [plague], <em>Francisella tularensis</em> [tularemia], <em>Bacteroides fragilis</em>, <em>Mycoplasma pneumoniae</em>, <em>Leptospira interrogans</em>, campylobacter serotypes).</td>
<td>A greater than fourfold rise in titer to &gt;1:128 in specimens gathered more than 3 weeks apart indicates recent infection. A single titer of &gt;1:256 is considered diagnostic. About 50–60% of cases of legionellosis may have a positive direct fluorescent antibody test. Culture can have a sensitivity of 50%. All three methods may increase sensitivity to 90%. This test is species-specific. Polyvalent antiserum is needed to test for all serogroups and species. Epidemiol Infect 1994;112:347. Clin Infect Dis 1996;23:656.</td>
</tr>
<tr>
<td>&lt;1:32 titer Marbled $$$ Submit paired sera, one collected within 2 weeks of illness and another 2–3 weeks later.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<p>| <strong>Leukocyte alkaline phosphatase</strong>, whole blood (LAP) | The test measures the amount of alkaline phosphatase in neutrophils in a semiquantitative fashion. Neutrophilic leukocytes on a peripheral blood smear are stained for alkaline phosphatase activity and then 100 are scored on a scale from 0 to 4+ on the basis of the intensity of the dye in their cytoplasm. | <strong>Increased in:</strong> Leukemoid reaction (eg, severe infections), polycythemia vera, myelofibrosis with myeloid metaplasia. <strong>Decreased in:</strong> Chronic myeloid leukemia, paroxysmal nocturnal hemoglobinuria. | Test may be helpful for distinguishing leukemoid reactions (high-normal or increased LAP) from chronic myeloid leukemia (decreased LAP), but it is poorly reproducible. Br J Haematol 1997;96:815. |
| 40–130 Based on 0–4+ rating of 100 PMNs Green $$ | | |
| Blood smear from finger stick preferred. If collecting venous blood, make smear as soon as possible. | | |</p>
<table>
<thead>
<tr>
<th>Leukocyte (white blood cell) count, total, whole blood (WBC count)</th>
<th>Measure of the total number of leukocytes in whole blood. Counted on automated instruments using light scattering or electrical impedance after lysis of red blood cells. WBCs are distinguished from platelets by size.</th>
<th><strong>Increased in:</strong> Infection, inflammation, hematologic malignancy, leukemia, lymphoma. Drugs: corticosteroids. <strong>Decreased in:</strong> Aplastic anemia (decreased production), B₁2 or folate deficiency (maturation defect), sepsis (decreased survival). Drugs: phenothiazines, chloramphenicol, aminopyrine.</th>
<th>A spurious increase may be seen when there are a large number of nucleated red cells. WBC count is a poor predictor of severity of disease in the diagnosis of appendicitis. Lab Med 1983;14:509. J Clin Pathol 1996;49:664. Am Surg 1998;64:983.</th>
</tr>
</thead>
<tbody>
<tr>
<td>3.4–10 × 10³/µL [× 10⁶/L]</td>
<td>Panc: &lt;1.5 × 10³/µL Lavender $</td>
<td><strong>Lipase, serum</strong></td>
<td>0–160 U/L [0–2.66 µkat/L] (laboratory-specific) Marbled $$</td>
</tr>
<tr>
<td>Lipases are responsible for hydrolysis of glycerol esters of long-chain fatty acids to produce fatty acids and glycerol. Lipases are produced in the liver, intestine, tongue, stomach, and many other cells. Assays are highly dependent on the substrate used.</td>
<td><strong>Increased in:</strong> Acute, recurrent, or chronic pancreatitis, pancreatic pseudocyst, pancreatic malignancy, peritonitis, biliary disease, hepatic disease, diabetes mellitus (especially diabetic ketoacidosis), intestinal disease, gastric malignancy or perforation.</td>
<td>The sensitivity of lipase in acute pancreatitis is similar to that of amylase; lipase remains elevated longer than amylase. The specificity of lipase and amylase in acute pancreatitis is similar, though both are poor. Test sensitivity is not very good for chronic pancreatitis or pancreatic cancer. Lipase to amylase ratio is not useful in distinguishing alcoholic from non-alcoholic pancreatitis. Arch Pathol Lab Med 1991;115:325. Clin Chem 1991;37:447. Am J Gastroenterol 1995;90:67.</td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Luteinizing hormone, serum (LH)</strong></td>
<td>LH is stimulated by the hypothalamic hormone gonadotropin-releasing hormone (GnRH). It is secreted from the anterior pituitary and acts on the gonads. LH is the principal regulator of steroid biosynthesis in the ovary and testis.</td>
<td><strong>Increased in:</strong> Primary hypogonadism, polycystic ovary syndrome, post-menopause. <strong>Decreased in:</strong> Pituitary or hypothalamic failure, anorexia nervosa, severe stress, malnutrition, Kallman’s syndrome (gonadotropin deficiency associated with anosmia). Drugs: digoxin, oral contraceptives, phenothiazines.</td>
<td>Intact human chorionic gonadotropin (hCG) cross-reacts with LH in most immunoassays so that LH levels appear to be falsely elevated in pregnancy or in individuals with hCG-secreting tumors. Repeated measurement may be required to diagnose gonadotropin deficiencies. Measurement of total testosterone is the test of choice to diagnose polycystic ovary syndrome. Br J Obstet Gynaecol 1992;99:232. J Clin Endocrinol Metab 1994;78:1208. Obstet Gynecol 1994;84:613.</td>
</tr>
<tr>
<td><strong>Lyme disease antibody, serum</strong></td>
<td>Test detects the presence of antibody to <em>Borrelia burgdorferi</em>, the etiologic agent in Lyme disease, an inflammatory disorder transmitted by the ticks <em>Ixodes dammini</em>, <em>I pacificus</em>, and <em>I scapularis</em> in the northeastern and midwestern, western, and southeastern USA, respectively. Detects IgM antibody, which develops within 3–6 weeks after the onset of rash; or IgG, which develops within 6–8 weeks after the onset of disease. IgG antibody may persist for months.</td>
<td><strong>Positive in:</strong> Lyme disease, asymptomatic individuals living in endemic areas, syphilis (<em>Treponema pallidum</em>), tick-borne relapsing fever (<em>Borrelia hermsii</em>). <strong>Negative</strong> during the first 5 weeks of infection or after antibiotic therapy.</td>
<td>Test is less sensitive in patients with only a rash. Since culture or direct visualization of the organism is difficult, serologic diagnosis (by ELISA) is indicated, though sensitivity and specificity and standardization of procedure between laboratories need improvement. Cross-reactions may occur with syphilis (should be excluded by RPR and treponemal antibody assays). N Engl J Med 1989;321:586. Ann Intern Med 1991;114:472. Ann Intern Med 1997;127:1106.</td>
</tr>
<tr>
<td><strong>Magnesium</strong>, serum (Mg²⁺)</td>
<td>Magnesium is primarily an intracellular cation (second most abundant, 60% found in bone); it is a necessary cofactor in numerous enzyme systems, particularly ATPases. In extracellular fluid, it influences neuromuscular response and irritability. Magnesium concentration is determined by intestinal absorption, renal excretion, and exchange with bone and intracellular fluid.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1.8–3.0 mg/dL [0.75–1.25 mmol/L]</td>
<td><strong>Panic:</strong> &lt;0.5 or &gt;4.5 mg/dL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

```
1.8–3.0 mg/dL [0.75–1.25 mmol/L]
```

Increased in: Dehydration, tissue trauma, renal failure, hypoadrenocorticism, hypothyroidism. Drugs: aspirin (prolonged use), lithium, magnesium salts, progesterone, triamterene. **Decreased in:** Chronic diarrhea, enteric fistula, starvation, chronic alcoholism, total parenteral nutrition with inadequate replacement, hypoparathyroidism (especially post parathyroid surgery), acute pancreatitis, chronic glomerulonephritis, hyperaldosteronism, diabetic ketoacidosis. Drugs: albuterol, amphotericin B, calcium salts, cisplatin, citrates (blood transfusion), cyclosporine, diuretics, ethacrynic acid.

MCH indicates the amount of hemoglobin per red blood cell in absolute units. Low MCH can mean hypochromia or microcytosis or both. High MCH is evidence of macrocytosis.

<table>
<thead>
<tr>
<th><strong>Mean corpuscular hemoglobin</strong>, blood (MCH)</th>
</tr>
</thead>
<tbody>
<tr>
<td>26–34 pg</td>
</tr>
<tr>
<td>Lavender $</td>
</tr>
</tbody>
</table>


MCH is calculated from measured values of hemoglobin (Hb) and red cell count (RBC) by the formula:

\[
MCH = \frac{Hb}{RBC}
\]


<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean corpuscular hemoglobin concentration, blood (MCHC)</td>
<td>MCHC describes how fully the erythrocyte volume is filled with hemoglobin and is calculated from measurement of hemoglobin (Hb), mean corpuscular volume (MCV), and red cell count (RBC) by the formula: MCHC = ( \frac{Hb}{MCV \times RBC} )</td>
<td><strong>Increased in:</strong> Marked spherocytosis. Spuriously increased in autoagglutination, hemolysis (with spuriously high Hb or low MCV or RBC), lipemia. Cellular dehydration syndromes, xerocytosis. <strong>Decreased in:</strong> Hypochromic anemia (iron deficiency, thalassemia, lead poisoning), sideroblastic anemia, anemia of chronic disease. Spuriously decreased with high white blood cell count, low Hb, or high MCV or RBC.</td>
<td>Lab Med 1983;14:509.</td>
</tr>
<tr>
<td>31–36 g/dL [310–360 g/L] Lavender $</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean corpuscular volume, blood (MCV)</td>
<td>Average volume of the red cell is measured by automated instrument, by electrical impedance, or by light scatter.</td>
<td><strong>Increased in:</strong> Liver disease, megaloblastic anemia (folate, B12 deficiencies), reticulocytosis, newborns. Spurious increase in autoagglutination, high white blood cell count. Drugs: methotrexate, phenytoin, zidovudine. <strong>Decreased in:</strong> Iron deficiency, thalassemia; decreased or normal in anemia of chronic disease.</td>
<td>MCV can be normal in combined iron and folate deficiency. In patients with two red cell populations (macrocytic and microcytic), MCV may be normal. MCV is an insensitive test in the evaluation of anemia. Patients with iron deficiency anemia or pernicious anemia commonly have a normal MCV. J Gen Intern Med 1990;5:187. Br J Haematol 1994;88:443. Am J Clin Pathol 1996;106:201.</td>
</tr>
<tr>
<td>80–100 fL Lavender $</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Common Laboratory Tests: Selection and Interpretation</strong></td>
<td><strong>Methanol, whole blood</strong></td>
<td><strong>Methanephrines, urine</strong></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td><strong>Increased in:</strong> Methanol intoxication.</td>
<td>Serum methanol levels &gt;20 mg/dL are toxic and levels &gt;40 mg/dL are life-threatening.</td>
<td>Catecholamines, secreted in excess by pheochromocytomas, are metabolized by the enzyme catechol-O-methyltransferase to methanephrines, and these are excreted in the urine.</td>
<td></td>
</tr>
<tr>
<td><strong>Increased in:</strong> Methanephrines (96% sensitivity, 98% specificity), neuroblastoma, ganglioneuroma. Drugs: monoamine oxidase inhibitors.</td>
<td>Methanephrines, urine 0.3–0.9 mg/24 h [1.6–4.9 µmol/24 h]</td>
<td>Urine bottle containing hydrochloric acid $$$ Collect 24-hour urine.</td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Methemoglobin</strong>, whole blood (MetHb)</td>
<td>Methemoglobin has its heme iron in the oxidized ferric state and thus cannot combine with and transport oxygen. Methemoglobin can be assayed spectrophotometrically by measuring the decrease in absorbance at 630–635 nm due to the conversion of methemoglobin to cyanmethemoglobin with cyanide.</td>
<td><strong>Increased in:</strong> Hemoglobin variants (HbM) (rare), methemoglobin reductase deficiency. Oxidant drugs such as sulfonamides (dapsone, sulfasalazine), nitrites and nitrates, aniline dyes, phenacetin, anesthetics such as benzocaine.</td>
<td>Levels of 1.5 g/dL (10% of total Hb) result in visible cyanosis. Patients with levels of about 35% have headache, weakness, and breathlessness. Levels in excess of 70% are usually fatal. Fetal methemoglobin is accurately measured using newer multiple-wavelength spectrophotometers. Am J Med Sci 1985;289:200. Am J Hematol 1993;42:7. Clin Chem 1998;44:1569.</td>
</tr>
<tr>
<td>&lt;0.005 g/dL [&lt;0.5 g/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lavender $$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Analyze promptly.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Methylmalonic acid</strong>, serum</td>
<td>Elevation of serum methylmalonic acid in cobalamin deficiency results from impaired conversion of methylmalonyl-CoA to succinyl-CoA, a pathway involving methylmalonyl-CoA mutase as enzyme and adenosylcobalamin as coenzyme.</td>
<td><strong>Increased in:</strong> Vitamin $B_{12}$ (cobalamin) deficiency (95%), pernicious anemia, renal insufficiency, elderly (5–15%).</td>
<td>Explanation of high frequency (5–15%) of increased serum methylmalonic acid in the elderly with low or normal serum cobalamin is unclear. Only a small number have pernicious anemia confirmed. Normal levels can exclude vitamin $B_{12}$ deficiency in the presence of low unexplained cobalamin levels found in lymphoid disorders. Test is usually normal in HIV patients who may have low vitamin $B_{12}$ levels without cobalamin deficiency, because of low vitamin $B_{12}$ binding protein. Semin Hematol 1999;36:29. Semin Hematol 1999;36:35. Am J Clin Nutr 1997;66:741.</td>
</tr>
<tr>
<td>0–0.4 µmol/L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Metyrapone test (overnight), plasma or serum</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 AM cortisol: &lt; 10 µg/dL [ &lt; 280 nmol/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8 AM 11-deoxycortisol: &gt; 7 µg/dL [ &gt; 202 nmol/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Marbled, lavender, or green $$$

Give 2.0–2.5 g of metyrapone orally at 12:00 midnight. Draw serum cortisol and 11-deoxycortisol levels at 8:00 AM.

The metyrapone stimulation test assesses both pituitary and adrenal reserve and is mainly used to diagnose secondary adrenal insufficiency (see Adrenocortical Insufficiency algorithm, p 338).

Metyrapone is a drug that inhibits adrenal 11 β-hydroxylase and blocks cortisol synthesis. The consequent fall in cortisol increases release of ACTH and hence production of steroids formed proximal to the block (eg, 11-deoxycortisol).

**Decreased in:** An 8 AM 11-deoxycortisol level ≤ 7 µg/dL indicates primary or secondary adrenal insufficiency.

The metyrapone test can be useful in steroid-treated patients to assess the extent of suppression of the pituitary-adrenal axis.

The use of an extended metyrapone test in the differential diagnosis of ACTH-dependent Cushing’s syndrome (pituitary versus ectopic) has been questioned.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>β₂-Microglobulin,</td>
<td>β₂-Microglobulin is a portion of the HLA molecule on cell surfaces synthesized by all nucleated cell types and is present in all body fluids. It is increased in many conditions that are accompanied by high cell turnover.</td>
<td><strong>Increased in:</strong> Any type of inflammation, autoimmune disorders, lymphoid malignancies, multiple myeloma, viral infections (HIV, CMV). Marked elevation in patients with amyloidosis and renal failure.</td>
<td>Of tests used to predict progression to AIDS in HIV-infected patients, CD4 cell number has the most predictive power, followed closely by β₂-microglobulin. Asymptomatic HIV patients with elevated β₂-microglobulin levels have a two- to threefold increased chance of disease progression. However, β₂-microglobulin does not provide information significantly more useful than the combination of serial CD4 count and serum IgA in predicting onset of AIDS. β₂-Microglobulin is of prognostic value in multiple myeloma: serum level increases with increasing tumor mass. AIDS 1994;8:911. Semin Hematol 1997;34(1 Suppl):29. J Am Soc Nephrol 1998;9:1723.</td>
</tr>
<tr>
<td>serum (β₂-M)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;0.2 mg/dL [&lt;2.0 mg/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mitochondrial antibody, serum</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Mitochondrial antibody, serum</strong></td>
<td>The MHA-TP test measures specific antibody against <em>T. pallidum</em> in a patient’s serum by agglutination of <em>T. pallidum</em> antigen-coated erythrocytes. Antibodies to nonpathogenic treponemes are first removed by binding to nonpathogenic treponemal antigens.</td>
<td><strong>Increased in:</strong> Syphilis: primary (64–87%), secondary (96–100%), late latent (96–100%), tertiary (94–100%); infectious mononucleosis, collagen-vascular diseases, hyperglobulinemia and dysglobulinemia.</td>
<td>Test is used to confirm reactive serologic tests for syphilis (RPR or VDRL). Compared to FTA-ABS, MHA-TP is slightly less sensitive in all stages of syphilis and becomes reactive somewhat later in the disease. Because test usually remains positive for long periods of time regardless of therapy, it is not useful in assessing the effectiveness of therapy. In one study, 36 months after treatment of syphilis, 13% of patients had non-reactive MHA-TP tests. Ann Intern Med 1986;104:368. J Infect Dis 1990;162:862. Ann Intern Med 1991;114:1005. Clin Microbiol Rev 1995;8:1.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td>Neutrophil cytoplasmic antibodies, serum (ANCA)</td>
<td>Measurement of autoantibodies in serum against cytoplasmic constituents of neutrophils. (See also Autoantibodies table, p 367.)</td>
<td><strong>Positive in:</strong> Wegener’s granulomatosis, systemic vasculitis, crescentic glomerulonephritis, paraneoplastic vasculitis, ulcerative colitis.</td>
<td>Test sensitivity for Wegener’s granulomatosis ranges from 56% to 96%, depending on the population studied. Test specificity for Wegener’s granulomatosis is claimed to be high (99%) when requiring diffuse cytoplasmic staining for a positive result, but interpretation is highly technique-dependent. In the patient with systemic vasculitis, elevated ANCA levels imply active disease and high likelihood of recurrence. However, ANCA levels can be persistently elevated and should be used in conjunction with other clinical indices in treatment decisions. N Engl J Med 1988;318:1651. Ann Intern Med 1989;111:28. Am J Kidney Dis 1995;25:380. Ann Intern Med 1995;123:925.</td>
</tr>
<tr>
<td>Negative Marbled $$$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


<table>
<thead>
<tr>
<th><strong>Nuclear antibody</strong>, serum (ANA)</th>
<th>Heterogeneous antibodies to nuclear antigens (DNA and RNA, histone and nonhistone proteins). Nuclear antibody is measured in serum by layering the patient’s serum over human epithelial cells and detecting the antibody with fluorescein-conjugated polyvalent antihuman immunoglobulin.</th>
<th><strong>Elevated in:</strong> Patients over age 65 (35–75%, usually in low titers), systemic lupus erythematosus (98%), drug-induced lupus (100%), Sjögren’s syndrome (80%), rheumatoid arthritis (30–50%), scleroderma (60%), mixed connective tissue disease (100%), Felty’s syndrome, mononucleosis, hepatic or biliary cirrhosis, hepatitis, leukemia, myasthenia gravis, dermatomyositis, polymyositis, chronic renal failure.</th>
<th>A negative ANA test does not completely rule out SLE, but alternative diagnoses should be considered. Pattern of ANA staining may give some clues to diagnoses, but since the pattern also changes with serum dilution, it is not routinely reported. Only the rim (peripheral) pattern is highly specific (for SLE). Not useful as a screening test. Should be used only when there is clinical evidence of a connective tissue disease. West J Med 1987;147:210. Arch Intern Med 1996;156:1421. Clin Chem 1997;43:1981. Test is indicated only when multiple sclerosis is suspected clinically. Test interpretation is very subjective. IgG index is a more reliable test analytically, but neither test is specific for multiple sclerosis. Neurology 1985;35:212. Mayo Clin Proc 1989;64:577. Am J Clin Pathol 1998;109:585.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marbled</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Oligoclonal bands</strong>, serum and CSF</th>
<th>Electrophoretic examination of IgG found in CSF may show oligoclonal bands not found in serum. This suggests local production in CSF of limited species of IgG.</th>
<th><strong>Positive in:</strong> Multiple sclerosis (88%), CNS syphilis, subacute sclerosing panencephalitis, other CNS inflammatory diseases.</th>
<th>Test is indicated only when multiple sclerosis is suspected clinically. Test interpretation is very subjective. IgG index is a more reliable test analytically, but neither test is specific for multiple sclerosis. Neurology 1985;35:212. Mayo Clin Proc 1989;64:577. Am J Clin Pathol 1998;109:585.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled and glass or plastic tube for CSF</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Collect serum and CSF simultaneously.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Osmolality</strong>, serum (Osm)</td>
<td>Test measures the osmotic pressure of serum by the freezing point depression method. Plasma and urine osmolality are more useful indicators of degree of hydration than BUN, hematocrit, or serum proteins. Serum osmolality can be estimated by the following formula: ( \text{Osm} = 2\left(\text{Na}^+\right) + \frac{\text{BUN}}{2.8} + \frac{\text{Glucose}}{18} ) where ( \text{Na}^+ ) is in meq/L and BUN and glucose are in mg/dL.</td>
<td><strong>Increased in:</strong> Diabetic ketoacidosis, nonketotic hyperosmolar hyperglycemic coma, hypernatremia secondary to dehydration (diarrhea, severe burns, vomiting, fever, hyperventilation, inadequate water intake, central or nephrogenic diabetes insipidus, or osmotic diuresis), hypernatremia with normal hydration (hypothalamic disorders, defective osmostat), hypernatremia with overhydration (iatrogenic or accidental excessive NaCl or NaHCO(_3) intake), alcohol or other toxic ingestion (see Comments), hypercalcemia; tube feedings. Drugs: corticosteroids, mannitol, glycerin.</td>
<td>If the difference between calculated and measured serum osmolality is greater than 10 mosm/kg H(_2)O, suspect the presence of a low-molecular-weight toxin (alcohol, methanol, isoprophyl alcohol, ethylene glycol, acetone, ethyl ether, paraldehyde, or mannitol), ethanol being the most common. (See p 381 for further explanation.) Every 100 mg/dL of ethanol increases serum osmolality by 22 mosm/kg H(_2)O. While the osmolal gap may overestimate the blood alcohol level, a normal serum osmolality excludes ethanol intoxication. Clin Chem 1990;36:2004. J Emerg Med 1992;10:129. Pharmacotherapy 1993;13:60. Clin Chem 1998;44:1582.</td>
</tr>
<tr>
<td><strong>Panic:</strong> &lt;240 or &gt;320 mosm/kg H(_2)O</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Marbled $$</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

285–293 mosm/kg H\(_2\)O [mmol/kg H\(_2\)O]
<table>
<thead>
<tr>
<th>Common Laboratory Tests: Selection and Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Osmolality, urine</strong> (Urine Osm)</td>
</tr>
<tr>
<td>Random: 100–900 mosm/kg H₂O [mmol/kg H₂O]</td>
</tr>
<tr>
<td>Urine container $$</td>
</tr>
<tr>
<td>Test measures renal tubular concentrating ability.</td>
</tr>
<tr>
<td><strong>Increased in:</strong> Hypovolemia. Drugs: anesthetic agents (during surgery), carbamazepine, chlorpropamide, cyclophosphamide, metolazone, vincristine.</td>
</tr>
<tr>
<td><strong>Decreased in:</strong> Diabetes insipidus, primary polydipsia, exercise, starvation. Drugs: acetohexamide, demeclocycline, glyburide, lithium, tolazamide.</td>
</tr>
<tr>
<td>With average fluid intake, normal random urine osmolality is 100–900 mosm/kg H₂O.</td>
</tr>
<tr>
<td>After 12-hour fluid restriction, normal random urine osmolality is &gt;850 mosm/kg H₂O.</td>
</tr>
<tr>
<td><strong>Oxygen, partial pressure, whole blood</strong> (Po₂)</td>
</tr>
<tr>
<td>83–108 mm Hg [11.04–14.36 kPa]</td>
</tr>
<tr>
<td>Heparinized syringe $$$</td>
</tr>
<tr>
<td>Collect arterial blood in a heparinized syringe.</td>
</tr>
<tr>
<td>Send to laboratory immediately on ice.</td>
</tr>
<tr>
<td>Test measures the partial pressure of oxygen (oxygen tension) in arterial blood. Partial pressure of oxygen is critical since it determines (along with hemoglobin and blood supply) tissue oxygen supply.</td>
</tr>
<tr>
<td><strong>Increased in:</strong> Oxygen therapy.</td>
</tr>
<tr>
<td><strong>Decreased in:</strong> Ventilation/perfusion mismatching (asthma, COPD, atelectasis, pulmonary embolism, pneumonia, interstitial lung disease, airway obstruction by foreign body, shock); alveolar hypoventilation (kyphoscoliosis, neuromuscular disease, head injury, stroke); right-to-left shunt (congenital heart disease). Drugs: barbiturates, opioids.</td>
</tr>
<tr>
<td>% saturation of hemoglobin (So₂) represents the oxygen content divided by the oxygen carrying capacity of hemoglobin. % saturation on blood gas reports is calculated not measured. It is calculated from Po₂ and pH using reference oxyhemoglobin dissociation curves for normal adult hemoglobin (lacking methemoglobin, carboxyhemoglobin, etc). At Po₂ &lt;60 mm Hg, the oxygen saturation (and content) cannot be reliably estimated from the Po₂. Therefore, oximetry should be used to determine % saturation directly. JAMA 1990;264:244. Am J Clin Pathol 1995;(1 Suppl):579. Obstet Gynecol Surv 1998;53:645.</td>
</tr>
</tbody>
</table>

---

---
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Parathyroid hormone</strong>, serum (PTH)</td>
<td>PTH is secreted from the parathyroid glands. It mobilizes calcium from bone, increases distal renal tubular reabsorption of calcium, decreases proximal renal tubular reabsorption of phosphorus, and stimulates 1,25-hydroxy vitamin D synthesis from 25-hydroxy vitamin D by renal 1α-hydroxylase. The “intact” PTH molecule (84 amino acids) has a circulating half-life of about 5 minutes. Carboxyl terminal and mid-molecule fragments make up 90% of circulating PTH. They are biologically inactive, cleared by the kidney, and have half-lives of about 1–2 hours. The amino terminal fragment is biologically active and has a half-life of 1–2 minutes. Measurement of PTH by immunoassay depends on the specificity of the antibodies used.</td>
<td><strong>Increased in:</strong> Primary hyperparathyroidism, secondary hyperparathyroidism due to renal disease, vitamin D deficiency. Drugs: lithium, furosemide, phosphates. <strong>Decreased in:</strong> Hypoparathyroidism, sarcoidosis, hyperthyroidism, hypomagnesemia, malignancy with hypercalcemia, non-parathyroid hypercalcemia.</td>
<td>PTH results must always be evaluated in light of concurrent serum calcium levels. PTH tests differ in sensitivity and specificity from assay to assay and from laboratory to laboratory. Carboxyl terminal antibody measures intact, carboxyl terminal and mid-molecule fragments. It is 85% sensitive and 95% specific for primary hyperparathyroidism. Amino terminal antibody measures intact and amino terminal fragments. It is about 75% sensitive for hyperparathyroidism. Intact PTH assays are preferred because they detect PTH suppression in nonparathyroid hypercalcemia. Sensitivity of immunometric assays is 85–90% for primary hyperparathyroidism. Endocrinol Metab Clin North Am 1989;18:647. Mayo Clin Proc 1992;67:637. Recent Prog Horm Res 1998;53:283.</td>
</tr>
<tr>
<td>Intact PTH: 11–54 pg/mL [1.2–5.7 pmol/L] (laboratory-specific)</td>
<td>Fasting sample preferred; simultaneous measurement of serum calcium and phosphorus is also required.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Parathyroid hormone-related protein (PTHrP), plasma

Assay-specific (pmol/L or undetectable)

Tube containing anticoagulant and protease inhibitors; specimen drawn without a tourniquet.

Parathyroid hormone-related protein (PTHrP) is a 139- to 173-amino-acid protein with amino terminal homology to parathyroid hormone (PTH). The homology explains the ability of PTHrP to bind to the PTH receptor and have PTH-like effects on bone and kidney. PTHrP induces increased plasma calcium, decreased plasma phosphorus, and increased urinary cAMP.

PTHrP is found in keratinocytes, fibroblasts, placenta, brain, pituitary gland, adrenal gland, stomach, liver, testicular Leydig cells, and mammary glands. Its physiologic role in these diverse sites is unknown.

PTHrP is secreted by solid malignant tumors (lung, breast, kidney; other squamous tumors) and produces humoral hypercalcemia of malignancy.

PTHrP analysis is by immunoradiometric assay (IRMA). Assay of choice is amino terminal-specific IRMA. Two-site IRMA assays require sample collection in protease inhibitors because serum proteases destroy immunoreactivity.

Increased in: Humoral hypercalcemia of malignancy (80% of solid tumors).

Assays directed at the amino terminal portion of PTHrP are not influenced by renal failure.

Increases in PTHrP concentrations are readily detectable with most current assays in the majority of patients with humoral hypercalcemia of malignancy. About 20% of patients with malignancy and hypercalcemia will have low PTHrP levels because their hypercalcemia is caused by local osteolytic processes.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Partial thromboplastin time, activated, plasma (PTT)</td>
<td>Patient’s plasma is activated to clot in vitro by mixing it with phospholipid and an activator substance. Test screens the intrinsic coagulation pathway and adequacy of all coagulation factors except XIII and VII. PTT is usually abnormal if any factor level drops below 30–40% of normal.</td>
<td><strong>Increased in:</strong> Deficiency of any individual coagulation factor except XIII and VII; presence of nonspecific inhibitors (eg, lupus anticoagulant), specific factor inhibitors, von Willebrand’s disease (PTT may also be normal), hemophilia A and B, disseminated intravascular coagulation (DIC). Drugs: heparin, warfarin. <strong>Decreased in:</strong> Hypercoagulable states, DIC.</td>
<td>PTT is the best test to monitor adequacy of heparin therapy, but it does not reliably predict the risk of bleeding. Test is not always abnormal in von Willebrand’s disease. Test may be normal in chronic DIC. A very common cause of PTT prolongation is the spurious presence of heparin in the plasma sample. Sensitivity and degree of prolongation of PTT depend on particular reagents used. Therapeutic levels of heparin are best achieved using a weight-based dosing nomogram with dose adjustment based on the PTT at 6 hours. JAMA 1989;262:2428. Ann Intern Med 1993;119:874. Thromb Haemost 1995;73:73.</td>
</tr>
<tr>
<td>25–35 seconds (range varies)</td>
<td>Panic: ≥60 seconds (off heparin) Blue $$ Fill tube adequately. Do not contaminate specimen with heparin.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**pH, whole blood**

- **Arterial:** 7.35–7.45
- **Venous:** 7.31–7.41

**Heparinized syringe**

Specimen must be collected in heparinized syringe and immediately transported on ice to lab without exposure to air.

**pH** assesses the acid-base status of blood, an extremely useful measure of integrated cardiorespiratory function.

The essential relationship between pH, P\textsubscript{CO\textsubscript{2}} and bicarbonate (HCO\textsubscript{3}–) is expressed by the Henderson–Hasselbalch equation (at 37 °C):

$$\text{pH} = 6.1 + \log \left( \frac{\text{HCO}_3^-}{\text{P}_\text{CO}_2 \times 0.03} \right)$$

Arteriovenous pH difference is 0.01–0.03 but is greater in patients with congestive heart failure and shock.

**Increased in:** *Respiratory alkalosis:* hyperventilation (eg, anxiety), sepsis, liver disease, fever, early salicylate poisoning, and excessive artificial ventilation.

*Metabolic alkalosis:* Loss of gastric HCl (eg, vomiting), potassium depletion, excessive alkali administration (eg, bicarbonate, antacids), diuretics, volume depletion.

**Decreased in:** *Respiratory acidosis:* decreased alveolar ventilation (eg, COPD, respiratory depressants), neuromuscular diseases (eg, myasthenia).

*Metabolic acidosis* (bicarbonate deficit): increased formation of acids (eg, ketosis [diabetes mellitus, alcohol, starvation], lactic acidosis); decreased H\textsuperscript{+} excretion (eg, renal failure, renal tubular acidosis, Fanconi’s syndrome); increased acid intake (eg, ion-exchange resins, salicylates, ammonium chloride, ethylene glycol, methanol); and increased loss of alkaline body fluids (eg, diarrhea, fistulas, aspiration of gastrointestinal contents, biliary drainage).

The pH of a standing sample decreases because of cellular metabolism. The correction of pH (measured at 37°C), based on the patient’s temperature, is not clinically useful.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.5–4.5 mg/dL [0.8–1.45 mmol/L]</td>
<td>Panic: &lt;1.0 mg/dL</td>
<td>Marbled</td>
<td>Avoid hemolysis.</td>
</tr>
</tbody>
</table>


Platelet aggregation, whole blood
Aggregation by adenosine diphosphate (ADP), collagen, epinephrine, thrombin, ristocetin, and arachidonic acid

Drawn by lab
Whole blood in citrate is drawn into a plastic tube. Platelet-rich plasma (PRP) is obtained by centrifuging at 100 × g for 10–15 minutes.

Platelet aggregometry can provide information concerning possible qualitative platelet defects. Aggregation is measured as an increase in light transmission through stirred platelet-rich plasma (PRP) when a specific agonist is added. Test examines platelet aggregation response to various agonists (eg, ADP, collagen, epinephrine, thrombin, ristocetin, arachidonic acid).

Newer lumiaggregation measures aggregation and simultaneous platelet ATP release—the so-called “platelet release reaction.”

Abnormal in: Acquired defects in the platelet release reaction (eg, drugs, following cardiopulmonary bypass, uremia, paraproteinemias, myeloproliferative disorders), congenital release abnormalities, Glanzmann’s thrombasthenia (absent aggregation to ADP, collagen, epinephrine), essential athrombia (similar to Glanzmann’s disease except clot retraction is normal), storage pool disease (no secondary wave with ADP, epinephrine, and decreased aggregation with collagen), cyclooxygenase and thromboxane synthetase deficiencies (rare hereditary aspirin-like defects), von Willebrand’s disease (normal aggregation with all factors except ristocetin). Drugs: aspirin (absent aggregation curves to ADP and epinephrine, collagen, arachidonate).

Acquired platelet dysfunction is more common than the hereditary form.
Hereditary storage pool disease is common enough to be suspected in a child with easy or spontaneous bruising.
Test should not be done if the patient has taken aspirin within the previous 10 days.
Direct PRP aggregation by ristocetin (1.5 mg/mL) may be normal or abnormal in von Willebrand’s disease (vWD). Because this test has limited sensitivity for detection of vWD, it is no longer used for that purpose (see instead Bleeding time, p 59, and von Willebrand factor protein, p 184).
Thromb Haemost 1998;79:211.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Platelet-associated IgG, whole blood</strong></td>
<td>Antibody screening involves direct testing of a patient’s platelets to demonstrate platelet-associated IgG (which may be directed against specific platelet antigens or may represent immune complexes nonspecifically absorbed to the platelet surface) in idiopathic (autoimmune) thrombocytopenic purpura (ITP). It also involves indirect testing of the patient’s serum against a panel of reagent platelets to detect circulating antiplatelet antibodies. In allo-immune thrombocytopenia, the patient’s direct test is negative and the patient’s serum reacts with reagent platelets. Antibody specificity can be identified, and platelets lacking the involved antigen can be transfused.</td>
<td><strong>Positive in:</strong> Some autoimmune thrombocytopenias (eg, ITP) (90–95%).</td>
<td>In ITP, the direct antiplatelet antibody test may be useful to confirm the diagnosis and monitor subsequent response to therapy. It is also useful in diagnosing posttransfusion purpura and suspected neonatal isoimmune thrombocytopenia. Platelet-associated IgG is also useful for patients with thrombocytopenia or as part of a platelet cross-match prior to transfusion of patients who have repeatedly failed to respond to random donor platelet transfusions. N Engl J Med 1991;324:27. Br J Haematol 1997;96:204.</td>
</tr>
<tr>
<td>Negative</td>
<td>Yellow</td>
<td>$$$$$ 17 mL of blood is needed.</td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td>Porphobilinogen, urine (PBG)</td>
<td>Negative</td>
<td><strong>Positive in:</strong> Acute intermittent porphyria, variegate porphyria, coproporphyria, lead poisoning (rare). <strong>Negative in:</strong> 20–30% of patients with hepatic porphyria between attacks.</td>
<td>Positive qualitative urinary PBG tests should be followed up by quantitative measurements. Many labs report frequent false positives with the Watson-Schwartz test. A screening PBG test is insensitive, and a negative test does not rule out porphyria between attacks or the carrier state. Specific porphyrias can be better defined by quantitative measurement of urine PBG and by measurement of erythrocyte uroporphyrinogen-1-synthetase. Mayo Clin Proc 1994;69:289. J Inherit Metab Dis 1997;20:237. Semin Liver Dis 1998;18:57.</td>
</tr>
<tr>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protect from light.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Porphyrias are characterized clinically by neurologic and cutaneous manifestations and chemically by overproduction of porphyrin and other precursors of heme production. PBG is a water-soluble precursor of heme whose urinary excretion is increased in symptomatic hepatic porphyrias. PBG is detected qualitatively by a color reaction with Ehrlich’s reagent and confirmed by extraction into chloroform (Watson-Schwartz test).
<table>
<thead>
<tr>
<th>Potassium, serum (K⁺)</th>
<th>Potassium is predominantly an intracellular cation whose plasma level is regulated by renal excretion. Plasma potassium concentration determines neuromuscular irritability. Elevated or depressed potassium concentrations interfere with muscle contraction.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Panic:</strong> &lt;3.0 or &gt;6.0 meq/L</td>
<td><strong>Increased in:</strong> Massive hemolysis, severe tissue damage, rhabdomyolysis, acidosis, dehydration, acute or chronic renal failure, Addison’s disease, renal tubular acidosis type IV (hyporeninemic hypaldosteronism), hyperkalemic familial periodic paralysis, exercise (transient). Drugs: potassium salts, potassium-sparing diuretics (eg, spironolactone, triamterene), non-steroidal anti-inflammatory drugs, beta-blockers, ACE inhibitors, high-dose trimethoprim-sulfamethoxazole. <strong>Decreased in:</strong> Low potassium intake, prolonged vomiting or diarrhea, renal tubular acidosis types I and II, hyperaldosteronism, Cushing’s syndrome, osmotic diuresis (eg, hyperglycemia), alkalosis, familial periodic paralysis, trauma (transient). Drugs: adrenergic agents (isoproterenol), diuretics.</td>
</tr>
<tr>
<td>Marbled $</td>
<td>Spurious hyperkalemia can occur with hemolysis of sample, delayed separation of serum from erythrocytes, prolonged fist clenching during blood drawing, and prolonged tourniquet placement. Very high white blood cell or platelet counts may cause spurious elevation of serum potassium, but plasma potassium levels are normal.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
</tr>
<tr>
<td>-----------------------</td>
<td>------------------</td>
</tr>
<tr>
<td><strong>Prolactin</strong>, serum (PRL)</td>
<td>Prolactin is a polypeptide hormone secreted by the anterior pituitary. It functions in the initiation and maintenance of lactation in the postpartum period. PRL secretion is inhibited by hypothalamic secretion of dopamine. Prolactin levels increase with renal failure, hypothyroidism, and drugs that are dopamine antagonists.</td>
</tr>
</tbody>
</table>

<p>| <strong>Prostate-specific antigen</strong>, serum (PSA) | Prostate-specific antigen is a glycoprotein produced by cells of the prostatic ductal epithelium and is present in the serum of all men. It is absent from the serum of women. | <strong>Increased in:</strong> Prostate carcinoma, benign prostatic hypertrophy (BPH), following prostate examination. <strong>Negative in:</strong> Metastatic prostate carcinoma treated with antiandrogen therapy, postprostatectomy. | PSA is used to monitor recurrence of treated prostate cancer. Decrease in mortality rates resulting from use for cancer screening is unproved, and the risks of early therapy are significant. PSA is often increased in BPH, and the predictive value of a positive test in healthy older men is low. PSA replaces the acid phosphatase test. Hematol Oncol Clin North Am 1996;10:346. Urology 1998;51:789. JAMA 1999;281:1591. |</p>
<table>
<thead>
<tr>
<th>Protein C, plasma</th>
</tr>
</thead>
<tbody>
<tr>
<td>71–176%</td>
</tr>
<tr>
<td>Blue $$$</td>
</tr>
</tbody>
</table>

Protein C is a vitamin K-dependent proenzyme synthesized in the liver. Following its activation by thrombin, it exerts an anticoagulant effect through inactivation of factors Va and VIIIa using protein S as cofactor. Tests to assay quantitative (antigenic) or functional activity are available. Deficiency is inherited in an autosomal dominant fashion with incomplete penetrance or is acquired. Deficient patients may present with a hypercoagulable state, with recurrent thrombophlebitis or pulmonary emboli.

**Decreased in:** Congenital deficiency, liver disease, cirrhosis (13–25%), warfarin use (28–60%), vitamin K deficiency, disseminated intravascular coagulation (DIC).

Homozygous deficiency of protein C (<1% activity) is associated with fatal neonatal purpura fulminans and massive venous thrombosis. Heterozygous patients (one in 200–300 of the population, with levels 25–50% of normal) may be at risk for venous thrombosis. Interpretation of an abnormally low protein C must be tempered by the clinical setting. Anticoagulant therapy, DIC, and liver disease must not be present. There is overlap between lower limits of normal values and values found in heterozygotes. Kindred with dysfunctional protein C of normal quantity have been identified.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Protein electrophoresis, serum</strong></td>
<td>Electrophoresis of serum will separate serum proteins into albumin, $\alpha_1$, $\alpha_2$, $\beta_2$, and $\gamma$ fractions. Albumin is the principal serum protein (see Albumin, p 47). The term “globulin” generally refers to the non-albumin fraction of serum protein. The $\alpha_1$ fraction contains $\alpha_1$-antiprotease (90%), $\alpha_1$-lipoprotein and $\alpha_1$-acid glycoprotein. The $\alpha_2$ fraction contains $\alpha_2$-macroglobulin, haptoglobin, and ceruloplasmin. The $\beta$ fraction contains transferrin, hemopexin, complement C3, and $\beta$-lipoproteins. The $\gamma$ fraction contains immunoglobulins G, A, D, E, and M.</td>
<td>↑ $\alpha_1$: inflammatory states ($\alpha_1$-antiprotease), pregnancy. ↑ $\alpha_2$: nephrotic syndrome, inflammatory states, oral contraceptives, steroid therapy, hyperthyroidism. ↑ $\beta$: hyperlipidemia, hemoglobinemia, iron deficiency anemia. ↑ $\gamma$ polyclonal gammopathies (liver disease, cirrhosis [associated with $\beta-\gamma$ “bridging”], chronic infections, autoimmune disease); monoclonal gammopathies (multiple myeloma, Waldenström’s macroglobulinemia, lymphoid malignancies, monoclonal gammopathy of undetermined significance). ↓ $\alpha_1$: $\alpha_1$-antiprotease deficiency. ↓ $\alpha_2$: in vivo hemolysis, liver disease. ↓ $\gamma$: hypo-$\beta$-lipoproteinemia. ↓ $\gamma$: immune deficiency.</td>
<td>Presence of “spikes” in $\alpha_2$, $\beta_2$, or $\gamma$ regions necessitates the use of immunoelectrophoresis to verify the presence of a monoclonal gammopathy (see Immunoelectrophoresis, p 112). If Bence Jones proteins (light chains) are suspected, urine protein electrophoresis needs to be done. Test is insensitive for detection of decreased levels of immunoglobulins and $\alpha_1$-antiprotease. Specific quantitation is required (see Immunoglobulins, p 113 and $\alpha_1$-Antiprotease, p 55). If plasma is used, fibrinogen will be detected in the $\beta-\gamma$ region. The “acute-phase protein pattern” seen with acute illness, surgery, infarction or trauma is characterized by an ↑$\alpha_2$ (haptoglobin) and ↑$\alpha_1$ ($\alpha_1$-antiprotease). Arch Pathol Lab Med 1999;123:114.</td>
</tr>
<tr>
<td>Adults: Albumin:</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3.3–5.7 g/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha_1$: 0.1–0.4 g/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\alpha_2$: 0.3–0.9 g/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\beta_2$: 0.7–1.5 g/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\gamma$: 0.5–1.4 g/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\text{Marbled} \quad $$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Protein, total, plasma or serum</strong></td>
<td>Plasma protein concentration is determined by nutritional state, hepatic function, renal function, hydration, and various disease states. Plasma protein concentration determines the colloidal osmotic pressure.</td>
<td><strong>Increased in:</strong> Polyclonal or monoclonal gammopathies, marked dehydration. Drugs: anabolic steroids, androgens, corticosteroids, epinephrine. <strong>Decreased in:</strong> Protein-losing enteropathy, acute burns, nephrotic syndrome, severe dietary protein deficiency, chronic liver disease, malabsorption syndrome, agammaglobulinemia.</td>
<td>Serum total protein consists primarily of albumin and globulin. Serum globulin level is calculated as total protein minus albumin. Hypoproteinemia usually indicates hypoalbuminemia, since albumin is the major serum protein. Ann Thorac Surg 1999;67:236.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
</tbody>
</table>
| **Prothrombin time,** whole blood (PT) | PT screens the extrinsic pathway of the coagulation system. It is performed by adding calcium and tissue thromboplastin to a sample of citrated, platelet-poor plasma and measuring the time required for fibrin clot formation. It is most sensitive to deficiencies in the vitamin K-dependent clotting factors II, VII, IX, and X. It is also sensitive to deficiencies of factor V. It is insensitive to fibrinogen deficiency and not affected by heparin. PT is also used to monitor warfarin therapy. In liver disease, the PT reflects the hepatic capacity for protein synthesis. PT responds rapidly to altered hepatic function because the serum half-lives of factors II and VII are short (hours). | **Increased in:** Liver disease, vitamin K deficiency, intravascular coagulation, circulating anticoagulant, massive transfusion. Drugs: warfarin. | Routine preoperative measurement of PT is unnecessary unless there is clinical history of a bleeding disorder. Efforts to standardize and report the prothrombin time as an INR (International Normalized Ratio) depend on assigning reagents an International Sensitivity Index (ISI) so that: \[
\text{INR} = \left( \frac{\text{PT patient}}{\text{PT normal}} \right)^{\text{ISI}}
\]
However, assignment of incorrect ISI by reagent manufacturers has caused a greater lack of standardization. Bleeding has been reported to be three times more common in patients with INRs of 3.0–4.5 than in patients with INRs of 2.0–3.0. PT is quite insensitive to individual decreases in factors VII, IX, and X to 50% of normal but is much more sensitive to mild deficiencies in two or more factors. Thus, patients starting warfarin therapy or with liver disease may have elevated prothrombin times with no significant in vivo coagulation defects. JAMA 1989;262:2428. J Lab Clin Med 1996;128:214. J Clin Pathol 1998;51:356. |

**Blue $**

Fill tube completely.
| Q fever antibody, serum | **Increased in:** Acute or chronic Q fever (CF antibodies are present by the second week in 65% of cases and by the fourth week in 90%; acute and convalescent titers [IFA or ELISA] detect infection with 89–100% sensitivity and 100% specificity), and recent vaccination for Q fever. | Clinical presentation is similar to that of severe influenza. Typically, there is no rash. Tests are usually performed in large reference labs or public health centers. Occasionally, titers do not rise for 4–6 weeks, especially if antimicrobial therapy has been given. Patients with Q fever have a high prevalence of antiphospholipid antibody (81%), especially as measured by lupus anticoagulant test or measurement of antibodies to cardiolipin. These tests may be useful in diagnosing patients presenting with fever alone. Recent Q fever vaccination causes a rise in antibody titers similar to that seen with acute infection. Antibodies to Q fever do not cross-react with other rickettsial antibodies. | }
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rapid plasma reagin, serum (RPR)</strong></td>
<td>Measures nontreponemal antibodies that are produced when <em>Treponema pallidum</em> interacts with host tissue. The card test is a flocculation test performed by using a cardiolipin-lecithin-cholesterol carbon-containing antigen reagent mixed on a card with the patient’s serum. A positive test (presence of antibodies) is indicated when black carbon clumps produced by flocculation are seen by the naked eye.</td>
<td><strong>Increased in:</strong> Syphilis: primary (78%), secondary (97%), symptomatic late (74%). Biologic false-positives occur in a wide variety of conditions, including leprosy, malaria, intravenous drug abuse, aging, infectious mononucleosis, HIV infection (≤ 15%), autoimmune diseases (SLE, rheumatoid arthritis), pregnancy.</td>
<td>RPR is used as a screening test and in suspected primary and secondary syphilis. Since the test lacks specificity, positive tests should be confirmed with the FTA-ABS or MHA-TP test (see pp 92 and 129, respectively). RPR titers can be used to follow serologic response to treatment. (See Syphilis test table, Table 8-20, p 391.) Ann Intern Med 1991;114:1005. J Clin Microbiol 1995;33:1829. Sex Trans Dis 1998;25:569.</td>
</tr>
<tr>
<td>Nonreactive Marbled $</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Red cell volume, whole blood (RCV)**

**Male:** 24–32 mL/kg  
**Female:** 22–28 mL/kg

Yellow Lavender (for Hct) $$$

A sample of the patient’s whole blood is labeled with radioactive $^{51}$Cr (which is taken up into red cells) and reinjected into the patient. Blood is sampled 10 and 60 minutes later to measure radioactivity.

Test measures absolute volume of red cells based on hemodilution of a known quantity of radioactivity in the circulation. Test can distinguish between absolute polycythemia (increased hematocrit [Hct], increased RCV) and relative polycythemia (hemoconcentration) (increased Hct, normal RCV). Alternative techniques can be used to measure RCV without exposing the patient to radiation, including use of biotin-, $^{53}$Cr-, and sodium fluorescein-labeled red cells.

**Increased in:** Polycythemia vera, secondary polycythemia due to tissue hypoxemia (pulmonary disease, congenital heart disease, carboxyhemoglobinemia [cigarette smoking], methemoglobinemia), or neoplasms (renal cell carcinoma, hepatoma, large uterine leiomyomas), high altitude, pregnancy.

Test is clinically indicated (but not always required) in the diagnosis of polycythemia vera.  
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Renin activity, plasma (PRA)</strong></td>
<td>The renal juxtaglomerular apparatus generates renin, an enzyme that converts angiotensinogen to angiotensin I. The inactive angiotensin I is then converted to angiotensin II, which is a potent vasopressor. Renin activity is measured by the ability of patient’s plasma to generate angiotensin I from substrate (angiotensinogen). Normal values depend on the patient’s hydration, posture, and salt intake.</td>
<td><strong>Increased in:</strong> Dehydration, some hypertensive states (e.g., renal artery stenosis); edematous states (cirrhosis, nephrotic syndrome, congestive heart failure); hypokalemic states (gastrointestinal sodium and potassium loss, Bartter’s syndrome); adrenal insufficiency, chronic renal failure, left ventricular hypertrophy. Drugs: ACE inhibitors, estrogen, hydralazine, nifedipine, minoxidil, oral contraceptives. <strong>Decreased in:</strong> Hyporeninemic hypoaldosteronism, some hypertensive states (e.g., primary aldosteronism, severe preeclampsia). Drugs: beta-blockers, aspirin, clonidine, prazosin, reserpine, methyldopa, indomethacin.</td>
<td>PRA alone is not a satisfactory screening test for hyperaldosteronism because suppressed PRA has only 64% sensitivity and 83% specificity for primary hyperaldosteronism. However, when plasma aldosterone and PRA testing are combined, the sensitivity for primary hyperaldosteronism increases to 95% (see Aldosterone, plasma, p 48). Test is also useful in evaluation of hypoaldosteronism (low-sodium diet, patient standing). Measurement of peripheral vein renin activity is not useful in classification of hypertensive patients or in diagnosis of renal artery stenosis. Bilateral renal vein sampling has been used to investigate renal artery stenosis. In general, a renal vein renin (RVR) ratio of 1.5 or more (affected/nonaffected side) is predictive of response to revascularization in &gt;90% of cases, but 60% of cases with RVR ratios &lt;1.5 will also respond. Therefore, the test cannot reliably predict therapeutic response to a surgical procedure. Mayo Clin Proc 1994;69:1172. Acta Obstet Gynecol Scand 1998;77:609. J Hum Hypertens 1998;12:455. Am J Nephrol 1996;16:471.</td>
</tr>
<tr>
<td><strong>High-sodium diet</strong> (75–150 meq Na⁺/d): supine, 0.2–2.3; standing, 1.3–4.0 ng/mL/h</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Low-sodium diet</strong> (30–75 meq Na⁺/d): standing, 4.0–7.7 ng/mL/h</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lavender $$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Reptilase clotting time, plasma</strong></td>
<td>Reptilase is an enzyme derived from the venom of <em>Bothrops atrox</em> or <em>Bothrops jararaca</em>, South American pit vipers. Reptilase cleaves a fibrinopeptide from fibrinogen directly, bypassing the heparin-antithrombin system, and produces a fibrin clot. The reptilase time will be normal in heparin toxicity, even when the thrombin time is infinite.</td>
<td><strong>Increased in:</strong> Hypofibrinogenemia, dysfibrinogenemia, afibrinogenemia, and disseminated intravascular coagulation (DIC). <strong>Normal in:</strong> Presence of heparin.</td>
<td>When the thrombin time is prolonged, the reptilase time is useful in distinguishing the presence of an anti-thrombin (normal reptilase time) from hypo- or dysfibrinogenemia (prolonged reptilase time). The reptilase time is normal when heparin is the cause of a prolonged thrombin time. The reptilase time is only slightly prolonged by fibrin degradation products. Br J Haematol 1971;21:43.</td>
</tr>
<tr>
<td>Reticulocyte count, whole blood</td>
<td>Reticulocytes are immature red blood cells that contain cytoplasmic mRNA.</td>
<td><strong>Increased in:</strong> Hemolytic anemia, blood loss; recovery from iron, B₁₂, or folate deficiency or drug-induced anemia. <strong>Decreased in:</strong> Iron deficiency anemia, aplastic anemia, anemia of chronic disease, megaloblastic anemia, sideroblastic anemia, bone marrow suppression.</td>
<td>This test is indicated in the evaluation of anemia to distinguish hypoproliferative from hemolytic anemia or blood loss. The old method of measuring reticulocytes (manual staining and counting) has poor reproducibility. It has been replaced by automated methods (eg, flow cytometry), which are more precise. Method-specific reference ranges must be used. Am J Hematol 1990;33:13. Am J Clin Pathol 1994;102:623. Clin Lab Haematol 1996;18(Suppl 1):1</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Rh grouping, red cells (Rh)</td>
<td>The Rhesus blood group system is second in importance only to the ABO system. Anti-Rh antibodies are the leading cause of hemolytic disease of the newborn and may also cause hemolytic transfusion reactions. Although there are other Rhesus antigens, only tests for the D antigen are performed routinely in pretransfusion testing, since the D antigen is the most immunogenic. The terms Rh-positive and -negative refer to the presence or absence of the red cell antigen, D, on the cell surface. Persons whose red cells lack D do not regularly have anti-D in their serum. Formation of anti-D almost always results from exposure through transfusion or pregnancy to red cells possessing the D antigen.</td>
<td>Sixty percent of US whites are Rh(D)-positive, 40% negative; 72% of African-Americans are Rh(D)-positive, 28% negative; 95% of Asian-Americans are Rh(D)-positive, 5% negative.</td>
<td>Of D⁻ persons receiving a single D⁺ unit, 50–75% will develop anti-D. The blood of all donors and recipients is therefore routinely tested for D, so that D⁻ recipients can be given D⁻ blood. Donor bloods must also be tested for a weak form of D antigen, called D⁺, and must be labeled D⁺ if the D⁺ test is positive. Recipient blood need not be tested for D⁺. Technical Manual of the American Association of Blood Banks, 11th ed. American Association of Blood Banks, 1993.</td>
</tr>
<tr>
<td><strong>Rheumatoid factor</strong>, serum (RF)</td>
<td>Rheumatoid factor consists of heterogeneous autoantibodies usually of the IgM class that react against the Fc region of human IgG.</td>
<td><strong>Positive in</strong>: Rheumatoid arthritis (75–90%), Sjögren’s syndrome (80–90%), scleroderma, dermatomyositis, SLE (30%), sarcoidosis, Waldenström’s macroglobulinemia. Drugs: methyldopa, others. Low-titer RF can be found in healthy older patients (20%), in 1–4% of normal individuals, and in a variety of acute immune responses (eg, viral infections, including infectious mononucleosis and viral hepatitis), chronic bacterial infections (tuberculosis, leprosy, subacute infective endocarditis), and chronic active hepatitis. Rheumatoid factor can be useful in differentiating rheumatoid arthritis from other chronic inflammatory arthritides. However, a positive RF test is only one of several criteria needed to make the diagnosis of rheumatoid arthritis. (See also Autoantibodies table, p 367.) RF must be ordered selectively because its predictive value is low (34%) if it is used as a screening test. The test has poor positive predictive value because of its lack of specificity. The subset of patients with seronegative rheumatic disease limits its sensitivity and negative predictive value.</td>
<td><strong>Negative (&lt;1 : 16)</strong> Marbled $</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td><strong>Ribonucleoprotein antibody</strong>, serum (RNP)</td>
<td>This is an antibody to a ribonucleoprotein-extractable nuclear antigen.</td>
<td><strong>Increased in</strong>: Scleroderma (20–30% sensitivity, low specificity), mixed connective tissue disease (MCTD) (95–100% sensitivity, low specificity), SLE (30%), Sjögren’s syndrome, rheumatoid arthritis (10%), discoid lupus (20–30%).</td>
<td><strong>Negative</strong></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td>Rubella antibody, serum</td>
<td>Rubella (German measles) is a viral infection that causes fever, malaise, coryza, lymphadenopathy, fine maculopapular rash, and congenital birth defects when infection occurs in utero. Antibodies to rubella can be detected by hemagglutination inhibition (HI), complement fixation (CF), indirect hemagglutination (IHA), ELISA, or latex agglutination (LA). Tests can detect IgG and IgM antibody. Titers usually appear as rash fades (1 week) and peak at 10–14 days for HI and 2–3 weeks for other techniques. Baseline titers may remain elevated for life.</td>
<td><strong>Increased in:</strong> Recent rubella infection, congenital rubella infection, previous rubella infection or vaccination (immunity). Spuriously increased IgM antibody occurs in the presence of rheumatoid factor.</td>
<td>Rubella titers of $\leq 1 : 8$ indicate susceptibility and need for immunization to prevent infection during pregnancy. Titers of $&gt; 1 : 32$ indicate immunity from prior infection or vaccination. Definitive diagnosis is based on a fourfold rise in titer or the presence of IgM antibody. To diagnose congenital infection, submit a single specimen for IgM. If positive, submit a second specimen 2–3 months later to rule out maternal antibody transmission across the placenta. The recent resurgence of congenital rubella can largely be prevented with improved rubella testing and vaccination programs. Rev Infect Dis 1985;7(Suppl 1):S108. Am J Clin Pathol 1996;106:170. J Infect Dis 1997;175:749.</td>
</tr>
</tbody>
</table>
Russell’s viper venom clotting time (dilute), plasma (RVVT) 24–37 seconds

Blue $$

Russell viper venom is extracted from a pit viper (Vipera russelli), which is common in Southeast Asia (especially Burma) and which causes a rapidly fatal syndrome of consumptive coagulopathy with hemorrhage, shock, rhabdomyolysis, and renal failure.

Approximately 70% of the protein content of the venom is phospholipase A\(_2\), which activates factor X in the presence of phospholipid, bypassing factor VII.

RVVT is a phospholipid-dependent coagulation test used in detection of antiphospholipid antibodies (so-called lupus anticoagulants). It should be noted that the anticoagulant detected in vitro may be associated with thrombosis (and not bleeding) in vivo.

Increased in: Circulating lupus anticoagulants (LAC), severe fibrinogen deficiency (< 50 mg/dL), deficiencies in prothrombin, factor V, factor X, and heparin therapy.

Normal in: Factor VII deficiency and all intrinsic pathway factor deficiencies.

The lupus anticoagulant may be associated with a prolonged PTT and a positive inhibitor screen (mixing study). If heparin is not present, a dilute Russell viper venom test may be indicated to confirm that the inhibitor is an LAC.

Since specific factor inhibitors against factors VIII and IX are associated with clinically significant bleeding and require specific treatment, they must not be missed.

The LAC is associated with an increased risk of thrombosis (venous > arterial), recurrent spontaneous abortion, and the primary antiphospholipid syndrome of arterial thrombosis.

Haemostasis 1990;20:208.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Salicylate, serum</strong></td>
<td>At high concentrations, salicylate stimulates hyperventilation, uncouples oxidative phosphorylation, and impairs glucose and fatty acid metabolism. Salicylate toxicity is thus marked by respiratory alkalosis and metabolic acidosis.</td>
<td><strong>Increased in:</strong> Acute or chronic salicylate intoxication.</td>
<td>The potential toxicity of salicylate levels after acute ingestion can be determined by using the Salicylate nomogram, p 360. Nomograms have become less valid with the increasing popularity of enteric-coated slow-release aspirin preparations. Pediatrics 1960;26:800. Ann Pharmacother 1996;30:935. Am J Emerg Med 1996;14:443.</td>
</tr>
<tr>
<td>(aspirin)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>20–30 mg/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>[200–300 mg/L]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Panic:</strong> &gt;35 mg/dL</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(Scl-70 antibody), serum</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Semen analysis, ejaculate

Sperm count: >20 $\times$ 10⁶/mL [10⁹/L]
Motility score: >60% motile
Volume: 2–5 mL
Normal morphology: >60%

Semen is collected in a urine container after masturbation following 3 days of abstinence from ejaculation. Specimen must be examined promptly.

Smith (anti-Sm) antibody, serum

Negative
Marbled $$

This antibody to Smith antigen (an extractable nuclear antigen) is a marker antibody for SLE.

Positive in: SLE (30–40% sensitivity, high specificity).

A positive test substantially increases posttest probability of SLE. Test rarely needed for the diagnosis of SLE. (See also Autoantibodies table, p 367.)

Decreased in: Primary or secondary testicular failure, cryptorchidism, following vasectomy, drugs.

A low sperm count should be confirmed by sending two other appropriately collected semen specimens for evaluation. Functional and computer-assisted sperm analyses increase diagnostic accuracy but are not yet widely available.

J Androl 1996;17:718.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smooth muscle antibo-</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled $$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Sodium, serum (Na+)**

135—145 meq/L [mmol/L]

**Panic:** <125 or >155 meq/L

Marbled $\$

Sodium is the predominant extracellular cation. The serum sodium level is primarily determined by the volume status of the individual. Hyponatremia can be divided into hypovolemia, euvoemlacia, and hypervolemia categories. (See Hyponatremia algorithm, p 350.)

**Increased in:** Dehydration (excessive sweating, severe vomiting or diarrhea), polyuria (diabetes mellitus, diabetes insipidus), hyperaldosteronism, inadequate water intake (coma, hypotalamic disease). Drugs: steroids, licorice, oral contraceptives.

**Decreased in:** Congestive heart failure, cirrhosis, vomiting, diarrhea, excessive sweating (with replacement of water but not salt), salt-losing nephropathy, adrenal insufficiency, nephrotic syndrome, water intoxication, SIADH. Drugs: thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, antidepressants (selective serotonin reuptake inhibitors), antipsychotics.

Spurious hyponatremia may be produced by severe lipemia or hyperproteinemia if sodium analysis involves a dilution step. The serum sodium falls about 1.6 meq/L for each 100 mg/dL increase in blood glucose.

Hyponatremia in a normovolemic patient with urine osmolality higher than plasma osmolality suggests the possibility of SIADH, myxedema, hypopituitarism, or reset osmostat. Treatment of disorders of sodium balance relies on clinical assessment of the patient’s extracellular fluid volume rather than the serum sodium.

Sodium is commonly measured by ion-selective electrode.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Somatomedin C, plasma</td>
<td>Somatomedin C is a growth hormone-dependent plasma peptide produced by the liver. It is believed to mediate the growth-promoting effect of growth hormone (GH). It has an anabolic, insulin-like action on fat and muscle and stimulates collagen and protein synthesis. Its level is relatively constant throughout the day.</td>
<td><strong>Increased in:</strong> Acromegaly (level correlates with disease activity better than GH level). <strong>Decreased in:</strong> Pituitary dwarfism, hypopituitarism, Laron dwarfism (end-organ resistance to GH), fasting for 5–6 days, poor nutrition, hypothyroidism, cirrhosis. Values may be normal in growth hormone-deficient patients with hyperprolactinemia or craniopharyngioma.</td>
<td>A normal somatomedin C level in children is strong evidence that GH deficiency is not present and precludes the need for extensive pituitary function testing. A low level does not prove that GH deficiency is present, since levels may be reduced in malnutrition, malabsorption, chronic systemic illness, and hypothyroidism. Reference range here is for an immunoassay done following displacement of somatomedin C from its binding protein (acid-ethanol extraction). N Engl J Med 1979;301:1138. J Pediatr 1981;99:720. J Clin Endocrinol 1988;66:538. Endocrinol Metab Clin North Am 1992;21:649.</td>
</tr>
<tr>
<td><strong>SS-A/Ro antibody, serum</strong></td>
<td><strong>Antibodies to Ro (SSA) cellular ribonucleoprotein complexes are found in connective tissue diseases such as Sjögren’s syndrome (SS), SLE, rheumatoid arthritis (RA), and vasculitis.</strong></td>
<td><strong>Increased in:</strong> Sjögren’s (60–70% sensitivity, low specificity), SLE (30–40%), RA (10%), subacute cutaneous lupus, vasculitis.</td>
<td><strong>Useful in counseling women of childbearing age with known connective tissue disease, since a positive test is associated with a small but real risk of neonatal SLE and congenital heart block. The few (&lt;10%) patients with SLE who do not have a positive ANA commonly have antibodies to SS-A. (See also Autoantibodies table, p 367.)</strong> Medicine 1995;74:109. J Rheumatol 1996;23:1897. J Am Acad Dermatol 1996;35 (2 Part 1):147. J Autoimmun 1998;11:29. Br J Dermatol 1998;138:114. Clin Exper Rheumatol 1999;17:63,130.</td>
</tr>
<tr>
<td><strong>SS-B/La antibody, serum</strong></td>
<td><strong>Antibodies to La (SSB) cellular ribonucleoprotein complexes are found in Sjögren’s syndrome (SS) and appear to be relatively more specific for SS than are antibodies to SSA. They are quantitated by immunoassay.</strong></td>
<td><strong>Increased in:</strong> Sjögren’s (50% sensitivity, higher specificity than anti-SSA), SLE (10%).</td>
<td><strong>Direct pathogenicity and usefulness of autoantibody test in predicting disease exacerbation not proved. (See also Autoantibodies table, p 367.)</strong> Arthritis Rheum 1996;39:1055. Ann Rheum Dis 1997;156:272. J Autoimmun 1998;11:29. Clin Exper Rheumatol 1999;17:130.</td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>T cell receptor gene rearrangement</td>
<td>In general, the percentage of T lymphocytes with identical T cell receptors is very low; in malignancies, however, the clonal expansion of one population leads to a large number of cells with identical T cell receptor gene rearrangement. Southern blot is used to identify a monoclonal population.</td>
<td>Positive test results may be seen in T cell neoplasms such as T cell lymphocytic leukemia and cutaneous or nodal T cell lymphomas.</td>
<td>Samples with &gt;10% of cells showing a given T cell rearrangement are considered positive. However, a large monoclonal population is not absolutely diagnostic of malignancy. Am J Hematol 1996;52:171. Mol Pathol 1997;50:77. Arch Dermatol 1998;134:15. J Am Acad Dermatol 1998;39 (4 Part 1):554. Leukemia 1998;12:1081.</td>
</tr>
</tbody>
</table>
**Testosterone, serum**

<table>
<thead>
<tr>
<th>Males: 3.0–10.0 ng/mL</th>
<th>Females: 0.3–0.7 ng/mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marbled $$$$</td>
<td></td>
</tr>
</tbody>
</table>

- **Testosterone** is the principal male sex hormone, produced by the Leydig cells of the testes. Dehydroepiandrosterone (DHEA) is produced in the adrenal cortex, testes and ovaries and is the main precursor for serum testosterone in women. In normal males after puberty, the testosterone level is twice as high as all androgens in females. In serum, it is largely bound to albumin (38%) and to a specific steroid hormone-binding globulin (SHBG) (60%), but it is the free hormone (2%) that is physiologically active. The total testosterone level measures both bound and free testosterone in the serum (by immunoassay).

- **Increased in:** Idiopathic sexual precocity (in boys, levels may be in adult range), adrenal hyperplasia (boys), adrenocortical tumors, trophoblastic disease during pregnancy, idiopathic hirsutism, virilizing ovarian tumors, arrhenoblastoma, virilizing luteoma, testicular feminization (normal or moderately elevated), cirrhosis (through increased SHBG), hyperthyroidism.
- **Decreased in:** Hypogonadism (primary and secondary, orchidectomy, Klinefelter’s, uremia, hemodialysis, hepatic insufficiency, ethanol [men]). Drugs: digoxin, spironolactone, acarbose.
- Serum testosterone levels decrease in men after age 50.
- A free testosterone level is indicated when a normal total testosterone level is thought not to reflect free testosterone levels because of increases in SHBG.
- In men, there is a small diurnal variation in serum testosterone with a 20% elevation in levels in the evenings.

**Thrombin time, plasma**

<table>
<thead>
<tr>
<th>24–35 seconds</th>
<th>(laboratory-specific)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blue $</td>
<td></td>
</tr>
</tbody>
</table>

- **Prolongation of the thrombin time** indicates a defect in conversion of fibrinogen to fibrin.
- **Increased in:** Low fibrinogen (<50 mg/dL), abnormal fibrinogen (dysfibrinogenemia), increased fibrin degradation products (eg, disseminated intravascular coagulation), heparin, fibrinolytic agents (streptokinase, urokinase, tissue plasminogen activator), primary systemic amyloidosis (40%).
- Thrombin time can be used to monitor fibrinolytic therapy and to screen for dysfibrinogenemia or circulating anticoagulants.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Thyroglobulin, serum  | Thyroglobulin is a large protein specific to the thyroid gland from which thyroxine is synthesized and cleaved. Highly sensitive immunoradiometric assays (IRMAs) have minimal interference from autoantibodies. | **Increased in:** Hyperthyroidism, subacute thyroiditis, untreated thyroid carcinomas (except medullary carcinoma): follicular cancer (sensitivity 72%, specificity 81%), Hürthle cell cancer (sensitivity 56%, specificity 84%). **Decreased in:** Factitious hyperthyroidism, presence of thyroglobulin autoantibodies, after (>25 days) total thyroidectomy. | Thyroglobulin is useful to follow patients after treatment of non-medullary thyroid carcinomas. Levels fall after successful therapy and rise when metastases develop. Sensitivity of the test is increased if patients are off thyroid replacement for 6 weeks prior to testing or if given T₃ (Cytomel) for the first 4 weeks, then no medication for the last 2 weeks. Athyrotic patients on T₄ (levothyroxine) should have values <5 ng/mL and those off T₄ should have values <10 ng/mL. Clin Chem 1996;42:164. Clin Chem 1996;42:258. Eur J Nucl Med 1997;24:722. Eur J Surg Oncol 1998;24:553. Eur J Endocrinol 1998;138:249. |}
<p>| Thyroglobulin antibody, serum | Antibodies against thyroglobulin are produced in autoimmune diseases of the thyroid and other organs. Ten percent of the normal population have slightly elevated titers (especially women and the elderly). | <strong>Increased in:</strong> Hashimoto’s thyroiditis (&gt;90%), thyroid carcinoma (45%), thyrotoxicosis, pernicious anemia (50%), SLE (20%), subacute thyroiditis, Graves’ disease. <strong>Not Increased in:</strong> Multinodular goiter, thyroid adenomas, and some carcinomas. | The antithyroid peroxidase antibody test is more sensitive than the thyroglobulin antibody test in autoimmune thyroid disease. There is little indication for this test. (See Thyroid Peroxidase Antibody, below.) Am J Med 1983;74:941. Med Clin North Am 1991;75:1. J Clin Endocrinol Metab 1998;83:1121. |</p>
<table>
<thead>
<tr>
<th>Thyroperoxidase antibody, serum</th>
<th>Increased in: Hashimoto’s thyroiditis (&gt;99%), idiopathic myxedema (&gt;99%), Graves’ disease (75–85%), Addison’s disease (50%), and Riedel’s thyroiditis. Low titers are present in approximately 10% of normal individuals and patients with nonimmune thyroid disease.</th>
<th>Thyroperoxidase antibody is an antibody to the main autoantigenic component of microsomes and is a more sensitive and specific test than hemagglutination assays for microsomal antibodies in the diagnosis of autoimmune thyroid disease. Thyroperoxidase antibody testing alone is almost always sufficient to detect autoimmune thyroid disease.</th>
<th>J Clin Endocrinol Metab 1990;71:661. Arch Intern Med 1993;153:862. J Clin Endocrinol Metab 1996;81:2595. Thyroid 1997;7:471.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thyroperoxidase (TPO) is a membrane-bound glycoprotein. This enzyme mediates the oxidation of iodide ions and incorporation of iodine into tyrosine residues of thyroglobulin. Its synthesis is stimulated by thyroid-stimulating hormone (TSH). TPO is the major antigen involved in thyroid antibody-dependent cell-mediated cytotoxicity. Anti-thyroperoxidase antibody assays are performed by ELISA or radioimmunoassay.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td><strong>Thyroid-stimulating hormone, serum (TSH; thyrotropin)</strong> 0.4–6 µU/mL [mU/L] Marbled $$</td>
<td>TSH is an anterior pituitary hormone that stimulates the thyroid gland to produce thyroid hormones. Secretion is stimulated by thyrotropin-releasing hormone from the hypothalamus. There is negative feedback on TSH secretion by circulating thyroid hormone.</td>
<td><strong>Increased in:</strong> Hypothyroidism. Mild increases in recovery phase of acute illness. <strong>Decreased in:</strong> Hyperthyroidism, acute medical or surgical illness, pituitary hypothyroidism. Drugs: dopamine, high-dose corticosteroids.</td>
<td>Newer sensitive assays can detect low enough levels of TSH to be useful in the diagnosis of hyperthyroidism as well as hypothyroidism and in distinguishing hyperthyroidism from sub-normal TSH values occasionally found in euthyroid sick patients. (See also Thyroid function table, p 393.) Test is useful for following patients taking thyroid medication. Neonatal and cord blood levels are 2–4 times higher than adult levels. J Nucl Med 1985;26:1248. Endocrinol Metab Clin North Am 1992;21:903. Postgrad Med 1993;94:81. Clin Chem 1996;42:140. Clin Chem 1997;43:2428. J R Soc Med 1997;90:547.</td>
</tr>
<tr>
<td>Thyroid-stimulating hormone receptor antibody, serum (TSH-R [stim] Ab)</td>
<td>Test detects heterogeneous IgG antibodies directed against the TSH receptor on thyroid cells. Frequently, they cause excess release of hormone from the thyroid. Test measures antibodies indirectly by their stimulation of adenyl cyclase to produce cAMP.</td>
<td>Increased in: Graves’ disease.</td>
<td>Although TSH-R [stim] Ab is a marker of Graves’ disease, the test is not necessary for the diagnosis in most cases. Test is very rarely indicated but may be helpful in (1) pregnant women with a history of Graves’ disease, because TSH-R [stim] Ab may have some predictive value for neonatal thyrotoxicosis; (2) patients presenting with exophthalmos who are euthyroid, to confirm Graves’ disease. Use of the test to predict relapse of hyperthyroidism at the end of a course of antithyroid drugs is controversial. J Clin Endocrinol Metab 1989;69:1093.</td>
</tr>
</tbody>
</table>

| Thyroxine, total, serum (T₄) | 5.0–11.0 µg/dL [64–142 nmol/L] | | |
| | Marbled $ | | |

Marbled $$$$
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Thyroxine, free, serum (FT₄)</strong></td>
<td>FT₄ (if done by equilibrium dialysis or ultrafiltration method) is a more direct measure of the free T₄ hormone concentration (biologically available hormone) than the free T₄ index. FT₄ done by a two-step immunoassay is similar to the free thyroxine index. The presence of rheumatoid factor or drug treatment with furosemide, intravenous heparin, and subcutaneous low-molecular-weight heparin may interfere with newer assays for free thyroxine.</td>
<td><strong>Increased in:</strong> Hyperthyroidism, non-thyroidal illness, especially psychiatric. Drugs: amiodarone, beta-blockers (high dose). <strong>Decreased in:</strong> Hypothyroidism, non-thyroidal illness. Drugs: phenytoin.</td>
<td>FT₄ is functionally equivalent to the FT₄I (see below). The free thyroxine and sensitive TSH assays have similar sensitivities for detecting clinical hyperthyroidism and hypothyroidism. The TSH assay detects subclinical dysfunction and monitors thyroxine treatment better; the free thyroxine test detects central hypothyroidism and monitors rapidly changing function better. JAMA 1990;263:1529. Arch Intern Med 1996;156:2333. Clin Chem 1996;42:146. Arch Intern Med 1998;158:266.</td>
</tr>
<tr>
<td><strong>Thyroxine index, free, serum (FT₄I)</strong></td>
<td>Free thyroxine index is expressed as total T₄ × T₃ (or T₄) resin uptake and provides an estimate of the level of free T₄, since the T₃ (or T₄) resin uptake (ie, thyroid hormone binding ratio) is an indirect estimate of the thyroid binding globulin (TBG) concentration. (TBG binds 70% of circulating thyroid hormone.) The unbound form of circulating T₄, normally 0.03% of total serum T₄, determines the amount of T₄ available to cells.</td>
<td><strong>Increased in:</strong> Hyperthyroidism, non-thyroidal illness, especially psychiatric. Drugs: amiodarone, beta-blockers (high dose). <strong>Decreased in:</strong> Hypothyroidism, non-thyroidal illness. Drugs: phenytoin.</td>
<td>Test is useful in patients with clinically suspected hyper- or hypothyroidism, in elderly patients admitted to geriatric units, or in women over 40 with one or more somatic complaints. (See Thyroid function table, p 393.) Screening for thyroid disease is not indicated in younger women, men, or patients admitted with acute medical or psychiatric illnesses because transient abnormalities are indistinguishable from true thyroid disease. FT₄I is functionally equivalent to the FT₄ (see above). Ann Intern Med 1990;112:840.</td>
</tr>
<tr>
<td><strong>Toxoplasma antibody</strong>, serum or CSF (Toxo)</td>
<td><strong>Toxoplasma gondii</strong> is an obligate intracellular protozoan that causes human infection via ingestion, transplacental transfer, blood products, or organ transplantation. Cats are the definitive hosts of <em>T. gondii</em> and pass oocysts in their feces. Human infection occurs through ingestion of sporulated oocysts or via the transplacental route. In the immunodeficient host, acute infection may progress to lethal meningoencephalitis, pneumonitis, or myocarditis. In acute primary infection, IgM antibodies develop 1–2 weeks after onset of illness, peak in 6–8 weeks, and then decline. IgG antibodies develop on a similar time-course but persist for years. In adult infection, the disease usually represents a reactivation, not a primary infection. Therefore, the IgM test is less useful. Approximately 30% of all US adults have antibodies to <em>T. gondii.</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td></td>
<td></td>
</tr>
<tr>
<td>IgG: $&lt;1:16$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infant $&lt;1:2$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adult $&lt;1:8$ titer</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Marbled or CSF $$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Submit paired sera, one collected within 1 week of illness and another 2–3 weeks later.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Increased in:</strong> Acute or congenital toxoplasmosis (IgM), previous toxoplasma exposure (IgG), and false-positive (IgM) reactions (SLE, HIV infection, rheumatoid arthritis).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------------</td>
<td>----------------</td>
<td>----------</td>
</tr>
<tr>
<td>&lt;165 mg/dL [&lt;1.65 g/L]</td>
<td>Marbled $</td>
<td>Fasting specimen required.</td>
<td></td>
</tr>
<tr>
<td>Common Laboratory Tests: Selection and Interpretation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------------------------------------------------</td>
<td>---</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Triiodothyronine, total (T₃)**<br>**serum** 95–190 ng/dL [1.5–2.9 nmol/L]<br>

Marbled

---

**Triiodothyronine, total (T₃)**<br>**reflects the metabolically active form of thyroid hormone and is influenced by thyroid hormone binding activity.**

**Increased in:** Hyperthyroidism (some), increased thyroid-binding globulin.<br>**Decreased in:** Hypothyroidism, nonthyroidal illness, decreased thyroid-binding globulin.<br>**Drugs:** Amiodarone.

---

**T₃** may be increased in approximately 5% of hyperthyroid patients in whom T₄ is normal (T₃ toxicosis). Therefore, test is useful when hyperthyroidism is suspected and T₄ value is normal. Test is of no value in the diagnosis of hypothyroidism.

---


*JAMA 1990;263:1529.*

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin-I, cardiac, serum (cTnI) &lt; 1.5 ng/mL</td>
<td>Troponin is the contractile regulatory protein of striated muscle. It contains three subunits: T, C, and I. Subunit I consists of three forms, which are found in slow-twitch skeletal muscle, fast-twitch skeletal muscle, and cardiac muscle, respectively. Troponin I is predominantly a structural protein and is released into the circulation after cellular necrosis. Cardiac troponin I is expressed only in cardiac muscle, throughout development and despite pathology, and thus its presence in serum can distinguish between myocardial injury and skeletal muscle injury. cTnI is measured by immunoassay using monoclonal antibodies.</td>
<td><strong>Increased in:</strong> Myocardial infarction (sensitivity 50% at 4 hours, 97% at 6 hours; specificity 95%), cardiac trauma, cardiac surgery, myocardial damage following PTCA, defibrillations, and other cardiac interventions, nonischemic dilated cardiomyopathy. Slight elevations noted in patients with recent aggravated unstable angina, muscular disorders, CNS disorders, HIV infection, chronic renal failure, cirrhosis, sepsis, lung diseases, and endocrine disorders. <strong>Not Increased in:</strong> Skeletal muscle disease (myopathy, myositis, dystrophy), noncardiac trauma or surgery, rhabdomyolysis, severe muscular exertion, chronic renal failure.</td>
<td>Cardiac troponin I is a more specific marker for myocardial infarction than CKMB with roughly equivalent sensitivity early in the course of infarction (4–36 hours). Sensitivity and specificity for peak concentrations of cTnI (100%; 96%) are equivalent to or better than those for CK-MB (88%; 93%) and total CK (73%; 85%). cTnI appears in serum approximately 4 hours after onset of chest pain, peaks at 8–12 hours, and persists for 5–7 days. This prolonged persistence gives it much greater sensitivity than CKMB for diagnosis of myocardial infarction beyond the first 36–48 hours. Minor elevations of cardiac troponin I should be interpreted with caution, particularly in patients suffering from acute illnesses who do not have chest pain or prior myocardial infarction. Clin Chem 1994;40:1291. N Engl J Med 1994;330:670. Clin Chem 1995;41:1266. N Engl J Med 1997;337:1648. Am Heart J 1999;137:332. Am J Emerg Med 1999;17:225.</td>
</tr>
<tr>
<td><strong>Tularemia agglutinins</strong>, serum</td>
<td><em>Francisella tularensis</em> is an organism of wild rodents (rabbits and hares) that infects humans (eg, trappers and skinners) via contact with animal tissues, by the bite of certain ticks and flies, and by consumption of undercooked meat or contaminated water. Agglutinating antibodies appear in 10–14 days and peak in 5–10 weeks. A four-fold rise in titers is typically needed to prove acute infection. Titers decrease over years.</td>
<td><strong>Increased in:</strong> Tularemia; cross-reaction with brucella antigens and proteus OX-19 antigen (but at lower titers).</td>
<td>Single titers of &gt;1:160 are indicative of infection. Maximum titers are &gt;1:1280. A history of exposure to rabbits, ticks, dogs, cats, or skunks is suggestive of—but is not a requirement for—the diagnosis. Most common presentation is a single area of painful lymphadenopathy with low-grade fever. Initial treatment should be empiric. Culture of the organism is difficult, requiring special media, and hazardous to laboratory personnel. Serologic tests are the mainstay of diagnosis. Medicine 1985;64:251. N Engl J Med 1993;329:936. Semin Respir Infect 1997;12:61.</td>
</tr>
<tr>
<td>Type and cross-match, serum and red cells (Type and cross)</td>
<td>A type and cross-match involves ABO and Rh grouping (see pp 44 and 154, respectively), antibody screen (see p 53), and cross-match. (Compare with Type and Screen, below.) A major cross-match involves testing recipient serum against donor cells. It uses antihuman globulin to detect recipient’s antibodies on donor red cells. If the recipient’s serum contains a clinically significant alloantibody by antibody screen, a cross-match is required.</td>
<td>A type and screen is adequate preparation for operative procedures unlikely to require transfusion. Unnecessary type and cross-match orders reduce blood availability and add to costs. In addition, a preordering system should be in place, indicating the number of units of blood likely to be needed for each operative procedure. <em>Technical Manual of the American Association of Blood Banks</em>, 11th ed. American Association of Blood Banks, 1993.</td>
<td></td>
</tr>
<tr>
<td>Test/Range/Collection</td>
<td>Physiologic Basis</td>
<td>Interpretation</td>
<td>Comments</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Type and screen, serum and red cells</strong></td>
<td>Type and screen includes ABO and Rh grouping (see pp 44 and 154, respectively) and antibody screen (see p 53). (Compare with Type and Cross-Match, above.)</td>
<td>A negative antibody screen implies that a recipient can receive un-cross-matched type-specific blood with minimal risk. If the recipient’s serum contains a clinically significant alloantibody by antibody screen, a cross-match is required.</td>
<td>Type and screen is indicated for patients undergoing operative procedures unlikely to require transfusion. However, in the absence of preoperative indications, routine preoperative blood type and screen testing is not cost-effective and may be eliminated for some procedures, such as laparoscopic cholecystectomy, expected vaginal delivery, and vaginal hysterectomy. Technical Manual of the American Association of Blood Banks, 11th ed. American Association of Blood Banks, 1993. Am J Obstet Gynecol 1996;175:1201. Obstet Gynecol 1998;94(4 Part 1):493. Surg Endosc 1999;13:146.</td>
</tr>
<tr>
<td><strong>Red or lavender</strong> $$$</td>
<td>Specimen label must be signed by the person drawing the blood. A second “check” specimen is needed at some hospitals.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

Type and screen includes ABO and Rh grouping (see pp 44 and 154, respectively) and antibody screen (see p 53). (Compare with Type and Cross-Match, above.)
<table>
<thead>
<tr>
<th><strong>Uric acid, serum</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Males: 2.4–7.4 mg/dL</td>
</tr>
<tr>
<td>Females: 1.4–5.8 mg/dL</td>
</tr>
<tr>
<td>[Males: 140–440 µmol/L]</td>
</tr>
<tr>
<td>Females: 80–350 µmol/L</td>
</tr>
</tbody>
</table>

Marbled $\$

Uric acid is an end product of nucleoprotein metabolism and is excreted by the kidney. An increase in serum uric acid concentration occurs with increased nucleoprotein synthesis or catabolism (blood dyscrasias, therapy of leukemia) or decreased renal uric acid excretion (e.g., thiazide diuretic therapy or renal failure).

**Increased in:** Renal failure, gout, myeloproliferative disorders (leukemia, lymphoma, myeloma, polycythemia vera), psoriasis, glycogen storage disease (type I), Lesch-Nyhan syndrome (X-linked hypoxanthine-guanine phosphoribosyltransferase deficiency), lead nephropathy, hypertensive diseases of pregnancy, menopause. Drugs: antimetabolite and chemotherapeutic agents, diuretics, ethanol, nicotinic acid, salicylates (low dose), theophylline.

**Decreased in:** SIADH, xanthine oxidase deficiency, low-purine diet, Fanconi’s syndrome, neoplastic disease (various, causing increased renal excretion), liver disease. Drugs: salicylates (high dose), allopurinol (xanthine oxidase inhibitor).

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vanillylmandelic acid, urine (VMA)</td>
<td>Catecholamines secreted in excess by pheochromocytomas are metabolized by the enzymes monoamine oxidase and catechol-O-methyltransferase to VMA, which is excreted in urine.</td>
<td><strong>Increased in:</strong> Pheochromocytoma (96% sensitivity, 100% specificity), neuroblastoma, ganglioneuroma, generalized anxiety. <strong>Decreased in:</strong> Drugs: monoamine oxidase inhibitors.</td>
<td>A 24-hour urine metanephrine test (p 125) is the recommended test for the diagnosis of pheochromocytoma. (See also Pheochromocytoma algorithm, p 355.) A special diet is not needed when VMA test is done by the usual method. &lt;0.1% of hypertensive patients have a pheochromocytoma. Am J Cardiol 1970;26:270. Ann Surg 1974;179:740. Neuropsychobiology 1995;31:6. Psychiatr Res 1995;57:1.</td>
</tr>
<tr>
<td>Vanillylmandelic acid, urine (VMA)</td>
<td>Catecholamines secreted in excess by pheochromocytomas are metabolized by the enzymes monoamine oxidase and catechol-O-methyltransferase to VMA, which is excreted in urine.</td>
<td><strong>Increased in:</strong> Pheochromocytoma (96% sensitivity, 100% specificity), neuroblastoma, ganglioneuroma, generalized anxiety. <strong>Decreased in:</strong> Drugs: monoamine oxidase inhibitors.</td>
<td>A 24-hour urine metanephrine test (p 125) is the recommended test for the diagnosis of pheochromocytoma. (See also Pheochromocytoma algorithm, p 355.) A special diet is not needed when VMA test is done by the usual method. &lt;0.1% of hypertensive patients have a pheochromocytoma. Am J Cardiol 1970;26:270. Ann Surg 1974;179:740. Neuropsychobiology 1995;31:6. Psychiatr Res 1995;57:1.</td>
</tr>
<tr>
<td>Venereal Disease Research Laboratory test, serum (VDRL)</td>
<td>This syphilis test measures nontreponemal antibodies that are produced when Treponema pallidum interacts with host tissues. The VDRL usually becomes reactive at a titer of &gt;1:32 within 1–3 weeks after the genital chancre appears.</td>
<td><strong>Increased in:</strong> Syphilis: primary (59–87%), secondary (100%), late latent (79–91%), tertiary (37–94%); collagen-vascular diseases (rheumatoid arthritis, SLE), infections (mononucleosis, leprosy, malaria), pregnancy, drug abuse.</td>
<td>VDRL is used as a syphilis screening test and in suspected cases of primary and secondary syphilis. Positive tests should be confirmed with an FTA-ABS or MHA-TP test (see pp 92 and 129, respectively). The VDRL has similar sensitivity and specificity to the RPR (see Syphilis test table, p 391). Ann Intern Med 1986;104:368. Ann Intern Med 1991;114:1005. Sex Trans Dis 1998;26:12.</td>
</tr>
</tbody>
</table>
**Venereal Disease Research Laboratory test, CSF (VDRL)**

**Nonreactive**

$$
\text{Deliver in a clean plastic or glass tube.}
$$

The CSF VDRL test measures nontreponemal antibodies that develop in the CSF when *Treponema pallidum* interacts with the central nervous system.

**Increased in:** Tertiary neurosyphilis (10–27%).

The quantitative VDRL is the test of choice for CNS syphilis. Since the sensitivity of CSF VDRL is very low, a negative test does not rule out neurosyphilis. Clinical features, CSF white cell count, and CSF protein should be used together to make the diagnosis (see CSF profiles, p 369). Because the specificity of the CSF VDRL test is high, a positive test confirms the presence of neurosyphilis.

Patients being screened for neurosyphilis with CSF VDRL testing should have a positive serum RPR, VDRL, FTA-ABS, MHA-TP test or other evidence of infection.

Repeat testing may be indicated in HIV-infected patients in whom neurosyphilis is suspected.

When the CSF VDRL is negative but suspicion of CNS syphilis is high, other commonly used laboratory tests (CSF FTA-ABS, serum FTA-ABS, CSF Treponema Pallidum hemagglutination [TPHA], serum TPHA, and CSF cells) can, in combination, identify 87% of patients with neurosyphilis with 94% specificity.

Gen Hosp Psychiatry 1995;17:305.
Sex Trans Dis 1996;23:392.
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vitamin B&lt;sub&gt;12&lt;/sub&gt;, serum</strong>&lt;br&gt;140–820 pg/mL&lt;br&gt;[100–600 pmol/L]</td>
<td>Vitamin B&lt;sub&gt;12&lt;/sub&gt; is a necessary cofactor for three important biochemical processes: conversion of methylmalonyl-CoA to succinyl-CoA and methylation of homocysteine to methionine and demethylation of methylenetetrahydrofolate to tetrahydrofolate (THF). Consequent deficiency of folate coenzymes derived from THF is probably the crucial lesion caused by B&lt;sub&gt;12&lt;/sub&gt; deficiency. All vitamin B&lt;sub&gt;12&lt;/sub&gt; comes from ingestion of foods of animal origin. Vitamin B&lt;sub&gt;12&lt;/sub&gt; in serum is protein-bound, 70% to transcobalamin I (TC I) and 30% to transcobalamin II (TC II). The B&lt;sub&gt;12&lt;/sub&gt; bound to TC II is physiologically active; that bound to TC I is not.</td>
<td><strong>Increased in:</strong> Leukemia (acute myelocytic, chronic myelocytic, chronic lymphocytic, monocytic), marked leukocytosis, polycythemia vera. (Increased B&lt;sub&gt;12&lt;/sub&gt; levels are not diagnostically useful.)&lt;br&gt;<strong>Decreased in:</strong> Pernicious anemia, gastrectomy, gastric carcinoma, malabsorption (sprue, celiac disease, steatorrhea, regional enteritis, fistulas, bowel resection, <em>Diphyllobothrium latum</em> [fish tapeworm] infestation, small bowel bacterial overgrowth), pregnancy, dietary deficiency, HIV infection (with or without malabsorption), chronic high-flux hemodialysis, Alzheimer’s disease, drugs (eg, omeprazole, metformin, carbamazepine).</td>
<td>Differentiation among the causes of vitamin B&lt;sub&gt;12&lt;/sub&gt; deficiency can be accomplished by a vitamin B&lt;sub&gt;12&lt;/sub&gt; absorption (Schilling’s) test (see below). The commonly available competitive protein binding assay measures total B&lt;sub&gt;12&lt;/sub&gt;. It is insensitive to significant decreases in physiologically significant B&lt;sub&gt;12&lt;/sub&gt; bound to TC II. Specificity of the serum vitamin B&lt;sub&gt;12&lt;/sub&gt; test (approximately 73%) has not been systematically studied. Neuropsychiatric disorders caused by low serum B&lt;sub&gt;12&lt;/sub&gt; level can occur in the absence of anemia or macrocytosis. Br J Haematol 1993;83:643. Essays Biochem 1994;28:63. JAMA 1994;272:1233. Ann Intern Med 1994;120:211. Ann Clin Lab Sci 1997;27:249. Nephron 1997;75:259. Am J Med 1998;104:422.</td>
</tr>
<tr>
<td>Marbled $$</td>
<td>Serum vitamin B&lt;sub&gt;12&lt;/sub&gt; specimens should be frozen if not analyzed immediately.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**Vitamin B<sub>12</sub>**

---

**Vitamin B<sub>12</sub>**
**Vitamin B₁₂ absorption test**, 24-hour urine (Schilling’s test)

Excretion of >8% of administered dose

Stage I: 0.5–1.0 µCi of $^{52}$Co-B₁₂ is given orally, followed by 1.0 mg of unlabeled B₁₂ IM 2 hours later. A 24-hour urine is collected.

Stage II: After 5 days, test is repeated with 60 mg active hog intrinsic factor added to the oral labeled B₁₂.

Absorption of vitamin B₁₂ is dependent on two factors: adequate intrinsic factor produced by the stomach antrum and normal ileal absorption. Lack of either can lead to B₁₂ deficiency.

**Decreased in:** Ileal disease or resection, bacterial overgrowth, B₁₂ deficiency (because megaloblastosis of the intestinal wall leads to decreased B₁₂ absorption, pernicious anemia (<2.5% excretion of administered dose), post-gastrectomy, chronic pancreatitis, cystic fibrosis, giardiasis, Crohn’s disease.

Previously administered diagnostic and therapeutic radiopharmaceuticals may interfere with performance of the Schilling test for prolonged periods of time.

If the patient’s creatinine clearance is <60 mL/min, a 48-hour urine should be collected.

Pernicious anemia is suggested by an abnormal stage I test, followed by a normal stage II test (ie, addition of intrinsic factor leads to normal intestinal absorption and urinary excretion).

Ileal malabsorption gives abnormal results in stages I and II.

Low intrinsic factor contributing to B₁₂ deficiency is common in AIDS.

Egg yolk-bound B₁₂ should be used rather than crystalline B₁₂ to avoid false negative tests.

**References:**
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Vitamin D₃, 25-hydroxy, serum or plasma (25[OH]D₃) 10–50 ng/mL [25–125 nmol/L] Marbled or green $$$ | The vitamin D system functions to maintain serum calcium levels. Vitamin D is a fat-soluble steroid hormone. Two molecular forms exist: D₃ (cholecalciferol), synthesized in the epidermis, and D₂ (ergocalciferol), derived from plant sources. To become active, both need to be further metabolized. Two sequential hydroxylations occur: in the liver to 25(OH)D₃ and then, in the kidney, to 1,25(OH)₂D₃. Plasma levels increase with sun exposure. | **Increased in:** Heavy milk drinkers (up to 64 ng/mL), vitamin D intoxication, sun exposure.  
| Vitamin D₃, 1,25-dihydroxy, serum or plasma (1,25[OH]₂D₃) | 20–76 pg/mL | Marbled or green $$$$$ |

1,25-Dihydroxy vitamin D₃ is the most potent form of vitamin D. The main actions of vitamin D are the acceleration of calcium and phosphate absorption in the intestine and stimulation of bone resorption.

**Increased in:** Primary hyperparathyroidism, idiopathic hypercalciuria, sarcoidosis, some lymphomas, 1,25(OH)₂D₃-resistant rickets, normal growth (children), pregnancy, lactation, vitamin D toxicity.

**Decreased in:** Chronic renal failure, anephric patients, hypoparathyroidism, pseudohypoparathyroidism, 1α-hydroxylase deficiency, postmenopausal osteoporosis.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>von Willebrand’s factor protein (immunologic), plasma (vWF)</strong></td>
<td>von Willebrand’s factor (vWF) is produced by endothelial cells, circulates in the plasma complexed to factor VIII coagulant protein, and mediates platelet adhesion. vWF is a marker of endothelial injury. Both quantitative and qualitative changes can cause disease. vWF can be measured as protein antigen (immunologic measure) or by ristocetin cofactor activity (functional assay).</td>
<td><strong>Increased in:</strong> Inflammatory states (acute phase reactant). <strong>Decreased in:</strong> von Willebrand’s disease.</td>
<td>In von Willebrand’s disease, the platelet count and morphology are generally normal and the bleeding time is usually prolonged (markedly prolonged by aspirin). Variant forms associated with mild thrombocytopenia and angiodysplasia are described. The PTT may not be prolonged if factor VIII coagulant level is &gt;30%. Diagnosis is suggested by bleeding symptoms and family history. Laboratory diagnosis of von Willebrand’s disease has become more difficult because of the identification of numerous variant forms. In the classic type I disease, vWF antigen is decreased. Blood 1987;70:895. Mayo Clin Proc 1991;66:832. Thromb Haemost 1998;80:4095.</td>
</tr>
<tr>
<td>Blue $$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>d-Xylose absorption test, urine</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&gt;5 g per 5-hour urine (&gt;20% excreted in 5 hours)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
| $$$
  Fasting patient is given d-xylose, 25 g in two glasses of water, followed by four glasses of water over the next 2 hours. Urine is collected for 5 hours and refrigerated. |

| **Xylose is normally easily absorbed from the small intestine. Measuring xylose in serum or its excretion in urine after ingestion evaluates the carbohydrate absorption ability of the proximal small intestine.** |
|**Decreased in:** Intestinal malabsorption, small intestinal bacterial overgrowth, renal insufficiency, small intestinal HIV enteropathy, cryptosporidiosis, cytotoxic therapy-related malabsorption. |
| Test can be helpful in distinguishing intestinal malabsorption (decreased d-xylose absorption) from pancreatic insufficiency (normal d-xylose absorption). Urinary xylose excretion may be spuriously decreased in renal failure, thus limiting the specificity and usefulness of the test. In this case, a serum xylose level (gray top tube) obtained 1 hour after administration of a 25-g dose of d-xylose can be used to evaluate xylose absorption. The normal level should be > 29 mg/dL (1.9 mmol/L). |
This page intentionally left blank.
UNDERLYING ASSUMPTIONS

The basic assumptions underlying therapeutic drug monitoring are that drug metabolism varies from patient to patient and that the plasma level of a drug is more closely related to the drug’s therapeutic effect or toxicity than is the dosage.

INDICATIONS FOR DRUG MONITORING

Drugs with a narrow therapeutic index (where therapeutic drug levels do not differ greatly from levels associated with serious toxicity) should be monitored. Example: Lithium.

Patients who have impaired clearance of a drug with a narrow therapeutic index are candidates for drug monitoring. The clearance mechanism of the drug involved must be known. Example: Patients with renal failure have decreased clearance of gentamicin and therefore are at a higher risk for gentamicin toxicity.
Drugs whose toxicity is difficult to distinguish from a patient’s underlying disease may require monitoring. Example: Theophylline in patients with chronic obstructive pulmonary disease.

Drugs whose efficacy is difficult to establish clinically may require monitoring of plasma levels. Example: Phenytoin.

SITUATIONS IN WHICH DRUG MONITORING MAY NOT BE USEFUL

Drugs that can be given in extremely high doses before toxicity is apparent are not candidates for monitoring. Example: Penicillin.

If there are better means of assessing drug effects, drug level monitoring may not be appropriate. Example: Warfarin is monitored by prothrombin time and INR (International Normalized Ratio) determinations, not by serum levels.

Drug level monitoring to assess compliance is limited by the inability to distinguish noncompliance from rapid metabolism without direct inpatient scrutiny of drug administration.

Drug toxicity cannot be diagnosed with drug levels alone; it is a clinical diagnosis. Drug levels within the usual therapeutic range do not rule out drug toxicity in a given patient. Example: Digoxin, where other physiologic variables (eg, hypokalemia) affect drug toxicity.

In summary, therapeutic drug monitoring may be useful to guide dosage adjustment of certain drugs in certain patients. Patient compliance is essential if drug monitoring data are to be correctly interpreted.

OTHER INFORMATION REQUIRED FOR EFFECTIVE DRUG MONITORING

Reliability of the Analytic Method

The analytic sensitivity of the drug monitoring method must be adequate. For some drugs, plasma levels are in the nanogram per milliliter range. Example: Tricyclic antidepressants, digoxin.

The specificity of the method must be known, since the drug’s metabolites or other drugs may interfere. Interference by metabolites—which may or may not be pharmacologically active—is of particular concern in immunologic assay methods using antibodies to the parent drug.

The precision of the method must be known in order to assess whether changes in levels are caused by method imprecision or by clinical changes.
Reliability of the Therapeutic Range

Establishing the therapeutic range for a drug requires a reliable clinical assessment of its therapeutic and toxic effects, together with plasma drug level measurements by a particular analytic method. In practice, as newer, more specific analytic methods are introduced, the therapeutic ranges for those methods are estimated by comparing the old and new methodologies—without clinical correlation.

Pharmacokinetic Parameters

Five pharmacokinetic parameters that are important in therapeutic drug monitoring include:

1. **Bioavailability.** The bioavailability of a drug depends in part on its formulation. A drug that is significantly metabolized as it first passes through the liver exhibits a marked “first-pass effect,” reducing the effective oral absorption of the drug. A reduction in this first-pass effect (eg, because of decreased hepatic blood flow in heart failure) could cause a clinically significant increase in effective oral drug absorption.

2. **Volume of distribution and distribution phases.** The volume of distribution of a drug determines the plasma concentration reached after a loading dose. The distribution phase is the time taken for a drug to distribute from the plasma to the periphery. Drug levels drawn before completion of a long distribution phase may not reflect levels of pharmacologically active drug at sites of action. Examples: Digoxin, lithium.

3. **Clearance.** Clearance is either renal or nonrenal (usually hepatic). Whereas changes in renal clearance can be predicted on the basis of serum creatinine or creatinine clearance, there is no routine liver function test for assessment of hepatic drug metabolism. For most therapeutic drugs measured, clearance is independent of plasma drug concentration, so that a change in dose is reflected in a similar change in plasma level. If, however, clearance is dose-dependent, dosage adjustments produce disproportionately large changes in plasma levels and must be made cautiously. Example: Phenytoin.

4. **Half-life.** The half-life of a drug depends on its volume of distribution and its clearance and determines the time taken to reach a steady state level. In three or four half-lives, the drug level will be 87.5% to 93.75% of the way to steady state. Patients with decreased drug clearance and therefore increased drug half-lives will take longer to reach a higher steady state level. In general, since non-steady state drug levels are potentially misleading and can be difficult to interpret, it is recommended that most clinical monitoring be done at steady state.
5. **Protein binding of drugs.** All routine drug level analysis involves assessment of both protein-bound and free drug. However, pharmacologic activity depends on only the free drug level. Changes in protein binding (eg, in uremia or hypoalbuminemia) may significantly affect interpretation of reported levels for drugs that are highly protein-bound. *Example:* Phenytoin. In such cases, where the ratio of free to total measured drug level is increased, the usual therapeutic range based on total drug level will not apply.

**Drug Interactions**

For patients receiving several medications, the possibility of drug interactions affecting drug elimination must be considered. *Example:* Quinidine, verapamil, and amiodarone decrease digoxin clearance.

**Time to Draw Levels**

In general, the specimen should be drawn after steady state is reached (at least 3 or 4 half-lives after a dosage adjustment) and just before the next dose (trough level).

Peak and trough levels may be indicated to evaluate the dosage of drugs whose half-lives are much shorter than the dosing interval. *Example:* Gentamicin.

**Reference**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effective Concentrations</th>
<th>Half-Life (hours)</th>
<th>Dosage Adjustment</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amikacin</td>
<td>Peak: 10–25 µg/mL; Trough: &lt;10 µg/mL</td>
<td>2–3</td>
<td>↓ in renal dysfunction</td>
<td>Concomitant kanamycin or tobramycin therapy may give falsely elevated amikacin results by immunoassay.</td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>160–240 ng/mL</td>
<td>9–46</td>
<td></td>
<td>Drug is highly protein-bound. Patient-specific decrease in protein binding may invalidate quoted range of effective concentration.</td>
</tr>
<tr>
<td>Carbamazepine</td>
<td>4–8 µg/mL</td>
<td>10–30</td>
<td></td>
<td>Induces its own metabolism. Metabolite 10,11-epoxide exhibits 13% cross-reactivity by immunoassay. Toxicity: diplopia, drowsiness, nausea, vomiting, and ataxia.</td>
</tr>
<tr>
<td>Cyclosporine</td>
<td>150–400 mg/mL(ng/L) whole blood</td>
<td>6–12</td>
<td>Need to know specimen and methodology used</td>
<td>Cyclosporine is lipid-soluble (20% bound to leukocytes; 40% to erythrocytes; 40% in plasma, highly bound to lipoproteins). Binding is temperature-dependent, so whole blood is preferred to plasma or serum as specimen. High-performance liquid chromatography or monoclonal fluorescence polarization immunoassay measures cyclosporine reliably. Polyclonal fluorescence polarization immunoassays cross-react with metabolites, so the therapeutic range used with those assays is higher. Anticonvulsants and rifampin increase metabolism. Erythromycin, ketoconazole, and calcium channel blockers decrease metabolism.</td>
</tr>
<tr>
<td>Desipramine</td>
<td>100–250 ng/mL</td>
<td>13–23</td>
<td></td>
<td>Drug is highly protein-bound. Patient-specific decrease in protein binding may invalidate quoted range of effective concentration.</td>
</tr>
</tbody>
</table>

↔ = unchanged; ↑ = increased; ↓ = decreased; CHF = congestive heart failure
TABLE 4–1 (CONTINUED).  

<table>
<thead>
<tr>
<th>Drug</th>
<th>Effective Concentrations</th>
<th>Half-Life (hours)</th>
<th>Dosage Adjustment</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digoxin</td>
<td>0.8–2 ng/mL</td>
<td>42</td>
<td>↓ in renal dysfunc-</td>
<td>Bioavailability of digoxin tablets is 50–90%. Specimen must not be drawn tion, CHF</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>tion, CHF</td>
<td>relieved within 6 hours of dose. Dialysis does not remove a signific</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>ant amount. Hypokalemia potentiates toxicity. Digitalis toxicity is a</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>clinical and not a laboratory diagnosis. Digibind (digoxin-specific</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>antibody) therapy of digoxin overdose can interfere with measureme</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>nt of digoxin levels depending on the digoxin assay. Elimination is</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>reduced by quinidine, verapamil, and amiodarone.</td>
</tr>
<tr>
<td>Ethosuximide</td>
<td>40–100 mg/L</td>
<td>Child: 30 Adult: 50</td>
<td></td>
<td>Levels used primarily to assess compliance. Toxicity is rare and</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>does not correlate well with plasma concentrations.</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>Peak: 4–8 µg/mL Trough: &lt;2 µg/mL</td>
<td>2–5</td>
<td>↓ in renal dysfun-</td>
<td>Draw peak specimen 30 minutes after end of infusion. Draw trough</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ction</td>
<td>just before next dose. In uremic patients, carbenicillin may reduce</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>gentamicin half-life from 46 hours to 22 hours. If a once-daily regimen</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>(5 mg/kg) is used to maximize bacterial killing by optimizing the peak</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>concentration/MIC ratio and to reduce the potential for toxicity, dosag</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>e should be reduced if trough concentration is &gt;1 µg/mL (1 mg/L). Meas</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>urement of peak concentrations is not recommended with this regimen.</td>
</tr>
<tr>
<td>Imipramine</td>
<td>180–350 ng/mL</td>
<td>10–16</td>
<td>↓ in CHF, liver di-</td>
<td>Drug is highly protein-bound. Patient-specific decrease in protein</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ease</td>
<td>binding may invalidate quoted range of effective concentration.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Levels increased with cimetidine therapy. CNS toxicity common i</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1–5 µg/mL</td>
<td>1.8</td>
<td></td>
<td>n the elderly.</td>
</tr>
</tbody>
</table>

↔ = unchanged; ↑ = increased; ↓ = decreased; CHF = congestive heart failure
<table>
<thead>
<tr>
<th>Drug</th>
<th>Normal Range</th>
<th>86% Increase</th>
<th>Decreased</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lithium</td>
<td>0.7–1.5 meq/L</td>
<td>↑ in uremia</td>
<td>↓ in renal dysfunction</td>
<td>Thiazides and loop diuretics may increase serum lithium levels.</td>
</tr>
<tr>
<td>Methotrexate</td>
<td>50–40 ng/mL</td>
<td>↑ in uremia</td>
<td>↓ in renal dysfunction</td>
<td>7-Hydroxymethotrexate cross-reacts 1.5% in immunoassay. To minimize toxicity, leucovorin should be continued if methotrexate level is &gt;0.1 µmol/L at 48 hours after start of therapy. Methotrexate &gt;1 µmol/L at &gt;48 hours requires an increase in leucovorin rescue therapy.</td>
</tr>
<tr>
<td>Nortriptyline</td>
<td>10–30 µg/mL</td>
<td>↑ in cirrhosis</td>
<td>↓ in liver disease</td>
<td>Drug is highly protein-bound. Patient-specific decrease in protein binding may invalidate quoted range of effective concentration.</td>
</tr>
<tr>
<td>Phenobarbital</td>
<td>10–20 µg/mL</td>
<td>↑ in uremia, hypoalbuminemia</td>
<td>Dose-dependent</td>
<td>Metabolized principally by the hepatic microsomal enzyme system. Many drug-drug interactions.</td>
</tr>
<tr>
<td>Phenytoin</td>
<td>10–20 µg/mL</td>
<td>↓ in uremia, hypoalbuminemia</td>
<td>Dose-dependent</td>
<td>Metabolite cross-reacts 10% in immunoassay. Metabolism is capacity-limited. Increase dose cautiously when level approaches therapeutic range, since new steady state level may be disproportionately higher. Drug is very highly protein-bound, and when protein-binding is decreased in uremia and hypoalbuminemia, the usual therapeutic range does not apply. In this situation, use a reference range of 5–10 µg/mL.</td>
</tr>
<tr>
<td>Primidone</td>
<td>5–10 µg/mL</td>
<td>↑ in uremia</td>
<td>↓ in renal dysfunction</td>
<td>Phenobarbital cross-reacts 0.5%. Metabolized to phenobarbital. Primidone/phenobarbital ratio &gt;1:2 suggests poor compliance.</td>
</tr>
<tr>
<td>Procainamide</td>
<td>4–8 µg/mL</td>
<td>↑ in uremia</td>
<td>↓ in renal dysfunction</td>
<td>Thirty percent of patients with plasma levels of 12–16 µg/mL have ECG changes; 40% of patients with plasma levels of 16 µg/mL have severe toxicity. Metabolite N-acetylprocainamide is active.</td>
</tr>
<tr>
<td>Drug</td>
<td>Effective Concentrations</td>
<td>Half-Life (hours)</td>
<td>Dosage Adjustment</td>
<td>Comments</td>
</tr>
<tr>
<td>------------</td>
<td>--------------------------</td>
<td>------------------</td>
<td>------------------</td>
<td>--------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Quinidine</td>
<td>1–5 µg/L</td>
<td>7 ↔ in CHF</td>
<td>↓ in liver disease, CHF</td>
<td>Effective concentration is lower in chronic liver disease and nephrosis where binding is decreased.</td>
</tr>
<tr>
<td>Salicylate</td>
<td>150–300 µg/mL (15–30 mg/dL)</td>
<td>Dose-dependent</td>
<td></td>
<td>See Figure 8–23, p 360, for nomogram of salicylate toxicity.</td>
</tr>
<tr>
<td>Theophylline</td>
<td>5–20 µg/mL</td>
<td>9</td>
<td>↓ in CHF, cirrhosis, and with cimetidine</td>
<td>Caffeine cross-reacts 10%. Elimination is increased 1.5–2 times in smokers. 1,3-Dimethyl uric acid metabolite increased in uremia and because of cross-reactivity may cause an apparent slight increase in serum theophylline.</td>
</tr>
<tr>
<td>Tobramycin</td>
<td>Peak: 5–10 µg/mL Trough: &lt;2 µg/mL</td>
<td>2–3 ↑ in uremia</td>
<td>↓ in renal dysfunction</td>
<td>Tobramycin, kanamycin, and amikacin may cross-react in immunoassay. If a once-daily regimen is used to maximize bacterial killing by optimizing the peak concentration/MIC ratio and to reduce the potential for toxicity, dosage should be reduced if trough concentration is &gt;1 µg/mL (1 mg/L). Measurement of peak concentrations is not recommended with this regimen.</td>
</tr>
<tr>
<td>Valproic acid</td>
<td>55–100 µg/mL</td>
<td>13–19</td>
<td></td>
<td>Ninety-five percent protein-bound. Reduced binding in uremia and cirrhosis.</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>Trough: 5–15 µg/mL</td>
<td>6 ↑ in uremia</td>
<td>↓ in renal dysfunction</td>
<td>Toxicity in uremic patients leads to irreversible deafness. Keep peak level &lt;30–40 µg/mL to avoid toxicity.</td>
</tr>
</tbody>
</table>

↔ = unchanged; ↑ = increased; ↓ = decreased; CHF = congestive heart failure
Microbiology: Test Selection

Mary K. York, PhD

HOW TO USE THIS SECTION

This section displays information about clinically important infectious diseases in tabular form. Included in these tables are the Organisms involved in the disease/syndrome listed; Specimens/Diagnostic Tests that are useful in the evaluation; and Comments regarding the tests and diagnoses discussed. Topics are listed by body area/organ system: Central Nervous System, Eye, Ear, Sinus, Upper Airway, Lung, Heart and Vessels, Abdomen, Genitourinary, Bone, Joint, Muscle, Skin, and Blood.

Organisms

This column lists organisms that are known to cause the stated illness. Scientific names are abbreviated according to common usage (eg, *Streptococcus pneumoniae* as *S pneumoniae* or pneumococcus). Specific age or risk groups are listed in order of increasing age or frequency (eg, Infant, Child, Adult, HIV).

When bacteria are listed, Gram stain characteristics follow the organism name in parentheses—eg, “*S pneumoniae* (GPDC).” The following abbreviations are used:

Copyright 2001 The McGraw-Hill Companies. Click Here for Terms of Use.
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>GPC</td>
<td>Gram-positive cocci</td>
</tr>
<tr>
<td>GPD</td>
<td>Gram-positive diplococci</td>
</tr>
<tr>
<td>GPCB</td>
<td>Gram-positive coccobacilli</td>
</tr>
<tr>
<td>GPR</td>
<td>Gram-positive rods</td>
</tr>
<tr>
<td>GVCB</td>
<td>Gram-variable coccobacilli</td>
</tr>
<tr>
<td>GNC</td>
<td>Gram-negative cocci</td>
</tr>
<tr>
<td>GNDC</td>
<td>Gram-negative diplococci</td>
</tr>
<tr>
<td>GNCB</td>
<td>Gram-negative coccobacilli</td>
</tr>
<tr>
<td>GNR</td>
<td>Gram-negative rods</td>
</tr>
<tr>
<td>AFB</td>
<td>Acid-fast bacilli</td>
</tr>
</tbody>
</table>

When known, the frequency of the specific organism’s involvement in the disease process is also provided in parentheses—eg, “*S* pneumoniae (GPDC) (50%).”

**Specimen Collection/Diagnostic Tests**

This column describes the collection of specimens, laboratory processing, useful radiographic procedures, and other diagnostic tests. Culture or test sensitivities with respect to the diagnosis in question are placed in parentheses immediately following the test when known—eg, “Gram stain (60%).” Pertinent serologic tests are also listed. Keep in mind that few infections can be identified by definitive diagnostic tests and that clinical judgment is critical to making difficult diagnoses when test results are equivocal.

**Comments**

This column includes general information about the utility of the tests and may include information about patient management. Appropriate general references are also listed.

**Syndrome Name/Body Area**

In the last two columns the syndrome name and body area are placed perpendicular to the rest of the table to allow for quick referencing.
<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen /Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Brain Abscess</strong></td>
<td>Blood for bacterial cultures. Brain abscess aspirate for Gram stain (82%), bacterial (88%), AFB, fungal cultures, and cytology. Lumbar puncture is dangerous and contraindicated. Sources of infection in the ears, sinuses, lungs or bloodstream should be sought for culture when abscess is found. CT scan and MRI are the most valuable imaging procedures and can guide biopsy if a specimen is needed. (See CT scan, MRI of head, p 245.) Serum toxoplasma antibody in HIV-infected patients may not be positive at outset of presumptive therapy. If negative or if no response to empiric therapy, biopsy may be needed to rule out lymphoma, fungal infection, or tuberculosis. Biopsy material should be sent for toxoplasma antigen (DFA). Detection of toxoplasma DNA in blood or CSF samples by PCR techniques is now available from specialized or reference laboratories. A positive PCR result must be interpreted in the context of the clinical presentation. Active or recent infection is indicated by a positive IgM antibody test. (See also toxoplasma antibody, p 171.)</td>
<td>Occurs in patients with otitis media and sinusitis. Also seen in patients with cyanotic congenital heart disease and right-to-left shunting (eg, tetralogy of Fallot) or arteriovenous vascular abnormalities of the lung (eg, Osler-Weber-Rendu). Majority of toxoplasmosis abscesses are multiple and are seen on MRI in the basal ganglia, parietal and frontal lobes. 99mTcTechnetium brain scan is a very sensitive test for abscess and the test of choice where CT and MRI are unavailable. J Child Neurol 1995;10:283. Clin Infect Dis 1996;23:1061. Clin Infect Dis 1997;25:763. Neurol Clin 1998;16:419.</td>
</tr>
</tbody>
</table>

- **Brain Abscess**:
  - Usually polymicrobial
  - Child: anaerobes (40%), *S aureus* (GPC), *S pneumoniae* (GPDC), *S pyogenes* (GPC in chains), viridans streptococci (GPC in chains), less common, Enterobacteriaceae (GNR), *P aeruginosa* (GNR), *H influenzae* (GNDC), *N meningitidis* (GNDC)
  - Adults: Viridans and anaerobic streptococci (GPC in chains) (60–70%), bacteroides (GNR) (20–40%), Enterobacteriaceae (GNR) (23–33%), *S aureus* (GPC) (10–15%), other anaerobes, including fusobacterium (GNR) and actinomycyes (GPR), *T solium* (cysticerci)
  - Immunocompromised: *T gondii*, *C neoformans*, nocardia (GPR), mycobacteria (AFB), fungi, *E histolytica*.
  - Posttraumatic: *S aureus* (GPC), Enterobacteriaceae (GNR), coagulase-negative staphylococci (GPC), *P acnes* (GPR)
<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Encephalitis</strong></td>
<td>CSF for pressure (elevated), cell count (WBCs elevated but variable [10–2000/µL], mostly lymphocytes), protein (elevated, especially IgG fraction), glucose (normal), RBCs (especially in herpesvirus). Repeat examination of CSF after 24 hours often useful. (See CSF profiles, p 369.) CSF cultures for viruses and bacteria (low yield). CSF PCR in reference laboratories for CMV (33%), HSV (98%), VZV, and enterovirus. Identification of HSV DNA in CSF by PCR techniques is now the definitive diagnostic test. Throat swab for enterovirus, mumps. Stool culture for enterovirus, which is frequently shed for weeks (especially in children). Urine culture for mumps. Culture of both skin biopsy from hairline and saliva for rables. Single serum for bartonella (cat-scratch disease) IgM and IgG. Paired sera for arboviruses, mumps, or rables should be drawn acutely and after 1–3 weeks of illness. Serologic tests are often of academic interest only. Not indicated for herpes simplex. CT scan with contrast or MRI with gadolinium showing temporal lobe lesions suggests herpes simplex. Polyradiculopathy is highly suggestive of CMV in AIDS.</td>
<td>Pocket Guide to Diagnostic Tests CENTRAL NERVOUS SYSTEM Encephalitis</td>
</tr>
<tr>
<td>Arboviruses (California group, St. Louis, western equine), enteroviruses (coxsackie, echo, polio), herpes simplex (HSV), B henselae, lymphocytic choriomeningitis, mumps, tick-borne encephalitis virus, post-infectious (following influenza, human herpes virus 6 [HHV-6], measles, mumps, rubella, varicella-zoster [VZV]), rables, Creutzfeldt-Jakob Postvaccination: Rabies, pertussis. Immunocompromised: Cytomegalovirus (CMV), toxoplasmosis, papovavirus (PML)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Aseptic Meningitis

Acute: Enteroviruses (coxsackie, echo, polio) (90%), mumps, herpes simplex (HSV), HIV (primary HIV seroconversion), varicella-zoster (VZV), lymphocytic choriomeningitis virus (rare).

Recurrent: Herpes simplex type 2 (Mollaret's syndrome)

CSF for pressure (elevated), cell count (WBCs 10–100/µL, PMNs early, lymphocytes later), protein (normal or slightly elevated), and glucose (normal). (See CSF profiles, p 369.)

CSF viral culture can be negative despite active viral infection. Enteroviruses can be isolated from the CSF in the first few days after onset but only rarely after the first week.

Detection of enteroviral RNA in CSF by PCR from specialized or reference laboratories.

Urine viral culture for mumps.

Vesicle direct fluorescent antibody (DFA) or culture for HSV or VZV.

Paired sera for viral titers: poliovirus, mumps, and VZV. Not practical for other organisms unless actual isolate known and then only useful epidemiologically.

Detection of VZV or HSV in CSF by PCR.

Aseptic meningitis is acute meningeal irritation in the absence of pyogenic bacteria or fungi. Diagnosis is usually made by the examination of the CSF and by ruling out other infectious causes (eg, syphilis, tuberculosis). Consider nonsteroidal anti-inflammatory drugs as a noninfectious cause.

Enteroviral aseptic meningitis is rare after age 40. Patients with deficiency of the complement regulatory protein factor I may have recurrent aseptic meningitis.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacterial Meningitis</td>
<td>CSF for pressure (&gt;180 mm H&lt;sub&gt;2&lt;/sub&gt;O), cell count (WBCs 1000–100,000/µL, &gt;50% PMNs), protein (150–500 mg/dL), glucose (&lt;40% of serum). (See CSF profiles, p 369.) CSF for Gram stain of cytocentrifuged material (positive in 70–80%). CSF culture for bacteria. Blood culture positive in 40–60% of patients with pneumococcal, meningococcal, and ( H ) influenzae meningitis. CSF antigen tests are no longer considered useful because of their low sensitivity and false-positive results.</td>
<td>The first priority in the care of the patient with suspected acute meningitis is therapy, then diagnosis. Antibiotics should be started within 30 minutes of presentation. The death rate for meningitis is about 50% for pneumococcal, less for others. With recurrent ( N ) meningitidis meningitis, suspect a terminal complement component deficiency. With other recurrent bacterial meningitides, suspect a CSF leak. Postgrad Med 1998;103:102. Medicine (Baltimore) 1998;77:313. Infect Dis Clin North Am 1999;13:711. Infect Dis Clin North Am 1999;13:579.</td>
</tr>
</tbody>
</table>

Neonate: \( E \) coli (GNR), group B or D streptococci (GPC), \( L \) monocytogenes (GPR).

Infant: Group B streptococci, \( S \) pneumoniae (GPC), \( N \) meningitidis (GNDC), \( L \)isteria monocytogenes (GPR), \( H \) influenzae (GNCB).

Child: \( S \) pneumoniae, \( N \) meningitidis, \( H \) influenzae.

Adult: \( S \) pneumoniae, \( N \) meningitidis, \( L \) monocytogenes.

Postneurosurgical: \( S \) aureus (GPC), \( S \) pneumoniae, \( P \) acnes (GPR), coagulase-negative staphylococci (GPC), pseudomonas (GNR), \( E \) coli (GNR), other Enterobacteriaceae.

Alcoholic patients and the elderly: In addition to the adult organisms, Enterobacteriaceae, pseudomonas, \( H \) influenzae.
Fungal Meningitis

*C neoformans* (spherical, budding yeast). *C immitis* (spherules), *H capsulatum*.


<table>
<thead>
<tr>
<th>Test Selection</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Fungal Meningitis</strong></td>
</tr>
<tr>
<td>CSF for pressure (normal or elevated), cell count (WBCs 50–1000/µL, mostly lymphocytes), protein (elevated), and glucose (decreased). Serum cryptococcal antigen (CrAg) for <em>C neoformans</em> (99%). For other fungi, collect at least 5 mL of CSF for fungal culture. Initial cultures are positive in 40% of coccidioides cases and 27–65% of histoplasma cases. Repeat cultures are frequently needed. Culture of bone marrow, skin lesions, or other involved organs should also be performed if clinically indicated. CSF India ink preparation for cryptococcus is not recommended because it is positive in only 50% of cases. Serum coccidioidal serology is a concentrated serum immunodiffusion test for the organism (75–95%). CSF serologic testing is rarely necessary. (See coccidioides serology, p 74.) Complement fixation test for histoplasma is available from public health department laboratories (see p 109). Histoplasma antigen can be detected in urine, blood, or CSF in 61% of cases of histoplasma meningitis.</td>
</tr>
<tr>
<td>The clinical presentation of fungal meningitis in immunocompromised patients is that of an indolent chronic meningitis. Prior to AIDS, cryptococcal meningitis was seen both in patients with cellular immunologic deficiencies and in patients who lacked obvious defects (about 50% of cases). Cryptococcus is the most common cause of meningitis in AIDS patients and may present with normal CSF findings. Titer of CSF CrAg can be used to monitor therapeutic success (falling titer) or failure (unchanged or rising titer) or to predict relapse during suppressive therapy (rising titer). Clin Microbiol Rev 1995;8:515. Emerg Infect Dis 1996;2:109. Clin Infect Dis 1996;22:240. Scand J Infect Dis 1998;30:485.</td>
</tr>
</tbody>
</table>

---

**Fungal Meningitis**

- *C neoformans* (spherical, budding yeast).
- *C immitis* (spherules), *H capsulatum*.


- CSF for pressure (normal or elevated), cell count (WBCs 50–1000/µL, mostly lymphocytes), protein (elevated), and glucose (decreased).
- Serum cryptococcal antigen (CrAg) for *C neoformans* (99%).
- For other fungi, collect at least 5 mL of CSF for fungal culture. Initial cultures are positive in 40% of coccidioides cases and 27–65% of histoplasma cases. Repeat cultures are frequently needed.
- Culture of bone marrow, skin lesions, or other involved organs should also be performed if clinically indicated.
- CSF India ink preparation for cryptococcus is not recommended because it is positive in only 50% of cases.
- Serum coccidioidal serology is a concentrated serum immunodiffusion test for the organism (75–95%).
- CSF serologic testing is rarely necessary. (See coccidioides serology, p 74.)
- Complement fixation test for histoplasma is available from public health department laboratories (see p 109).
- Histoplasma antigen can be detected in urine, blood, or CSF in 61% of cases of histoplasma meningitis. 

---

**Fungal Meningitis**

- *C neoformans* (spherical, budding yeast).
- *C immitis* (spherules), *H capsulatum*.


- CSF for pressure (normal or elevated), cell count (WBCs 50–1000/µL, mostly lymphocytes), protein (elevated), and glucose (decreased).
- Serum cryptococcal antigen (CrAg) for *C neoformans* (99%).
- For other fungi, collect at least 5 mL of CSF for fungal culture. Initial cultures are positive in 40% of coccidioides cases and 27–65% of histoplasma cases. Repeat cultures are frequently needed.
- Culture of bone marrow, skin lesions, or other involved organs should also be performed if clinically indicated.
- CSF India ink preparation for cryptococcus is not recommended because it is positive in only 50% of cases.
- Serum coccidioidal serology is a concentrated serum immunodiffusion test for the organism (75–95%).
- CSF serologic testing is rarely necessary. (See coccidioides serology, p 74.)
- Complement fixation test for histoplasma is available from public health department laboratories (see p 109).
- Histoplasma antigen can be detected in urine, blood, or CSF in 61% of cases of histoplasma meningitis. 

---

**Fungal Meningitis**

- *C neoformans* (spherical, budding yeast).
- *C immitis* (spherules), *H capsulatum*.


- CSF for pressure (normal or elevated), cell count (WBCs 50–1000/µL, mostly lymphocytes), protein (elevated), and glucose (decreased).
- Serum cryptococcal antigen (CrAg) for *C neoformans* (99%).
- For other fungi, collect at least 5 mL of CSF for fungal culture. Initial cultures are positive in 40% of coccidioides cases and 27–65% of histoplasma cases. Repeat cultures are frequently needed.
- Culture of bone marrow, skin lesions, or other involved organs should also be performed if clinically indicated.
- CSF India ink preparation for cryptococcus is not recommended because it is positive in only 50% of cases.
- Serum coccidioidal serology is a concentrated serum immunodiffusion test for the organism (75–95%).
- CSF serologic testing is rarely necessary. (See coccidioides serology, p 74.)
- Complement fixation test for histoplasma is available from public health department laboratories (see p 109).
- Histoplasma antigen can be detected in urine, blood, or CSF in 61% of cases of histoplasma meningitis. 

<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
</table>
| Spirochetal Meningitis/ Neurologic diseases | **Neuroborreliosis:** CSF for pressure (normal or elevated), cell count (WBCs elevated, mostly lymphocytes), protein (may be elevated), and glucose (normal). Serum and CSF for serologic testing. False-positive serologic tests may occur. Western blots should be used to confirm borderline or positive results. CSF serology for anti-*B burgdorferi* IgM (90%). Culture and PCR less specific. For Lyme disease serologies, see p 122.  
**Acute syphilitic meningitis:** CSF for pressure (elevated), cell count (WBCs 25–2000/µL, mostly lymphocytes), protein (elevated), and glucose (normal or low). (See CSF profiles, p 369.) Serum VDRL. (See VDRL, serum, p 178.) CSF VDRL is the preferred test (see p 179), but is only 66% sensitive for acute syphilitic meningitis. **Neurosyphilis:** CSF for pressure (normal), cell count (WBCs normal or slightly increased, mostly lymphocytes), protein (elevated), glucose (normal), and CSF VDRL. Serum VDRL, FTA-ABS, or MHA-TP should be done. **Leptospirosis:** CSF cell count (WBCs <500/µL, mostly monocytes), protein (slightly elevated), and glucose (normal). Urine for dark-field examination of sediment. Blood and CSF dark-field examination only positive in acute phase prior to meningitis. Serum for serology for IgM. | Neurosyphilis is a late stage of infection and can present with meningo-vascular (hemiparesis, seizures, aphasia), parenchymal (general paresis, tabes dorsalis), or asymptomatic (latent) disease. Because there is no single highly sensitive or specific test for neurosyphilis, the diagnosis must depend on a combination of clinical and laboratory data. Therapy of suspected neurosyphilis should not be withheld on the basis of a negative CSF VDRL if clinical suspicion is high. In HIV neurosyphilis, treatment failures may be common. Lyme disease can present as a lymphocytic meningitis, facial palsy, or painful radiculitis. Leptospirosis follows exposure to rats.  
<table>
<thead>
<tr>
<th>Parasitic Meningoencephalitis</th>
<th>Tuberculous Meningitis</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>T. gondii, E. chaffeensis</em> (human monocytic ehrlichiosis) (HME) and other species of human granulocytic ehrlichiosis (HGE), <em>E. histolytica, N. fowleri, T. solium</em> (cysticerci).</td>
<td><em>M. tuberculosis</em> (MTb) (acid-fast bacilli [AFB])</td>
</tr>
<tr>
<td>CSF for pressure (normal or elevated), cell count (WBCs 100–1000/µL, chiefly monocytes, lymphocytes), protein (elevated), glucose (normal). Serology as for brain abscess.</td>
<td>CSF for pressure (elevated), cell count (WBCs 100–500/µL, PMNs early, lymphocytes later), protein (elevated), glucose (decreased). (See CSF profiles, p 369.)</td>
</tr>
<tr>
<td>Ehrlichiosis: White blood cell count low (1300–4000/µL), platelets low (50,000–140,000/µL), hepatic aminotransferases (tenfold above normal). Buffy coat for Giemsa (1% in HME, 18–80% in HGE), PCR of blood available (50–90% depending on prior therapy). Serum IgG and IgM usually not positive until the third week.</td>
<td>CSF for AFB stain. Stain is positive in only 30%. Cytocentrifugation and repeat smears increase yield. CSF for AFB culture (positive in &lt;70%). Repeated sampling of the CSF during the first week of therapy is recommended; ideally, 3 or 4 specimens of 5–10 mL each should be obtained (87% yield with 4 specimens). PCR available but not yet validated. DNA probes are available for rapid confirmation from mycobacterial growth.</td>
</tr>
<tr>
<td>Cysticercosis: Characteristic findings on CT and MRI are diagnostic. Serology is less sensitive.</td>
<td></td>
</tr>
<tr>
<td>Organism</td>
<td>Specimen / Diagnostic Tests</td>
</tr>
<tr>
<td>----------</td>
<td>---------------------------</td>
</tr>
</tbody>
</table>

### Keratitis

**Bacteria:** *P. aeruginosa* (GNR), staphylococci (GPC), *S. pneumoniae* (GPDC), moraxella sp.  
**Virus:** Herpes simplex (HSV) (dendritic pattern on fluorescein slitlamp examination), varicella-zoster virus (VZV)  
**Contact lens:** Acanthamoeba, Enterobacteriaceae (GNR).  
**Fungus:** Candida, fusarium, aspergillus, rhodotorula, and other filamentous fungi.  
**Parasite:** *O. volvulus* (river blindness), microsporidia (HIV)

**Corneal scrapings for Gram stain, KOH, and culture.**  
Routine bacterial culture is used for most bacterial causes, viral culture for herpes, and special media for acanthamoeba (can be detected with trichrome or Giemsa stain of smears).  
**T**reatment depends on Gram stain appearance and culture.  
**Corneal biopsy** may be needed if initial cultures are negative.

### Endophthalmitis

**Spontaneous or postoperative:** *S. aureus* (GPC), coagulase-negative staphylococci (GPC), *S. pneumoniae* (GPDC), candida sp; streptococci, non-group B (GPC in chains).  
**Trauma:** Bacillus sp (GPR), fungi.  
**Post-filtering bleb:** Viridans group streptococcus (57%), *S. pneumoniae* (GPDC), *H. influenzae* (GNCB).  
**IV drug abuse:** Add *B. cereus.*

**Culture material from anterior chamber, vitreous cavity, and wound abscess for bacteria, mycobacteria, and fungi.**  
Traumatic and postoperative cases should have aqueous and vitreous aspiration for culture and smear (56%).  
**Conjunctival cultures** are inadequate and misleading.

**Prompt ophthalmologic consultation is mandatory.**  
Acanthamoeba infection occurs in soft contact (extended-wear) lens wearers and may resemble HSV infection on fluorescein examination (dendritic [“branching”] ulcer).  
**Bacterial keratitis** is usually caused by contact lens use or trauma. Fungal keratitis is usually caused by trauma.  
CLA O J 1998;24:52.  
Cornea 1998;17:3.  
Cornea 1999;18:144.

**Endophthalmitis** is an inflammatory process of the ocular cavity and adjacent structures. Rapid diagnosis is critical, since vision may be compromised.  
**Bacterial endophthalmitis** usually occurs as a consequence of ocular surgery. Prophylactic antibiotic use is of unproved benefit, though topical antibiotics are widely used.  
Also consider retinitis in immunocompromised patients, caused by CMV, HSV, VZV, and toxoplasma (retinochoroiditis), which is diagnosed by retinal examination.  
Clin Infect Dis 1997;24:1172.  
<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Otitis Media</td>
<td>Infant, child, and adult: <em>S. pneumoniae</em> (30%) (GPDC), <em>H. influenzae</em> (30%) (GNCB), <em>M. catarrhalis</em> (10%) (GNDC), <em>S. aureus</em> (GPC), <em>S. pyogenes</em> (GPC in chains), <em>M. pneumoniae</em>, <em>C. pneumoniae</em>, “sterile.”</td>
<td>Peak incidence of otitis media occurs in the first 3 years of life, especially between 6 and 24 months of age.</td>
</tr>
<tr>
<td></td>
<td>Neonate: Same as above plus Enterobacteriaceae (GNR), group B streptococcus (GPC).</td>
<td>In neonates, predisposing factors include cleft palate, hypotonia, mental retardation (Down’s syndrome).</td>
</tr>
<tr>
<td></td>
<td>Endotracheal intubation: Pseudomonas sp (GNR), klebsiella (GNR), Enterobacteriaceae (GNR).</td>
<td>Tympanocentesis is indicated if the patient fails to improve after 48 hours or develops fever. It may hasten resolution and decrease sterile effusion.</td>
</tr>
<tr>
<td></td>
<td>Chronic: <em>S. aureus</em> (GPC), <em>P. aeruginosa</em> (GNR), anaerobes, <em>M. tuberculosis</em> (AFB).</td>
<td>Persistent middle ear effusion may require placement of ventilating or tympanostomy tubes.</td>
</tr>
<tr>
<td>Tympanocentesis aspirate for Gram stain and bacterial culture in the patient who has a toxic appearance. Otherwise, microbiologic studies of effusions are so consistent that empiric treatment is acceptable.</td>
<td>Bullous myringitis suggests mycoplasma.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>CSF examination if clinically indicated.</td>
<td>Emerging antibiotic resistance should be considered in choice of empiric antibiotic therapy.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Organism</td>
<td>Specimen/Diagnostic Tests</td>
<td>Comments</td>
</tr>
<tr>
<td>---------------------</td>
<td>------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| Sinusitis           | Nasal aspirate for bacterial culture is not usually helpful. Maxillary sinus aspirate for bacterial culture may be helpful in severe or atypical cases. | Diagnosis and treatment of sinusitis is usually based on clinical and radiologic features. Microbiologic studies can be helpful in severe or atypical cases. Sinus CT scan (or MRI) is better than plain x-ray for diagnosing sinusitis, particularly if sphenoid sinusitis is suspected. However, sinus CT scans should be interpreted cautiously, since abnormalities are also seen in patients with the common cold. 
Acute and chronic sinusitis occur frequently in HIV-infected patients, may be recurrent or refractory, and may involve multiple sinuses (especially when the CD4 cell count is <200/µL). Acute sinusitis often results from bacterial superinfection following viral upper respiratory infection.  

Acute: *S. pneumoniae* (GPC) (31%), *H. influenzae* (GNCB) (21%), *M. catarrhalis* (GNDC), *S. pyogenes* (2–5%) (GPC), anaerobes (2–5%), viruses (adenovirus, influenza, parainfluenza), *S. aureus* (GPC) (rare).  
Chronic (child): Viridans and anaerobic streptococci (GPC in chains) (23%), *S. aureus* (19%), *S. pneumoniae*, *H. influenzae*, *M. catarrhalis*, *P. aeruginosa* (GNR) in cystic fibrosis.  
Chronic (adult): Coagulase-negative staphylococci (GPC) (36%), *S. aureus* (GPC) (25%), viridans streptococci (GPC in chains) (8%), corynebacteria (GPR) (5%), anaerobes (6%), including bacteroides sp, prevotella sp (GNR), peptostreptococcus (GPC), fusobacterium sp (GNR).  
Hospitalized with nasogastric tube or nasotracheal intubation: Enterobacteriaceae (GNR), pseudomonas sp (GNR).  
Fungal: Zygomycetes (rhizopus), aspergillus, *P. boydii*.  
Immunocompromised: *P. aeruginosa* (GNR), cytomegalovirus (CMV), aspergillus sp. and other filamentous fungi plus microsporidia, *Cryptosporidium parvum*, acanthamoeba in HIV-infected patients.
<table>
<thead>
<tr>
<th><strong>Pharyngitis</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Throat swab for culture. Place in sterile tube or transport medium. If <em>N. gonorrhoeae</em> suspected, use chocolate agar or Thayer-Martin media. If <em>C. diphtheriae</em> suspected, use Tinsdale or blood agar. Throat swabs are routinely cultured for group A streptococcus only. If other organisms are suspected, this must be stated. Throat culture is about 70% sensitive for group A streptococcus. “Rapid” tests for group A streptococcus can speed diagnosis and aid in the treatment of family members. However, false-negative results may lead to underdiagnosis and failure to treat.</td>
</tr>
<tr>
<td>Controversy exists over how to evaluate patients with sore throat. Some authors suggest culturing all patients and then treating only those with positive cultures. In patients with compatible histories, be sure to consider pharyngeal abscess or epiglottitis, both of which may be life-threatening. Complications include pharyngeal abscess and Lemierre’s syndrome (infection with fusobacterium sp.), which can progress to sepsis and multi-organ failure.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th><strong>Laryngitis</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Virus (90%) (influenza, rhinovirus, adenovirus, parainfluenza, Epstein-Barr virus), <em>S. pyogenes</em> (GPC) (10%), <em>M. catarrhalis</em> (GNDC) (55% of adults), <em>M. tuberculosis</em>, fungus (cryptococcosis, histoplasmosis). Immunocompromised: Candida sp, cytomegalovirus, herpes simplex (HSV)</td>
</tr>
<tr>
<td>Diagnosis is made by clinical picture of upper respiratory infection with hoarseness.</td>
</tr>
<tr>
<td>Laryngitis usually occurs with common cold or influenzal syndromes. Fungal laryngeal infections occur most commonly in immunocompromised patients (AIDS, cancer, organ transplants, corticosteroid therapy, diabetes mellitus). Consider acid reflux for chronic cases.</td>
</tr>
</tbody>
</table>

---

**References:**
<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
</table>

**Infant/child:** Respiratory syncytial virus (RSV) (50–75%) (bronchiolitis), adenovirus, parainfluenza virus (croup), *B pertussis* (GNCB) (whooping cough), other viruses, including rhinovirus, coronavirus, influenza. **Adolescent/adult:** Usually viruses, *M pneumoniae*, *C pneumoniae*, *B pertussis*. **Chronic adult:** *S pneumoniae* (GPDC), *H influenzae* (GNCB), *M catarrhalis* (GNDC), klebsiella (GNR), other Enterobacteriaceae (GNR), viruses (eg, influenza), aspergillus (allergic bronchopulmonary aspergillosis). **Chronic obstructive airway disease:** Viral (25–50%), *S pneumoniae* (GPC), *H influenzae* (GNCB), *S aureus* (GPC), Enterobacteriaceae (GNR), anaerobes (<10%).
**Epiglottitis**

Child: *H influenzae* type B (GNCB).
Adult: *S pyogenes* (GPC), *H influenzae*.
HIV: Candida

Blood for bacterial culture: positive in 50–100% of children with *H influenzae*. Lateral neck x-ray may show an enlarged epiglottis but has a low sensitivity (31%).

Acute epiglottitis is a rapidly moving cellulitis of the epiglottis and represents an airway emergency. Epiglottitis can be confused with croup, a viral infection of gradual onset that affects infants and causes inspiratory and expiratory stridor. Airway management is the primary concern, and an endotracheal tube should be placed or tracheostomy performed as soon as the diagnosis of epiglottitis is made in children. A tracheostomy set should be at the bedside for adults.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen /Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Community-Acquired Pneumonia</strong></td>
<td>Sputum for Gram stain desirable; culture, if empiric therapy fails or patient is seriously ill. An adequate specimen should have &lt;10 epithelial cells and &gt;25 PMNs per low-power field. Special sputum cultures for legionella are available. DFA for legionella sp has a sensitivity of 25–70% and a specificity of 95%. (Positive predictive value is low in areas of low disease prevalence.) Blood for bacterial cultures, especially in ill patients. Pleural fluid for bacterial culture if significant effusion is present. Bronchoalveolar lavage or brushings for bacterial, fungal, and viral antigen tests and AFB culture in immunocompromised patients and atypical cases. Paired sera for <em>M pneumoniae</em> complement fixation testing can diagnose infection retrospectively. Serologic tests for <em>C pneumoniae</em>, <em>C psittaci</em> strains, and Q fever are available. Serologic tests and PCR for hantavirus (IgM and IgG) are available. Other special techniques (bronchoscopy with telescoping plugged catheter on protected brush, transtracheal aspiration, transthoracic fine-needle aspiration, or, rarely, open lung biopsy) can be used to obtain specimens for culture in severe cases, in immunocompromised patients, or in cases with negative conventional cultures and progression despite empiric antibiotic therapy.</td>
<td>About 60% of cases of community-acquired pneumonia have an identifiable microbial cause. Pneumatoceles suggest <em>S aureus</em> but are also reported with pneumococcus, group A streptococcus, <em>H influenzae</em>, and Enterobacteriaceae (in neonates). An “atypical pneumonia” presentation (diffuse pattern on chest x-ray with lack of organisms on Gram stain of sputum) should raise suspicion of mycoplasma, Legionella, or chlamydial infection. Consider hantavirus pulmonary syndrome if pulmonary symptoms follow afebrile illness. Aspirations are most commonly associated with stroke, alcoholism, drug abuse, sedation, and periodontal disease. Am Rev Resp Dis 1993;148:1418. Clin Infect Dis 1998;27:566. Clin Infect Dis 1998;26:811. Lancet 1998;352:1295. Infect Dis Clin North Am 1998;12:689. Can Respir J 1999;6(Suppl A):15.</td>
</tr>
</tbody>
</table>
### Anaerobic Pneumonia/Lung Abscess

**Usually polymicrobial:** bacteroides sp (15% *B fragilis*), peptostreptococcus, microaerophilic streptococcus, veillonella, *S aureus*, *P aeruginosa*, type 3 *S pneumoniae* (rare), klebsiella (rare).

- Sputum Gram stain and culture for anaerobes are of little value because of contaminating oral flora.
- Bronchoalveolar sampling (brush or aspirate) for Gram stain will usually make an accurate diagnosis. As contamination is likely with a bronchoscope alone, a Bartlett tube should be used. Percutaneous transthoracic needle aspiration may be useful for culture and for cytology to demonstrate coexistence of an underlying carcinoma. Blood cultures are usually negative.

- Aspiration is the most important background feature of lung abscess. Without clear-cut risk factors such as alcoholism, coma, or seizures, bronchoscopy is often performed to rule out neoplasm. Am J Ment Retard 1995;99:579. J Periodontol 1996;67:1114. Curr Opin Pulm Med 1997;3:120.

### Hospital-Acquired Pneumonia

*P aeruginosa* (GNR), klebsiella (GNR), *S aureus* (GPC), acinetobacter (GNR), Enterobacteriaceae (GNR), *S pneumoniae* (GPDC), *H influenzae* (GNCB), influenza virus, respiratory syncytial virus (RSV), legionella (GNR), oral anaerobes.

- Mendelson’s syndrome (see comments): No organisms initially, then pseudomonas, Enterobacteriaceae, *S aureus*, *S pneumoniae*.

- Sputum Gram stain and culture for bacteria (aerobic and anaerobic) and fungus (if suspected).
- Blood cultures for bacteria are often negative. Endotracheal aspirate or bronchoalveolar sample for bacterial and fungal culture in selected patients.

- Most cases are related to aspiration. Hospital-acquired aspiration pneumonia is associated with intubation and the use of broad-spectrum antibiotics.
<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen/Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pneumonia in the Immunocompromised Host</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Child with HIV infection: Lymphoid interstitial pneumonia (LIP). AIDS: <em>M avium</em> (31%), <em>P carinii</em> (13%), cytomegalovirus (CMV) (11%), <em>H capsulatum</em> (7%), <em>S pneumoniae</em> (GPDC), <em>H influenzae</em> (GNCB), <em>P aeruginosa</em> (GNR), Enterobacteriaceae (GPR), <em>C neoformans</em>, <em>C pseudodiptheriticum</em> (GPR), <em>M tuberculosis</em> (AFB), <em>C immitis</em>, <em>P marneffei</em>, <em>Rhodococcus equi</em> (GPR). Neutropenic: <em>Pseudomonas sp</em> (GNR), klebsiella, enterobacter (GNR), bacteroides sp and other oral anaerobes, legionella, candida, aspergillus, mucor. Transplant recipients: <em>Cytomegalovirus</em> (CMV) (60–70%), <em>P aeruginosa</em> (GNR), <em>S aureus</em> (GPC), <em>S pneumoniae</em> (GPDC), legionella (GNR), respiratory syncytial virus (RSV), influenza virus, <em>P carinii</em>, aspergillus, <em>P boydii</em>, nocardia sp, strongyloides.</td>
<td>Expectorated sputum for Gram stain and bacterial culture, if purulent. Sputum induction or bronchiolar lavage for Giemsa or methenamine silver staining or direct fluorescent antibody (DFA) for <em>P carinii</em> trophozoites or cysts; for mycobacterial, fungal staining and culture, for legionella culture, and for CMV culture. Blood for CMV antigenemia or PCR from transplant patients. Blood or bone marrow fungal culture for histoplasmosis (positive in 50%), coccidioidomycosis (positive in 30%). Blood culture for bacteria. Blood cultures are more frequently positive in HIV-infected patients with bacterial pneumonia and often are the only source where a specific organism is identified; bacteremic patients have higher mortality rates. Histoplasma polysaccharide antigen positive in 90% of AIDS patients with disseminated histoplasmosis; antigen increases ≥2 RIA units with relapse. Immunodiffusion or CIE is useful for screening for, and CF for confirmation of, suspected histoplasmosis or coccidioidomycosis. Serum cryptococcal antigen when pulmonary cryptococcosis is suspected. Serum lactate dehydrogenase (LDH) levels are elevated in 63% and hypoxemia with exercise (<em>Pao2 &lt; 75 mm Hg</em>) occurs in 57% of PCP cases. In PCP, the sensitivities of the various diagnostic tests are: sputum induction 80% (in experienced labs), bronchoscopy with lavage 90–97%, transbronchial biopsy 94–97%. In PCP, chest x-ray may show interstitial (36%) or alveolar (25%) infiltrates or may be normal (39%), particularly if leukopenia is present. Recurrent episodes of bacterial pneumonia are common. Kaposi’s sarcoma of the lung is a common neoplastic process that can imitate infection in homosexual and African HIV-infected patients. J Antimicrob Chemother 1995;36(Suppl B):59. Semin Respir Infect 1996;11:119. Infect Dis Clin North Am 1998;12:781. J Thorac Imaging 1998;13:247. Haematologica 1999;84:71. Clin Infect Dis 1999;28:341.</td>
<td></td>
</tr>
<tr>
<td>Mycobacterial Pneumonia</td>
<td>Sputum for AFB stain and culture. First morning samples are best, and at least three samples are required. Culture systems detect mycobacterial growth in as little as several days to 6 weeks. Bronchoalveolar lavage for AFB stain and culture or gastric washings for AFB culture can be used if sputum tests are negative. Sputum or bronchoalveolar lavage for PCR to MTb available for confirmation of smear positive (99%), less sensitive for smear negative (75%). CT- or ultrasound-guided transthoracic fine-needle aspiration cytology can be used if clinical or radiographic features are nonspecific or if malignancy is suspected.</td>
<td>AFB found on sputum stain do not necessarily make the diagnosis of tuberculosis, because <em>M. kansasii</em> and <em>M. avium-intracellulare</em> look identical. Tuberculosis is very common in HIV-infected patients, in whom the chest x-ray appearance may be atypical and occasionally (4%) may mimic PCP (especially in patients with CD4 cell counts &lt;200/µL). In one study, only 2% of patients sent for sputum induction for PCP had tuberculosis. Consider HIV testing if MTb is diagnosed. Delayed diagnosis of pulmonary tuberculosis is common (up to 20% of cases), especially among patients who are older or who do not have respiratory symptoms. In any patient with suspected tuberculosis, respiratory isolation is required. Chest 1998;114:317. Respiration 1998;65:163. CMAJ 1999;160:1725. Chest Surg Clin North Am 1999;9:227.</td>
</tr>
<tr>
<td>Organism</td>
<td>Specimen/Diagnostic Tests</td>
<td>Comments</td>
</tr>
<tr>
<td>---------------</td>
<td>-------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Empyema</td>
<td>Pleural fluid for cell count (WBCs 25,000–100,000/µL, mostly PMNs), protein (&gt; 50% of serum), glucose (&lt; serum, often very low), pH (&lt;7.20), LDH (&gt;60% of serum). (See Pleural fluid profiles, p 382.) Blood cultures for bacteria. Sputum for Gram stain and bacterial culture. Special culture can also be performed for legionella when suspected. Pleural fluid for Gram stain and bacterial culture (aerobic and anaerobic).</td>
<td>Chest tube drainage is paramount. The clinical presentation of empyema is nonspecific. Chest CT with contrast is helpful in demonstrating pleural fluid accumulations due to mediastinal or subdiaphragmatic processes and can identify loculated effusions, bronchopleural fistulae, and lung abscesses. About 25% of cases result from trauma or surgery. Bronchoscopy is indicated when the infection is unexplained. Occasionally, multiple thoracenteses may be needed to diagnose empyema. Curr Opin Pulm Med 1998;4:185. Clin Chest Med 1998;19:363. Semin Respir Infect 1999;14:18. Semin Respir Infect 1999;14:82.</td>
</tr>
</tbody>
</table>

Neonate: E coli (GNR), group A or B streptococcus (GPC), S aureus (GPC), pseudomonas sp (GNR). Infant/child (<5 years): S aureus (GPC), S pneumoniae (GPC), H influenzae (GNCB), anaerobes. Child (>5 years)/adult, Acute: S pneumoniae (GPC), group A streptococcus (GPC), S aureus (GPC), H influenzae (GNCB), legionella. Child (>5 years)/adult, chronic: Anaerobic streptococci, bacteroides sp, prevotella sp, porphyromonas sp, fusobacterium sp, Enterobacteriaceae, E coli, Klebsiella pneumoniae, M tuberculosis.
<table>
<thead>
<tr>
<th>Pericarditis</th>
<th>Tuberculous Pericarditis</th>
</tr>
</thead>
</table>
| **Viruses:** Enteroviruses (coxsackie, echo), influenza, Epstein-Barr, herpes zoster, mumps, HIV, CMV.  
**Bacteria:** *S. aureus* (GPC), *S. pneumoniae* (GPC), mycoplasma, *S. pyogenes* (GPC), Enterobacteriaceae (GNR), *N. meningitidis* (GNDC).  
**Fungi:** Candida (immunocompromised) | **PPD skin testing should be performed (negative in a sizable minority).**  
**Pericardial fluid obtained by needle aspiration can show AFB by smear (rare) or culture (low yield). The yield is improved by obtaining three or four repeated specimens for smear and culture.**  
**Pericardial biopsy for culture and histologic examination has highest diagnostic yield.**  
**Other sources of culture for MTb besides pericardium are available in 50% of patients.**  
**Pericardial fluid may show markedly elevated levels of adenosine deaminase.** |
| In acute pericarditis, specific bacterial diagnosis is made in only 19%.  
**Pericardial fluid aspirate for Gram stain and bacterial culture (aerobic and anaerobic).** In acute pericarditis, only 54% have pericardial effusions.  
**Blood for buffy coat, stool or throat for enteroviral culture.** PCR available in reference laboratories.  
**Surgical pericardial drainage with biopsy of pericardium for culture (22%) and histologic examination.**  
**Paired sera for enterovirus (coxsackie) and mycoplasma.** | Viral pericarditis is usually diagnosed clinically (precordial pain, muffled heart sounds, pericardial friction rub, cardiomegaly). The diagnosis is rarely aided by microbiologic tests.  
**CT and MRI may demonstrate pericardial thickening.**  
**Bacterial pericarditis is usually secondary to surgery, immunosuppression (including HIV), esophageal rupture, endocarditis with ruptured ring abscess, extension from lung abscess, aspiration pneumonia or empyema, or sepsis with pericarditis.**  
| **Spread from nearby caseous mediastinal lymph nodes or pleurisy is the most common route of infection. Acutely, serofibrinous pericardial effusion develops with substernal pain, fever, and friction rub. Tamponade may occur.**  
**Tuberculosis accounts for 4% of cases of acute pericarditis, 7% of cases of cardiac tamponade, and 6% of cases of constrictive pericarditis.**  
**One-third to one-half of patients develop constrictive pericarditis despite drug therapy. Constrictive pericarditis occurs 2–4 years after acute infection.**  
<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious Myocarditis</td>
<td>Endomyocardial biopsy for pathologic examination, PCR, and culture in selected cases. Indium-111 antimyosin antibody imaging is more sensitive than endomyocardial biopsy. Stool or throat swab for enterovirus culture. Blood for enterovirus PCR (reference labs) and culture of white cells. Paired sera for coxsackie B, Mycoplasma pneumoniae, Chlamydia pneumoniae, C diphtheriae (GPR), Trichinella spiralis (trichinosis), Trypanosoma cruzi (Chagas’ disease), toxoplasma.</td>
<td>Acute infectious myocarditis should be suspected in a patient with dynamically evolving changes in ECG, echocardiography, and serum CK levels and symptoms of an infection. The value of endomyocardial biopsy in such cases has not been established. In contrast, an endomyocardial biopsy is needed to diagnose lymphocytic or giant cell myocarditis. The incidence of myocarditis in AIDS may be as high as 46%. Many patients with acute myocarditis progress to dilated cardiomyopathy.</td>
</tr>
</tbody>
</table>

Enteroviruses (especially coxsackie B), adenovirus, influenza virus, HIV, *Borrelia burgdorferi* (Lyme disease), scrub typhus, *Rickettsia rickettsii* (Rocky Mountain spotted fever), *Coxiella burnetii* (Q fever), Mycoplasma pneumoniae, Chlamydia pneumoniae, C diphtheriae (GPR), Trichinella spiralis (trichinosis), Trypanosoma cruzi (Chagas’ disease), toxoplasma.

**Infective Endocarditis**

<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prosthetic Valve Infective Endocarditis (PVE)</td>
<td>Blood cultures for bacteria and yeast. Three sets of blood cultures are sufficient in 97% of cases. Draw before temperature spike. While more invasive, transesophageal echocardiography is superior in predicting which patients with infective endocarditis have perivalvular abscess or prosthetic valve dysfunction and which are most susceptible to systemic embolism.</td>
<td>In a large series using perioperative prophylaxis, the incidences of early-onset and late-onset prosthetic valve endocarditis were 0.78% and 1.1%, respectively. The portals of entry of early-onset PVE are intraoperative contamination and postoperative wound infections. The portals of entry of late-onset PVE appear to be the same as those of native valve endocarditis, and the microbiologic profiles are also similar. Clinically, patients with late-onset PVE resemble those with native valve disease. However, those with early-onset infection are often critically ill, more often have other complicating problems, are more likely to go into shock and are more likely to have conduction abnormalities due to ring abscess. Medicine (Baltimore) 1997;76:94. Clin Infect Dis 1997;24:884. J Infect 1999;39:27.</td>
</tr>
<tr>
<td>Infectious Thrombophlebitis</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Associated with venous catheters:</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>S. aureus</em> (GPC) (65–78%),</td>
<td></td>
<td></td>
</tr>
<tr>
<td>coagulase-negative staphylococci (GPC), candida sp (yeast),</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pseudomonas sp (GNR), Enterobacteriaceae (GNR).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperalimentation with catheter:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indwelling venous catheter (e.g., Broviac, Hickman, Gershorn):</td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>S. aureus</em>, coagulase-negative staphylococci, diphtheroids (GPR), pseudomonas sp, Enterobacteriaceae, candida sp.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postpartum or post-abortion pelvic thrombophlebitis: Bacteroides (GNR), Enterobacteriaceae, clostridium (GPR), streptococcus (GPC).</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Blood cultures for bacteria (positive in 80–90%). Catheter tip for bacterial culture to document etiology. More than 15 colonies (CFUs) suggests colonization or infection. CT and MRI are the studies of choice in the evaluation of puerperal septic pelvic thrombophlebitis. Blood cultures for bacteria (positive in 80–90%). Catheter tip for bacterial culture to document etiology. More than 15 colonies (CFUs) suggests colonization or infection. CT and MRI are the studies of choice in the evaluation of puerperal septic pelvic thrombophlebitis. 

<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Helicobacter pylori</em></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Infectious Esophagitis</td>
<td>Barium esophagram reveals abnormalities in the majority of cases of candidal esophagitis. Endoscopy with biopsy and brushings for culture and cytology has the highest diagnostic yield (57%) and should be performed if clinically indicated or if empiric antifungal therapy is unsuccessful.</td>
<td>Thrush and odynophagia in an immunocompromised patient warrants empiric therapy for candida. Factors predisposing to infectious esophagitis include HIV infection, exposure to radiation, cytotoxic chemotherapy, recent antibiotic therapy, corticosteroid therapy, and neutropenia. Gastrointest Endosc 1996;44:587. Med Pediatr Oncol 1997;28:299. Am J Gastroenterol 1998;93:394, 2239. Am J Gastroenterol 1999;94:339.</td>
</tr>
<tr>
<td><em>Candida sp</em> (yeast), herpes simplex (HSV), cytomegalovirus (CMV), varicella-zoster (VZV), <em>Helicobacter pylori</em> (GNR), cryptosporidium. (Rare causes: <em>Mycobacterium tuberculosis</em> [AFB], aspergillus, histoplasma, blastomycetes, HIV).</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Infectious Colitis/Dysentery</strong></td>
<td><strong>Stool for occult blood helpful to diagnosis of <em>E coli</em> O157:H7, salmonella, <em>E histolytica</em>.</strong> Stool collected culture, ova and parasites examination. Two samples are often needed. The sensitivity of the stool culture is only 72%, but its specificity is 100% (using PCR as standard). One repeat culture may increase sensitivity. Special culture technique is needed for <em>yersinia</em>, <em>E coli</em>, and vibrio. Cultures for salmonella or shigella are not helpful in patients hospitalized more than 72 hours. Proctosigmoidoscopy is indicated in patients with chronic or recurrent diarrhea or in diarrhea of unknown cause for smears of aspirates (may show organisms) and biopsy. Cultures of biopsy specimens have somewhat higher sensitivities than stool cultures. Rectal and jejunal biopsies may be necessary in HIV-infected patients. Need modified acid-fast stain for cryptosporidium. Immunodiagnosis of <em>G lamblia</em>, cryptosporidium, or <em>E histolytica</em> cysts in stool is highly sensitive and specific. <strong>Acute dysentery is diarrhea with bloody, mucoid stools, tenesmus, and pain on defecation and implies an inflammatory invasion of the colonic mucosa. BUN and serum electrolytes may be indicated for supportive care. Severe dehydration is a medical emergency. Necrotizing enterocolitis is a fulminant disease of premature newborns; cause is unknown but human breast milk is protective. Air in the intestinal wall (pneumatosis intestinalis), in the portal venous system, or in the peritoneal cavity seen on plain x-ray can confirm diagnosis. 30–50% of these infants will have bacteremia or peritonitis. Risk factors for infectious colitis include poor hygiene and immune compromise (infancy, advanced age, corticosteroid or immunosuppressive therapy, HIV infection).</strong></td>
<td></td>
</tr>
<tr>
<td>Infant: <em>E coli</em> (enteropathogenic). Child/Adult without travel, afebrile, no gross blood or WBCs in stool: Rotavirus, caliciviruses (eg, Norwalk agent), <em>E coli</em> (GNR). Child/Adult with fever, bloody stool or history of travel to subtropics/tropics (varies with epidemiology): <em>Campylobacter jejuni</em> (GNR), <em>E coli</em> (GNR), (enterotoxigenic, enteroinvasive, enterohemorrhagic O157:H7), shigella (GNR), salmonella (GNR), <em>Yersinia enterocolitica</em> (GPR), <em>Clostridium difficile</em> (GPR), aeromonas (GNR), vibrio (GNR), cryptosporidium, Entamoeba histolytica, Giardia lamblia, cyclospora, strongyloides, edwardsiella (GNR). Child/Adult with vomiting and no fever: <em>S aureus</em> (GPC), <em>Bacillus cereus</em> (GPR).</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ABDOMEN</strong></td>
<td><strong>Infectious Colitis/Dysentery</strong></td>
<td></td>
</tr>
<tr>
<td>Microbiology: Test Selection</td>
<td>223</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Antibiotic-Associated Pseudomembranous Colitis</strong>&lt;br&gt; <em>Clostridium difficile</em> (GPR) toxin, <em>Clostridium perfringens</em> (GPR), <em>Staphylococcus aureus</em> (GPC), <em>Klebsiella oxytoca</em> (GNR).</td>
<td>Send stool for <em>C. difficile</em>, cytotoxin A by tissue culture or toxin A or A and B by less sensitive immuno-assay. Testing two stools on different days will increase sensitivity; toxin testing for test-of-cure is not recommended. Fecal WBCs are present in 30–50% of cases. The toxin is very labile and can be present in infants with no disease. Stool culture is not recommended because non-toxigenic strains occur. Colonoscopy and visualization of characteristic 1–5 mm raised yellow plaques provides the most rapid diagnosis. However, an ultrasound appearance of grossly thickened bowel wall with luminal narrowing or CT findings of thickened bowel wall, presence of an “accordion” sign, heterogeneous contrast enhancement pattern (“target sign”), pericolic stranding, ascites, pleural effusion, and subcutaneous edema can suggest the diagnosis of pseudomembranous colitis.</td>
<td>Antibiotics cause changes in normal intestinal flora, allowing overgrowth of <em>C. difficile</em> and elaboration of toxin. Other risk factors for <em>C. difficile</em>-induced colitis are GI manipulations, advanced age, female sex, inflammatory bowel disease, HIV, chemotherapy, and renal disease. <em>C. difficile</em> nosocomial infection can be controlled by handwashing. Antibiotic-associated diarrhea may include uncomplicated diarrhea, colitis, or pseudomembranous colitis. Only 10–20% of cases are caused by infection with <em>C. difficile</em>. Most clinically mild cases are due to functional disturbances of intestinal carbohydrate or bile acid metabolism, to allergic and toxic effects of antibiotics on intestinal mucosa, or to their pharmacologic effects on motility. Radiolog 1996;198:1.&lt;br&gt;Clin Infect Dis 1998;27:702.&lt;br&gt;Dig Dis 1998;16:292.&lt;br&gt;Dis Colon Rectum 1998;41:1435.&lt;br&gt;J Antimicrob Chemother 1998;41 (Suppl C):29, 59.&lt;br&gt;Digestion 1999;60:91.&lt;br&gt;Hepatogastroenterology 1999;46:343.</td>
</tr>
</tbody>
</table>
### Diarrhea in the HIV-Infected Host

Same as Child-Adult Infectious Colitis with addition of cyto
tomegalovirus, cryptosporidium, *Isospora belli*, microsporidia
(*Enterocytozoon bieneusi*), *C difficile*, *Giardia intestinalis*,
*Mycobacterium avium-intracellularare* complex (AFB),
herpes simplex (HSV). *Ent-
amoeba histolytica*, HIV.

| Test Selection | Stool for stain for fecal leukocytes, culture (especially for salmonella, shigella, yersinia, and campylobac
ter), *C difficile* toxin, ova and parasite examination, and AFB smear. Multiple samples are often needed. Proctosigmoidoscopy with fluid aspiration and biopsy is indicated in patients with chronic or recurrent diarrhea or in diarrhea of unknown cause for smears of aspirates (may show organisms) and histologic examination and culture of tissue. Rectal and jejunal biopsies may be necessary, especially in patients with tenesmus or bloody stools. Need modified acid-fast stain for cryptosporidium. Intranuclear inclusion bodies on histologic exam suggest CMV. Immunodiagnosis of giardia, cryptosporidium, and *E histolytchal* cysts in stool is highly sensitive and specific. | Most patients with HIV infection will develop diarrhea at some point in their illness. Cryptosporidium causes a chronic debilitating diarrheal infection that rarely remits spontaneously and is still without effective treatment. Diarrhea seems to be the result of malabsorption and produces a cholera-like syndrome. Between 15% and 50% of HIV-infected patients with diarrhea have no identifiable pathogen. J Clin Microbiol 1995;33:745. Gastroenterology 1996;111:1724. Gastrointest Endosc Clin North Am 1998;8:857. J Infect Dis 1999;179(Suppl 3):S454. Am J Gastroenterol 1999;94:596. Arch Intern Med 1999;159:1473. |
<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peritonitis</td>
<td>Peritoneal fluid sent for WBC (&gt;1000/µL in SPB, &gt;100/µL in CAPD) with PMN (≥250/µL SBP and secondary peritonitis, 50% PMN in CAPD); total protein (&gt;1 g/dL); glucose (&lt;50 mg/dL) and lactate dehydrogenase (&gt;225 units/mL) in secondary; pH (&lt;7.35 in 57% for SBP). Gram stain (22–77% for SBP), and culture of large volumes often in blood culture bottles. (See Ascitic fluid profiles, p 365.) Blood cultures for bacteria positive in 75% of SBP cases. Catheter-related infection is associated with a WBC &gt;500/µL.</td>
<td>In nephrotic patients, Enterobacteriaceae and <em>S. aureus</em> are most frequent. In cirrhotics, 69% of cases are due to Enterobacteriaceae. Cirrhotic patients with low ascitic fluid protein levels (≤1 g/dL) and high bilirubin level or low platelet count are at high risk of developing spontaneous bacterial peritonitis. “Bacterascites,” a positive ascitic fluid culture without an elevated PMN count, is seen in 8% of cases of SBP and probably represents early infection. In secondary peritonitis, factors influencing the incidence of postoperative complications and death include age, presence of certain concomitant diseases, site of origin of peritonitis, type of admission, and the ability of the surgeon to eliminate the source of infection. Clin Infect Dis 1998;27:669. Eur J Clin Microbiol Infect Dis 1998;17:542. J Am Soc Nephrol 1998;9:1956. Langenbecks Arch Surg 1999;384:24. Gastroenterology 1999;117:414.</td>
</tr>
</tbody>
</table>
### Tuberculous Peritonitis/Enterocolitis

*Mycobacterium tuberculosis* (MTb, AFB, acid-fast beaded rods).

**Ascitic fluid for appearance (clear, hemorrhagic or chylous), RBCs (can be high), WBCs (>1000/µL, >70% lymphs), protein (>2.5 g/dL), serum/ascites albumin gradient (<1.1), LDH (>90 units/L), AFB culture (<50% positive). (See Ascitic fluid profiles, p 365.) With coexistent chronic liver disease, protein level and SAAG are usually not helpful, but LDH >90 units/L is a useful predictor.**

Culture or AFB smear from other sources (especially from respiratory tract) can help confirm diagnosis.

Abdominal ultrasound may demonstrate free or loculated intra-abdominal fluid, intra-abdominal abscess, ileocecal mass, and retroperitoneal lymphadenopathy. Ascites with fine, mobile septations shown by ultrasound and peritoneal and omental thickening detected by CT strongly suggest tuberculous peritonitis.

Marked elevations of serum CA 125 have been noted; levels decline to normal with antituberculous therapy.

Diagnosis of enterocolitis rests on biopsy of colonic lesions via endoscopy if pulmonary or other extrapulmonary infection cannot be documented. Diagnosis is best confirmed by laparoscopy with peritoneal biopsy and culture. Operative procedure may be needed to relieve obstruction or for diagnosis.

**Infection of the intestines can occur anywhere along the GI tract but occurs most commonly in the ileocecal area or mesenteric lymph nodes. It often complicates pulmonary infection. Peritoneal infection usually is an extension of intestinal disease. Symptoms may be minimal even with extensive disease. In the US, 29% of patients with abdominal tuberculosis have a normal chest x-ray. Presence of AFB in the feces does not correlate with intestinal involvement.**

<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Diverticulitis</strong></td>
<td>Identification of organism is not usually sought. Ultrasoundography (US) or flat and upright x-rays of abdomen are crucial to rule out perforation (free air under diaphragm) and to localize abscess (air-fluid collections). Barium enema can (82%) show presence of diverticula. Avoid enemas in acute disease because increased intraluminal pressure may cause perforation. Ultrasound (85%) and CT (79–98%) have greater accuracy in the evaluation of patients with diverticulitis. Specificities of barium enema, ultrasound, and CT are 81–84%. Thin-section helical CT is also able to reveal inflamed diverticula in acute diverticulitis by demonstrating an enhancing pattern of the colonic wall. Urinalysis will reveal urinary tract involvement, if present.</td>
<td>Pain usually is localized to the left lower quadrant because the sigmoid and descending colon are the most common sites for diverticula. It is important to rule out other abdominal disease (e.g., colon carcinoma, Crohn’s disease, ischemic colitis). Acta Radiologica 1997;38:313. Radiology 1997;205:503. Dis Colon Rect 1998;41:1023. Radiology 1998;208:611. N Engl J Med 1998;338:1521. AJR Am J Roentgenol 1999;172:601. Surg Endosc 1999;13:430.</td>
</tr>
<tr>
<td><strong>Liver Abscess</strong></td>
<td>CT scan with contrast and ultrasonography are the most accurate tests for the diagnosis of liver abscess. Antibodies against <em>E histolytica</em> should be obtained on all patients. (See Amebic serology, p 50.) Complete removal of abscess material obtained via surgery or percutaneous aspiration is recommended for culture and direct examination for <em>E histolytica</em>. <em>E histolytica</em> has been described with modern techniques as a complex of two species, the commensal parasite <em>E dispar</em> and the pathogenic parasite. Stool for antigen detection is sensitive and can distinguish the two species. Chest x-ray is often useful with raised hemidiaphragm, right pleural effusion, or right basilar atelectasis in 41% of patients. Elevation of serum alkaline phosphatase level in 78%. Travel to and origin in an endemic area are important risk factors for amebic liver abscess. 60% of patients have a single lesion; 40% have multiple lesions. Biliary tract disease is the most common underlying disease, followed by malignancy (biliary tract or pancreatic), colonic disease (diverticulitis), diabetes mellitus, liver disease, and alcoholism. Clin Radiol 1997;52:912. South Med J 1997;90:23. Ann Emerg Med 1999;34:351. World J Surg 1999;23:102. West J Med 1999;170:104.</td>
<td></td>
</tr>
<tr>
<td>Cholangitis/Cholecystitis</td>
<td>Ultrasonography is the best test to quickly demonstrate gallstones or phlegmon around the gallbladder or dilation of the biliary tree. (See Abdominal Ultrasound, p 258.) CT scanning is useful in cholangitis in detecting the site and cause of obstruction but may fail to detect stones in the common bile duct. In acute cholecystitis, ultrasonography is superior to MR cholangiography in evaluating gallbladder wall thickening. However, MR cholangiography is superior to ultrasound in depicting cystic duct and gallbladder neck stones and in evaluating cystic duct obstruction. Radionuclide scans can demonstrate cystic duct obstruction. (See p 266.) Blood cultures for bacteria.</td>
<td>90% of cases of acute cholecystitis are calculous, 10% are acalculous. Risk factors for acalculous disease include prolonged illness, fasting, hyper-alimentation, HIV infection, and carcinoma of the gallbladder or bile ducts. Biliary obstruction and cholangitis can develop before biliary dilation is detected. Common bile duct obstruction secondary to tumor or pancreatitis seldom results in infection (0–15%). There is a high incidence of acalculous cholecystitis in AIDS patients with CD4 counts &lt; 200/µL, due to cryptosporidium, cytomegalovirus, yeast, tuberculosis, and <em>Mycobacterium avium-intracellulare</em>. Observation of gallbladder contraction on hepatobiliary scintigraphy after intravenous cholecystokinin excludes acalculous cholecystitis. Radiology 1998;209:781. Mayo Clin Proc 1998;73:473, 479. Radiology 1999;211:373.</td>
</tr>
<tr>
<td>--------------------------</td>
<td>---------------------------------------------------------------------------------</td>
<td>--------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Enterobacteriaceae (GNR) (68%), enterococcus (GPC in chains) (14%), <em>Pseudomonas aeruginosa</em> (GNR), bacteroides (GNR) (10%), clostridium sp (GPR) (7%), microsporidia (<em>Enterocyttozoon bieneusi</em>), <em>Ascaris lumbricoides</em>, <em>Opisthorchis viverrini</em>, <em>O felineus</em>, <em>Clonorchis sinensis</em>, <em>Fasciola hepatica</em>, <em>Echinococcus granulosus</em>, <em>E multilocularis</em>, hepatitis C virus, hepatitis B virus, cytomegalovirus.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Organism</td>
<td>Specimen / Diagnostic Tests</td>
<td>Comments</td>
</tr>
<tr>
<td>----------</td>
<td>----------------------------</td>
<td>----------</td>
</tr>
<tr>
<td>Urinary Tract Infection (UTI)/Cystitis/Pyuria-Dysuria Syndrome</td>
<td>Urinalysis and culture reveal the two most important signs: bacteriuria and pyuria (&gt;10 WBCs/µL). 30% of patients have hematuria. Cystitis (95%) is diagnosed by ≥10^2 CFU/mL of bacteria; other urinary infections (90%) by ≥10^5 CFU/mL. Culture is generally not necessary for uncomplicated cystitis in women. However, pregnant women should be screened for asymptomatic bacteriuria and promptly treated. Both Gram stain for bacteria and dipstick analysis for nitrite and leukocyte esterase perform similarly in detecting UTI in children and are superior to microscopic analysis for pyuria. Intravenous pyelogram and cystoscopy should be performed in women with recurrent or childhood infections, all young boys with UTI, men with recurrent or complicated infection, and patients with symptoms suggestive of obstruction or renal stones. (See Intravenous pyelogram, p 273.)</td>
<td>Most men with urinary tract infections have a functional or anatomic genitourinary abnormality. In catheter-related UTI, cure is unlikely unless the catheter is removed. In asymptomatic catheter-related UTI, antibiotics should be given only if patients are at risk for sepsis (old age, underlying disease, diabetes mellitus, pregnancy). Up to one-third of cases of acute cystitis have “silent” upper tract involvement. Infect Dis Clin North Am 1997;11:13 Infect Dis Clin North Am 1997;11:609. Pediatrics 1999;103(4 Part 1):843. Urol Clin North Am 1999;26:821. Nephrol Dial Transplant 1999;14:2746. Am J Med 1999;106:636. Postgrad Med 1999;105:181. BMJ 1999;318:770.</td>
</tr>
<tr>
<td>Enterobacteriaceae (GNR, especially E coli), Chlamydia trachomatis, Staphylococcus saprophyticus (GPC) (in young women), enterococcus (GPC), candida sp (yeast), N gonorrhoeae (GNCB), HSV, adeno-virus, Corynebacterium glucuronolyticum (GPR), Urea plasma, urealyticum (GPR).</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prostatitis</td>
<td>Urinalysis shows pyuria. Urine culture usually identifies causative organism. Prostatic massage is useful in chronic prostatitis to retrieve organisms but is contraindicated in acute prostatitis (it may cause bacteremia). Bacteriuria is first cleared by antibiotic treatment. Then urine cultures are obtained from first-void, bladder, and post-prostatic massage urine specimens. A higher organism count in the post-prostatic massage specimen localizes infection to the prostate (91%).</td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>Acute and Chronic: Enterobacteriaceae (GNR), pseudomonas sp (GNR), enterococcus (GPC in chains), cytomegalovirus (CMV). Acute prostatitis is a severe illness characterized by fever, dysuria, and a boggy or tender prostate. Chronic prostatitis often has no symptoms of dysuria or perineal discomfort and a normal prostate examination. Nonbacterial prostatitis (prostatodynia) represents 90% of prostatitis cases. Its etiology is unknown, although chlamydia antigen can be found in up to 25% of patients.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Organism</td>
<td>Specimen/Diagnostic Tests</td>
<td>Comments</td>
</tr>
<tr>
<td>------------------</td>
<td>--------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td><strong>Perinephric Abscess</strong></td>
<td>Associated with staphylococcal bacteremia: <em>Staphylococcus aureus</em> (GPC). Associated with pyelonephritis: Enterobacteriaceae (GNR), candida sp (yeast), coagulase-negative staphylococci (GPC).</td>
<td>CT scan with contrast is more sensitive than ultrasound in imaging abscess and confirming diagnosis. (See Abdominal CT, p 259.) Urinalysis may be normal or may show pyuria. Urine culture (positive in 60%). Blood cultures for bacteria (positive in 20–40%). Bacterial culture of abscess fluid via needle aspiration or drainage (percutaneous or surgical). Most perinephric abscesses are the result of extension of an ascending urinary tract infection. Often they are very difficult to diagnose. They should be considered in patients who fail to respond to antibiotic therapy, in patients with anatomic abnormalities of the urinary tract, and in patients with diabetes mellitus. Hosp Pract (Off Ed) 1997;32(6):40. Infect Dis Clin North Am 1997;11:663.</td>
</tr>
</tbody>
</table>
Urethritis (Gonococcal and Nongonococcal)

Gonococcal (GC): *Neisseria gonorrhoeae* (GNDC).
Nongonococcal (NGU): *Chlamydia trachomatis* (50%), *Ureaplasma urealyticum*, *Trichomonas vaginalis*, herpes simplex (HSV), *Mycoplasma genitalium*, unknown (35%).

Urethritis (Gonococcal and Nongonococcal)

Gonococcal (GC): *Neisseria gonorrhoeae* (GNDC).
Nongonococcal (NGU): *Chlamydia trachomatis* (50%), *Ureaplasma urealyticum*, *Trichomonas vaginalis*, herpes simplex (HSV), *Mycoplasma genitalium*, unknown (35%).

Epididymitis/Orchitis

Age <35 years, homosexual men: *Chlamydia trachomatis, N gonorrhoeae* (GNDC).
Age >35 years, or children: Enterobacteriaceae (especially *E coli*) (GNR), pseudomonas sp (GNR), salmonella (*GNR, Haemophilus influenzae* (GNCB), varicella (VZV), mumps.

Immunosuppression: *H influenzae, Mycobacterium tuberculosis* (AFB), candida sp (yeast), cytomegalovirus (CMV).

Urethral discharge collected with urethral swab usually shows ≥4 WBCs per oil immersion field, Gram stain (identify gonococcal organisms as gram-negative intracellular diplococci), PMNs (in GC, >95% of WBCs are PMNs, in NGU usually <80% are PMNs).

Urethral discharge for culture (80%) or nucleic acid assay (97%) for GC (usually not needed for diagnosis); urine (80–92%) or urethral discharge (97%) for detection of *C trachomatis* by nucleic acid amplification or wet mount for *T vaginalis*. Culture or nonamplified assays are considerably less sensitive for diagnosis of *C trachomatis*. VDRL should be checked in all patients because of high incidence of associated syphilis.

Testicular torsion is a surgical emergency that is often confused with orchitis or epididymitis. Sexual partners should be examined for signs of sexually transmitted diseases. In non-sexually transmitted disease, evaluation for underlying urinary tract infection or structural defect is recommended.

About 50% of patients with GC will have concomitant NGU infection.
Always treat sexual partners. Recurrence may be secondary to failure to treat partners. Half of the cases of nongonococcal urethritis (NGU) are not due to *Chlamydia trachomatis*; frequently, no pathogen can be isolated.
Persistent or recurrent episodes with adequate treatment of patient and partners may warrant further evaluation for other causes (eg, prostatitis).

Urine analysis may reveal pyuria. Patients aged >35 years will often have midstream pyuria and scrotal edema.
Culture urine and expressible urethral discharge when present.
Prostatic secretions for Gram stain and bacterial culture are helpful in older patients.
When testicular torsion is considered, Doppler ultrasound or radionuclide scan can be useful in diagnosis.

Ultrasoundography in tuberculous epididymitis shows enlargement of the epididymis (predominantly in the tail) and marked heterogeneity in texture. Other sonographic findings include a hypoechoic lesion of the testis with associated sinus tract or extratesticular calcifications.

About 50% of patients with GC will have concomitant NGU infection.
Always treat sexual partners. Recurrence may be secondary to failure to treat partners. Half of the cases of nongonococcal urethritis (NGU) are not due to *Chlamydia trachomatis*; frequently, no pathogen can be isolated.
Persistent or recurrent episodes with adequate treatment of patient and partners may warrant further evaluation for other causes (eg, prostatitis).

Urine analysis may reveal pyuria. Patients aged >35 years will often have midstream pyuria and scrotal edema.
Culture urine and expressible urethral discharge when present.
Prostatic secretions for Gram stain and bacterial culture are helpful in older patients.
When testicular torsion is considered, Doppler ultrasound or radionuclide scan can be useful in diagnosis.

Ultrasoundography in tuberculous epididymitis shows enlargement of the epididymis (predominantly in the tail) and marked heterogeneity in texture. Other sonographic findings include a hypoechoic lesion of the testis with associated sinus tract or extratesticular calcifications.

About 50% of patients with GC will have concomitant NGU infection.
Always treat sexual partners. Recurrence may be secondary to failure to treat partners. Half of the cases of nongonococcal urethritis (NGU) are not due to *Chlamydia trachomatis*; frequently, no pathogen can be isolated.
Persistent or recurrent episodes with adequate treatment of patient and partners may warrant further evaluation for other causes (eg, prostatitis).

Urine analysis may reveal pyuria. Patients aged >35 years will often have midstream pyuria and scrotal edema.
Culture urine and expressible urethral discharge when present.
Prostatic secretions for Gram stain and bacterial culture are helpful in older patients.
When testicular torsion is considered, Doppler ultrasound or radionuclide scan can be useful in diagnosis.

Ultrasoundography in tuberculous epididymitis shows enlargement of the epididymis (predominantly in the tail) and marked heterogeneity in texture. Other sonographic findings include a hypoechoic lesion of the testis with associated sinus tract or extratesticular calcifications.
<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaginitis/Vaginosis</td>
<td>Vaginal discharge for appearance (in candidiasis, area is pruritic with thick “cheesy” discharge: in trichomoniasis, copious foamy discharge), pH (about 4.5 for candida; 5.0–7.0 in trichomonas; 5.0–6.0 with bacterial), saline (“wet”) preparation (motile organisms seen in trichomonas; cells covered with organisms—“clue” cells—in gardnerella; yeast and hyphae in candida, “fishy” odor on addition of KOH with gardnerella infection). Vaginal fluid pH as a screening test for bacterial vaginosis showed a sensitivity of 74.3%, but combined with clinical symptoms and signs its sensitivity increased to 81.3%.</td>
<td>Bacterial vaginosis results from massive overgrowth of anaerobic vaginal bacterial flora (especially gardnerella). Serious infectious sequelae associated with bacterial vaginosis include abscesses, endometritis and pelvic inflammatory disease. There is also a danger of miscarriage, premature rupture of the membranes, and premature labor.</td>
</tr>
</tbody>
</table>

Candida sp, *Trichomonas vaginalis*, *Gardnerella vaginalis* (GNR), bacteroides (non-fragilis (GPR), mobiluncus (GPR), peptostreptococcus (GPC), *Mycoplasma hominis*, groups A and B streptococci (GPC), herpes simplex (HSV).

Atrophic vaginitis is seen in postmenopausal patients, often with bleeding, scant discharge, and pH 6.0–7.0

Cultures for gardnerella are not useful and are not recommended. Culture for *T vaginalis* has greater sensitivity than wet mount. Culture for groups A and B streptococci and rare causes of bacterial vaginosis may be indicated.

| Cervicitis, Mucopurulent | Cervical swab specimen for appearance (yellow or green purulent material), cell count (>10 WBCs per high-power oil immersion field and culture (58–80%) or nucleic acid assay (93%) for GC; urine for nucleic acid assay (93%) for GC; urine (80–92%) or cervical swab (97%) for detection of *C trachomatis* by nucleic acid amplification. Culture (52%) or nonamplified assays (50–80%) are considerably less sensitive for diagnosis of *C trachomatis*. | Because of the danger of false-positive amplified nucleic acid assays, culture is the preferred method in cases of suspected child abuse. In one study of pregnant women, a wet mount preparation of endocervical secretions with <10 PMNs per high-power field had a negative predictive value of 99% for gonococcus-induced cervicitis and of 96% for *C trachomatis*-induced cervicitis. In family planning clinics, however, a mucopurulent discharge with >10 PMNs/hpf had a low positive predictive value of 29.2% for *C trachomatis*-related cervicitis. Mucopurulent discharge may persist for 3 months or more even after appropriate therapy. Curr Probl Dermatol 1996;24:110. CMAJ 1998;158:41. J Clin Microbiol 1998;36:1630. Am J Obstet Gynecol 1999;181:283. Eur J Clin Microbiol Infect Dis 1999;18:142. |
## Organism | Specimen/Diagnostic Tests | Comments |
--- | --- | --- |
**Salpingitis/Pelvic Inflammatory Disease (PID)**


- Gram stain and culture or amplified nucleic acid assays of urethral or endocervical exudate.
- Ultrasonographic findings include thickened fluid-filled tubes, polycystic-like ovaries, and free pelvic fluid. MRI imaging findings for PID (95%) include fluid-filled tube, pyosalpinx, tubo-ovarian abscess, or polycystic-like ovaries and free fluid.
- Laparoscopy supplemented by microbiologic tests and fimbrial biopsy is the diagnostic standard for PID. Transvaginal ultrasonography (81%) has a lower specificity than MRI.
- Laparoscopy is the most specific test to confirm the diagnosis of PID.
- VDRL should be checked in all patients because of the high incidence of associated syphilis.

- PID typically progresses from cervicitis to endometritis to salpingitis. PID is a sexually transmitted disease in some cases, not in others.
- All sexual partners should be examined.
- All IUDs should be removed.
- Some recommend that all patients with PID be hospitalized.

**Chorioamnionitis/Endometritis**

- Group B streptococcus (GPC), *Escherichia coli* (GNR), *Listeria monocytogenes* (GPR), *Mycoplasma hominis*, *Ureaplasma urealyticum*, *Gardnerella vaginalis*, enterococci (GPC), viridans streptococci (GPC), bacteroides (GNR), prevotella (GNR), and other anaerobic flora, *Chlamydia trachomatis*, group A streptococcus (GPC)

- Amniotic fluid for Gram stain, leukocyte esterase, glucose levels <10–20 mg/dL, and aerobic and anaerobic culture; blood for culture. Sonographic evaluation of fetus can be helpful, but findings are nonspecific.

**Osteomyelitis**

*Staphylococcus aureus* (GPC) (about 60% of all cases).  
Infant: *S. aureus*, Enterobacteriaceae (GNR), groups A and B streptococci (GPC).  
Child (>3 years) to Adult: *S. aureus*, *Pseudomonas aeruginosa*.  
Postoperative: *S. aureus*, Enterobacteriaceae, pseudomonas sp (GNR), *Bartonella henselae* (GNR).  
Joint prosthesis: Coagulase-negative staphylococci, peptostreptococcus (GPC), *Propionibacterium acnes* (GPR), viridans streptococci (GPC in chains).

Blood cultures for bacteria are positive in about 60%. Cultures of percutaneous needle biopsy or open bone biopsy are needed if blood cultures are negative and osteomyelitis is suspected. Imaging with bone scan or gallium/indium scan (sensitivity 95%, specificity 60–70%) can localize areas of suspicion. Technetium (99mTc)-Methylene diphosphonate (MDP) bone scan can suggest osteomyelitis days or weeks before plain bone films. Plain bone films are abnormal in acute cases after about 2 weeks of illness (33%). Indium-labeled WBC scan is useful in detecting abscesses. Ultrasound to detect subperiosteal abscesses and ultrasound-guided aspiration can assist in diagnosis and management of osteomyelitis. Ultrasound can differentiate acute osteomyelitis from vaso-occlusive crisis in patients with sickle cell disease. CT scan aids in detecting sequestra. When bone x-rays and scintigraphy are negative, MRI (98%) is useful for detecting early osteomyelitis (specificity 89%), in defining extent, and in distinguishing osteomyelitis from cellulitis. Myelography, CT, or MRI is indicated to rule out epidural abscess in vertebral osteomyelitis.

Hematogenous or contiguous infection (eg, infected prosthetic joint, chronic cutaneous ulcer) may lead to osteomyelitis in children (metaphyses of long bones) or adults (vertebrae, metaphyses of long bones). Hematogenous osteomyelitis in drug addicts occurs in unusual locations (vertebrae, clavicle, ribs). In infants, osteomyelitis is often associated with contiguous joint involvement.  
### Bacterial/Septic Arthritis

**Infant (<3 months):** *S. aureus* (GPC), Enterobacteriaceae (GNR), *Kingella kingae* (GNCB), *Haemophilus influenzae* (GNCB).

Child (3 months to 6 years): *S. aureus* (35%), *H. influenzae*, group A streptococcus (GPC), (10%), Enterobacteriaceae (6%), *Borrelia burgdorferi* (Lyme).

**Adult, STD not likely:** *S. aureus* (40%), group A streptococcus (27%), Enterobacteriaceae (23%), *Streptobacillus moniliformis* (GNR), brucella (GNR), *Mycobacterium marinum* (AFB).

Adult, STD likely: *N. gonorrhoeae* (GNDC) (disseminated gonococcal infection, DGI).

Prosthetic joint, postoperative or following intraarticular injection: Coagulase-negative staphylococci (40%), *S. aureus* (20%), viridans streptococci (GPC in chains), enterococci (GPC), peptostreptococcus (GPC), *Propionibacterium acnes* (GPR), Enterobacteriaceae, pseudomonas sp.

<table>
<thead>
<tr>
<th>Organism</th>
<th>Specimen / Diagnostic Tests</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Joint aspiration (synovial) fluid for WBCs (in nongonococcal infection, mean WBC is 100,000/µL), Gram stain (best on centrifuged concentrated specimen; positive in one-third of cases), culture (non gonococcal infection in adults [85–95%], DGI [25%]).</strong> (See Synovial fluid profiles, p 389.) Yield of culture is greatest if 10 mL of synovial fluid is inoculated into a large volume of culture media, such as a blood culture bottle, within 1 hour after collection. Blood cultures for bacteria may be useful, especially in infants; nongonococcal infection in adults (50%); DGI (13%). <em>B. burgdorferi</em> serology for Lyme disease. Genitourinary, throat, or rectal culture: DGI may be diagnosed by positive culture from a nonarticular source and by a compatible clinical picture. In difficult cases, MRI can help differentiate septic arthritis from transient synovitis.</td>
<td>It is important to obtain synovial fluid and blood for culture before starting antimicrobial treatment. Septic arthritis is usually hematogenously acquired. Prosthetic joint and diminished host defenses secondary to cancer, HIV, liver disease, or hypogammaglobulinemia are common predisposing factors. Nongonococcal bacterial arthritis is usually monarticular (and typically affects one knee joint). DGI is the most common cause of septic arthritis in urban centers and is usually polyarticular with associated tenosynovitis. Radiol Clin North Am 1996;34:293. Rheumat Dis Clin North Am 1997;23:239. Lancet 1998;351:197. Am J Orthop 1999;28:168. Radiology 1999;211:459. Pediatr Emerg Care 1999;15:40.</td>
<td></td>
</tr>
<tr>
<td><strong>Impetigo</strong></td>
<td><strong>Gas Gangrene</strong></td>
<td></td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>Gram stain, culture, and smear for HSV and VZV antigen detection by direct fluorescent antibody (DFA) of scrapings from lesions may be useful in differentiating impetigo from other vesicular or pustular lesions (HSV, VZV, contact dermatitis). DFA smear can be performed by scraping the contents, base, and roof of vesicle and applying to glass slide. After fixing, the slide is stained with direct fluorescent antibody (DFA) for identification of HSV or VZV.</td>
<td>Gas gangrene occurs in the setting of a contaminated wound. <em>Clostridium perfringens</em> produces potent exotoxins, including alpha toxin and theta toxin, which depresses myocardial contractility, induces shock, and causes direct vascular injury at the site of infection. Infections with enterobacter or <em>E coli</em> and anaerobic infections can also cause gas formation. These agents cause cellulitis rather than myonecrosis. Postgrad Med 1996;99:217. Clin Infect Dis 1999;28:159.</td>
<td></td>
</tr>
<tr>
<td><em>Clostridium perfringens</em> (GPR), (80–95%), other clostridium sp.</td>
<td>Diagnosis should be suspected in areas of devitalized tissue when gas is discovered by palpation (subcutaneous crepitation) or x-ray. Gram stain of foul-smelling, brown or blood-tinged watery exudate can be diagnostic with gram-positive rods and a remarkable absence of neutrophils. Anaerobic culture of discharge is confirmatory.</td>
<td></td>
</tr>
<tr>
<td>Organism</td>
<td>Specimen / Diagnostic Tests</td>
<td>Comments</td>
</tr>
<tr>
<td>------------------</td>
<td>---------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Cellulitis</td>
<td>Skin culture: In spontaneous cellulitis, isolation of the causative organism is difficult. In traumatic and postoperative wounds, Gram stain may allow rapid diagnosis of staphylococcal or clostridial infection. Culture of wound or abscess material after disinfection of the skin site will almost always yield the diagnosis. MRI can aid in diagnosis of secondary abscess formation, necrotizing fasciitis, or pyomyositis. Frozen section of biopsy specimen may be useful.</td>
<td>Cellulitis has long been considered to be the result of an antecedent bacterial invasion with subsequent bacterial proliferation. However, the difficulty in isolating putative pathogens from cellulitic skin has cast doubt on this theory. Predisposing factors for cellulitis include diabetes mellitus, edema, peripheral vascular disease, venous insufficiency, leg ulcer or wound, tinea pedis, dry skin, obesity, and prior history of cellulitis. Consider updating antitetanus prophylaxis for all wounds. In the diabetic, and in postoperative and traumatic wounds, consider prompt surgical debridement for necrotizing fasciitis. With abscess formation, surgical drainage is the mainstay of therapy and may be sufficient. Hemolytic streptococcal gangrene may follow minor trauma and involves specific strains of streptococcus.</td>
</tr>
</tbody>
</table>
Bacteremia of Unknown Source

Neonate (<4 days): Group B streptococcus (GPC), *E coli* (GNR), klebsiella (GNR), enterobacter (GNR), *S aureus* (GPC), coagulase-negative staphylococci (GPC).

Neonate (>5 days): Add *H influenzae* (GNCB).


Adult (IV drug use): *S aureus* or viridans streptococci (GPC).


Adult (splectomized): *S pneumoniae, H influenzae, N meningitidis*.

Neutropenia (<500 PMN): Enterobacteriaceae, pseudomonas sp, *S aureus*, coagulase-negative staphylococci, viridans group streptococcus.

Parasites: Babesia, ehrlichia, plasmodium sp, filarial worms.

Immunocompromised: Bartonella sp (GNR), herpesvirus 8 (HHV8), *Mycobacterium avium-intracellulare* (AFB).

Blood cultures are mandatory for all patients with fever and no obvious source of infection. Often they are negative, especially in neonates. Cultures should be drawn at onset of febrile episode. Culture should never be drawn from an IV line or from a femoral site.

Culture and Gram stain of urine, wounds, and other potentially infected sites provide a more rapid diagnosis than blood cultures.

Occult bacteremia affects approximately 5% of febrile children ages 2–36 months. In infants, the findings of an elevated total WBC count (>15,000) and absolute neutrophil count (ANC >10,000) were equally sensitive in predicting bacteremia, but the ANC was more specific. Predisposing factors in adults include IV drug use, neutropenia, cancer, diabetes mellitus, venous catheterization, hemodialysis, and plasmapheresis.

Catheter-related infection in patients with long-term venous access (Broviac, Hickman, etc) may be treated successfully without removal of the line, but recurrence of bacteremia is frequent.

Switching needles during blood cultures does not decrease contamination rates and increases the risk of needle-stick injuries.

This page intentionally left blank.
Diagnostic Imaging: Test Selection and Interpretation

Sean Perini, MD, and Susan D. Wall, MD

HOW TO USE THIS SECTION

Information in this chapter is arranged anatomically from superior to inferior. It would not be feasible to include all available imaging tests in one chapter in a book this size, but we have attempted to summarize the essential features of those examinations that are most frequently ordered in modern clinical practice or those that may be associated with difficulty or risk. Indications, advantages and disadvantages, contraindications, and patient preparation are presented. Costs of the studies are approximate and represent averages reported from several large medical centers.

$ = <$250
$$ = $250–$750
$$$ = $750–$1000
$$$$ = >$1000
RISKS OF INTRAVENOUS CONTRAST STUDIES

While intravenous contrast is an important tool in radiology, it is not without substantial risks. Minor reactions (nausea, vomiting, hives) occur with an overall incidence between 1% and 12%. Major reactions (laryngeal edema, bronchospasm, cardiac arrest) occur in 0.16 to 2 cases per 1000 patients. Deaths have been reported in 1:170,000 to 1:40,000 cases. Patients with an allergic history (asthma, hay fever, allergy to foods or drugs) are at increased risk. A history of reaction to contrast material is associated with an increased risk of a subsequent severe reaction. Prophylactic measures that may be required in such cases include H₁ and H₂ blockers and corticosteroids.

In addition, there is a risk of contrast-induced renal failure, which is usually mild and reversible. Persons at increased risk for potentially irreversible renal damage include patients with preexisting renal disease (particularly diabetics with high serum creatinine concentrations), multiple myeloma, and severe hyperuricemia.

In summary, intravenous contrast should be viewed in the same manner as other medications—ie, risks and benefits must be balanced before an examination using this pharmaceutical is ordered.
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>HEAD CT</strong></td>
<td>Evaluation of acute craniofacial trauma, acute neurologic dysfunction (&lt;72 hours) from suspected intracranial or subarachnoid hemorrhage. Further characterization of intracranial masses identified by MRI (presence or absence of calcium or involvement of the bony calvarium). Evaluation of sinus disease and temporal bone disease.</td>
<td>Rapid acquisition makes it the modality of choice for trauma. Superior to MRI in detection of hemorrhage within the first 24–48 hours.</td>
<td>Artifacts from bone may interfere with detection of disease at the skull base and in the posterior fossa. Generally limited to transaxial views. Direct coronal images of paranasal sinuses and temporal bones are routinely obtained if patient can lie prone. <strong>Contraindications and risks:</strong> Contraindicated in pregnancy because of the potential harm of ionizing radiation to the fetus. See Risks of Intravenous Contrast Studies, page 244.</td>
<td>Normal hydration. Sedation of agitated patients. Recent serum creatinine determination if intravenous contrast is to be used.</td>
</tr>
<tr>
<td><strong>HEAD MRI</strong></td>
<td>Evaluation of essentially all intracranial disease except those listed above for CT.</td>
<td>Provides excellent tissue contrast resolution, multiplanar capability. Can detect flowing blood and cryptic vascular malformations. Can detect demyelinating and dysmyelinating disease. No beam-hardening artifacts such as can be seen with CT. No ionizing radiation. Subject to motion artifacts. Inferior to CT in the setting of acute trauma because it is insensitive to acute hemorrhage, incompatible with traction devices, inferior in detection of bony injury and foreign bodies, and requires longer imaging acquisition time. Special instrumentation required for patients on life support. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves.</td>
<td>Sedation of agitated patients. Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye.</td>
<td></td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
<td>Advantages</td>
<td>Disadvantages/Contraindications</td>
<td>Preparation</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>BRAIN</td>
<td>Evaluation of cerebral arteriovenous malformations, intracranial aneurysm, and blood supply of vascular tumors as aid to operative planning (MRA). Evaluation of dural sinus thrombosis (MRV).</td>
<td>No ionizing radiation. No iodinated contrast needed.</td>
<td>Subject to motion artifacts. Special instrumentation required for patients on life support.</td>
<td>Sedation of agitated patients. Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye.</td>
</tr>
<tr>
<td>Magnetic resonance angiography/venography (MRA/MRV)</td>
<td></td>
<td></td>
<td><strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves.</td>
<td></td>
</tr>
<tr>
<td>$$$</td>
<td></td>
<td></td>
<td><strong>Contraindications and risks:</strong> Caution in pregnancy because of the potential harm of ionizing radiation to the fetus.</td>
<td></td>
</tr>
<tr>
<td>BRAIN</td>
<td>Confirmation of brain death.</td>
<td>Confirmation of brain death not impeded by hypothermia or barbiturate coma. Can be portable.</td>
<td>Limited resolution. Delayed imaging required with some agents. Cannot be used alone to establish diagnosis of brain death. Must be used in combination with clinical examination or cerebral angiography to establish diagnosis.</td>
<td>Sedation of agitated patients. Premedicate with potassium perchlorate when using TcO₄⁻ in order to block choroid plexus uptake.</td>
</tr>
<tr>
<td>Brain scan (radio-nuclide)</td>
<td></td>
<td></td>
<td><strong>Contraindications and risks:</strong></td>
<td></td>
</tr>
<tr>
<td>$$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brain Positron emission tomography (PET)/single photon emission (SPECT) brain scan</td>
<td>Evaluation of suspected dementia. Evaluation of medically refractory seizures.</td>
<td>Provide functional information. Can localize seizure focus prior to surgical excision. Up to 82% positive predictive value for Alzheimer’s dementia in appropriate clinical settings. Provide cross-sectional images and therefore improved lesion localization compared with planar imaging techniques.</td>
<td>Limited resolution compared with MRI and CT. Limited application in workup of dementia due to low specificity of images and fact that test results do not alter clinical management. <strong>Contraindications and risks:</strong> Caution in pregnancy because of potential harm of ionizing radiation to the fetus.</td>
<td>Requires lumbar puncture to deliver radiopharmaceutical.</td>
</tr>
<tr>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Brain Cisternography (radio-nuclide)</td>
<td>Evaluation of hydrocephalus (particularly normal pressure), CSF rhinorrhea or otorrhea, and ventricular shunt patency.</td>
<td>Provides functional information. Can help distinguish normal pressure hydrocephalus from senile atrophy. Can detect CSF leaks.</td>
<td>Requires multiple delayed imaging sessions up to 48–72 hours after injection. <strong>Contraindications and risks:</strong> Caution in pregnancy because of the potential harm of ionizing radiation to the fetus.</td>
<td>Sedation of agitated patients. For suspected CSF leak, pack the patient’s nose or ears with cotton pledgets prior to administration of dose. Must follow strict sterile precautions for intrathecal injection.</td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
<td>Advantages</td>
<td>Disadvantages/Contraindications</td>
<td>Preparation</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------</td>
</tr>
</tbody>
</table>
| NECK **Magnetic resonance imaging** (MRI) | Evaluation of the upper aerodigestive tract.  
Staging of neck masses.  
Differentiation of lymphadenopathy from blood vessels.  
Evaluation of head and neck malignancy, thyroid nodules, parathyroid adenoma, lymphadenopathy, retropharyngeal abscess, brachial plexopathy. | Provides excellent tissue contrast resolution.  
Tissue differentiation of malignancy or abscess from benign tumor often possible.  
Sagittal and coronal planar imaging possible.  
Multiplanar capability especially advantageous regarding brachial plexus.  
No iodinated contrast needed to distinguish lymphadenopathy from blood vessels. | Subject to motion artifacts, particularly those of carotid pulsation and swallowing.  
Special instrumentation required for patients on life support.  
**Contraindications and risks:** Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves. | Sedation of agitated patients.  
Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye. |
| NECK **Magnetic resonance angiography** (MRA) | Evaluation of carotid bifurcation atherosclerosis, cervicocranial arterial dissection.                                                                                                                     | No ionizing radiation.  
No iodinated contrast needed.  
MRA of the carotid arteries can be a sufficient preoperative evaluation regarding critical stenosis when local expertise exists. | Subject to motion artifacts, particularly from carotid pulsation and swallowing.  
Special instrumentation required for patients on life support.  
**Contraindications and risks:** Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves. | Sedation of agitated patients.  
Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye. |
<table>
<thead>
<tr>
<th>Test</th>
<th>Description</th>
<th>Preferences</th>
<th>Contraindications and Risks</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>NECK</strong>&lt;br&gt;Computed tomography (CT)</td>
<td>Evaluation of the upper aero-digestive tract. Staging of neck masses for patients who are not candidates for MRI. Evaluation of suspected abscess.</td>
<td>Rapid. Superb spatial resolution. Can guide percutaneous fine-needle aspiration of possible tumor or abscess.</td>
<td>Adequate intravenous contrast enhancement of vascular structures is mandatory for accurate interpretation. <strong>Contraindications and risks:</strong> Contraindicated in pregnancy because of the potential harm of ionizing radiation to the fetus. See Risks of Intravenous Contrast Studies, page 244.</td>
</tr>
<tr>
<td><strong>THYROID</strong>&lt;br&gt;Ultrasound (US)</td>
<td>Determination as to whether a palpable nodule is a cyst or solid mass and whether single or multiple nodules are present. Assessment of response to suppressive therapy. Screening patients with a history of prior radiation to the head and neck. Guidance for biopsy.</td>
<td>Noninvasive. No ionizing radiation. Can be portable. Can image in all planes.</td>
<td>Cannot distinguish between benign and malignant lesions unless local invasion is demonstrated. <strong>Contraindications and risks:</strong> None.</td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
<td>Advantages</td>
<td>Disadvantages/Contraindications</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>THYROID</td>
<td>Uptake indicated for evaluation of clinical hypothyroidism, hyperthyroidism, thyroiditis, effects of thyroid-stimulating and suppressing medications, and for calculation of therapeutic radiation dosage. Scanning indicated for above as well as evaluation of palpable nodules, mediastinal mass, and screening of patients with history of head and neck irradiation. Total body scanning used for postoperative evaluation of thyroid metastases.</td>
<td>Demonstrates both morphology and function. Can identify ectopic thyroid tissue and “cold” nodules that have a greater risk of malignancy. Imaging of total body with one dose ($^{131}$I).</td>
<td>Substances interfering with test include iodides in vitamins and medicines, antithyroid drugs, steroids, and intravascular contrast agents. Delayed imaging is required with iodides ($^{123}$I, 6 hours and 24 hours; $^{131}$I total body, 72 hours). Test may not visualize thyroid gland in subacute thyroiditis. <strong>Contraindications and risks:</strong> Not advised in pregnancy because of the risk of ionizing radiation to the fetus (iodides cross placenta and concentrate in fetal thyroid). Significant radiation exposure occurs in total body scanning with $^{131}$I; patients should be instructed about precautionary measures by nuclear medicine personnel.</td>
</tr>
<tr>
<td>Thyroid uptake and scan (radio-nuclide)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procedure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------</td>
<td>----------------</td>
<td>----------------</td>
<td>----------------</td>
</tr>
<tr>
<td><strong>THYROID</strong></td>
<td><strong>Thyroid therapy</strong> (radio-nuclide)</td>
<td>Hyperthyroidism and some thyroid carcinomas (papillary and follicular types are amenable to treatment, whereas medullary and anaplastic types are not).</td>
<td>Noninvasive alternative to surgery.</td>
</tr>
<tr>
<td><strong>THYROID</strong></td>
<td><strong>Parathyroid scan (radio-nuclide)</strong></td>
<td>Evaluation of suspected parathyroid adenoma.</td>
<td>Identifies hyperfunctioning tissue, which is useful when planning surgery.</td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
<td>Advantages</td>
<td>Disadvantages/Contraindications</td>
</tr>
<tr>
<td>----------------------</td>
<td>-----------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>CHEST Chest radiograph</td>
<td>Evaluation of pleural and parenchymal pulmonary disease, mediastinal disease, cardiogenic and noncardiogenic pulmonary edema, congenital and acquired cardiac disease. Screening for traumatic aortic rupture (though angiogram is the standard and spiral computed tomography is playing an increasing role). Evaluation of possible pneumothorax (expiratory upright film) or free flowing fluid (decubitus views).</td>
<td>Inexpensive. Widely available.</td>
<td>Difficult to distinguish between causes of hilar enlargement (ie, vasculature versus adenopathy). <strong>Contraindications and risks:</strong> Caution in pregnancy because of the potential harm of ionizing radiation to the fetus.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHEST Chest radiograph</td>
<td>Evaluation of pleural and parenchymal pulmonary disease, mediastinal disease, cardiogenic and noncardiogenic pulmonary edema, congenital and acquired cardiac disease. Screening for traumatic aortic rupture (though angiogram is the standard and spiral computed tomography is playing an increasing role). Evaluation of possible pneumothorax (expiratory upright film) or free flowing fluid (decubitus views).</td>
<td>Inexpensive. Widely available.</td>
<td>Difficult to distinguish between causes of hilar enlargement (ie, vasculature versus adenopathy). <strong>Contraindications and risks:</strong> Caution in pregnancy because of the potential harm of ionizing radiation to the fetus.</td>
<td>None.</td>
</tr>
</tbody>
</table>
## CHEST Magnetic resonance imaging (MRI)

<table>
<thead>
<tr>
<th>Evaluation of mediastinal masses. Discrimination between hilar vessels and enlarged lymph nodes. Tumor staging (especially when invasion of vessels or pericardium is suspected). Evaluation of aortic dissection, aortic aneurysm, congenital and acquired cardiac disease. Provides excellent tissue contrast resolution and multiplanar capability. No beam-hardening artifacts such as can be seen with CT. No ionizing radiation. Subject to motion artifacts. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves. Sedation of agitated patients. Screening CT of the orbits if history suggests possible metallic foreign body in the eye.</th>
</tr>
</thead>
</table>

## LUNG Ventilation-perfusion scan (radio-nuclide)

| Evaluation of pulmonary embolism or burn inhalation injury. Preoperative evaluation of patients with chronic obstructive pulmonary disease and of those who are candidates for pneumonectomy. Noninvasive. Provides functional information in preoperative assessment. Permits determination of differential and regional lung function in preoperative assessment. Documented pulmonary embolism is extremely rare with normal perfusion scan. Patients must be able to cooperate for ventilation portion of the examination. There is a high proportion of intermediate probability studies in patients with underlying lung disease. The likelihood of pulmonary embolism ranges from 20% to 80% in these cases. A patient who has a low probability scan still has a chance ranging from nil to 19% of having a pulmonary embolus. **Contraindications and risks:** Patients with severe pulmonary artery hypertension or significant right-to-left shunts should have fewer particles injected. Caution advised in pregnancy because of risk of ionizing radiation to the fetus. Sedation of agitated patients. Screening CT of the orbits if history suggests possible metallic foreign body in the eye. Current chest radiograph is mandatory for interpretation. |

\[
\dot{V} = \text{radio-nuclide}
\]

\[
\dot{Q} = \text{radio-nuclide}
\]

\[
\dot{V} + \dot{Q} = \text{radio-nuclide}
\]
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>LUNG Spiral computed tomography</td>
<td>Evaluation of clinically suspected pulmonary embolism.</td>
<td>Rapid. Sensitivity and specificity values likely about 90% for the CT diagnosis of pulmonary emboli involving main to segmental artery branches in unselected patients. Overall, spiral CT sensitivity may be higher than ventilation/perfusion scintigraphy.</td>
<td>Accuracy of spiral CT in diagnosing pulmonary embolism depends on the size of the pulmonary artery involved and the size of the thrombus. Sensitivity and accuracy of CT decreases for small, subsegmental emboli (sensitivity rates of 53–63% have been reported). Respiratory motion artifacts can be a problem in dyspneic patients. High-quality study requires breath-holding of approximately 20 seconds. Specific imaging protocol utilized which limits diagnostic information for other abnormalities. <strong>Contraindications and risks:</strong> Contraindicated in pregnancy because of potential harm of ionizing radiation to fetus. See Risks of Intravenous Contrast Studies, page 244.</td>
<td>Large gauge intravenous access (minimum 20-gauge) required. Prebreathing oxygen may help dyspneic patients perform adequate breath hold. Normal hydration. Preferably NPO for 2 hours prior to study. Recent serum creatinine determination.</td>
</tr>
<tr>
<td>LUNG Pulmonary angiography</td>
<td>Suspected pulmonary embolism with equivocal results on ventilation/perfusion scan or when definitive diagnosis especially important because of contraindication to anticoagulation. Arteriovenous malformation, pulmonary sequestration, vasculitides, vascular occlusion by tumor or inflammatory disease.</td>
<td>Remains the standard for diagnosis of acute and chronic pulmonary embolism.</td>
<td>Invasive. Requires catheterization of the right heart and pulmonary artery. <strong>Contraindications:</strong> Elevated pulmonary artery pressure (&gt; 70 mm Hg) or elevated right ventricular end-diastolic pressure (&gt; 20 mm Hg). Pulmonary artery hypertension.</td>
<td>Ventilation/perfusion scan for localization of right versus left lung. Electrocardiogram, especially to exclude left bundle branch block (in such cases, temporary cardiac pacemaker should be placed before the catheter is introduced into the pulmonary artery).</td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
<td>Advantages</td>
<td>Disadvantages/Contraindications</td>
<td>Preparation</td>
</tr>
<tr>
<td>------------</td>
<td>------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------</td>
<td>-------------</td>
</tr>
<tr>
<td>BREAST Mammogram</td>
<td>Screening for breast cancer in asymptomatic women: (1) every 1–2 years between ages 40 and 49; (2) every year after age 50. If prior history of breast cancer, mammogram should be performed yearly at any age. Indicated at any age for symptoms (palpable mass, bloody discharge) or before breast surgery.</td>
<td>Newer film screen techniques generate lower radiation doses (0.1–0.2 rad per film, mean glandular dose). A 23% lower mortality has been demonstrated in patients screened with combined mammogram and physical exam compared to physical exam alone. In a screening population, more than 40% of cancers are detected by mammography alone and cannot be palpated on physical exam.</td>
<td>Detection of breast masses is more difficult in patients with radiographically dense breasts. Breast compression may cause patient discomfort. In a screening population, 9% of cancers are detected by physical examination alone and are not detectable by mammography. <strong>Contraindications and risks:</strong> Radiation from repeated mammograms can theoretically cause breast cancer; however, the benefits of screening mammograms greatly outweigh the risks.</td>
<td>None.</td>
</tr>
<tr>
<td>HEART</td>
<td>Myocardial perfusion scan (thallium scan, technetium-99m methoxyisobutylisonitrile (sestamibi) scan, others)</td>
<td>Evaluation of atypical chest pain. Detection of presence, location, and extent of myocardial ischemia.</td>
<td>Highly sensitive for detecting physiologically significant coronary stenosis. Noninvasive. Able to stratify patients according to risk for myocardial infarction. Normal examination associated with average risk of cardiac death or nonfatal myocardial infarction of &lt;1% per year.</td>
<td>The patient must be carefully monitored during treadmill or pharmacologic stress—optimally, under the supervision of a cardiologist. False-positive results may be caused by exercise-induced spasm, aortic stenosis, or left bundle branch block; false-negative results may be caused by inadequate exercise, mild or distal disease, or balanced diffuse ischemia. <strong>Contraindications and risk:</strong> Aminophylline (inhibitor of dipyridamole) is a contraindication to the use of dipyridamole. Treadmill or pharmacologic stress carries a risk of arrhythmia, ischemia, infarct, and, rarely, death. Caution in pregnancy because of the risk of ionizing radiation to the fetus.</td>
</tr>
</tbody>
</table>

<p>| HEART | Radionuclide ventriculography (multigated acquisition [MUGA]) | Evaluation of patients with ischemic heart disease and other cardiomyopathies. Evaluation of response to pharmacologic therapy and effects of cardiotoxic drugs. | Noninvasive. Ejection fraction is a reproducible index that can be used to follow course of disease and response to therapy. | Gated data acquisition may be difficult in patients with severe arrhythmias. <strong>Contraindications and risks:</strong> Recent infarct is a contraindication to exercise ventriculography (arrhythmia, ischemia, infarct, and rarely death may occur with exercise). Caution is advised in pregnancy because of the risk of ionizing radiation to the fetus. | Requires harvesting, labeling, and re-injecting the patient’s red blood cells. Sterile technique required in handling of red cells. |</p>
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>ABDOMEN Abdominal plain radiograph (KUB [kidneys, ureters, bladder] x-ray)</td>
<td>Assessment of bowel gas patterns (eg, to distinguish ileus from obstruction). To rule out pneumoperitoneum, order an upright abdomen and chest radiograph (acute abdominal series).</td>
<td>Inexpensive. Widely available.</td>
<td>Supine film alone is inadequate to rule out pneumoperitoneum (see indications). Obstipation may obscure lesions. <strong>Contraindications and risks:</strong> Contraindicated in pregnancy because of the risk of ionizing radiation to the fetus.</td>
<td>None.</td>
</tr>
<tr>
<td>ABDOMEN Ultrasound (US)</td>
<td>Differentiation of cystic versus solid lesions of the liver and kidneys, intra- and extrahepatic biliary ductal dilation, cholelithiasis, gall-bladder wall thickness, pericholecystic fluid, peripancreatic fluid and pseudocyst, primary and metastatic liver carcinoma, hydronephrosis, abdominal aortic aneurysm, appendicitis, ascites.</td>
<td>Noninvasive. No ionizing radiation. Can be portable. Imaging in all planes. Can guide percutaneous fine-needle aspiration of tumor or abscess.</td>
<td>Technique very operator-dependent. Organs (particularly pancreas and distal aorta) may be obscured by bowel gas. Presence of barium obscures sound waves. <strong>Contraindications and risks:</strong> None.</td>
<td>NPO for 6 hours.</td>
</tr>
<tr>
<td>Procedure</td>
<td>Description</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
<td>Advantages</td>
<td>Disadvantages/Contraindications</td>
<td>Preparation</td>
</tr>
<tr>
<td>-------------------------------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>ABDOMEN Magnetic resonance imaging (MRI)</td>
<td>Clarification of CT findings when surgical clip artifacts are present.</td>
<td>Provides excellent tissue contrast resolution, multiplanar capability.</td>
<td>Subject to motion artifacts. Gastrointestinal opacification not yet readily available. Special instrumentation required for patients on life support. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves.</td>
<td>NPO for 4–6 hours. Intramuscular glucagon to inhibit peristalsis. Sedation of agitated patients. Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye.</td>
</tr>
<tr>
<td></td>
<td>Differentiation of retroperitoneal lymphadenopathy from blood vessels or the diaphragmatic crus. Preoperative staging of renal cell carcinoma.</td>
<td>No beam-hardening artifacts such as can be seen with CT. No ionizing radiation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Differentiation of benign nonhyperfunctioning adrenal adenoma from malignant adrenal mass. Complementary to CT in evaluation of liver lesions (especially metastatic disease and possible tumor invasion of hepatic or portal veins). Differentiation of benign cavernous hemangioma (&gt;2 cm in diameter) from malignancy.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ABDOMEN Mesenteric angiography</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$$$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gastrointestinal hemorrhage that does not resolve with conservative therapy and cannot be treated endoscopically.</td>
<td>Therapeutic embolization of gastrointestinal hemorrhage is often possible.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Localization of gastrointestinal bleeding site. Acute mesenteric ischemia, intestinal angina, splenic or other splanchnic artery aneurysm. Evaluation of possible vasculitis, such as polyarteritis nodosa. Detection of islet cell tumors not identified by other studies. Abdominal trauma.</td>
<td>Invasive. Patient must remain supine with leg extended for 6 hours following the procedure in order to protect the common femoral artery at the catheter entry site. <strong>Contraindications and risks:</strong> Allergy to iodinated contrast material may require corticosteroid and H$_1$ blocker or H$_2$ blocker premedication. Contraindicated in pregnancy because of the potential harm of ionizing radiation to the fetus. Contrast nephrotoxicity, especially with preexisting impaired renal function due to diabetes mellitus or multiple myeloma; however, any creatinine elevation following the procedure is usually reversible (see page 244).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>NPO for 4–6 hours. Good hydration to limit possible renal insult due to iodinated contrast material. Recent serum creatinine determination, assessment of clotting parameters, reversal of anti-coagulation. Performed with conscious sedation. Requires cardiac, respiratory, blood pressure, and pulse oximetry monitoring.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
<td>Advantages</td>
<td>Disadvantages/Contraindications</td>
<td>Preparation</td>
</tr>
<tr>
<td>---------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td>-----------------------------------------------------------------------------------------------</td>
<td>---------------------------------------------------------------------------------------------</td>
<td>--------------------------</td>
</tr>
<tr>
<td>GI Upper GI study (UGI)</td>
<td>Double-contrast barium technique demonstrates esophageal, gastric, and duodenal mucosa for evaluation of inflammatory disease and other subtle mucosal abnormalities. Single-contrast technique is suitable for evaluation of possible outlet obstruction, peristalsis, gastro-esophageal reflux and hiatal hernia, esophageal cancer and varices. Water-soluble contrast (Gastrografin) is suitable for evaluation of anastomotic leak or gastrointestinal perforation.</td>
<td>Good evaluation of mucosa with double-contrast examination. No sedation required. Less expensive than endoscopy.</td>
<td>Aspiration of water-soluble contrast material may occur, resulting in severe pulmonary edema. Leakage of barium from a perforation may cause granulomatous inflammatory reaction. Identification of a lesion does not prove it to be the site of blood loss in patients with gastrointestinal bleeding. Barium precludes endoscopy and body CT examination. Retained gastric secretions prevent mucosal coating with barium. <strong>Contraindications and risks:</strong> Contraindicated in pregnancy because of the potential harm of ionizing radiation to the fetus.</td>
<td>NPO for 8 hours.</td>
</tr>
<tr>
<td>GI Enteroclysis</td>
<td>Barium fluoroscopic study for location of site of intermittent partial small bowel obstruction. Evaluation of extent of Crohn’s disease or small bowel disease in patient with persistent gastrointestinal bleeding and normal upper gastrointestinal and colonic evaluations. Evaluation of metastatic disease to the small bowel.</td>
<td>Clarifies lesions noted on more traditional barium examination of the small bowel. Best means of establishing small bowel as normal. Controlled high rate of flow of barium can dilate a partial obstruction.</td>
<td>Requires nasogastric or orogastric tube placement and manipulation to beyond the ligament of Treitz. <strong>Contraindications and risks:</strong> Radiation exposure is substantial, since lengthy fluoroscopic examination is required. Therefore, the test is contraindicated in pregnant women and should be used sparingly in children and women of childbearing age.</td>
<td>Clear liquid diet for 24 hours. Colonic cleansing.</td>
</tr>
<tr>
<td>GI</td>
<td>Peroral pneumocolon</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>----</td>
<td>---------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$</td>
<td>Fluoroscopic evaluation of the terminal ileum by insufflating air per rectum after orally ingested barium has reached the cecum.</td>
<td>Best evaluation of the terminal ileum. Can be performed concurrently with upper GI series.</td>
<td>Undigested food in the small bowel interferes with the evaluation. <strong>Contraindications and risk:</strong> Contraindicated in pregnancy because of the potential harm of ionizing radiation to the fetus.</td>
<td>Clear liquid diet for 24 hours.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>GI</th>
<th>Barium enema (BE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>$$</td>
<td>Double-contrast technique for evaluation of colonic mucosa in patients with suspected inflammatory bowel disease or neoplasm. Single-contrast technique for investigation of possible fistulous tracts, bowel obstruction, large palpable masses in the abdomen, and diverticulitis and for examination of debilitated patients. Least invasive colon cancer screening technique.</td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
</tr>
<tr>
<td>----------------------</td>
<td>-----------------------------------------------------------------------------</td>
</tr>
<tr>
<td>GI Hypaque enema</td>
<td>Water-soluble contrast for fluoroscopic evaluation of sigmoid or cecal volvulus, anastomotic leak or other perforation. Differentiation of colonic versus small bowel obstruction. Therapy for obstipation.</td>
</tr>
<tr>
<td>GI Esophageal reflux study (radio-nuclide)</td>
<td>Evaluation of heartburn, regurgitation, recurrent aspiration pneumonia.</td>
</tr>
<tr>
<td>GI</td>
<td>Gastric emptying study (radio-nuclide)</td>
</tr>
<tr>
<td>GI</td>
<td>GI bleeding scan (labeled red cell scan, radio-nuclide)</td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
</tr>
<tr>
<td>------------------------------</td>
<td>-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>GALL-BLADDER Hepatic imino-diacid acid scan (HIDA)</td>
<td>Evaluation of suspected acute cholecystitis or common bile duct obstruction. Evaluation of bile leaks, biliary atresia, and biliary enteric bypass patency.</td>
</tr>
<tr>
<td>Procedure</td>
<td>Description</td>
</tr>
<tr>
<td>-----------</td>
<td>-------------</td>
</tr>
<tr>
<td><strong>LIVER</strong></td>
<td><strong>Ultrasound (US)</strong></td>
</tr>
<tr>
<td><strong>PANCREAS/BILIARY TREE</strong></td>
<td><strong>Endoscopic retrograde cholangio-pancreatography (ERCP)</strong></td>
</tr>
<tr>
<td></td>
<td>Primary sclerosing cholangitis, AIDS-associated cholangitis, and cholangiocarcinomas. Demonstrates cause, location, and extent of extrahepatic biliary obstruction (eg, choledocho-lithiasis). Can diagnose chronic pancreatitis.</td>
</tr>
<tr>
<td></td>
<td>Ultrasound (US)</td>
</tr>
<tr>
<td>Test</td>
<td>Indications</td>
</tr>
<tr>
<td>------</td>
<td>-------------</td>
</tr>
<tr>
<td>LIVER</td>
<td>Suspected metastatic or primary tumor, gallbladder carcinoma, biliary obstruction, abscess.</td>
</tr>
<tr>
<td><strong>Computed tomography (CT)</strong></td>
<td></td>
</tr>
<tr>
<td>$$$–$$$$</td>
<td></td>
</tr>
<tr>
<td>LIVER</td>
<td>Assessment of number, location, and resectability of metastatic liver tumors.</td>
</tr>
<tr>
<td><strong>Computed tomographic arterial portography (CTAP)</strong></td>
<td></td>
</tr>
<tr>
<td>$$$</td>
<td></td>
</tr>
</tbody>
</table>

CTAP
<p>| LIVER MRI | Characterization of focal hepatic lesion, including suspected cyst, hepatocellular carcinoma, focal nodular hyperplasia, and metastasis. Suspected metastatic or primary tumor. Differentiation of benign cavernous hemangioma from malignant tumor. Evaluation of hemochromatosis, hemosiderosis, fatty liver, and suspected focal fatty infiltration. | Requires no iodinated contrast material. Provides excellent tissue contrast resolution, multiplanar capability. | Subject to motion artifacts, particularly those of respiration. Special instrumentation required for patients on life support. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, some artificial heart valves. | Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye. Intramuscular glucagon is used to inhibit intestinal peristalsis. |</p>
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIVER/ BILIARY TREE</td>
<td>Evaluation of biliary obstruction in patients in whom ERCP has failed or patients with Roux-en-Y hepaticojejunostomy.</td>
<td>Best examination to assess site and morphology of obstruction close to the hilum (as opposed to endoscopic retrograde cholangiopancreatography [ERCP], which is better for distal obstruction). Can characterize the nature of diffuse intrahepatic biliary disease such as primary sclerosing cholangitis. Provides guidance and access for percutaneous transhepatic biliary drainage (PTBD) and possible stent placement to treat obstruction.</td>
<td>Invasive; requires special training. Performed with conscious sedation. Ascites may present a contraindication.</td>
<td>NPO for 4–6 hours. Sterile technique, assessment of clotting parameters, correction of coagulopathy. Performed with conscious sedation.</td>
</tr>
<tr>
<td>Percuta- neous trans- hepatic cholangio- gram (PTC)</td>
<td>$$$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

Pocket Guide to Diagnostic Tests
<p>| LIVER | Hepatic angiography | Preoperative evaluation for liver transplantation, vascular malformations, trauma, Budd-Chiari syndrome, portal vein patency (when ultrasound equivocal) prior to transjugular intrahepatic portosystemic shunt (TIPS) procedure. In some cases, evaluation of hepatic neoplasm or transcatheter embolotherapy of hepatic malignancy. | Best assessment of hepatic arterial anatomy, which is highly variable. More accurate than ultrasound with respect to portal vein patency when the latter suggests occlusion. | Invasive. Patient must remain supine with leg extended for 6 hours following the procedure in order to protect the common femoral artery at the catheter entry site. <strong>Contraindications and risks:</strong> Allergy to iodinated contrast material may require corticosteroid and H&lt;sub&gt;1&lt;/sub&gt; blocker or H&lt;sub&gt;2&lt;/sub&gt; blocker premedication. Contraindicated in pregnancy because of the potential harm of ionizing radiation to the fetus. Contrast nephrotoxicity may occur, especially with preexisting impaired renal function due to diabetes mellitus or multiple myeloma; however, any creatinine elevation following the procedure is usually reversible. | NPO for 4–6 hours. Good hydration to limit possible renal insult due to iodinated contrast material. Recent serum creatinine determination, assessment of clotting parameters, reversal of anticoagulation. Performed with conscious sedation. Requires cardiac, respiratory, blood pressure, and pulse oximetry monitoring. |
| LIVER, SPLEEN | Liver, spleen scan (radio-nuclide) | Identification of functioning splenic tissue to localize an accessory spleen or evaluate suspected functional asplenia. Assessment of size, shape, and position of liver and spleen. Characterization of a focal liver mass with regard to inherent functioning reticuloendothelial cell activity (with the exception of focal nodular hyperplasia mass lesions, which are more often “cold” than “hot”). Confirmation of patency and distribution of hepatic arterial perfusion catheters. | May detect isodense lesions missed by CT. | Diminished sensitivity for small lesions (less than 1.5–2 cm) and deep lesions. Single photon emission computed tomography (SPECT) increases sensitivity (can detect lesions of 1–1.5 cm). Nonspecific; unable to distinguish solid versus cystic or inflammatory versus neoplastic tissue. Lower sensitivity for diffuse hepatic tumors. <strong>Contraindications and risks:</strong> Caution in pregnancy advised because of the risk of ionizing radiation to the fetus. | None. |</p>
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Computed tomography (CT)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$$$–$$$$$$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Ultrasound (US)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ADRENAL MIBG (metaiodobenzylguanidine) (radio-nuclide)</td>
<td>Suspected pheochromocytoma when CT is negative or equivocal. Also useful in evaluation of neuroblastoma, carcinoid, and medullary carcinoma of thyroid.</td>
<td>Test is useful for localization of pheochromocytomas (particularly extra-adrenal). Eighty to 90 percent sensitive for detection of pheochromocytoma.</td>
<td>High radiation dose to adrenal gland. High cost and limited availability of MIBG. Delayed imaging (at 1, 2, and 3 days) necessitates return of patient. <strong>Contraindications and risks:</strong> Contraindicated in pregnancy because of the risk of ionizing radiation to the fetus. Because of the relatively high dose of $^{131}$I, patients should be instructed about precautionary measures by nuclear medicine personnel.</td>
<td>Administration of Lugol’s iodine solution (to block thyroid uptake) prior to and following administration of MIBG.</td>
</tr>
<tr>
<td>$$$$$</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procedure</td>
<td>Description</td>
<td>Indications</td>
<td>Contraindications and Risks</td>
<td></td>
</tr>
<tr>
<td>-----------</td>
<td>-------------</td>
<td>-------------</td>
<td>-----------------------------</td>
<td></td>
</tr>
<tr>
<td>Fluoroscopic evaluation of uroepithelial neoplasm, calculus, papillary necrosis, and medullary sponge kidney. Screening for urinary system injury after trauma.</td>
<td>FLUOROSCOPIC EPILOG</td>
<td>Permits evaluation of collecting system in less invasive manner than retrograde pyelogram. Can assess both renal morphology and function.</td>
<td>Suboptimal evaluation of the renal parenchyma. Does not adequately evaluate cause of ureteral deviation. <strong>Contraindications and risks:</strong> Caution in pregnancy is advised because of the risk of ionizing radiation to the fetus. See Risks of Intravenous Contrast Studies, page 244. Adequate hydration. Colonic cleansing is preferred but not essential. Recent serum creatinine determination.</td>
<td></td>
</tr>
<tr>
<td>Staging of cancers of the uterus, cervix, and prostate. Can provide information additional to what is obtained by CT in some cases of cancer of the kidney and urinary bladder.</td>
<td>GENITO-URINARY MAGNETIC RESONANCE IMAGING</td>
<td>Provides excellent tissue contrast resolution, multiplanar capability. No beam-hardening artifacts such as can be seen with CT. No ionizing radiation.</td>
<td>Subject to motion artifacts. Gastrointestinal opacification not yet readily available. Special instrumentation required for patients on life support. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves. Sedation of agitated patients. Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye.</td>
<td></td>
</tr>
</tbody>
</table>

- **IVP:** Intravenous pyelogram
- **US:** Ultrasound
- **MRI:** Magnetic resonance imaging
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>GENITOURINARY Renal scan (radio-nuclide)</td>
<td>Evaluation of suspected renal vascular hypertension. Differentiation of a dilated but non-obstructed system from one that has a urodynamically significant obstruction. Evaluation of renal blood flow and function in acute or chronic renal failure. Evaluation of both medical and surgical complications of renal transplant. Estimation of glomerular filtration rate (GFR) and effective renal plasma flow (ERPF). Determination of relative renal function prior to nephrectomy.</td>
<td>Provides functional information without risk of iodinated contrast used in IVP. Provides quantitative information not available by other means.</td>
<td>Finding of poor renal blood flow does not pinpoint an etiologic diagnosis. Limited utility when renal function is extremely poor. Estimation of glomerular filtration rate and renal plasma flow often is inaccurate. <strong>Contraindications and risks:</strong> Caution in pregnancy because of the risk of ionizing radiation to the fetus.</td>
<td>Normal hydration needed for evaluation of suspected obstructive uropathy since dehydration may result in false-positive examination. Blood pressure should be monitored and an intravenous line started when an angiotensin-converting enzyme (ACE) inhibitor is used to enhance test sensitivity in the evaluation of renal vascular hypertension. Patient should discontinue ACE inhibitor medication for at least 48 hours prior to examination if possible.</td>
</tr>
<tr>
<td>Test</td>
<td>Description</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>------</td>
<td>-------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PELVIS Ultrasound (US) $$$</td>
<td>Evaluation of palpable ovarian mass, enlarged uterus, vaginal bleeding, pelvic pain, possible ectopic pregnancy, and infertility. Monitoring of follicular development. Localization of intrauterine device. Use of a vaginal probe enables very early detection of intrauterine pregnancy and ectopic pregnancy and does not require a full bladder. Transabdominal scan has limited sensitivity for uterine or ovarian pathology. Vaginal probe has limited field of view and therefore may miss large masses outside the pelvis. <strong>Contraindications and risks:</strong> None. Use of a vaginal probe enables very early detection of intrauterine pregnancy and ectopic pregnancy and does not require a full bladder. Distended bladder required (only in transabdominal examination).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>PELVIS Magnetic resonance imaging (MRI) $$ $$</td>
<td>Evaluation of gynecologic malignancies, particularly endometrial, cervical, and vaginal carcinoma. Evaluation of prostate, bladder, and rectal carcinoma. Evaluation of congenital anomalies of the genitourinary tract. Useful in distinguishing lymphadenopathy from vasculature. Provides excellent tissue contrast resolution, multiplanar capability. No beam-hardening artifacts such as can be seen with CT. No ionizing radiation. Subject to motion artifacts. Special instrumentation required for patients on life support. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves. Intramuscular glucagon is used to inhibit intestinal peristalsis. Sedation of agitated patients. Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye. An endorectal device (radiofrequency coil) is used for prostate MRI.</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

---

**Distended bladder required (only in transabdominal examination).**
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bone scan, whole body</strong></td>
<td>Evaluation of primary or metastatic neoplasm, osteomyelitis, arthritis, metabolic disorders, trauma, avascular necrosis, joint prosthesis, and reflex sympathetic dystrophy. Evaluation of clinically suspected but radiographically occult fractures. Identification of stress fractures.</td>
<td>Can examine entire osseous skeleton or specific area of interest. Highly sensitive compared with plain film radiography for detection of bone neoplasm. In osteomyelitis, bone scan may be positive much earlier (24 hours) than plain film (10–14 days).</td>
<td>Nonspecific. Correlation with plain film radiographs often necessary. Limited utility in patients with poor renal function. Poor resolution in distal extremities, head, and spine; in these instances, single photon emission computed tomography (SPECT) is often useful. Sometimes difficult to distinguish osteomyelitis from cellulitis or septic joint; dual imaging with gallium or with indium-labeled leukocytes can be helpful. False-negative results for osteomyelitis can occur following antibiotic therapy and within the first 24 hours after trauma. In avascular necrosis, bone scan may be “hot,” “cold,” or normal, depending on the stage. <strong>Contraindications and risks:</strong> Caution in pregnancy because of the risk of ionizing radiation to the fetus.</td>
<td>Patient should be well hydrated and void frequently after the procedure.</td>
</tr>
</tbody>
</table>

**Test**

Bone scan

**Indications**


**Advantages**

Can examine entire osseous skeleton or specific area of interest. Highly sensitive compared with plain film radiography for detection of bone neoplasm. In osteomyelitis, bone scan may be positive much earlier (24 hours) than plain film (10–14 days).

**Disadvantages/Contraindications**

Nonspecific. Correlation with plain film radiographs often necessary. Limited utility in patients with poor renal function. Poor resolution in distal extremities, head, and spine; in these instances, single photon emission computed tomography (SPECT) is often useful. Sometimes difficult to distinguish osteomyelitis from cellulitis or septic joint; dual imaging with gallium or with indium-labeled leukocytes can be helpful. False-negative results for osteomyelitis can occur following antibiotic therapy and within the first 24 hours after trauma. In avascular necrosis, bone scan may be “hot,” “cold,” or normal, depending on the stage. **Contraindications and risks:** Caution in pregnancy because of the risk of ionizing radiation to the fetus.

**Preparation**

Patient should be well hydrated and void frequently after the procedure.
<p>| SPINE Computed tomography (CT) | Evaluation of structures that are not well visualized on MRI, including ossification of the posterior longitudinal ligament, tumoral calcification, osteophytic spurring, retropulsed bone fragments after trauma. Also used for patients in whom MRI is contraindicated. | Rapid. Superb spatial resolution. Can guide percutaneous fine-needle aspiration of possible tumor or abscess. | Generally limited to transaxial views. Coronal and sagittal reformation images can be generated. MRI unequivocally superior in evaluation of the spine and cord except for conditions mentioned in Indications. Artifacts from metal prostheses degrade images. <strong>Contraindications and risks:</strong> Contraindicated in pregnancy because of the potential harm of ionizing radiation to the fetus. See Risks of Intravenous Contrast Studies, page 244. | Normal hydration. Sedation of agitated patients. |
| SPINE Magnetic resonance imaging (MRI) | Diseases involving the spine and cord except where CT is superior (ossification of the posterior longitudinal ligament, tumoral calcification, osteophytic spurring, retropulsed bone fragments after trauma). | Provides excellent tissue contrast resolution, multiplanar capability. No beam-hardening artifacts such as can be seen with CT. No ionizing radiation. | Less useful in detection of calcification, small spinal vascular malformations, acute spinal trauma (because of longer acquisition time, incompatibility with life support devices, and inferior detection of bony injury). Subject to motion artifacts. Special instrumentation required for patients on life support. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves. | Sedation of agitated patients. Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye. |</p>
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>MUSCULOSKELETAL SYSTEM</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnetic resonance imaging (MRI)</td>
<td>Evaluation of joints except where a prosthesis is in place. Extent of primary or malignant tumor (bone and soft tissue). Evaluation of aseptic necrosis, bone and soft tissue infections, marrow space disease, and traumatic derangements.</td>
<td>Provides excellent tissue contrast resolution, multiplanar capability. No beam-hardening artifacts such as can be seen with CT. No ionizing radiation.</td>
<td>Subject to motion artifacts. Less able than CT to detect calcification, ossification, and periosteal reaction. Special instrumentation required for patients on life support. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves.</td>
<td>Sedation of agitated patients. Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye.</td>
</tr>
<tr>
<td><strong>VASCULATURE</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ultrasound (US)</td>
<td>Evaluation of deep venous thrombosis, extremity grafts, patency of inferior vena cava, portal vein, and hepatic veins. Carotid doppler indicated for symptomatic carotid bruit, atypical transient ischemic attack, monitoring after endarterectomy, and baseline prior to major vascular surgery. Surveillance of transjugular intrahepatic portosystemic shunt (TIPS) patency and flow.</td>
<td>Noninvasive. No radiation. Can be portable. Imaging in all planes.</td>
<td>Technique operator-dependent. Ultrasound not sensitive to detection of ulcerated plaque. May be difficult to diagnose tight stenosis versus occlusion (catheter angiography may be necessary). May be difficult to distinguish acute from chronic deep venous thrombosis. <strong>Contraindications and risks:</strong> None.</td>
<td>None.</td>
</tr>
</tbody>
</table>

**MUSCULOSKELETAL**

**MRI**

**VASCULATURE**

**Ultrasound**
<p>| AORTA AND ITS BRANCHES | Peripheral vascular disease, abdominal aortic aneurysm, renal artery stenosis (atherosclerotic and fibromuscular disease), polyarteritis nodosa, visceral ischemia, thoracic aortic dissection, gastrointestinal hemorrhage, thromboangiitis obliterans (Buerger’s disease), popliteal entrapment syndrome, cystic adventitial disease, abdominal tumors, arteriovenous malformations, abdominal trauma. Preoperative evaluation for aorto-femoral bypass reconstructive surgery. Postoperative assessment of possible graft stenosis, especially femoral to popliteal or femoral to distal (foot or ankle). | Can localize atherosclerotic stenosis and assess the severity by morphology, flow, and pressure gradient. Provides assessment of stenotic lesions and access for percutaneous transluminal balloon dilation as well as stent treatment of iliac stenoses. Provides access for thrombolytic therapy of acute or subacute occlusion of native artery or bypass graft. | Invasive. Patient must remain supine with leg extended for 6 hours following the procedure in order to protect the common femoral artery at the catheter entry site. Contraindications and risks: Allergy to iodinated contrast material may require corticosteroid and H$_2$ blocker or H$_2$ blocker premedication. Contraindicated in pregnancy because of the potential harm of ionizing radiation to the fetus. Contrast nephrotoxicity may occur, especially with preexisting impaired renal function due to diabetes mellitus or multiple myeloma; however, any creatinine elevation that occurs after the procedure is usually reversible. | NPO for 4–6 hours. Good hydration to limit possible renal insult due to iodinated contrast material. Recent serum creatinine determination, assessment of clotting parameters, reversal of anti-coagulation. Performed with conscious sedation. Requires cardiac, respiratory, blood pressure, and pulse oximetry monitoring as well as non-invasive studies of peripheral vascular disease to verify indication for angiography and to guide the examination. |</p>
<table>
<thead>
<tr>
<th>Test</th>
<th>Indications</th>
<th>Advantages</th>
<th>Disadvantages/Contraindications</th>
<th>Preparation</th>
</tr>
</thead>
<tbody>
<tr>
<td>AORTA AND ITS BRANCHES Magnetic resonance angiography (MRA)</td>
<td>Can provide preoperative assessment of abdominal aortic aneurysm to determine aneurysm size, proximal and distal extent, relationship to renal arteries, and presence of anatomic anomalies. Permits evaluation of the hemodynamic and functional significance of renal artery stenosis.</td>
<td>No ionizing radiation. No iodinated contrast needed.</td>
<td>Subject to motion artifacts. Special instrumentation required for patients on life support. <strong>Contraindications and risks:</strong> Contraindicated in patients with cardiac pacemakers, intraocular metallic foreign bodies, intracranial aneurysm clips, cochlear implants, and some artificial heart valves.</td>
<td>Sedation of agitated patients. Screening CT or plain radiograph images of orbits if history suggests possible metallic foreign body in the eye.</td>
</tr>
<tr>
<td>Blood Leukocyte Scan</td>
<td>Evaluation of fever of unknown origin, suspected abscess, pyelonephritis, osteomyelitis, and inflammatory bowel disease. Examination of choice for evaluation of suspected vascular graft infection.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Highly specific (98%) for infection (in contrast to gallium). Highly sensitive in detecting abdominal source of infection. In patients with fever of unknown origin, total body imaging is advantageous compared with CT scan or ultrasound. Preliminary imaging as early as 4 hours is possible with indium but less sensitive (30–50% of abscesses are detected at 24 hours).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>24-hour delayed imaging may limit the utility of indium scan in critically ill patients. False-negative scans occur with antibiotic administration or in chronic infection. Perihepatic or splenic infection can be missed because of normal leukocyte accumulation in these organs; liver and spleen scan is necessary adjunct in this situation. False-positive scans occur with swallowed leukocytes, bleeding, indwelling tubes and catheters, surgical skin wound uptake, and bowel activity due to inflammatory processes. Pulmonary uptake is nonspecific and has low predictive value for infection. Patients must be able to hold still during relatively long acquisition times (5–10 minutes). Tc99m-HMPAO WBC may be suboptimal for detecting infection involving the genitourinary and gastrointestinal tracts because of normal distribution of the agent to these organs. <strong>Contraindications and risks:</strong> Contraindicated in pregnancy because of the hazard of ionizing radiation to the fetus. High radiation dose to spleen.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Leukocytes from the patient are harvested, labeled in vitro, and then reinjected; process requires 12 hours. Scanning takes place 24 hours after injection of indium-labeled WBC and 1–2 hours after injection of Tc99m-HMPAO WBC. Homologous donor leukocytes should be used in neutropenic patients.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Scanning takes place 24 hours after injection of indium-labeled WBC and 1–2 hours after injection of Tc99m-HMPAO WBC. Homologous donor leukocytes should be used in neutropenic patients.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>$$–$$ $$–$$ $$–$$ $$–$$ $$–$$</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Basic Electrocardiography*

G. Thomas Evans, Jr., MD

HOW TO USE THIS SECTION

This chapter includes criteria for the diagnosis of basic electrocardiographic waveforms and cardiac arrhythmias. It is intended for use as a reference and assumes a basic understanding of the electrocardiogram (ECG).

Electrocardiographic interpretation is a “stepwise” procedure, and the first steps are to study and characterize the cardiac rhythm.

Step One

Categorize what you see in the 12-lead ECG or rhythm strip, using the three major parameters that allow for systematic analysis and subsequent diagnosis of the rhythm:

1. Mean rate of the QRS complexes (slow, normal, or fast).
2. Width of the QRS complexes (wide or narrow).
3. Rhythmicity of the QRS complexes (characterization of spaces between QRS complexes) (regular or irregular).

Based upon this categorization, refer to pages 286–299 for specific categories of rhythms. If the rhythm is irregularly irregular, go directly to page 288 (atrial fibrillation). For specific criteria for atrial flutter, go to page 288.

**Step Two**

Step 2 consists of examining and characterizing the morphology of the cardiac waveforms.

1. Examine for atrial abnormalities and bundle branch blocks (BBBs) (pages 301–302).
3. Examine for signs of left ventricular hypertrophy (pages 306–307).
4. Examine for signs of right ventricular hypertrophy (pages 307–308).
5. Examine for signs of myocardial infarction, if present (pages 310–320).
6. Bear in mind conditions that may alter the ability of the ECG to diagnose a myocardial infarction (page 320).
7. Examine for abnormalities of the ST segment or T wave (pages 320–323).
8. Assess the QT interval (pages 324–327).

**STEP 1: DIAGNOSIS OF THE CARDIAC RHYTHM**

**A. APPROACH TO DIAGNOSIS OF THE CARDIAC RHYTHM**

Most electrocardiograph machines display 10 seconds of data in a standard tracing. A rhythm is defined as three or more successive P waves or QRS complexes.

Categorize the patterns seen in the tracing according to a systematic method. This method proceeds in three steps that lead to a diagnosis based upon the most likely rhythm producing a particular pattern:

1. What is the mean rate of the QRS complexes?
   - **Slow** (<60 bpm): The easiest way to determine this is to count the total number of QRS complexes in a 10-second period. If there are no more than 9, the rate is slow.
Normal (60–100 bpm): If there are 10–16 complexes in a 10-second period, the rate is normal.

Fast (>100 bpm): If there are ≥17 complexes in a 10-second period, the rate is fast.

2. Is the duration of the dominant QRS morphology narrow (≤0.119 s) or wide (≥0.12 s)? (Refer to the section below on the QRS duration.)

3. What is the “rhythmicity” of the QRS complexes (defined as the spacing between QRS complexes)? Regular or irregular? (Any change in the spacing of the R-R intervals defines an irregular rhythm.)

Using the categorization above, refer to Tables 7–1 and 7–2 to select a specific diagnosis for the cardiac rhythm.

**TABLE 7–1. SUSTAINED REGULAR RHYTHMS.**

<table>
<thead>
<tr>
<th>Rate</th>
<th>Fast</th>
<th>Normal</th>
<th>Slow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Narrow QRS duration</td>
<td>Sinus tachycardia Atrial tachycardia Atrial flutter (2:1 AV conduction)</td>
<td>Sinus rhythm Ectopic atrial rhythm Atrial flutter (4:1 conduction)</td>
<td>Sinus bradycardia Ectopic atrial bradycardia</td>
</tr>
<tr>
<td>Wide QRS duration</td>
<td>All rhythms listed above under narrow QRS duration, but with BBB or intraventricular conduction delay (IVCD) patterns</td>
<td>Ventricular tachycardia Accelerated ventricular rhythm</td>
<td>Ventricular escape rhythm</td>
</tr>
</tbody>
</table>

**TABLE 7–2. SUSTAINED IRREGULAR RHYTHMS.**

<table>
<thead>
<tr>
<th>Rate</th>
<th>Fast</th>
<th>Normal</th>
<th>Slow</th>
</tr>
</thead>
<tbody>
<tr>
<td>Narrow QRS duration</td>
<td>Atrial fibrillation Atrial flutter (variable AV conduction) Multifocal atrial tachycardia Atrial tachycardia with AV block (rare)</td>
<td>Atrial fibrillation Atrial flutter (variable AV conduction) Multifocal atrial tachycardia</td>
<td>Atrial fibrillation Atrial flutter (variable AV conduction) Multiform atrial rhythm</td>
</tr>
<tr>
<td>Wide QRS duration</td>
<td>All rhythms listed above under narrow QRS duration, but with BBB or IVCD patterns</td>
<td>Rarely, anterograde conduc- tion of atrial fibrillation over an accessory pathway in patients with WPW syndrome</td>
<td></td>
</tr>
</tbody>
</table>
B. CATEGORIES OF QRS RHYTHM IRREGULARITY

A Dominant Regular Rhythm With Interruptions

This is the most common category of irregularity. Diagnoses include the following:

A. An **atrial pause**, defined as occasional abrupt pauses not initiated by premature QRS activity (and accompanied by a change in the P-P interval). Causes include: a nonconducted premature atrial complex (PAC) (most common); sinus pause (less common); atypical sinus arrhythmia (less common); and sinoatrial exit block (rare).

B. During **tachycardia**, occasional lengthening of the R–R cycle, unmasking the presence of either regular atrial activity or flutter waves.

C. **Premature QRS activity** that initiates a pause, called a **post-extrasystolic pause**, with either (1) a narrow QRS complex (most common) due to either a normally conducted premature atrial complex (PAC) or, rarely, a premature junctional complex (PJC); or (2) a wide or abnormal QRS complex, due to a premature ventricular complex (PVC), the most common cause of a de novo wide QRS complex; aberrant ventricular conduction of a premature supraventricular impulse (either a PAC or PJC); or, rarely, a PAC that conducts over an accessory pathway.

Aberrant Ventricular Conduction

Aberrant ventricular conduction is defined as an abnormal QRS complex formed by premature activation of the His-Purkinje system that results in block of the impulse in one of the bundle branches. Aberrant conduction is usually a normal phenomenon and does not imply disease of the conduction system. The PR interval of a PAC that causes aberrant ventricular conduction is commonly prolonged.

An Irregularly Irregular Rhythm

Irregularly irregular rhythms have successive RR intervals that occur in random patterns. One method of ascertaining this pattern is to place calipers on the first RR interval at the start of the tracing and to precisely adjust the calipers to each successive RR interval throughout the 10-second period. If there are random changes in the intervals, the rhythm is irregularly irregular.

An irregularly irregular rhythm is usually the QRS “footprint” of (1) atrial fibrillation (most common sustained abnormal cardiac rhythm), (2) atrial flutter (with variable AV conduction), (3) multifocal atrial
tachycardia (MAT), (4) atrial tachycardia with AV block, or (5) other less common rhythms.

Regularly Irregular Rhythm ("Group Beating")

Group beating is defined as clusters of regularly spaced QRS complexes, separated by pauses of identical duration. Whenever there is group beating, consider some form of Wenckebach periodicity, either during AV block or during junctional tachycardia with exit block in digitalis toxicity. Causes of group beating include the following:

A. Second-degree AV block, type I (Wenckebach) or type II (Mobitz II): There are usually single—but rarely multiple—nonconducted P waves in the setting of a constant PP interval.
B. PVCs in a repetitive pattern (ventricular trigeminy, quadrigeminy).
C. PACs or PJCs in a repetitive pattern (atrial trigeminy, etc).

Accelerating-Decelerating Rhythm

Causes include sinus arrhythmia (most common), defined as PP intervals that vary by > 10%; and sinoatrial exit block in a Wenckebach pattern (rare).

C. SINUS RHYTHMS

Sinus rhythms are defined by upright P waves in leads I, II, and aVF (present in 94% of normals) and are classified in Table 7–3.

D. ATRIAL RHYTHMS

Atrial rhythms, by definition, have nonsinus P waves. Focal atrial arrhythmias are defined as arrhythmias with a single focus (Table 7–4).

Multifocal Atrial Tachycardia (MAT)

Defined as having P waves with three or more morphologies per lead, with variable P-R intervals, and a mean atrial rate > 100/min. There is

<table>
<thead>
<tr>
<th>TABLE 7–3. SINUS RHYTHMS.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus rhythm</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
</tr>
<tr>
<td>Sinus arrhythmia</td>
</tr>
</tbody>
</table>
commonly nonconducted atrial activity. The baseline between T waves and P waves is isoelectric. The cause is COPD (60% of cases).

**Atrial Fibrillation**

Atrial fibrillation is the most common sustained abnormal cardiac rhythm. It is defined by the presence of fibrillatory waves that have a small amplitude and very rapid rate and are characterized by an inconstancy of morphology. They are best seen in leads V₁, V₂, II, aVF, and III.

**Atrial Flutter**

Classic atrial flutter, seen in two-thirds of patients, produces the waveforms shown below, usually at an atrial rate of 250–350/min.

| II, aVF, III | “Sawtooth” morphology |
| V₁ | Discrete upright “P” waves |

**E. JUNCTIONAL RHYTHMS**

“Junctional rhythm” is not recommended terminology for a final rhythm diagnosis because the specific subtypes have clinical implications.

**Definition of Junctional Complexes**

There are three possible relationships between the P waves and QRS complexes during junctional complexes or rhythms:

A. A constant PR interval ≥0.08 s with a 1:1 AV ratio.
B. No discernible P wave activity (P waves buried in the QRS complexes).
C. A retrograde P wave following the QRS complex.

**Definition of an Escape Complex or Rhythm**

An escape complex or rhythm occurs when a lower down, subsidiary (secondary) pacemaking site assumes the role of cardiac pacemaker
because of failure of a primary pacer site anatomically superior to the escape focus. Note: “Escape” always implies normal function of the structure that is escaping and that an anatomically superior pacemaker has failed. Escape rhythms are usually very regular.

**Definition of an Accelerated Complex or Rhythm**

In contrast, an accelerated rhythm always implies abnormal function of the structure that is accelerated. The lower rate limit of accelerated rhythms equals the upper rate limit of the escape rate of the structure but is $<101$ bpm. Accelerated rhythms are usually very regular.

**Classification of Junctional Rhythms**

Table 7–5 summarizes a useful classification of junctional rhythms.

**F. VENTRICULAR RHYTHMS**

**Definition of Ventricular Complexes**

Ventricular complexes are not initiated by atrial activity and have a morphology that is inconsistent with that of typical RBBB or LBBB. The QRS duration is $\geq 0.12$ s, usually between 0.14 s and 0.16 s. There are three major types of ventricular rhythms (Table 7–6).

**TABLE 7–5. THREE TYPES OF JUNCTIONAL RHYTHM.**

<table>
<thead>
<tr>
<th>Rhythm</th>
<th>Rate</th>
<th>Clinical Correlates</th>
</tr>
</thead>
<tbody>
<tr>
<td>Junctional escape rhythm</td>
<td>Rate $&lt;60$ bpm</td>
<td>Sinus node dysfunction or drug side effects</td>
</tr>
<tr>
<td>Accelerated junctional rhythm</td>
<td>Rate 61–100 bpm</td>
<td>Digitalis toxicity, post cardiac surgery, rheumatic fever, infections of the AV node area, idiopathic</td>
</tr>
<tr>
<td>Junctional tachycardia</td>
<td>Rate $&gt;100$ bpm</td>
<td>Same as accelerated junctional rhythm</td>
</tr>
</tbody>
</table>

**TABLE 7–6. MAJOR TYPES OF VENTRICULAR RHYTHMS.**

<table>
<thead>
<tr>
<th>Rhythm</th>
<th>Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ventricular escape rhythm</td>
<td>Rate $25–40$ bpm</td>
</tr>
<tr>
<td>Accelerated ventricular rhythm</td>
<td>Rate $41–100$</td>
</tr>
<tr>
<td>Ventricular tachycardia</td>
<td>Rate $&gt;100$ bpm</td>
</tr>
</tbody>
</table>

$^1$ Refer to definitions of escape and accelerated rhythms, above.
G. WIDE QRS COMPLEX TACHYCARDIA WITH A REGULAR RHYTHM (WCT-RR)

The most common cause of a wide QRS complex tachycardia with a regular rhythm (WCT-RR) is sinus tachycardia with either RBBB or LBBB. However, if a patient with structural heart disease presents with WCT-RR, one assumes a worst-case scenario and the presumptive diagnosis becomes ventricular tachycardia (VT). If the QRS complex in WCT-RR does not fit the typical pattern of either LBBB or RBBB, the diagnosis defaults to VT.

VT usually originates in an area at the border of infarcted and normal myocardium. Therefore, it does not require normal activation via the bundle branches or Purkinje system and produces an abnormal QRS complex.

Diagnosis of VT

Many criteria will diagnose VT with good performance, but no method will diagnose VT with 100% accuracy. Three methods—the “Quick” method, the Brugada algorithm, and the Griffith method (see below)—are commonly used but may yield different answers (VT or SVT). In some methods, 83% of VTs can be diagnosed using only the morphology of the QRS complex.

AV Dissociation in WCT-RR

The presence of AV dissociation or VA block in WCT-RR supersedes all other QRS morphologic criteria and is diagnostic of VT. However, during wide QRS complex tachycardia, it may be very difficult to identify atrial activity.

Regularity of RR Intervals in Ventricular Tachycardia

In ventricular tachycardia induced in the electrophysiology laboratory, the RR intervals were noted to be regular after 30 QRS complexes in 50% of patients and regular after 50 QRS complexes in 93% of patients. The mean rate of VT in these patients was 170 bpm. Therefore, a fast, wide, irregularly irregular rhythm that persists after 50 QRS complexes (about 18 seconds) is not likely to be ventricular tachycardia.

1. METHOD 1: QUICK METHOD FOR DIAGNOSIS OF VT (REQUIRES LEADS I, V₁, AND V₂)

This method derives from an analysis of typical waveforms of RBBB or LBBB as seen in leads I, V₁, and V₂. If the waveforms do not con-
form to either the common or uncommon typical morphologic patterns, the diagnosis defaults to VT.

**Step One**

Determine the morphologic classification of the wide QRS complexes (RB type or LB type), using the criteria below.

A. **Determination of the Morphologic Type of Wide QRS Complexes:** Use lead V1 only to determine the type of bundle branch block morphology of abnormally wide QRS complexes.

1. **RBBB and RBB type QRS complexes as seen in lead V1:** A wide QRS complex with a net positive area under the QRS curve is called the right bundle branch "type" of QRS. This does not mean that the QRS conforms exactly to the morphologic criteria for RBBB. Typical morphologies seen in RBBB are shown in the box at left below. Atypical morphologies at the right are most commonly seen in PVCs or during VT.

2. **LBBB and LBB-type QRS complexes as seen in lead V1:** A wide QRS complex with a net negative area under the QRS curve is called a left bundle branch "type" of QRS. This does not mean that the QRS conforms exactly to the morphologic criteria for LBBB. Typical morphologies of LBBB are shown in the box at left below. Atypical morphologies at the right are most commonly seen in PVCs or during VT.
Step Two

Apply criteria for common and uncommon normal forms of either RBBB or LBBB, as described below. The waveforms may not be identical, but the morphologic descriptions must match. If the QRS complexes do not match, the rhythm is probably VT.

A. RBBB: Lead I must have a terminal broad S wave, but the R/S ratio may be <1.

![Lead I](image1)

In lead V1, the QRS complex is usually triphasic but sometimes is notched and monophasic. The latter must have notching on the ascending limb of the R wave, usually at the lower left.

![Lead V1](image2)

B. LBBB: Lead I must have a monophasic, usually notched R wave and may not have Q waves or S waves.

![Lead I](image3)

Both lead V1 and lead V2 must have a dominant S wave, usually with a small, narrow R wave. S descent must be rapid and smooth, without notching.

![Leads V1 AND V2](image4)
2. METHOD 2: THE BRUGADA ALGORITHM FOR DIAGNOSIS OF VT

(Requires All Six Precordial Leads)

Brugada and coworkers reported on a total of 554 patients with WCT-RR whose mechanism was diagnosed in the electrophysiology laboratory. Patients included 384 (69%) with VT and 170 (31%) with SVT with aberrant ventricular conduction.

1. **Is there absence of an RS complex in ALL precordial leads?**
   If Yes ($n = 83$), VT is established diagnosis. (Sensitivity 21%, Specificity 100%). *Note:* Only QR, Qr, qR, QS, QRS, monophasic R, or rSR' are present. qRs complexes were not mentioned in the Brugada study.
   If No ($n = 471$), proceed to next step.

2. **Is the RS interval >100 ms in ANY ONE precordial lead?**
   If Yes ($n = 175$), VT is established diagnosis. (Sensitivity 66%, Specificity 98%). *Note:* The onset of R to the nadir of S is >100 ms (>2.5 small boxes) in a lead with an RS complex.

   ![RS](image)

   If No ($n = 296$), proceed to next step.

3. **Is there AV dissociation?**
   If Yes ($n = 59$), VT is established diagnosis. (Sensitivity 82%, Specificity 98%). *Note:* VA block also implies the same diagnosis.
   If No ($n = 237$), proceed to next step. *Note:* Antiarrhythmic drugs were withheld from patients in this study. Clinically, drugs that prolong the QRS duration may give a false-positive sign of VT using this criterion.

4. **Are morphologic criteria for VT present?**
   If Yes ($n = 59$), VT is established diagnosis. (Sensitivity 99%, Specificity 97%). *Note:* RBBB type QRS in $V_1$ versus LBBB type QRS in $V_1$ should be assessed as shown in the boxes below.
If No (n = 169)—and if there are no matches for VT in the boxes below—the diagnosis is SVT with aberration. (Sensitivity 97%, Specificity 99%).
3. METHOD 3: THE GRIFFITH METHOD FOR DIAGNOSIS OF VT (REQUIRES LEADS V₁ AND V₆)

This method derives from an analysis of typical waveforms of RBBB or LBBB as seen in both leads V₁ and V₆. If the waveforms do not conform to the typical morphologic patterns, the diagnosis defaults to VT.

**Step One**

Determine the morphologic classification of the wide QRS complexes (RB type or LB type), using the criteria above.

**Step Two**

Apply criteria for normal forms of either RBBB or LBBB, as described below. A negative answer to any of the three questions is inconsistent with either RBBB or LBBB, and the diagnosis defaults to VT.

**A. For QRS Complexes With RBBB Categorization:**

1. Is there an rSR' morphology in lead V₁?

   ![Diagram showing V₁ with an rSR' morphology](image_url)
2. Is there an RS complex in V₆ (may have a small septal Q wave)?

3. Is the R/S ratio in lead V₆ > 1?

**B. For QRS Complexes With LBBB Categorization:**
1. Is there an rS or QS complex in leads V₁ and V₂?

2. Is the onset of the QRS to the nadir of the S wave in lead V₁ < 70 ms?
3. Is there an R wave in lead V₆, without a Q wave?

**Torsade de Pointes**

Torsade de pointes ("twisting of the points") is defined as a pause-dependent polymorphic VT with a characteristic shifting morphology of the QRS complex that occurs in the setting of a prolonged QT interval. Clinical correlations include drug-induced states, congenital long QT syndrome, and hypokalemia.
H. AV BLOCK AND AV DISSOCIATION

1. AV BLOCK

Definitions of AV Block

If an atrial impulse has an “opportunity” to conduct normally and does not, then there is AV block. The relationship of P waves to QRS complexes determines the degree of AV block. An “opportunity” to conduct normally occurs when the P wave or atrial impulse enters the conducting system at a time other than during the effective or relative refractory periods of either the AV node or the bundle branches. The end of the T wave usually delineates the end of this period.

A. First-degree AV block is defined as prolongation of the PR interval (>0.21 s) with a 1:1 atrioventricular ratio.

B. Second-degree AV block is defined as occasional failure of conduction of a P wave, usually during a period of regular PP intervals. There are two major types of second-degree AV block:

1. Second-degree AV block type I (Wenckebach type I):
   The alerting sign is the presence of group beating, defined as clusters of mathematically spaced QRS complexes separated by pauses of identical duration. Criteria include the following:
   - There is usually a constant PP interval.
   - There is some, but not necessarily progressive, prolongation of successive PR intervals, leading to a nonconducted P wave that initiates a pause.
   - Twice the immediate RR interval that precedes the pause is longer than the RR interval that includes the pause.
   - The PR interval that precedes the pause is usually the longest in that Wenckebach cycle.
   - The PR interval that terminates the pause is usually the shortest P-R interval in that heart.

2. Second-degree AV block type II (Mobitz type II) is defined as sudden failure of conduction of a P wave during a period of regular PP intervals. The PR intervals preceding each conducted P wave are constant, and some P waves do not conduct. Rarely, after prior long RR cycles, the PR intervals may shorten by ≤0.02 s.

3. Third-degree AV block is defined as complete failure of conduction of all atrial impulses. The escape pacemaker originates from either the AV junction or the ventricle.
2. AV DISSOCIATION

Complete AV Dissociation

The P waves and QRS complexes are without relationship all of the time.

Incomplete AV Dissociation

The P waves and QRS complexes are without relationship most of the time. The two electrocardiographic manifestations of incomplete AV dissociation are the presence of either (1) a fusion complex, a blending of waveforms in the same chamber, with usually a waveform produced by an atrial impulse at the same time as one produced by a ventricular impulse; or (2) a capture complex, ie, a premature QRS complex produced by a P wave when the majority of QRS complexes are not produced by P waves. The P wave producing the ventricular capture is usually superimposed upon the ST segment or T wave caused by the prior QRS complex.

There are four basic disorders of impulse formation or conduction producing incomplete AV dissociation:

A. Slowing of the Primary Pacemaker: An example is sinus bradycardia with a junctional escape rhythm.

B. Acceleration of a Subsidiary Pacemaker: Examples are ventricular tachycardia (common); accelerated ventricular rhythm (common; requires no AV block); junctional tachycardia (less common); or accelerated junctional rhythm (less common; requires no AV block).

C. Third-Degree AV Block: Defined as complete failure of conduction of all atrial impulses. The escape pacemaker originates from one of two sites: the AV junction, producing the normal QRS complex seen in that heart, including RBBB or LBBB; or the ventricle (sub-His), producing a wide QRS complex lacking the classic pattern of either RBBB or LBBB.

D. Combinations, Usually of A and B: An example is relative slowing of the sinus in association with an accelerated junctional rhythm. The diagnostic hallmark is a capture complex.

I. PAROXYSMAL SUPRAVENTRICULAR TACHYCARDIA (PSVT)

This category encompasses several types of tachycardias (rate >100 bpm) that, by definition, are paroxysmal, ie, have sudden onset. The QRS complex is usually narrow in these arrhythmias, but occasionally it can show preexistent BBB or rate-dependent BBB. Approximately 90% of all
PSVTs are due to either AVRT or AVNRT. The four most common mechanisms of PSVT are as follows.

**Atrioventricular Reentry Tachycardia (AVRT)**

In this common arrhythmia, also called orthodromic reciprocating tachycardia (ORT), there is a reentrant circuit in which an accessory atrioventricular pathway conducts the impulse, usually in a retrograde direction from the ventricle to the atrium. The AV node-His-Purkinje axis conducts the impulse in an anterograde (orthodromic) direction from atrium to ventricle. The onset of atrial activation occurs just after the end of the QRS complex, producing a P wave located a short distance from the J point (a short-RP tachycardia).

**AV Nodal Reentry Tachycardia (AVNRT)**

This is a common reentrant rhythm involving tissues in close proximity to the AV node or within the node, some of which conduct rapidly and have a relatively long refractory period (“fast” pathway) and some of which conduct slowly and have a relatively short refractory period (“slow” pathway). About half the time, the P wave is buried within the QRS complex and is hidden, while in the remainder the P wave distorts the end of the QRS complex, producing apparent S waves in the inferior leads (pseudo-S waves) and apparent R’ waves in V1 (pseudo-R’ waves).

**Atrial Tachycardia**

This arrhythmia is uncommon.

**Atrial Flutter (Usually Atypical Flutter)**

In this uncommon arrhythmia, there is either 1:1 or 2:1 AV conduction, which may occasionally be very difficult to diagnose from the surface ECG. In these cases, drugs that block AV nodal conduction and prolong the RR intervals may allow for unmasking of the flutter waves.

**STEP 2: MORPHOLOGIC DIAGNOSIS OF THE CARDIAC WAVEFORMS**

**A. THE NORMAL ECG: TWO BASIC QRST PATTERNS**

The most common pattern is illustrated below and is usually seen in leads I or II and V5–6. There is a small “septal” Q wave <30 ms in duration. The T wave is upright. The normal ST segment, which is never normally isoelectric except sometimes at slow rates (<60 bpm), slopes
upward into an upright T wave, whose proximal angle is more obtuse than the distal angle. The normal T wave is never symmetric.

The pattern seen in the right precordial leads, usually V1–3, is shown below. There is a dominant S wave. The J point, the junction between the end of the QRS complex and the ST segment, is usually slightly elevated, and the T wave is upright. The T wave in V1 may occasionally be inverted as a normal finding in up to 50% of young women and 25% of young men, but this finding is usually abnormal in adult males. V2 usually has the largest absolute QRS and T wave magnitude of any of the 12 electrocardiographic leads.

B. ATRIAL ABNORMALITIES

Right Atrial Enlargement (RAE)

Diagnostic criteria include a positive component of the P wave in lead V1 or V2 ≥1.5 mm. Another criterion is a P wave amplitude in lead II >2.5 mm. Note: A tall, peaked P in lead II may represent RAE but is more commonly due to either COPD or increased sympathetic tone. Clinical correlation: RAE is seen with RVH.

Left Atrial Enlargement (LAE)

The most sensitive lead for the diagnosis of LAE is lead V1, but the criteria for lead II are more specific. Criteria include a terminal negative wave ≥1 mm deep and ≥40 ms wide (one small box by one small box in
area) and >40 ms between the first (right) and second (left) atrial components of the P wave in lead II, or a P wave duration >110 ms in lead II.

Clinical correlations: LVH, coronary artery disease, mitral valve disease, or cardiomyopathy.

C. BUNDLE BRANCH BLOCK

The normal QRS duration in adults ranges from 67 ms to 114 ms (Glasgow cohort). If the QRS duration is ≥120 ms (three small boxes or more on the electrocardiographic paper), there is usually an abnormality of conduction of the ventricular impulse. The most common causes are either RBBB or LBBB, shown above, page 291. However, other conditions may also prolong the QRS duration.

RBBB is defined by delayed terminal QRS forces that are directed to the right and anteriorly, producing broad terminal positive waves in leads V1 and aVR and a broad terminal negative wave in lead I.

LBBB is defined by delayed terminal QRS forces that are directed to the left and posteriorly, producing wide R waves in leads that face the left ventricular free wall and wide S waves in the right precordial leads.

RIGHT BUNDLE BRANCH BLOCK (RBBB)

Diagnostic Criteria

The diagnosis of uncomplicated complete right bundle branch block is made when the following criteria are met:

1. Prolongation of the QRS duration to 120 ms or more.
2. An rsr', rsR', or rSR' pattern in lead V1 or V2. The R' is usually greater than the initial R wave. In a minority of cases, a wide and notched R pattern may be seen.
3. Leads V6 and I show a QRS complex with a wide S wave (S duration is longer than the R duration, or >40 ms in adults).

(See common and uncommon waveforms for RBBB under Step Two, page 292, above).

ST–T changes in RBBB

In uncomplicated RBBB, the ST–T segment is depressed and the T wave inverted in the right precordial leads with an R' (usually only in lead V1 but occasionally in V2). The T wave is upright in leads I, V5, and V6.
LEFT BUNDLE BRANCH BLOCK (LBBB)

Diagnostic Criteria

The diagnosis of uncomplicated complete left bundle branch block is made when the following criteria are met:

1. Prolongation of the QRS duration to 120 ms or more.
2. There are broad and notched or slurred R waves in left-sided precordial leads V5 and V6, as well as in leads I and aVL. Occasion-ally, an RS pattern may occur in leads V5 and V6 in uncomplicated LBBB associated with posterior displacement of the left ventricle.
3. With the possible exception of lead aVL, Q waves are absent in the left-sided leads, specifically in leads V5, V6, and I.
4. The R peak time is prolonged to >60 ms in lead V5 or V6 but is normal in leads V1 and V2 when it can be determined.
5. In the right precordial leads V1 and V3, there are small initial r waves in the majority of cases, followed by wide and deep S waves. The transition zone in the precordial leads is displaced to the left. Wide QS complexes may be present in leads V1 and V2 and rarely in lead V3.

(See common and uncommon waveforms for LBBB under Step Two, page 292, above).

ST–T changes in LBBB

In uncomplicated LBBB, the ST segments are usually depressed and the T waves inverted in left precordial leads V5 and V6 as well as in leads I and aVL. Conversely, ST segment elevations and positive T waves are recorded in leads V1 and V2. Only rarely is the T wave upright in the left precordial leads.

D. INCOMPLETE BUNDLE BRANCH BLOCKS

Incomplete LBBB

The waveforms are similar to those in complete LBBB, but the QRS duration is <120 ms. Septal Q waves are absent in I and V6. Incomplete LBBB is synonymous with LVH and commonly mimics a delta wave in leads V5 and V6.

Incomplete RBBB

The waveforms are similar to those in complete RBBB, but the QRS duration is <120 ms. This diagnosis suggests RVH. Occasionally, in a
normal variant pattern, there is an rSr’ waveform in lead V1. In this case, the r’ is usually smaller than the initial r wave; this pattern is not indicative of incomplete RBBB.

**Intraventricular Conduction Delay or Defect (IVCD)**

If the QRS duration is ≥120 ms but typical waveforms of either RBBB or LBBB are not present, there is an intraventricular conduction delay or defect (IVCD). This pattern is common in dilated cardiomyopathy. An IVCD with a QRS duration of ≥170 ms is highly predictive of dilated cardiomyopathy.

**E. FASCICULAR BLOCKS (HEMIBLOCKS)**

**1. LEFT ANTERIOR FASCICULAR BLOCK (LAFB)**

**Diagnostic Criteria**

1. Mean QRS axis from −45 degrees to −90 degrees (possibly −31 to −44 degrees).
2. A qR pattern in lead aVL, with the R peak time, ie, the onset of the Q wave to the peak of the R wave ≥45 ms (slightly more than one small box wide), as shown below.

![Diagram of qR pattern in lead aVL](image)

Clinical correlations: Hypertensive heart disease, coronary artery disease, or idiopathic conducting system disease.

**2. LEFT POSTERIOR FASCICULAR BLOCK (LPFB)**

**Diagnostic Criteria**

1. Mean QRS axis from +90 degrees to +180 degrees.
2. A qR complex in leads III and aVF, an rS complex in leads aVL and I, with a Q wave ≥40 ms in the inferior leads.

Clinical correlations: LPFB is a diagnosis of exclusion. It may be seen in the acute phase of inferior myocardial injury or infarction or may result from idiopathic conducting system disease.
F. DETERMINATION OF THE MEAN QRS AXIS

The mean electrical axis is the average direction of the activation or repolarization process during the cardiac cycle. Instantaneous and mean electrical axes may be determined for any deflection (P, QRS, ST–T) in the three planes (frontal, transverse, and sagittal). The determination of the electrical axis of a QRS complex is useful for the diagnosis of certain pathologic cardiac conditions.

The Mean QRS Axis in the Frontal Plane (Limb Leads)

Arzbaecher developed the hexaxial reference system that allowed for the display of the relationships among the six frontal plane (limb) leads. A diagram of this system is shown below.

![Diagram of hexaxial reference system](image)

The normal range of the QRS axis in adults is −30 degrees to +90 degrees.

It is rarely important to precisely determine the degrees of the mean QRS. However, the recognition of abnormal axis deviations is critical since it leads to a presumption of disease. The mean QRS axis is derived from the net area under the QRS curves. The most efficient method of determining the mean QRS axis uses the method of Grant, which requires only leads I and II (see below). If the net area under the QRS curves in these leads is positive, the axis falls between −30 degrees and +90 degrees, which is the normal range of axis in adults. (The only exception to this rule is in RBBB, in which the first 60 ms of the QRS is used. Alternatively,
one may use the maximal amplitude of the R and S waves in leads I and II to assess the axis in RBBB.) Abnormal axes are shown below.

**Left Axis Deviation (LAD)**

The four main causes of left axis deviation (LAD) are as follows:

**A. Left Anterior Fascicular Block (LAFB):** See criteria above.

**B. Inferior MI:** There is a pathologic Q wave ≥30 ms either in lead aVF or lead II in the absence of ventricular preexcitation.

**C. Ventricular Preexcitation (WPW Pattern):** LAD is seen with inferior paraseptal accessory pathway locations. This can mimic inferoposterior MI. The classic definition of the Wolff-Parkinson-White (WPW) pattern includes a short PR interval (<120 ms); an initial slurring of the QRS complex, called a delta wave; and prolongation of the QRS complex to >120 ms. However, since this pattern may not always be present despite the presence of ventricular preexcitation, a more practical definition is an absent PR segment and an initial slurring of the QRS complex in any lead. The diagnosis of the WPW pattern usually requires sinus rhythm.

**D. COPD:** LAD is seen in 10% of patients with COPD.

**Right Axis Deviation (RAD)**

The four main causes of right axis deviation (RAD) are as follows:

**A. Right Ventricular Hypertrophy:** This is the most common cause (refer to diagnostic criteria, below). However, one must
first exclude acute occlusion of the posterior descending coronary
artery, causing LPFB, and exclude also items B and C below.

B. **Extensive Lateral and Apical MI:** Criteria include QS or Qr
patterns in leads I and aVL and in leads V₄₋₆.

C. **Ventricular Preexcitation (WPW Pattern):** RAD seen with
left posterosuperior accessory pathway locations. This can mimic
lateral MI.

D. **Left Posterior Fascicular Block (LPFB):** This is a diagnosis
of exclusion (see criteria above).

Right Superior Axis Deviation

This category is rare. Causes include RVH, apical MI, ventricular tachy-
cardia, and hyperkalemia. Right superior axis deviation may rarely be
seen as an atypical form of LAFB.

G. **VENTRICULAR HYPERTROPHY**

1. **LEFT VENTRICULAR HYPERTROPHY (LVH)**

The ECG is very insensitive as a screening tool for LVH, but electro-
cardiographic criteria are usually specific. Echocardiography is the
major resource for this diagnosis.

The best electrocardiographic criterion for the diagnosis of LVH
is the Cornell voltage, the sum of the R wave amplitude in lead aVL and
the S wave depth in lead V₃, adjusted for sex:

1. RaVL + SV₃ >20 mm (females), >25 mm (males). The R wave
   height in aVL alone is a good place to start.
2. RaVL >9 mm (females), >11 mm (males).
   Alternatively, application of the following criteria will diagnose
   most cases of LVH.
3. Sokolow-Lyon criteria: SV₁ + RV₅ or RV₆ (whichever R wave
   is taller) >35 mm (in patients age >35).
4. Romhilt-Estes criteria: Points are scored for QRS voltage (1 point),
   the presence of LAE (1 point), typical repolarization abnormalities
   in the absence of digitalis (1 point), and a few other findings. The
   combination of LAE (see above) and typical repolarization abnor-
   malities (see below) (score ≥5 points) will suffice for the diagnosis
   of LVH even when voltage criteria are not met.
5. RV₆ > RV₅ (usually occurs with dilated LV). First exclude
   anterior MI and establish that the R waves in V₅ are >7 mm tall
   and that in V₆ they are >6 mm tall before using this criterion.
**Repolarization Abnormalities in LVH**

Typical repolarization abnormalities in the presence of LVH are an ominous sign of end-organ damage. In repolarization abnormalities in LVH, the ST segment and T wave are directed opposite to the dominant QRS waveform in all leads. However, this directional rule does not apply either in the transitional lead (defined as a lead having an R wave height equal to the S wave depth) or in the transitional zone (defined as leads adjacent to the transitional lead) or one lead to the left in the precordial leads.

**Spectrum of Repolarization Abnormalities in LVH**

The waveforms below, usually seen in leads I, aVL, V₅, and V₆ but more specifically in leads with dominant R waves, represent hypothetical stages in the progression of LVH.

<table>
<thead>
<tr>
<th>Normal</th>
<th>LVH voltage</th>
<th>LVH voltage with minor T wave flattening</th>
<th>LVH voltage with minor T wave inversion</th>
</tr>
</thead>
<tbody>
<tr>
<td><img src="image1" alt="Normal Waveform" /></td>
<td><img src="image2" alt="LVH Waveform" /></td>
<td><img src="image3" alt="LVH Waveform with T wave flattening" /></td>
<td><img src="image4" alt="LVH Waveform with T wave inversion" /></td>
</tr>
</tbody>
</table>

**CLASSICAL:** LVH voltage with typical repolarization abnormalities (“strain”)

<table>
<thead>
<tr>
<th><img src="image5" alt="LVH Waveform with typical repolarization abnormalities" /></th>
<th><img src="image6" alt="LVH Waveform with typical repolarization abnormalities and QRS widening" /></th>
<th><img src="image7" alt="Incomplete LBBB (absent septal Q in leads I and V₆)" /></th>
<th><img src="image8" alt="Complete LBBB" /></th>
</tr>
</thead>
</table>

2. **RIGHT VENTRICULAR HYPERTROPHY (RVH)**

The ECG is insensitive for the diagnosis of RVH. In 100 cases of RVH from one echocardiography laboratory, only 33% had RAD because of the confounding effects of LV disease. Published electrocardiographic criteria for RVH are listed below, all of which have ≥97% specificity.

With rare exceptions, right atrial enlargement is synonymous with RVH.
Diagnostic Criteria

Recommended criteria for the electrocardiographic diagnosis of RVH are as follows:

1. Right axis deviation (>90 degrees), or
2. An R/S ratio ≥1 in lead V1 (absent posterior MI or RBBB), or
3. An R wave >7 mm tall in V1 (not the R’ of RBBB), or
4. An rsR’ complex in V1 (R’ ≥10 mm), with a QRS duration of <0.12 s (incomplete RBBB), or
5. An S wave >7 mm deep in leads V5 or V6 (in the absence of a QRS axis more negative than +30 degrees), or
6. RBBB with RAD (axis derived from first 60 ms of the QRS). (Consider RVH in RBBB if the R/S ratio in lead I is <0.5.)

A variant of RVH (type C loop) may produce a false-positive sign of an anterior MI.

Repolarization Abnormalities in RVH

The morphology of repolarization abnormalities in RVH is identical to those in LVH, when a particular lead contains tall R waves reflecting the hypertrophied RV or LV. In RVH, these typically occur in leads V1-2 or V3 and in leads aVF and III. This morphology of repolarization abnormalities due to ventricular hypertrophy is illustrated above. In cases of RVH with massive dilation, all precordial leads may overlie the diseased RV and may exhibit repolarization abnormalities.

H. LOW VOLTAGE OF THE QRS COMPLEX

Low-Voltage Limb Leads Only

Defined as peak-to-peak QRS voltage <5 mm in all limb leads.

Low-Voltage Limb and Precordial Leads

Defined as peak-to-peak QRS voltage <5 mm in all limb leads and <10 mm in all precordial leads. Primary myocardial causes include multiple or massive infarctions; infiltrative diseases such as amyloidosis, sarcoidosis, or hemochromatosis; and myxedema. Extracardiac causes include pericardial effusion, COPD, pleural effusion, obesity, anasarca, and subcutaneous emphysema. When there is COPD, expect to see low voltage in the limb leads as well as in leads V5 and V6.
I. PROGRESSION OF THE R WAVE IN THE PRECORDIAL LEADS

The normal R wave height increases from V₁ to V₅. The normal R wave height in V₅ is always taller than that in V₆ because of the attenuating effect of the lungs. The normal R wave height in lead V₃ is usually >2 mm.

“Poor R Wave Progression”

The term “poor wave progression” (PRWP) is a nonpreferred term because most physicians use this term to imply the presence of an anterior MI, though it may not be present. Other causes of small R waves in the right precordial leads include LVH, LAFB, LBBB, cor pulmonale (with the type C loop of RVH), and COPD.

Reversed R Wave Progression (RRWP)

Reversed R wave progression is defined as a loss of R wave height between leads V₁ and V₂ or between leads V₂ and V₃ or between leads V₃ and V₄. In the absence of LVH, this finding suggests anterior MI or precordial lead reversal.

J. TALL R WAVES IN THE RIGHT PRECORDIAL LEADS

Etiology

Causes of tall R waves in the right precordial leads include the following:

A. Right Ventricular Hypertrophy: This is the most common cause. There is an R/S ratio ≥1 or an R wave height >7 mm in lead V₁.

B. Posterior MI: There is an R wave ≥6 mm in lead V₁ or ≥15 mm in lead V₂. One should distinguish the tall R wave of RVH from the tall R wave of posterior MI in lead V₁. In RVH, there is a downsloping ST segment and an inverted T wave, usually with right axis deviation. In contrast, in posterior MI, there is usually an upright, commonly tall T wave and, because posterior MI is usually associated with concomitant inferior MI, a left axis deviation.

C. Right Bundle Branch Block: The QRS duration is prolonged, and typical waveforms are present (see above).
D. The WPW Pattern: Left-sided accessory pathway locations produce prominent R waves with an R/S ratio ≥ 1 in V1, with an absent PR segment and initial slurring of the QRS complex, usually best seen in lead V4.

E. Rare or Uncommon Causes: The normal variant pattern of early precordial QRS transition (not uncommon); the reciprocal effect of a deep Q wave in leads V5–6 (very rare); Duchenne’s muscular dystrophy (very rare); and chronic constrictive pericarditis (very rare); and reversal of the right precordial leads.

K. MYOCARDIAL INJURY, ISCHEMIA, AND INFARCTION

Definitions

A. Myocardial Infarction: Pathologic changes in the QRS complex reflect ventricular activation away from the area of infarction.

B. Myocardial Injury: Injury always points outward from the surface that is injured.
   2. Endocardial injury: Diffuse ST segment depression, which is really reciprocal to the primary event, reflected as ST elevation in aVR.

C. Myocardial Ischemia: Diffuse ST segment depression, usually with associated T wave inversion. It usually reflects subendocardial injury, reciprocal to ST elevation in lead aVR. In ischemia, there may only be inverted T waves with a symmetric, sharp nadir.

D. Reciprocal Changes: Passive electrical reflections of a primary event viewed from either the other side of the heart, as in epicardial injury, or the other side of the ventricular wall, as in subendocardial injury.

Steps in the Diagnosis of Myocardial Infarction

The following pages contain a systematic method for the electrocardiographic diagnosis of myocardial injury or infarction, arranged in seven steps. Following the steps will achieve the diagnosis in most cases.

Step 1: Identify the presence of myocardial injury by ST segment deviations.

Step 2: Identify areas of myocardial injury by assessing lead groupings.
Step 3: Define the primary area of involvement and identify the culprit artery producing the injury.

Step 4: Identify the location of the lesion in the artery in order to risk-stratify the patient.

Step 5: Identify any electrocardiographic signs of infarction found in the QRS complexes.

Step 6: Determine the age of the infarction by assessing the location of the ST segment in leads with pathologic QRS abnormalities.

Step 7: Combine all observations into a final diagnosis.

**STEPS 1 AND 2**

Identify presence of and areas of myocardial injury.

The GUSTO study of patients with ST segment elevation in two contiguous leads defined four affected areas as set out in Table 7–7.

Two other major areas of possible injury or infarction were not included in the GUSTO categorization because they do not produce ST elevation in two contiguous standard leads. These are:

1. **Posterior Injury:** The most commonly used sign of posterior injury is ST depression in leads V1–3, but posterior injury may best be diagnosed by obtaining posterior leads V7, V8, and V9.

2. **Right Ventricular Injury:** The most sensitive sign of RV injury, ST segment elevation ≥1 mm, is found in lead V4R. A very specific—but insensitive—sign of RV injury or infarction is ST elevation in V1, with concomitant ST segment depression in V2 in the setting of ST elevation in the inferior leads.

**STEP 3**

Identify the primary area of involvement and the culprit artery.

**TABLE 7–7. GUSTO STUDY DEFINITIONS.**

<table>
<thead>
<tr>
<th>Area of ST Segment Elevation</th>
<th>Leads Defining This Area</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior (Ant)</td>
<td>V1–4</td>
</tr>
<tr>
<td>Apical (Ap)</td>
<td>V5–6</td>
</tr>
<tr>
<td>Lateral (Lat)</td>
<td>I, aVL</td>
</tr>
<tr>
<td>Inferior (Inf)</td>
<td>II, aVF, III</td>
</tr>
</tbody>
</table>
Primary Anterior Area

ST elevation in two contiguous V₁-₄ leads defines a primary anterior area of involvement. The left anterior descending coronary artery (LAD) is the culprit artery. Lateral (I and aVL) and apical (V₅ and V₆) areas are contiguous to anterior (V₁-₄), so ST elevation in these leads signifies more myocardium at risk and more adverse outcomes.

Primary Inferior Area

ST segment elevation in two contiguous leads (II, aVF, or III) defines a primary inferior area of involvement. The right coronary artery (RCA) is usually the culprit artery. Apical (V₅ and V₆), posterior (V₁-₃ or V₇-₉) and right ventricular (V₄R) areas are contiguous to inferior (II, aVF, and III), so ST elevation in these contiguous leads signifies more myocardium at risk and more adverse outcomes (see below).

The Culprit Artery

In the GUSTO trial, 98% of patients with ST segment elevation in any two contiguous V₁-₄ leads, either alone or with associated changes in leads V₅-₆ or I and aVL, had left anterior descending coronary artery obstruction. In patients with ST segment elevation only in leads II, aVF, and III, there was right coronary artery obstruction in 86%.

PRIMARY ANTERIOR PROCESS

Acute occlusion of the LAD coronary artery produces a sequence of changes in the anterior leads (V₁-₄).

Earliest Findings

A. “Hyperacute” Changes: ST elevation with loss of normal ST segment concavity, commonly with tall, peaked T waves.

B. Acute Injury: ST elevation, with the ST segment commonly appearing as if a thumb has been pushed up into it.
Evolutionary Changes

A patient who presents to the emergency department with chest pain and T wave inversion in leads with pathologic Q waves is most likely to be in the evolutionary or completed phase of infarction. Successful revascularization usually causes prompt resolution of the acute signs of injury or infarction and results in the electrocardiographic signs of a fully evolved infarction. The tracing below shows QS complexes in lead V2.

A. Development of Pathologic Q Waves (Infarction): Pathologic Q waves develop within the first hour after onset of symptoms in at least 30% of patients.

B. ST Segment Elevation Decreases: T wave inversion usually occurs in the second 24-hour period after infarction.

C. Fully Evolved Pattern: Pathologic Q waves, ST segment rounded upward, T waves inverted.
PRIMARY INFERIOR PROCESS

A primary inferior process usually develops after acute occlusion of the right coronary artery, producing changes in the inferior leads (II, III, and aVF).

Earliest Findings

The earliest findings are of acute injury (ST segment elevation). The J point may “climb up the back” of the R wave (a), or the ST segment may rise up into the T wave (b).

Evolutionary Changes

ST segment elevation decreases and pathologic Q waves develop. T wave inversion may occur in the first 12 hours of an inferior MI—in contrast to that in anterior MI.

Right Ventricular Injury or Infarction

With RV injury, there is ST segment elevation, best seen in lead V_4R. With RV infarction, there is a QS complex.

For comparison, the normal morphology of the QRS complex in lead V_4R is shown below. The normal J point averages +0.2 mm.
POSTERIOR INJURY OR INFARCTION

Posterior injury or infarction is commonly due to acute occlusion of the left circumflex coronary artery, producing changes in the posterior leads (V7, V8, V9) or reciprocal ST segment depression in leads V1–3.

**Acute Pattern**

Acute posterior injury or infarction is shown by ST segment depression in V1–3 and perhaps also V4, usually with upright (often prominent) T waves.

**Chronic Pattern**

Chronic posterior injury or infarction is shown by pathologic R waves with prominent tall T waves in leads V1–3.

**STEP 4**

Identify the location of the lesion within the artery in order to risk stratify the patient.
Primary Anterior Process

Aside from an acute occlusion of the left main coronary artery, occlusion of the proximal left anterior descending coronary artery conveys the most adverse outcomes. Four electrocardiographic signs indicate proximal LAD occlusion:

1. ST elevation > 1 mm in lead I, in lead aVL, or in both
2. New RBBB
3. New LAFB
4. New first-degree AV block

Primary Inferior Process

Nearly 50% of patients with IMI have distinguishing features that may produce complications or adverse outcomes unless successfully managed:

1. Precordial ST segment depression in V1–3 (suggests concomitant posterior wall involvement);
2. Right ventricular injury or infarction (identifies a proximal RCA lesion);
3. AV block (implies a greater amount of involved myocardium);
4. The sum of ST segment depressions in leads V4–6 exceeds the sum of ST segment depressions in leads V1–3 (suggests multi-vessel disease).

Reciprocal Changes in the Setting of Acute MI

ST depressions in leads remote from the primary site of injury are felt to be a purely reciprocal change. With successful reperfusion, the ST depressions usually resolve. If they persist, patients more likely have significant three-vessel disease and so-called ischemia at a distance. Mortality rates are higher in such patients.

STEP 5

Identify Electrocardiographic Signs of Infarction in the QRS Complexes

The 12-lead ECG shown below contains numbers corresponding to pathologic widths for Q waves and R waves for selected leads (see Table 7–8 for more complete criteria).
One can memorize the above criteria by mastering a simple scheme of numbers which represent the durations of pathological Q waves or R waves. Begin with lead V₁ and repeat the numbers in the box below in the following order. The numbers increase from “any” to 50.

| Any Q wave in lead V₄, for anterior MI |
| Q wave ≥ 20 ms in lead V₄, for anterior MI |
| Q wave ≥ 30 ms in lead V₅, for apical MI |
| Q wave ≥ 30 ms in lead V₆, for apical MI |
| Q wave ≥ 30 ms in lead I, for lateral MI |
| Q wave ≥ 30 ms in lead aVL, for lateral MI |
| Q wave ≥ 30 ms in lead II, for inferior MI |
| Q wave ≥ 30 ms in lead aVF, for inferior MI |
| R wave ≥ 40 ms in lead V₁, for posterior MI |
| R wave ≥ 50 ms in lead V₂, for posterior MI |

Test Performance Characteristics for Electrocardiographic Criteria in the Diagnosis of MI

Haisty and coworkers studied 1344 patients with normal hearts documented by coronary arteriography and 837 patients with documented MI (366 inferior, 277 anterior, 63 posterior, and 131 inferior and anterior) (Table 7–8). (Patients with LVH, LAFB, LPFB, RVH, LBBB, RBBB, COPD, or WPW patterns were excluded from analysis because these conditions can give false-positive results for MI.) Shown below are the
TABLE 7–8. DIAGNOSIS OF MYOCARDIAL INFARCTION.¹

<table>
<thead>
<tr>
<th>Infarct Location</th>
<th>ECG Lead</th>
<th>Criterion</th>
<th>Sensitivity</th>
<th>Specificity</th>
<th>Likelihood Ratio (+)</th>
<th>Likelihood Ratio (−)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inferior</td>
<td>II</td>
<td>Q ≥ 30 ms</td>
<td>45</td>
<td>98</td>
<td>22.5</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td>aVF</td>
<td>Q ≥ 30 ms</td>
<td>70</td>
<td>94</td>
<td>11.7</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Q ≥ 40 ms</td>
<td>40</td>
<td>98</td>
<td>20.0</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R/Q ≤ 1</td>
<td>50</td>
<td>98</td>
<td>25.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Anterior</td>
<td>V₁</td>
<td>Any Q</td>
<td>50</td>
<td>97</td>
<td>16.7</td>
<td>0.5</td>
</tr>
<tr>
<td></td>
<td>V₂</td>
<td>Any Q, or R ≤ 0.1 mV and R ≤ 10 ms, or RV₂ ≤ RV₁</td>
<td>80</td>
<td>94</td>
<td>13.3</td>
<td>0.2</td>
</tr>
<tr>
<td></td>
<td>V₃</td>
<td>Any Q, or R ≤ 0.2 mV, or R ≤ 20 ms</td>
<td>70</td>
<td>93</td>
<td>10.0</td>
<td>0.3</td>
</tr>
<tr>
<td></td>
<td>V₄</td>
<td>Q ≥ 20 ms</td>
<td>40</td>
<td>92</td>
<td>5.0</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R/Q ≤ 0.5, or R/S ≤ 0.5</td>
<td>40</td>
<td>97</td>
<td>13.3</td>
<td>0.6</td>
</tr>
<tr>
<td>Anterolateral (lateral)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td></td>
<td>Q ≥ 30 ms</td>
<td>10</td>
<td>98</td>
<td>5.0</td>
<td>0.9</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R/Q ≤ 1, or R ≤ 2 mm</td>
<td>10</td>
<td>97</td>
<td>3.3</td>
<td>0.9</td>
</tr>
<tr>
<td>aVL</td>
<td></td>
<td>Q ≥ 30 ms</td>
<td>7</td>
<td>97</td>
<td>0.7</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R/Q ≤ 1</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Apical</td>
<td>V₅</td>
<td>Q ≥ 30</td>
<td>5</td>
<td>99</td>
<td>5.0</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R/Q ≤ 2, or R ≤ 7 mm, or R/S ≤ 2, or notched R</td>
<td>60</td>
<td>91</td>
<td>6.7</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R/Q ≤ 1, or R/S ≤ 1</td>
<td>25</td>
<td>98</td>
<td>12.5</td>
<td>0.8</td>
</tr>
<tr>
<td></td>
<td>V₆</td>
<td>Q ≥ 30</td>
<td>3</td>
<td>98</td>
<td>1.5</td>
<td>1.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R/Q ≤ 3, or R ≤ 6 mm, or R/S ≤ 3, or notched R</td>
<td>40</td>
<td>92</td>
<td>25.0</td>
<td>0.7</td>
</tr>
<tr>
<td></td>
<td></td>
<td>R/Q ≤ 1, or R/S ≤ 1</td>
<td>10</td>
<td>99</td>
<td>10.0</td>
<td>0.9</td>
</tr>
</tbody>
</table>
sensitivity, specificity, and likelihood ratios for the best-performing infarct criteria. Notice that leads III and aVR are not listed: lead III may normally have a Q wave that is both wide and deep, and lead aVR commonly has a wide Q wave.

**Mimics of Myocardial Infarction**

Conditions that can produce pathologic Q waves, ST segment elevation, or loss of R wave height in the absence of infarction are set out in Table 7–9.

**STEP 6**

**Determine the Age of the Infarction**

An **acute infarction** manifests ST segment elevation in a lead with a pathologic Q wave. The T waves may be either upright or inverted.

An **old** or **age-indeterminate infarction** manifests a pathologic Q wave, with or without slight ST segment elevation or T wave abnormalities.
Persistent ST segment elevation $\geq 1$ mm after a myocardial infarction is a sign of dyskinetic wall motion in the area of infarct. Half of these patients have ventricular aneurysms.

**STEP 7**

**Combine Observations Into a Final Diagnosis**

There are two possibilities for the major electrocardiographic diagnosis: myocardial infarction or acute injury. If there are pathologic changes in the QRS complex, one should make a diagnosis of myocardial infarction—beginning with the primary area, followed by any contiguous areas—and state the age of the infarction. If there are no pathologic changes in the QRS complex, one should make a diagnosis of acute injury of the affected segments—beginning with the primary area and followed by any contiguous areas.

**L. ST SEGMENTS**

Table 7–10 summarizes major causes of ST segment elevations. Table 7–11 summarizes major causes of ST segment depressions or T wave inversions. The various classes and morphologies of ST–T waves as seen in lead V2 are shown in Table 7–12.
### TABLE 7–10. MAJOR CAUSES OF ST SEGMENT ELEVATION.

<table>
<thead>
<tr>
<th>Cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>LBBB</td>
</tr>
<tr>
<td>LVH (with repolarization abnormalities)</td>
</tr>
<tr>
<td>Ventricular pacemaker</td>
</tr>
<tr>
<td>Anterior injury, seen in leads V₁₋₄</td>
</tr>
</tbody>
</table>

- **Pulmonary embolism (acute cor pulmonale)**
  - New signs of any or all of the following: tachycardia; complete or incomplete RBBB; S₁, Q₃, T₃ pattern; right axis shift. There may be inferior or RV injury patterns. The most common cause of an S₁, Q₃, T₃ pattern is a completed inferior MI.

- **Inferior injury, seen in leads II, aVF,**
  - Acute pericarditis (stage I); diffuse ST elevation
  - Normal variant early repolarization, seen in aVL, I, II, aVF, V₄₋₆ (physiologic)

- **Hyperkalemia:** Patterns are best seen in leads V₄₋₅
  - Common: Tall, peaked, narrow-based symmetric T waves
  - The most common pattern: R/S ratio <1 in V₄, with broad, prominent S wave and symmetric, not necessarily peaked, T waves
  - An “M”-shaped QRS-ST-T morphology in leads V₄₋₅
Table 7–11. Major Causes of ST Segment Depression or T Wave Inversion.

<table>
<thead>
<tr>
<th>Whenever the ST segment or the T wave is directed counter to an expected repolarization abnormality, consider ischemia, healed MI, or drug or electrolyte effect.</th>
<th>In RBBB, there is an obligatory inverted T wave in right precordial leads with an R' (usually only in V1) or its equivalent (a qR complex in septal MI). An upright T in these leads suggests completed posterior MI.</th>
<th>Altered depolarization RBBB</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LBBB</strong></td>
<td><strong>LVH (with repolarization abnormality)</strong></td>
<td><strong>Subarachnoid hemorrhage</strong></td>
</tr>
<tr>
<td><img src="image" alt="V5" /></td>
<td><img src="image" alt="V6" /></td>
<td><img src="image" alt="V4" /></td>
</tr>
<tr>
<td><strong>RVH</strong></td>
<td><strong>Inferior subendocardial injury</strong></td>
<td><strong>Posterior subepicardial injury</strong></td>
</tr>
<tr>
<td><img src="image" alt="RVH" /></td>
<td><img src="image" alt="II" /></td>
<td><img src="image" alt="V2" /></td>
</tr>
<tr>
<td><strong>Anterior subendocardial injury or non-Q wave MI</strong></td>
<td><strong>Hypokalemia</strong></td>
<td><strong>Digitalis</strong></td>
</tr>
<tr>
<td><img src="image" alt="V4" /></td>
<td><img src="image" alt="V4" /></td>
<td><img src="image" alt="V4" /></td>
</tr>
<tr>
<td><strong>J point depression secondary to catecholamines</strong></td>
<td><strong>When K+ ≤ 2.8, 80% have ECG changes</strong></td>
<td><strong>PR interval and ST segment occupy the same curve</strong></td>
</tr>
</tbody>
</table>
### TABLE 7–12. VARIOUS CLASSES AND MORPHOLOGIES OF ST-T WAVES AS SEEN IN LEAD V2.1

<table>
<thead>
<tr>
<th>Description</th>
<th>Illustration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal ST segment (asymmetric upsloping ST segment with concavity, slight ST segment elevation)</td>
<td><img src="image" alt="Normal ST segment" /></td>
</tr>
<tr>
<td>Abnormal ST segment elevation or lack of normal upward concavity in the first part of the ST-T segment (as seen in LVH or acute ischemia or injury)</td>
<td><img src="image" alt="Abnormal ST segment" /></td>
</tr>
<tr>
<td>ST-T segment typical of acute or recent myocardial infarction, ie, the ST-T segment appears as though a thumb were pushed up into it</td>
<td><img src="image" alt="ST-T segment" /></td>
</tr>
<tr>
<td>Negative amplitudes in the latter part of the ST-T segment (may be seen in ischemia or old infarction)</td>
<td><img src="image" alt="Negative amplitudes" /></td>
</tr>
<tr>
<td>Negative T wave (may be a nonspecific sign, but may be seen in ischemia or old MI)</td>
<td><img src="image" alt="Negative T wave" /></td>
</tr>
<tr>
<td>Downward sloping in the first part of the ST-T segment (consider ischemia, digitalis, or hypokalemia)</td>
<td><img src="image" alt="Downward sloping" /></td>
</tr>
<tr>
<td>Flat ST-T segment (a nonspecific sign)</td>
<td><img src="image" alt="Flat ST-T segment" /></td>
</tr>
</tbody>
</table>

**Nonspecific ST segment or T wave abnormalities**

By definition, nonspecific abnormalities of either the ST segment (ones that are only slightly depressed or abnormal in contour) or T wave (ones that are either 10% the height of the R wave that produced it, or are either flat or slightly inverted) do not conform to the characteristic waveforms found above or elsewhere.

---

M. U WAVES

Normal U Waves
In many normal hearts, low-amplitude positive U waves < 1.5 mm tall that range from 160 ms to 200 ms in duration are seen in leads V2 or V3. Leads V2 and V3 are close to the ventricular mass, and small-amplitude signals may be best seen in these leads.
   Cause: Bradycardias.

Abnormal U Waves
Abnormal U waves have increased amplitude or merge with abnormal T waves and produce T–U fusion. Criteria include an amplitude ≥ 1.5 mm or a U wave that is as tall as the T wave that immediately precedes it.
   Causes: Hypokalemia, digitalis, antiarrhythmic drugs.

Inverted U Waves
These are best seen in leads V4–6.
   Causes: LVH, acute ischemia.
   Table 7–13 summarizes various classes and morphologies of ST–T–U abnormalities as seen in lead V4.

N. QT INTERVAL
A prolonged QT interval conveys adverse outcomes. The QT interval is inversely related to the heart rate. QT interval corrections for heart rate often use Bazett’s formula, defined as the observed QT interval divided by the square root of the RR interval in seconds. A corrected QT interval of ≥ 440 ms is abnormal.

Use of the QT Nomogram (Hodges Correction)
Measure the QT interval in either lead V2 or V3, where the end of the T wave can usually be clearly distinguished from the beginning of the U wave. If the rate is regular, use the mean rate of the QRS complexes. If the rate is irregular, calculate the rate from the immediately prior R-R cycle, because this cycle determines the subsequent QT interval. Use the numbers you have obtained to classify the QT interval using the
TABLE 7–13. VARIOUS CLASSES AND MORPHOLOGIES OF ST-T-U ABNORMALITIES AS SEEN IN LEAD V₄.

<table>
<thead>
<tr>
<th>Condition</th>
<th>ECG Appearance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Normal QT interval</td>
</tr>
<tr>
<td>Hypokalemia</td>
<td>Depressed, upsloping ST segment, low T wave, prominent U wave</td>
</tr>
<tr>
<td>Hypokalemia with T-U fusion (the most common pattern)</td>
<td>Depressed, upsloping ST segment, “tent-like” symmetric wide T wave, apparent long QT interval</td>
</tr>
<tr>
<td>Class Ia drug: quinidine, procainamide, disopyramide</td>
<td>Wide QRS, horizontally depressed ST segment, low T wave amplitude, prominent U, long QT</td>
</tr>
<tr>
<td>Digitalis</td>
<td>Bowl-shaped ST segment, low amplitude T wave, prominent U wave, short QT interval</td>
</tr>
<tr>
<td>Digitalis (possible toxicity)</td>
<td>“Checkmark”-shaped ST segment, T low to absent, first-degree AV block, short QT interval</td>
</tr>
<tr>
<td>Hypocalcemia</td>
<td>Long, straight ST segment, normal T wave, long QT interval</td>
</tr>
<tr>
<td>Hypercalcemia</td>
<td>Abbreviated ST segment, short or normal QT interval</td>
</tr>
</tbody>
</table>
nomogram below. Or remember that at heart rates of $\geq 40$ bpm, an observed QT interval $\geq 480$ ms is abnormal.

**Prolonged QT Interval**

The four major causes of a prolonged QT interval are as follows:

**A. Electrolyte Abnormalities:** Hypokalemia, hypocalcemia

**B. Drugs:** Also associated with torsade de pointes.

Class Ia antiarrhythmic agents: Quinidine, procainamide, disopyramide

Class Ic agents: Propafenone

Class III agents: Amiodarone, bretylium, N-acetylprocainamide, sotalol

Antihistamines: Astemizole, terfenadine

Antibiotics: Erythromycin, trimethoprim-sulfamethoxazole

Antifungals: Ketoconazole, itraconazole

Chemotherapeutics: Pentamidine, perhaps anthracyclines

Psychotropic agents: Tricyclic and heterocyclic antidepressants, phenothiazines, haloperidol

Toxins and poisons: Organophosphate insecticides

Miscellaneous: Cisapride, prednisone, probucol, chloral hydrate
C. Congenital Long QT Syndromes: Though rare, a congenital long QT syndrome should be considered in any young patient who presents with syncope or presyncope.

D. Miscellaneous Causes:
- Third-degree and sometimes second-degree A-V block
- At the cessation of ventricular pacing
- Left ventricular hypertrophy (usually minor degrees of lengthening)
- Myocardial infarction (in the evolutionary stages where there are marked repolarization abnormalities)
- Significant active myocardial ischemia
- Cerebrovascular accident (subarachnoid hemorrhage)
- Hypothermia

Short QT Interval

The four causes of a short QT interval are hypercalcemia, digitalis, thyrotoxicosis, and increased sympathetic tone.

O. MISCELLANEOUS ABNORMALITIES

Right-Left Arm Cable Reversal Versus Mirror Image Dextrocardia

Misplacement of the Right Leg Cable

This error should not occur but it does occur nevertheless. It produces a “far field” signal when one of the bipolar leads (I, II, or III) records the signal between the left and right legs. The lead appears to have no signal except for a tiny deflection representing the QRS complex. There
are usually no discernible P waves or T waves. RL–RA cable reversal is shown here.

**Early Repolarization Normal Variant ST–T Abnormality**

- Tall QRS voltage
- Sometimes sharp “fishhook” deformity at the J point, but usually slurring or notching
- Prominent T waves
- ST segment elevation, maximal in leads with tallest R waves

**Hypothermia**

Hypothermia is usually characterized on the ECG by a slow rate, a long QT, and muscle tremor artifact. An Osborn wave is typically present.
**Acute Pericarditis: Stage I**  
*(With PR Segment Abnormalities)*

There is usually widespread ST segment elevation with concomitant PR segment depression in the same leads. The PR segment in aVR protrudes above the baseline like a knuckle, reflecting atrial injury.

![Diagram of II and aVR leads showing PR segment abnormalities.](image)

**Differentiating Pericarditis From Early Repolarization**

Only lead V6 is used. If the indicated amplitude ratio A/B is ≥25%, suspect pericarditis. If A/B <25%, suspect early repolarization.

![Diagram of V6 leads showing amplitude ratio](image)

**Wolff-Parkinson-White Pattern**

The WPW pattern is most commonly manifest as an absent PR segment and initial slurring of the QRS complex in any lead. The lead with the best sensitivity is V4.

![Diagram of V4 lead showing tall R waves](image)
A. **Left Lateral Accessory Pathway:** This typical WPW pattern mimics lateral or posterior MI.

I, aVL

V₁

B. **Posteroseptal Accessory Pathway:** This typical WPW pattern mimics inferoposterior MI.

I

II, aVF

**COPD Pattern, Lead II**

The P wave amplitude in the inferior leads is equal to that of the QRS complexes.

Prominent P waves with low QRS voltage

**REFERENCES**


This page intentionally left blank.
HOW TO USE THIS SECTION

This section includes algorithms, nomograms, and tables, arranged alphabetically by subject, designed to be used in the selection and interpretation of appropriate laboratory tests.

A conventional algorithm layout is displayed below. Diagnostic tests are enclosed in ovals; diagnoses in italics; and treatment recommendations in rectangles.

SUSPECTED DIAGNOSIS/CLINICAL SITUATION

Diagnostic test

Test abnormal

Test normal

Diagnosis

Treatment

Diagnosis
Abbreviations used throughout this section include the following:

N  =  Normal
Abn  =  Abnormal
Pos  =  Positive
Neg  =  Negative
Occ  =  Occasional
↑  =  Increased or high
↓  =  Decreased or low

Contents
Acetaminophen toxicity: Nomogram (Figure 8–1)..........................336
Acid-base disturbances: Laboratory characteristics
   (Table 8–1)...................................................................................362
Acid-base nomogram (Figure 8–2)..................................................337
Adrenocortical insufficiency: Diagnostic algorithm
   (Figure 8–3)...................................................................................338
Amenorrhea: Diagnostic algorithm (Figure 8–4)............................339
Anemia: Diagnosis based on RBC indices (Table 8–2)...................363
Anemia, microcytic: Laboratory evaluation (Table 8–3)..................364
Ascites: Ascitic fluid profiles in various disease states
   (Table 8–4)...................................................................................365
Autoantibodies in connective tissue diseases (Table 8–5)..............367
Cerebrospinal fluid profiles in CNS diseases (Table 8–6)..............369
Child’s criteria for severity of hepatic dysfunction
   (Table 8–7)...................................................................................372
Cushing’s syndrome: Diagnostic algorithm (Figure 8–5)..............340
Dermatome charts (Figure 8–6).......................................................341
Genetic diseases diagnosed by molecular diagnostic techniques (Table 8–8).......................................................373
Hemostatic function: Laboratory evaluation (Table 8–9)...............377
Hepatic function tests (Table 8–10)................................................378
Hepatitis A: Serologic changes (Figure 8–7).................................343
Hepatitis B: Serologic changes (Figure 8–8).................................344
Hepatitis C: Acute and chronic typical course (Figure 8–9).........345
Hirsutism: Diagnostic algorithm (Figure 8–10).............................346
Hypercalcemia: Diagnostic approach (Figure 8–11).....................347
Hyperlipidemia: Laboratory findings (Table 8–11)......................379
Hypertension with hypokalemia: Diagnostic algorithm
   (Figure 8–12).............................................................................348
Hypoglycemia: Diagnostic algorithm (Figure 8–13).....................349
Hyponatremia: Diagnostic algorithm (Figure 8–14).....................350
Hypothyroidism: Diagnostic algorithm (Figure 8–15)..................351
Male infertility: Diagnostic algorithm (Figure 8–16) ......................352
Myocardial enzymes after acute myocardial infarction
(Figure 8–17) .................................................................................353
The osmolal gap in toxicology (Table 8–12).................................381
Parathyroid hormone and calcium nomogram (Figure 8–18)......354
Pheochromocytoma: Diagnostic algorithm (Figure 8–19) ..........355
Pleural fluid profiles in various disease states (Table 8–13).........382
Prenatal diagnostic methods: Amniocentesis and chorionic villus sampling (Table 8–14) .........................................................384
Pulmonary embolus: Diagnostic algorithm (Figures 8–20A
and 8–20B)...................................................................................356
Pulmonary function tests: Interpretation (Table 8–15) ...............385
Pulmonary function tests: Spirometry (Figure 8–21) ..................358
Ranson’s criteria for severity of acute pancreatitis
(Table 8–16) ..................................................................................386
Renal failure: Estimated creatinine clearance (Figure 8–22) ....359
Renal failure: Classification and differential diagnosis
(Table 8–17) ..................................................................................387
Renal tubular acidosis: Laboratory diagnosis (Table 8–18) ....388
Salicylate toxicity: Nomogram (Figure 8–23) ......................... 360
Synovial fluid: Classification of synovial (joint) fluid
(Table 8–19) ..................................................................................389
Syphilis: Laboratory diagnosis in untreated patients
(Table 8–20) ..................................................................................391
α-Thalassemia syndromes (Table 8–21) ......................................392
β-Thalassemia syndromes (Table 8–22) ......................................392
Thyroid function tests (Table 8–23) ............................................393
Urine composition in common diseases states (Table 8–24) .....395
Vaginal discharge: Laboratory evaluation (Table 8–25) ..........397
Valvular heart disease: Diagnostic evaluation (Table 8–26) ....398
White blood cell count and differential (Table 8–27) ...............400
Transfusion: Summary chart of blood components (Table 8–28)...401
Figure 8–1. ACETAMINOPHEN TOXICITY: Nomogram for prediction of acetaminophen hepatotoxicity following acute overdosage. The upper line defines serum acetaminophen concentrations known to be associated with hepatotoxicity; the lower line defines serum levels 25% below those expected to cause hepatotoxicity. To give a margin for error, the lower line should be used as a guide to treatment. (Modified and reproduced, with permission, from Rumack BH, Matthew H: Acetaminophen poisoning and toxicity. Pediatrics 1975;55:871. Reproduced by permission of Pediatrics. Copyright © 1975. Permission obtained also from Saunders CE, Ho MT [editors]: Current Emergency Diagnosis & Treatment, 4th ed. Originally published by Appleton & Lange. Copyright © 1992 by The McGraw-Hill Companies, Inc.)
Figure 8–2. ACID-BASE NOMOGRAM: Shown are the 95% confidence limits of the normal respiratory and metabolic compensations for primary acid-base disturbances. (Reproduced, with permission, from Cogan MG [editor]: Fluid and Electrolytes: Physiology & Pathophysiology. Originally published by Appleton & Lange. Copyright © 1991 by The McGraw-Hill Companies, Inc.)
ADRENOCORTICAL INSUFFICIENCY SUSPECTED

Rapid ACTH stimulation test

Cortisol abnormal
- Adrenal insufficiency
  - Obtain plasma ACTH; Perform 2-day ACTH stimulation test
    - Plasma ACTH elevated
      - No response to exogenous ACTH
        - Primary adrenocortical insufficiency
    - Plasma ACTH normal
      - Adequate response to exogenous ACTH
        - Secondary adrenocortical insufficiency

Cortisol normal
- Excludes primary adrenocortical insufficiency
  - Adrenal atrophy excluded, decreased ACTH reserve not excluded
    - Metyrapone blockade
      - 11-Deoxycortisol (Compound S) abnormal

---

1 In the rapid ACTH stimulation test, a baseline cortisol sample is obtained; cosyntropin, 10–25 µg, is given IM or IV; and plasma cortisol samples are obtained 30 or 60 minutes later.
2 The normal response is a cortisol increment >7 µg/dL. If a cortisol level of >18 µg/dL is obtained, the response is normal regardless of the increment.
3 Administer ACTH, 250 µg every 8 hours, as a continuous infusion for 48 hours, and measure daily urinary 17-hydroxycorticosteroids (17-OHCS) or free cortisol excretion and plasma cortisol. Urinary 17-OHCS excretion of >27 mg during the first 24 hours and >47 mg during the second 24 hours is normal. Plasma cortisol >20 µg/dL at 30 or 60 minutes after infusion is begun and >25 µg/dL 6–8 hours later is normal.
4 Metyrapone blockade is performed by giving 2–2.5 g metyrapone orally at 12 midnight. Draw cortisol and 11-deoxycortisol levels at 8 AM. 11-Deoxycortisol level <7 µg/dL indicates secondary adrenal insufficiency (as long as there is adequate blockade of cortisol synthesis [cortisol level <10 µg/dL]).

Figure 8–4. AMENORRHEA: Diagnostic evaluation of amenorrhea. PRL = prolactin; TSH = thyroid-stimulating hormone; FSH = follicle-stimulating hormone; LH = luteinizing hormone; CT = computed tomography; MRI = magnetic resonance imaging. (Modified, with permission, from Greenspan FS, Baxter JD [editors]: Basic & Clinical Endocrinology, 4th ed. Originally published by Appleton & Lange. Copyright © 1994 by The McGraw-Hill Companies, Inc.)
CUSHING’S SYNDROME SUSPECTED

24-hour urinary free cortisol
Low-dose dexamethasone suppression test¹

Abnormal
Plasma ACTH
High-dose dexamethasone suppression test²

ACTH undetectable
No suppression
Adrenal tumor
CT scan (adrenals)

ACTH normal to elevated
Dexamethasone suppression of serum cortisol to < 50% of baseline
Cushing’s disease
CT scan or MRI (pituitary)

ACTH elevated
No suppression
Ectopic ACTH syndrome

¹ Low dose: Give 1 mg dexamethasone at 11 PM; draw serum cortisol at 8 AM. Normally, AM cortisol is <5 µg/dL.
² High dose: Give 8 mg dexamethasone at 11 PM; draw serum cortisol at 8 AM or collect 24-hour urinary free cortisol. Normally, AM cortisol is <5 µg/dL or 24-hour urinary free cortisol is <20 µg.

Figure 8–5. CUSHING’S SYNDROME: Diagnostic evaluation of Cushing’s syndrome. ACTH = adrenocorticotropic hormone; CT = computed tomography; MRI = magnetic resonance imaging. (Modified, with permission, from Baxter JD, Tyrrell JB: The adrenal cortex. In: Endocrinology and Metabolism, 3rd ed. Felig P, Baxter JD, Frohman LA [editors]. McGraw-Hill, 1995; from Harvey AM et al [editors]: The Principles and Practice of Medicine, 22nd ed. Appleton & Lange, 1988; and from Greenspan FS, Baxter JD [editors]: Basic & Clinical Endocrinology, 4th ed. Originally published by Appleton & Lange. Copyright © 1994 by The McGraw-Hill Companies, Inc.)
Figure 8–6. DERMATOME CHART: Cutaneous innervation. The segmental or radicular (root) distribution is shown on the left side of the body and the peripheral nerve distribution on the right side. Above: posterior view; next page: anterior view. (Reproduced, with permission, from Aminoff MJ, Greenberg DA, Simon RP: Clinical Neurology, 3rd ed. Originally published by Appleton & Lange. Copyright © 1996 by The McGraw-Hill Companies, Inc.)
**Figure 8–7. HEPATITIS A:** Usual pattern of serologic changes in hepatitis A. HA = hepatitis A; AST = aspartate aminotransferase; ALT = alanine aminotransferase; Anti-HAV = hepatitis A virus antibody; IgM = immunoglobulin M; IgG = immunoglobulin G. (Reproduced, with permission, from Harvey AM et al [editors]: The Principles and Practice of Medicine, 22nd ed. Originally published by Appleton & Lange. Copyright © 1988 by The McGraw-Hill Companies, Inc.)
Usual Patterns of Hepatitis B Antigens and Antibodies

<table>
<thead>
<tr>
<th></th>
<th>HBsAg</th>
<th>Anti-HBc</th>
<th>Anti-HBs</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Very early</td>
<td>+</td>
<td>+ or −</td>
</tr>
<tr>
<td>B</td>
<td>Acute</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>C</td>
<td>Active HB with high titer Anti-HBc (“window”)</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>D</td>
<td>Convalescence</td>
<td>−</td>
<td>+</td>
</tr>
<tr>
<td>E</td>
<td>Recovery</td>
<td>−</td>
<td>+ or −</td>
</tr>
<tr>
<td>F</td>
<td>Chronic carrier</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

Figure 8–8. HEPATITIS B: Usual pattern of serologic changes in hepatitis B (HB). HBV = hepatitis B virus; HBsAg = hepatitis B surface antigen; Anti-HBc = hepatitis B core antibody; Anti-HBs = hepatitis B surface antibody; AST = aspartate aminotransferase; ALT = alanine aminotransferase. (Modified and reproduced, with permission, from Harvey AM et al [editors]: The Principles and Practice of Medicine, 22nd ed. Originally published by Appleton & Lange. Copyright © 1988 by The McGraw-Hill Companies, Inc.)
Figure 8–9. HEPATITIS C: The typical course of chronic hepatitis C. ALT = alanine aminotransferase; Anti-HCV = antibody to hepatitis C virus by enzyme immunoassay; HCV RNA [PCR] = hepatitis C viral RNA by polymerase chain reaction. (Reproduced, with permission, from Tierney LM Jr, McPhee SJ, Papadakis MA [editors]: Current Medical Diagnosis & Treatment 2000. McGraw-Hill, 2000.)
HIRSUTISM

Signs of Cushing’s syndrome?

No

Yes

Evaluate for Cushing’s syndrome (see Figure 8-5)

Serum DHEAS

High (>700 µg/dL)

Low (<700 µg/dL)

DHEAS suppressible?*

No

Yes

Adrenal neoplasm

Adrenal hyperplasia

Hypothalamic dysfunction

Polycystic ovary syndrome

Ovarian neoplasm

Serum testosterone

<2 x normal

>2 x normal

LH/FSH ratio

<2.0

>2.0

Pelvic CT scan

Figure 8–10. HIRSUTISM: Evaluation of hirsutism in females. Exceptions occur that do not fit this algorithm. CT = computed tomography; DHEAS = dehydroepiandrosterone sulfate; FSH = follicle-stimulating hormone; LH = luteinizing hormone. (*DHEAS <170 µg/dL after dexamethasone 0.5 mg orally every 6 hours for 5 days, with DHEAS repeated on the fifth day.) (Reproduced, with permission, from Fitzgerald PA [editor]: Handbook of Clinical Endocrinology, 2nd ed. Originally published by Appleton & Lange. Copyright © 1992 by The McGraw-Hill Companies, Inc.)
HYPERCALCEMIA

No obvious malignancy

Measure serum PTH

"Normal"¹ or high PTH

Measure calcium/creatinine clearance ratio

>0.02

Primary hyperparathyroidism

<0.01

Familial hypocalciuric hypercalcemia

Low PTH

Bone survey, bone scan

No obvious malignancy

Serum 1,25-OH vitamin D, 25-OH vitamin D levels

Normal or low

No diagnosis

Thyrotoxicosis

Immobilization

Milk-alkali syndrome

Myeloma/lymphoma

Sarcoidosis and other granulomatous disease (high 1,25-OH vitamin D)

Vitamin D intoxication (high 25-OH vitamin D)

High

Osteolytic malignancy

Obvious malignancy

Measure serum PTH to exclude coexistent primary hyperparathyroidism²

Lytic lesions

¹"Normal" PTH in presence of hypercalcemia is inappropriate and indicative of primary hyperparathyroidism.

²PTH-related protein is high in solid tumors that cause hypercalcemia.

Figure 8-11. HYPERCALCEMIA: Diagnostic approach to hypercalcemia. PTH = parathyroid hormone. (Modified, with permission, from Harvey AM et al [editors]: The Principles and Practice of Medicine, 22nd ed. Originally published by Appleton & Lange. Copyright © 1988 by The McGraw-Hill Companies, Inc.)
Figure 8–12. HYPERTENSION WITH HYPOKALEMIA: Evaluation of secondary causes of hypertension associated with hypokalemia. (*Studies are performed during a high-sodium intake [120 meq Na+/d.]) (**In addition, plasma aldosterone may be measured at 8 AM supine after overnight recumbency and after 4 hours of upright posture.) (Reproduced, with permission, from Fitzgerald PA [editor]: Handbook of Clinical Endocrinology, 2nd ed. Originally published by Appleton & Lange. Copyright © 1992 by The McGraw-Hill Companies, Inc.)
HYPOGLYCEMIA (Fasting plasma glucose <45 mg/dL)

- Serum insulin
  - <6 µU/mL
    - Rapid ACTH stimulation test
      - Normal
      - Low
        - Adrenal insufficiency
          - Normal
          - Low
            - Evaluate for growth hormone deficiency
              - Severe liver disease
              - Severe kidney disease
              - Inanition
              - ?Nonpancreatic neoplasm

- >6 µU/mL
  - Insulin antibody
    - Positive
    - Negative
      - Insulinoma
        - C-peptide during hypoglycemia
          - >2 ng/mL
          - <2 ng/mL
            - Insulinoma
            - Surreptitious insulin administration
              - Positive
              - Negative
                - C-peptide and proinsulin during hypoglycemia
                  - C-peptide <2 ng/mL
                    - Proinsulin <20%
                    - Surreptitious short-term or human insulin administration
                  - C-peptide >2 ng/mL
                    - Proinsulin >20%
                    - Insulinoma

Figure 8–13. HYPOGLYCEMIA: Evaluation of fasting hypoglycemia in adults. (Reproduced, with permission, from Fitzgerald PA [editor]: Handbook of Clinical Endocrinology, 2nd ed. Appleton & Lange, 1992.)
HYPONATREMIA

Serum osmolality

Low

Elevated

Isotonic hyponatremia

Serum glucose
Serum lipids
Total protein

Pseudohyponatremia

Hyperlipidemia
Hyperproteinemia

Isotonic infusion

Glucose
Mannitol

Clinically assess extracellular fluid volume

Low

Normal

Elevated

Hypovolemic hypotonic hyponatremia

Low (UNa⁺ < 10)

Extrarenal loss

GI losses (diarrhea, vomiting, NG suction, pancreatitis)
Skin losses (burns, sweating)
Lung losses (third-spacing)

Renal loss

Diuretics, osmotic diuretics (urea, mannitol, glucose)
Urinary obstruction
Salt-losing nephritis
Ketonuria
Bicarbonaturia (RTA, metabolic alkalosis)

Isotonic hyponatremia

Serum glucose
Serum lipids
Total protein

Hypertonic hyponatremia

Serum glucose

Hyperglycemia
Hypertonic infusion

Glucose
Mannitol
Contrast agents

Isotonic infusion

Glucose
Mannitol

Clinically assess extracellular fluid volume

Low

Normal

Elevated

Hypervolemic hypotonic hyponatremia

Low (UNa⁺ < 10)

Extrarenal loss

GI losses (diarrhea, vomiting, NG suction, pancreatitis)
Skin losses (burns, sweating)
Lung losses (third-spacing)

Renal loss

Diuretics, osmotic diuretics (urea, mannitol, glucose)
Urinary obstruction
Salt-losing nephritis
Ketonuria
Bicarbonaturia (RTA, metabolic alkalosis)

Pseudohyponatremia

Hyperlipidemia
Hyperproteinemia

Isotonic infusion

Glucose
Mannitol

Clinically assess extracellular fluid volume

Low

Normal

Elevated

Hypovolemic hypotonic hyponatremia

Low (UNa⁺ < 10)

Extrarenal loss

GI losses (diarrhea, vomiting, NG suction, pancreatitis)
Skin losses (burns, sweating)
Lung losses (third-spacing)

Renal loss

Diuretics, osmotic diuretics (urea, mannitol, glucose)
Urinary obstruction
Salt-losing nephritis
Ketonuria
Bicarbonaturia (RTA, metabolic alkalosis)

Figure 8–14. HYPONATREMIA: Evaluation of hyponatremia. SIADH = syndrome of inappropriate antidiuretic hormone; UNa⁺ = urinary sodium (mg/dL). (Adapted, with permission, from Narins RG et al: Diagnostic strategies in disorders of fluid, electrolyte and acid-base homeostasis. Am J Med 1982;72:496.)
HYPOTHYROIDISM SUSPECTED

---

**No**

Patient takes thyroid hormone

Stop medication for 6 weeks

**FT4 normal**

**TSH normal**

---

**Yes**

Patient takes no thyroid hormone

Serum FT4 and TSH

**FT4 low**

**TSH high**

---

Primary hypothyroidism

Secondary hypothyroidism

TRH test

Normal response

No response

Hypothalamic lesion

Pituitary lesion

---

*Figure 8–15. HYPOTHYROIDISM: Diagnostic approach to hypothyroidism. FT4 = free thyroxine index; TSH = thyroid-stimulating hormone; TRH = thyroid-releasing hormone. (Modified, with permission, from Greenspan FS, Strewler GJ editors: Basic & Clinical Endocrinology, 5th ed. Originally published by Appleton & Lange. Copyright © 1996 by The McGraw-Hill Companies, Inc.)*
Figure 8–16. MALE INFERTILITY: Evaluation of male factor infertility. FSH = follicle-stimulating hormone; LH = luteinizing hormone; PRL = prolactin; T = testosterone. (Adapted, with permission, from Swerdloff RS, Boyers SM: Evaluation of the male partner of an infertile couple: An algorithmic approach. JAMA 1982;247:2418. Copyright © 1982 by The American Medical Association.)
Figure 8–17. MYOCARDIAL ENZYMES: Time course of serum enzyme concentrations after a typical myocardial infarction. CKMB = isoenzyme of creatine kinase.
Figure 8–18. PARATHYROID HORMONE AND CALCIUM NOMOGRAM: Relationship between serum intact parathyroid hormone (PTH) and serum calcium levels in patients with hypoparathyroidism, pseudohypoparathyroidism, nonparathyroid hypercalcemia, primary hyperparathyroidism, and secondary hyperparathyroidism. HPT = hyperparathyroidism. (Courtesy of GJ Strewler.)
PHEOCHROMOCYTOMA SUSPECTED
(Suspected secondary hypertension)

24-hour urine metanephrines

- High: Suspicion very high
- Low: Suspicion low

Plasma catecholamines\(^1\)

- Very high: Pheochromocytoma
- Normal or low: No further workup

CT scan abdomen

- Negative: Selective venous sampling and/or \(^{131}\)I MIBG scan

- Adrenal tumor: Operate
- Paraadrenal tumor: Angiography

- Positive: Operate
- Negative: Medical therapy, then relocalize later

\(^1\) Plasma catecholamines must be measured under controlled conditions.

Figure 8–19. PHEOCHROMOCYTOMA: Flow chart for investigation and localization of a possible pheochromocytoma. \(^{131}\)I MIBG = \(^{131}\)I metaiodobenzylguanidine. (Modified, with permission, from Welbourne RM, Khan O: Tumors of the Neuroendocrine System. In: Current Problems in Surgery. Year Book, 1984; and Stobo JD et al [editors]: The Principles and Practice of Medicine, 23rd ed. Originally published by Appleton & Lange. Copyright © 1996 by The McGraw-Hill Companies, Inc.)
Figure 8–20A. ALGORITHM FOR THE CLINICAL MODEL TO DETERMINE THE PRETEST PROBABILITY OF PULMONARY EMBOLISM (PE): Respiratory points consist of dyspnea or worsening of chronic dyspnea, pleuritic chest pain, chest pain that is nonretrosternal and nonpleuritic, an arterial oxygen saturation <92% while breathing room air that corrects with oxygen supplementation <40%, hemoptysis, and pleural rub. Risk factors are surgery within 12 weeks, immobilization (complete bed rest) for 3 or more days in the 4 weeks before presentation, previous deep venous thrombosis or objectively diagnosed pulmonary embolism, fracture of a lower extremity and immobilization of the fracture within 12 weeks, strong family history of deep venous thrombosis or pulmonary embolism (two or more family members with objectively proved events or a first-degree relative with hereditary thrombophilia), cancer (treatment ongoing, within the past 6 months, or in the palliative stages), the postpartum period, and lower extremity paralysis. JVP = jugular venous pressure; RBBB = right bundle-branch block. See Figure 8–20B opposite. (Reproduced, with permission, from Wells PS et al: Use of a clinical model for safe management of patients with suspected pulmonary embolism. Ann Intern Med 1998;129:997.)
Figure 8–20B. DIAGNOSTIC STRATEGY USED IN PATIENTS WITH SUSPECTED PULMONARY EMBOLISM. See Figure 8–20A opposite. (Reproduced, with permission, from Wells PS et al: Use of a clinical model for safe management of patients with suspected pulmonary embolism. Ann Intern Med 1998;129:997.)
Figure 8–21. PULMONARY FUNCTION TESTS: SPIROMETRY. Representative spirograms (upper panel) and expiratory flow-volume curves (lower panel) for normal (A), obstructive (B), and restrictive (C) patterns. (Reproduced, with permission, from Tierney LM Jr, McPhee SJ, Papadakis MA [editors]: Current Medical Diagnosis & Treatment 2000. McGraw-Hill, 2000.)
Nomogram and Procedure for Rapid Evaluation of Endogenous Creatinine Clearance

Figure 8–22. RENAL FAILURE: ESTIMATED CREATININE CLEARANCE. Siersback-Nielsen nomogram for estimation of creatinine clearance from serum creatinine.

(1) Identify the axis point along the reference line (R) around which the relation between the patient’s serum creatinine and creatinine clearance rotates. To do so, place a straightedge so as to connect the patient’s age (in years, for male or female) with the patient’s weight (in kilograms).

(2) Put a dot along the reference line where the rule and line intersect.

(3) Rotate the ruler to connect the patient’s serum creatinine and this dot, and determine where the ruler falls along the line, estimating the patient’s creatinine clearance.

Note: This nomogram is based on the assumption that an increase in weight represents an increase in lean body mass. Substantial error in the estimate occurs when a weight increase reflects obesity rather than increased lean body mass. In addition, the nomogram yields a much more accurate estimate in the presence of moderate to moderately severe renal impairment than in the presence of normal renal function. It should also not be relied upon in severe renal insufficiency (eg, serum creatinine >5 mg/dL or creatinine clearance <15 mL/min).

(Modified, with permission, from Harvey AM et al [editors]: The Principles and Practice of Medicine, 22nd ed. Originally published by Appleton & Lange. Copyright © 1988 by The McGraw-Hill Companies, Inc.)
Figure 8–23. SALICYLATE TOXICITY: Nomogram for determining severity of salicylate intoxication. Absorption kinetics assume acute ingestion of non-enteric-coated aspirin preparation. (Modified and reproduced, with permission, from Done AK: Significance of measurements of salicylate in blood in cases of acute ingestion. Pediatrics 1960;26:800. Permission obtained also from Saunders CE, Ho MT [editors]: Current Emergency Diagnosis & Treatment, 4th ed. Originally published by Appleton & Lange. Copyright © 1992 by The McGraw-Hill Companies, Inc.)
THYROID NODULE

FNA Biopsy

Malignant

Follicular neoplasm or suspicious

Benign

"Cold"

High risk

Operate

"Hot"

"Hot"

Observe; perhaps T4 therapy

T4 therapy

Low risk

T4 therapy

Figure 8–24. THYROID NODULE: Laboratory evaluation of a thyroid nodule. FNA = fine-needle aspiration; T4 = thyroxine. (Modified, with permission, from Greenspan FS, Strewler GJ [editors]: Basic & Clinical Endocrinology, 5th ed. Originally published by Appleton & Lange. Copyright © 1997 by The McGraw-Hill Companies, Inc.)
### TABLE 8–1. ACID-BASE DISTURBANCES: LABORATORY CHARACTERISTICS OF PRIMARY SINGLE DISTURBANCES OF ACID-BASE BALANCE. ¹

<table>
<thead>
<tr>
<th>Disturbance</th>
<th>Acute Primary Change</th>
<th>Arterial pH</th>
<th>([K^+]) (meq/L)</th>
<th>Anion Gap²</th>
<th>Clinical Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>None</td>
<td>7.35–7.45</td>
<td>3.5–5.0</td>
<td>8–12</td>
<td>None.</td>
</tr>
<tr>
<td>Respiratory acidosis</td>
<td>(P_{CO_2}) retention</td>
<td>↓</td>
<td>↑</td>
<td>(N)</td>
<td>Dyspnea, polypnea, respiratory outflow obstruction, ↑ anterior-posterior chest diameter, musical rales, wheezes. In severe cases, stupor, disorientation, coma.</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>(P_{CO_2}) depletion</td>
<td>↑</td>
<td>↓</td>
<td>(N) or ↓</td>
<td>Anxiety, breathlessness, frequent sighing, lungs usually clear to examination, positive Chvostek and Trousseau signs.</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>(HCO_3^-) depletion</td>
<td>↓</td>
<td>↑ or ↓</td>
<td>(N) or ↑</td>
<td>Weakness, air hunger, Kussmaul respiration, dry skin and mucous membranes. In severe cases, poor skin turgor, coma, hypotension, death.</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>(HCO_3^-) retention</td>
<td>↑</td>
<td>↓</td>
<td>(N)</td>
<td>Weakness, positive Chvostek and Trousseau signs, hyporeflexia.</td>
</tr>
</tbody>
</table>

¹ Reproduced, with permission, from Harvey AM et al (editors): The Principles and Practice of Medicine, 22nd ed. Originally published by Appleton & Lange. Copyright © 1988 by The McGraw-Hill Companies, Inc.

² Anion gap = \([Na^+]-([HCO_3^-]+[Cl^-]) = 8–12\) meq normally.
## TABLE 8-2. ANEMIA: DIAGNOSIS OF COMMON ANEMIAS BASED ON RED BLOOD CELL (RBC) INDICES.

<table>
<thead>
<tr>
<th>Type of Anemia</th>
<th>MCV (fL)</th>
<th>MCHC (g/dL)</th>
<th>Common Causes</th>
<th>Common Laboratory Abnormalities</th>
<th>Other Clinical Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Microcytic, hypochromic</td>
<td>&lt;80</td>
<td>&lt;32</td>
<td>Iron deficiency</td>
<td>Low reticulocyte count, low serum and bone marrow iron, high TIBC.</td>
<td>Mucositis, blood loss.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thalassemias</td>
<td>Reticulocytosis, abnormal red cell morphology, normal serum iron levels.</td>
<td>Asian, African, or Mediterranean descent.</td>
</tr>
<tr>
<td>Chronic lead poisoning</td>
<td></td>
<td></td>
<td></td>
<td>Chronic lead poisoning</td>
<td>Basophilic stippling of RBCs, elevated lead and free erythrocyte protoporphyrin levels.</td>
</tr>
<tr>
<td>Sideroblastic anemia</td>
<td></td>
<td></td>
<td></td>
<td>Sideroblastic anemia</td>
<td>High serum iron, ringed sideroblasts in bone marrow.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Population of hypochromic RBCs on smear.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Hemolysis</td>
<td>Hemoglobin low or absent, reticulocytosis, hyperbilirubinemia.</td>
<td>Hemoglobinuria, splenomegaly.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Chronic disease&lt;sup&gt;2&lt;/sup&gt;</td>
<td>Low serum iron, TIBC low or low normal.</td>
<td>Depends on cause.</td>
</tr>
<tr>
<td>Macrocytic, normochromic</td>
<td>&gt;101&lt;sup&gt;3&lt;/sup&gt;</td>
<td>&gt;36</td>
<td>Vitamin B&lt;sub&gt;12&lt;/sub&gt; deficiency</td>
<td>Hypersegmented PMNs; low serum vitamin B&lt;sub&gt;12&lt;/sub&gt; levels; achlorhydria.</td>
<td>Peripheral neuropathy; glossitis.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Folate deficiency</td>
<td>Hypersegmented PMNs; low folate levels.</td>
<td>Alcoholism; malnutrition.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Liver disease</td>
<td>Mean corpuscular volume usually &lt;120 fL; normal serum vitamin B&lt;sub&gt;12&lt;/sub&gt; and folate levels.</td>
<td>Signs of liver disease.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Reticulocytosis</td>
<td>Marked (&gt;15%) reticulocytosis.</td>
<td>Variable.</td>
</tr>
</tbody>
</table>


<sup>2</sup> May be microcytic, hypochromic.

<sup>3</sup> If MCV > 120–130, vitamin B<sub>12</sub> or folate deficiency is likely.

MCV = mean corpuscular volume; MCHC = mean corpuscular hemoglobin concentration; TIBC = total iron-binding capacity, serum; PMN = polymorphonuclear cell.
## TABLE 8–3. ANEMIA, MICROCYTIC: LABORATORY EVALUATION OF MICROCYTIC, HYPOCHROMIC ANEMIAS.¹

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>MCV (fL)</th>
<th>Serum Iron (µg/dL)</th>
<th>Iron-binding Capacity (µg/dL)</th>
<th>Transferrin Saturation (%)</th>
<th>Serum Ferritin (µg/L)</th>
<th>Free Erythrocyte Protoporphyrin (µg/dL)</th>
<th>Basophilic Stippling</th>
<th>Bone Marrow Iron Stores</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>80–100</td>
<td>50–175</td>
<td>250–460</td>
<td>16–60</td>
<td>16–300</td>
<td>&lt;35</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Iron deficiency anemia</td>
<td>↓</td>
<td>&lt;30</td>
<td>↑</td>
<td>&lt;16</td>
<td>&lt;12</td>
<td>↑</td>
<td>Absent</td>
<td>Absent</td>
</tr>
<tr>
<td>Anemia of chronic disease</td>
<td>N or ↓</td>
<td>&lt;30</td>
<td>N or ↓</td>
<td>N or ↓</td>
<td>N or ↑</td>
<td>↑</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Thalassemia minor</td>
<td>↓</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Usually present</td>
<td>Present</td>
</tr>
</tbody>
</table>

¹ Modified, with permission, from Harvey AM et al (editors): The Principles and Practice of Medicine, 22nd ed. Originally published by Appleton & Lange. Copyright © 1988 by The McGraw-Hill Companies, Inc.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Appearance</th>
<th>Fluid Protein (g/dL)</th>
<th>Serum-Ascites Albumin Gradient (SAAG)</th>
<th>Fluid Glucose (mg/dL)</th>
<th>WBC and Differential (per µL)</th>
<th>RBC (per µL)</th>
<th>Bacteriologic Gram Stain and Culture</th>
<th>Cytology</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Clear</td>
<td>&lt;3.0</td>
<td>Equal to plasma glucose</td>
<td>&lt;250</td>
<td>Few or none</td>
<td>Neg</td>
<td>Neg</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>TRANSUDATES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cirrhosis</td>
<td>Clear</td>
<td>&lt;3.0</td>
<td>High(^3)</td>
<td>N</td>
<td>&lt;250, MN</td>
<td>Few</td>
<td>Neg</td>
<td>Neg</td>
<td>Occasionally turbid, rarely bloody. Fluid LDH/serum LDH ratio &lt;0.6</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Clear</td>
<td>&lt;2.5</td>
<td>High(^3)</td>
<td>N</td>
<td>&lt;250, MN</td>
<td>Few</td>
<td>Neg</td>
<td>Neg</td>
<td></td>
</tr>
<tr>
<td>Nephrotic syndrome</td>
<td>Clear</td>
<td>&lt;2.5</td>
<td>Low(^4)</td>
<td>N</td>
<td>&lt;250, MN</td>
<td>Few</td>
<td>Neg</td>
<td>Neg</td>
<td></td>
</tr>
<tr>
<td>Pseudomyxoma peritonei</td>
<td>Gelatinous</td>
<td>&lt;2.5</td>
<td>N</td>
<td>&lt;250</td>
<td>Few</td>
<td>Neg</td>
<td>Occ Pos</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>EXUDATES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bacterial peritonitis</td>
<td>Cloudy</td>
<td>&gt;3.0</td>
<td>&lt;50 with perforation</td>
<td>&gt;500, PMN</td>
<td>Few</td>
<td>Pos</td>
<td>Neg</td>
<td>Blood cultures frequently positive</td>
<td></td>
</tr>
<tr>
<td>Tuberculous peritonitis</td>
<td>Clear</td>
<td>&gt;3.0</td>
<td>Low(^4)</td>
<td>&gt;500, MN</td>
<td>Few, occasionally many</td>
<td>Stain Pos in 25%; culture Pos in 65%</td>
<td>Neg</td>
<td>Occasionally chylous. Peritoneal biopsy positive in 65%</td>
<td></td>
</tr>
<tr>
<td>Diagnosis</td>
<td>Appearance</td>
<td>Fluid Protein (g/dL)</td>
<td>Fluid Glucose (mg/dL)</td>
<td>WBC and Differential (per µL)</td>
<td>RBC (per µL)</td>
<td>Bacteriologic Gram Stain and Culture</td>
<td>Cytology</td>
<td>Comments</td>
<td></td>
</tr>
<tr>
<td>-----------------</td>
<td>------------</td>
<td>----------------------</td>
<td>-----------------------</td>
<td>------------------------------</td>
<td>--------------</td>
<td>--------------------------------------</td>
<td>-----------</td>
<td>----------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Malignancy</td>
<td>Clear or bloody</td>
<td>&gt;3.0 Low&lt;sup&gt;4&lt;/sup&gt;</td>
<td>&lt;60 &gt;500, MN, PMN</td>
<td>Many</td>
<td>Neg</td>
<td>Pos in 60–90%</td>
<td>Occasionally chylos. Fluid LDH/Serum LDH ratio &gt;0.6. Peritoneal biopsy diagnostic.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Clear or bloody</td>
<td>&gt;2.5 N</td>
<td>&gt;500, MN, PMN</td>
<td>Many</td>
<td>Neg</td>
<td>Neg</td>
<td>Occasionally chylos. Fluid amylase &gt; 1000 IU/L, sometimes &gt;10,000 IU/L Fluid amylase &gt; serum amylase</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chyloous ascites</td>
<td>Turbid</td>
<td>Varies, often &gt;2.5</td>
<td>N</td>
<td>Few</td>
<td>Few</td>
<td>Neg</td>
<td>Neg</td>
<td>Fluid TG &gt; 400 mg/dL (turbid) Fluid TG &gt; serum TG</td>
<td></td>
</tr>
</tbody>
</table>


Transudates have protein concentration <2.5–3 g/dL; fluid LDH/serum LDH ratio <0.6 (may be useful in difficult cases).

High = ≥1.1

Low = <1.1

Exudates have fluid protein concentration >2.5–3 g/dL; fluid LDH/serum LDH ratio >0.6 (may be useful in difficult cases).

MN = mononuclear cells; PMN = polymorphonuclear cells; TG = triglycerides.
### TABLE 8-5. AUTOANTIBODIES: ASSOCIATIONS WITH CONNECTIVE TISSUE DISEASES.¹

<table>
<thead>
<tr>
<th>Suspected Disease State</th>
<th>Test</th>
<th>Primary Disease Association (Sensitivity, Specificity)</th>
<th>Other Disease Associations (Sensitivity)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>CREST syndrome</td>
<td>Anti-centromere antibody</td>
<td>CREST (70–90%, high)</td>
<td>Scleroderma (10–15%), Raynaud’s disease (10–30%)</td>
<td>Predictive value of a positive test is &gt;95% for scleroderma or related disease (CREST, Raynaud’s). Diagnosis of CREST is made clinically.</td>
</tr>
<tr>
<td>Systemic lupus erythematosus (SLE)</td>
<td>Anti-nuclear antibody (ANA)</td>
<td>SLE (&gt;95%, low)</td>
<td>RA (30–50%), discoid lupus, scleroderma (60%), drug-induced lupus (100%), Sjögren’s syndrome (80%), miscellaneous inflammatory disorders.</td>
<td>Often used as a screening test; a negative test virtually excludes SLE; a positive test, while nonspecific, increases posttest probability. Titer does not correlate with disease activity.</td>
</tr>
<tr>
<td></td>
<td>Anti-double-stranded-DNA antibody (anti-ds-DNA)</td>
<td>SLE (60–70%, high)</td>
<td>Lupus nephritis, rarely RA, CTD, usually in low titer.</td>
<td>Predictive value of a positive test is &gt;90% for SLE if present in high titer; a decreasing titer may correlate with worsening renal disease. Titer generally correlates with disease activity.</td>
</tr>
<tr>
<td></td>
<td>Anti-Smith antibody (anti-SM)</td>
<td>SLE (30–40%, high)</td>
<td></td>
<td>SLE-specific. A positive test substantially increases posttest probability of SLE. Test rarely indicated.</td>
</tr>
<tr>
<td>Mixed connective tissue disease (MCTD)</td>
<td>Anti-ribonucleoprotein antibody (RNP)</td>
<td>MCTD (95–100%, low) Scleroderma (20–30%, low)</td>
<td>SLE (30%), Sjögren’s syndrome, RA (10%), discoid lupus (20–30%).</td>
<td>A negative test essentially excludes MCTD; a positive test in high titer, while nonspecific, increases posttest probability of MCTD.</td>
</tr>
</tbody>
</table>
TABLE 8–5 (CONTINUED).

<table>
<thead>
<tr>
<th>Suspected Disease State</th>
<th>Test</th>
<th>Primary Disease Association (Sensitivity, Specificity)</th>
<th>Other Disease Associations (Sensitivity)</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rheumatoid arthritis (RA)</td>
<td>Rheumatoid factor (RF)</td>
<td>Rheumatoid arthritis (50–90%)</td>
<td>Other rheumatic diseases, chronic infections, some malignancies, some healthy individuals, elderly patients.</td>
<td>Titer does not correlate with disease activity.</td>
</tr>
<tr>
<td>Scleroderma</td>
<td>Anti-Scl-70 antibody</td>
<td>Scleroderma (15–20%, high)</td>
<td></td>
<td>Predictive value of a positive test is &gt; 95% for scleroderma.</td>
</tr>
<tr>
<td>Sjögren’s syndrome</td>
<td>Anti-SS-A/Ro antibody</td>
<td>Sjögren’s (60–70%, low)</td>
<td>SLE (30–40%), RA (10%), subacute cutaneous lupus, vasculitis.</td>
<td>Useful in counseling women of child-bearing age with known CTD, since a positive test is associated with a small but real risk of neonatal SLE and congenital heart block.</td>
</tr>
<tr>
<td>Wegener’s granulomatosis</td>
<td>Anti-neutrophil cytoplasmic antibody (ANCA)</td>
<td>Wegener’s granulomatosis (systemic necrotizing vasculitis) (56–96%, high)</td>
<td>Crescentic glomerulonephritis or other systemic vasculitis (eg, polyarteritis nodosa).</td>
<td>Ability of this assay to reflect disease activity remains unclear.</td>
</tr>
</tbody>
</table>


RA = rheumatoid arthritis; SLE = systemic lupus erythematosus; CTD = connective tissue disease; MCTD = mixed connective tissue disease; SSA = Sjögren’s syndrome A antibody; CREST = calcinosis, Raynaud’s phenomenon, esophageal dysmotility, sclerodactyly and telangiectasia.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Appearance</th>
<th>Opening Pressure (mm H₂O)</th>
<th>RBC (per µL)</th>
<th>WBC &amp; Diff (per µL)</th>
<th>CSF Glucose (mg/dL)</th>
<th>CSF Protein (mg/dL)</th>
<th>Smears</th>
<th>Culture</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Clear, colorless</td>
<td>70–200</td>
<td>0</td>
<td>≤5 MN, 0 PMN</td>
<td>45–85</td>
<td>15–45</td>
<td>Neg</td>
<td>Neg</td>
<td>Neg</td>
</tr>
<tr>
<td>Bacterial meningitis</td>
<td>Cloudy</td>
<td>↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑</td>
<td>0</td>
<td>200–20,000, mostly PMN</td>
<td>&lt; 45</td>
<td>&gt; 50</td>
<td>Gram’s stain Pos</td>
<td>Pos</td>
<td>PMN predominance may be seen early in course.</td>
</tr>
<tr>
<td>Tuberculous meningitis</td>
<td>N or cloudy</td>
<td>↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑</td>
<td>0</td>
<td>100–1000, mostly MN</td>
<td>&lt; 45</td>
<td>&gt; 50</td>
<td>AFB stain Pos</td>
<td>±</td>
<td>Counterimmunoelectrophoresis or latex agglutination may be diagnostic. CSF and serum cryptococcal antigen positive in cryptococcal meningitis.</td>
</tr>
<tr>
<td>Fungal meningitis</td>
<td>N or cloudy</td>
<td>N or ↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑</td>
<td>0</td>
<td>100–1000, mostly MN</td>
<td>&lt; 45</td>
<td>&gt; 50</td>
<td>±</td>
<td>Viral (aseptic) meningitis</td>
<td>RBC count may be elevated in herpes simplex encephalitis. Glucose may be decreased in herpes simplex or mumps infections. Viral cultures may be helpful.</td>
</tr>
<tr>
<td>Viral (aseptic) meningitis</td>
<td>N</td>
<td>N or ↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑</td>
<td>0</td>
<td>100–1000, mostly MN</td>
<td>45–85</td>
<td>N or ↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑↑</td>
<td>Neg</td>
<td>Neg</td>
<td>Bacillus brevis contamination may be seen early in course.</td>
</tr>
</tbody>
</table>

TABLE 8-6. CEREBROSPINAL FLUID (CSF): CSF PROFILES IN CENTRAL NERVOUS SYSTEM DISEASE.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Appearance</th>
<th>Opening Pressure (mm H₂O)</th>
<th>RBC (per µL)</th>
<th>WBC &amp; Diff (per µL)</th>
<th>CSF Glucose (mg/dL)</th>
<th>CSF Protein (mg/dL)</th>
<th>Smears</th>
<th>Culture</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Parasitic meningitis</td>
<td>N or cloudy</td>
<td>N or ↑</td>
<td>0</td>
<td>100–1000, mostly MN</td>
<td>&lt; 45</td>
<td>N or ↑</td>
<td>Amebae may be seen on wet smear</td>
<td>±</td>
<td></td>
</tr>
<tr>
<td>Carcinomatous meningitis</td>
<td>N or cloudy</td>
<td>N or ↑</td>
<td>0</td>
<td>N or 100–1000, mostly MN</td>
<td>&lt; 45</td>
<td>N or ↑</td>
<td>Cytology Pos</td>
<td>Neg</td>
<td></td>
</tr>
<tr>
<td>Cerebral lupus erythematous</td>
<td>N</td>
<td>N or ↑</td>
<td>0</td>
<td>N or ↑, mostly MN</td>
<td>N</td>
<td>N or ↑</td>
<td>Neg</td>
<td>Neg</td>
<td></td>
</tr>
<tr>
<td>Subarachnoid hemorrhage</td>
<td>Pink-red, supernatant yellow</td>
<td>↑</td>
<td>↑ crenated or fresh</td>
<td>N or 100–1000, mostly PMN</td>
<td>N or ↓</td>
<td>N or ↑</td>
<td>Neg</td>
<td>Neg</td>
<td>Blood in all tubes equally. Pleocytosis and low glucose sometimes seen several days after subarachnoid hemorrhage, reflecting chemical meningitis caused by subarachnoid blood.</td>
</tr>
<tr>
<td>“Traumatic” tap</td>
<td>Bloody, supernatant clear</td>
<td>N</td>
<td>↑</td>
<td>N</td>
<td>↑</td>
<td>Neg</td>
<td>Neg</td>
<td>Most blood in tube #1, least blood in tube #4.</td>
<td></td>
</tr>
<tr>
<td>Condition</td>
<td>CSF Appearance</td>
<td>PMN</td>
<td>RBC</td>
<td>WBC</td>
<td>Protein</td>
<td>Glucose</td>
<td>Sugar</td>
<td>PMN/CNS</td>
<td>CSF VDRL</td>
</tr>
<tr>
<td>-----------------------------------------------</td>
<td>----------------</td>
<td>-----</td>
<td>-----</td>
<td>-----</td>
<td>---------</td>
<td>---------</td>
<td>-------</td>
<td>---------</td>
<td>----------</td>
</tr>
<tr>
<td>Spirochetal, early, acute syphilitic meningitis</td>
<td>Clear to turbid</td>
<td>↑</td>
<td>0</td>
<td>25–2000, mostly MN</td>
<td>15–75</td>
<td>&gt; 50</td>
<td>Neg</td>
<td>Neg</td>
<td>PMN may predominate early. Positive serum RPR or VDRL. CSF VDRL insensitive. If clinical suspicion is high, institute treatment despite negative CSF VDRL.</td>
</tr>
<tr>
<td>Late CNS syphilis</td>
<td>Clear</td>
<td>Usually N</td>
<td>0</td>
<td>N or ↑</td>
<td>N</td>
<td>N or ↑</td>
<td>Neg</td>
<td>Neg</td>
<td>CSF VDRL insensitive.</td>
</tr>
<tr>
<td>“Neighborhood” meningeal reaction</td>
<td>Clear or turbid, often xanthochromic</td>
<td>Variable, usually N</td>
<td>Variable</td>
<td>↑</td>
<td>N</td>
<td>N or ↑</td>
<td>Neg</td>
<td>Usually Neg</td>
<td>May occur in mastoiditis, brain abscess, sinusitis, septic thrombophlebitis, brain tumor, intrathecal drug therapy.</td>
</tr>
<tr>
<td>Hepatic encephalopathy</td>
<td>N</td>
<td>N</td>
<td>0</td>
<td>≤5</td>
<td>N</td>
<td>N</td>
<td>Neg</td>
<td>Neg</td>
<td>CSF glutamine &gt;15 mg/dL.</td>
</tr>
<tr>
<td>Uremia</td>
<td>N</td>
<td>Usually ↑</td>
<td>0</td>
<td>N or ↑</td>
<td>N or ↑</td>
<td>N or ↑</td>
<td>Neg</td>
<td>Neg</td>
<td></td>
</tr>
<tr>
<td>Diabetic coma</td>
<td>N</td>
<td>Low</td>
<td>0</td>
<td>N or ↑</td>
<td>↑</td>
<td>N</td>
<td>Neg</td>
<td>Neg</td>
<td></td>
</tr>
</tbody>
</table>

MN = mononuclear cells (lymphocytes or monocytes); PMN = polymorphonuclear cells; E = eosinophils; CNS = central nervous system.
TABLE 8–7. RELATIONSHIP OF HEPATIC FUNCTION AND NUTRITION OR PROTHROMBIN TIME TO OPERATIVE DEATH RATE AFTER PORTACAVAL SHUNT.

<table>
<thead>
<tr>
<th></th>
<th>Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
</tr>
<tr>
<td><strong>Child’s criteria</strong></td>
<td></td>
</tr>
<tr>
<td>Operative death rate</td>
<td>2%</td>
</tr>
<tr>
<td>Serum bilirubin (mg/dL)</td>
<td>&lt;2</td>
</tr>
<tr>
<td>Serum albumin (g/dL)</td>
<td>&gt;3.5</td>
</tr>
<tr>
<td>Ascites</td>
<td>None</td>
</tr>
<tr>
<td>Encephalopathy</td>
<td>None</td>
</tr>
<tr>
<td>Nutrition¹</td>
<td>Excellent</td>
</tr>
<tr>
<td><strong>Pugh modification¹</strong></td>
<td></td>
</tr>
<tr>
<td>Prothrombin time</td>
<td>1–4</td>
</tr>
</tbody>
</table>

¹ In the Pugh modification of Child’s criteria, prothrombin time is substituted for nutrition.
### TABLE 8–8. GENETIC DISEASES DIAGNOSED BY MOLECULAR DIAGNOSTIC TECHNIQUES.

<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cystic fibrosis mutation</strong></td>
<td>Cystic fibrosis is caused by a mutation in the cystic fibrosis transmembrane regulator gene (CFTR). Over 600 mutations have been found, with the most common being ∆F508, present in 68% of cases.</td>
<td>Test specificity approaches 100%, so a positive result should be considered diagnostic of a cystic fibrosis mutation. Because of the wide range of mutations, an assay for the ∆F508 mutation alone is 68% sensitive; a combined panel encompassing the 31 most common mutations is about 90% sensitive. The test can distinguish between heterozygous carriers and homozygous patients.</td>
<td>Cystic fibrosis is the most common inherited disease in North American Caucasians, affecting one in 2500 births. Caucasians have a carrier frequency of one in 25. The disease is autosomal recessive. Proc Natl Acad Sci U S A 1989;86:6230. Hum Mutations 1995;5:333. J Lab Clin Med 1995;125:421. J Pediatr 1998;132:589.</td>
</tr>
<tr>
<td>PCR + reverse dot blot</td>
<td>Blood</td>
<td>Lavender $$$$$</td>
<td></td>
</tr>
<tr>
<td><strong>Factor V (Leiden) mutation (activated protein C resistance)</strong></td>
<td>The Leiden mutation is a single nucleotide base substitution leading to an amino acid substitution (glutamine replaces arginine) at one of the sites where coagulation factor V is cleaved by activated protein C. The mutation causes factor V to be partially resistant to protein C, which is involved in inhibiting coagulation. Factor V mutations may be present in up to half of the cases of unexplained venous thrombosis and are seen in 96% of patients with activated protein C resistance.</td>
<td><strong>Positive in:</strong> Hypercoagulability secondary to factor V mutation (specificity approaches 100%).</td>
<td>The presence of mutation is only a risk factor for thrombosis, not an absolute marker for disease. Homozygotes have a 50- to 100-fold increase in risk of thrombosis (relative to the general population), and heterozygotes have a 7-fold increase in risk. The current PCR and reverse dot blot assay only detects the Leiden mutation of factor V; other mutations may yet be discovered. There is also increased risk of thrombosis in carriers of the prothrombin G → A20210 variant and in methylenetetrahydrofolate reductase deficiency. Thromb Hemost 1997;78:523. Ann Intern Med 1998;128:1000. Ann Intern Med 1998;129:89. Ann Intern Med 1999;130:643.</td>
</tr>
<tr>
<td>Blood</td>
<td>Lavender or blue $$$$$</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Test/Range/Collection</strong></td>
<td><strong>Physiologic Basis</strong></td>
<td><strong>Interpretation</strong></td>
<td><strong>Comments</strong></td>
</tr>
<tr>
<td>--------------------------</td>
<td>-----------------------</td>
<td>--------------------</td>
<td>--------------</td>
</tr>
<tr>
<td><strong>Fragile X syndrome</strong></td>
<td>Fragile X syndrome results from a mutation in the familial mental retardation-1 gene (<em>FMR1</em>), located at Xq27.3. Fully symptomatic patients have abnormal methylation of the gene (which blocks transcription) during oogenesis. The gene contains a variable number of repeating CGG sequences and, as the number of sequences increases, the probability of abnormal methylation increases. The number of copies increases with subsequent generations so that females who are unaffected carriers may have offspring who are affected.</td>
<td>Normal patients have 6–52 CGG repeat sequences. Patients with 52–230 repeat sequences are asymptomatic carriers. Patients with more than 230 repeat sequences are very likely to have abnormal methylation and to be symptomatic.</td>
<td>Fragile X syndrome is the most common cause of inherited mental retardation, occurring in one in 1000–1500 males and one in 2000–2500 females. Full mutations can show variable penetration in females, but most such females will be at least mildly retarded. N Engl J Med 1991;325:1673. Am J Hum Genet 1995;56:1147. Am J Med Genet 1996;64:191. Am J Hum Genet 1997;61:660.</td>
</tr>
<tr>
<td>Blood, cultured amniocytes</td>
<td>lavender</td>
<td>$$$$</td>
<td></td>
</tr>
</tbody>
</table>

<p>| <strong>Hemophilia A</strong>         | Approximately half of severe hemophilia A cases are caused by an inversion mutation within the factor VIII gene. The resulting rearrangement of BCL1 sites can be detected by Southern blot hybridization assays. | Test specificity approaches 100%, so a positive result should be considered diagnostic of a hemophilia A inversion mutation. Because of a variety of mutations, however, test sensitivity is only about 50%. | Hemophilia A is one of the most common X-linked diseases in humans, affecting one in 5000 males. Nat Genet 1993;5:236. Hematol Oncol Clin North Am 1998;12:1315. |
| Southern blot            |                    |                    |              |
| Blood, cultured amniocytes | lavender | $$$$ |
| <strong>Huntington's disease</strong> | Huntington's disease is an inherited neurodegenerative disorder associated with an autosomal dominant mutation on chromosome 4. The disease is highly penetrant, but symptoms (disordered movements, cognitive decline, and emotional disturbance) are often not expressed until middle age. The mutation results in the expansion of a CAG trinucleotide repeat sequence within the gene. | Normal patients will have fewer than 34 CAG repeats, while patients with disease usually have more than 37 repeats and may have 80 or more. Occasional affected patients can be seen with “high normal” (32–34) numbers of repeats. Tests showing 34–37 repeats are indeterminate. | Huntington's disease testing involves ethical dilemmas. Counseling is recommended prior to testing. Hum Molec Genet 1993;2:633. J Neurol 1998;245:709. |
| <strong>α-Thalassemia</strong> | A deletion mutation in the α-globin gene region of chromosome 16 due to unequal crossing-over events can lead to defective synthesis of the α-globin chain of hemoglobin. Normally, there are two copies of the α-globin gene on each chromosome 16, and the severity of disease increases with the number of defective genes. | This assay is highly specific (approaches 100%). Sensitivity, however, can vary since detection of different mutations may require the use of different probes. α-Thalassemia due to point mutations may not be detected. | Patients with one deleted gene are usually normal or very slightly anemic; patients with two deletions usually have a hypochromic microcytic anemia; patients with three deletions have elevated hemoglobin H and a moderately severe hemolytic anemia; patients with four deletions generally die in utero with hydrops fetalis (see Table 8–21). Eur J Clin Invest 1990;20:340. Prenat Diagn 1996;16:1181. |</p>
<table>
<thead>
<tr>
<th>Test/Range/Collection</th>
<th>Physiologic Basis</th>
<th>Interpretation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>β-Thalassemia</strong></td>
<td><strong>β</strong>-Thalassemia results from a mutation in the gene encoding the <strong>β</strong>-globin subunit of hemoglobin A (which is composed of a pair of <strong>α</strong>-chains and a pair of <strong>β</strong>-chains). A relative excess of <strong>α</strong>-globin chains precipitates within red blood cells, causing hemolysis and anemia. Over 100 different mutations have been described; testing usually covers a panel of the more common mutations. The test can distinguish between heterozygous and homozygous individuals.</td>
<td>Test specificity approaches 100%, so a positive result should be considered diagnostic of a thalassemia mutation. Because of the large number of mutations, sensitivity can be poor. A panel with the 41 most common mutations has a sensitivity that approaches 95%.</td>
<td><strong>β</strong>-Thalassemia is very common; about 3% of the world’s population are carriers. The incidence is increased in persons of Mediterranean, African, and Asian descent. The mutations may vary from population to population, and different testing panels may be needed for patients of different ethnicities (see Table 8–22). <em>JAMA</em> 1997;278:1273. <em>J Hum Genet</em> 1998;43:237.</td>
</tr>
</tbody>
</table>
### TABLE 8-9. HEMOSTATIC FUNCTION: LABORATORY EVALUATION.¹

<table>
<thead>
<tr>
<th>Suspected Diagnosis</th>
<th>Platelet Count</th>
<th>PT</th>
<th>PTT</th>
<th>TT</th>
<th>Further Diagnostic Tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic thrombocytopenic purpura, drug sensitivity, bone marrow depression</td>
<td>↓</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Platelet antibody, marrow aspirate.</td>
</tr>
<tr>
<td>Disseminated intravascular coagulation</td>
<td>↓</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>Fibrinogen assays, fibrin D-dimers.</td>
</tr>
<tr>
<td>Platelet function defect, salicylates, or uremia</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Bleeding time, platelet aggregation, blood urea nitrogen (BUN), creatinine.</td>
</tr>
<tr>
<td>von Willebrand’s disease</td>
<td>N</td>
<td>N</td>
<td>↑ or N</td>
<td>N</td>
<td>Bleeding time, factor VIII assay, factor VIII antigen.</td>
</tr>
<tr>
<td>Factor VII deficiency or inhibitor</td>
<td>N</td>
<td>↑</td>
<td>N</td>
<td>N</td>
<td>Factor VII assay (normal plasma should correct PT if no inhibitor is present).</td>
</tr>
<tr>
<td>Factor V, X, II, I deficiencies as in liver disease or with anticoagulants</td>
<td>N</td>
<td>↑</td>
<td>↑</td>
<td>N or ↑</td>
<td>Liver function tests.</td>
</tr>
<tr>
<td>Factor VIII (hemophilia), IX, XI, or XII deficiencies or inhibitor</td>
<td>N</td>
<td>N</td>
<td>↑</td>
<td>N</td>
<td>Inhibitor screen, individual factor assays.</td>
</tr>
<tr>
<td>Factor XIII deficiency</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Urea stabilizing test, factor XIII assay.</td>
</tr>
</tbody>
</table>


**Note:** In approaching patients with bleeding disorders, try to distinguish clinically between platelet disorders (eg, patient has petechiae, mucosal bleeding) and factor deficiency states (eg, patient has hemarthrosis).

PT = prothrombin time; PTT = activated partial thromboplastin time; TT = thrombin time.
### TABLE 8–10. HEPATIC FUNCTION TESTS.¹

<table>
<thead>
<tr>
<th>Clinical Condition</th>
<th>Direct Bilirubin (mg/dL)</th>
<th>Indirect Bilirubin (mg/dL)</th>
<th>Urine Bilirubin</th>
<th>Serum Albumin &amp; Total Protein (g/dL)</th>
<th>Alkaline Phosphatase (IU/L)</th>
<th>Prothrombin time (seconds)</th>
<th>ALT (SGPT); AST (SGOT) (IU/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>0.1–0.3</td>
<td>0.2–0.7</td>
<td>None</td>
<td>Albumin, 3.4–4.7 Total protein, 6.0–8.0</td>
<td>30–115 (lab-specific)</td>
<td>11–15 seconds. After vitamin K, 15% increase within 24 hours.</td>
<td>ALT, 5–35; AST, 5–40 (lab specific)</td>
</tr>
<tr>
<td>Hepatocellular jaundice (eg, viral, alcoholic hepatitis)</td>
<td>↑↑</td>
<td>↑</td>
<td>↑</td>
<td>↓ Albumin</td>
<td>N to ↑</td>
<td>Prolonged if damage is severe. Does not respond to parenteral vitamin K.</td>
<td>Increased in hepatocellular damage, viral hepatitides; AST/ALT ratio often &gt;2:1 in alcoholic hepatitis</td>
</tr>
<tr>
<td>Uncomplicated obstructive jaundice (eg, common bile duct obstruction)</td>
<td>↑↑</td>
<td>↑</td>
<td>↑</td>
<td>N</td>
<td>↑</td>
<td>Prolonged if obstruction marked but responds to parenteral vitamin K.</td>
<td>N to minimally ↑</td>
</tr>
<tr>
<td>Hemolysis</td>
<td>N</td>
<td>↑</td>
<td>None</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Gilbert's syndrome</td>
<td>N</td>
<td>↑</td>
<td>None</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Intrahepatic cholestasis (drug-induced)</td>
<td>↑↑</td>
<td>↑</td>
<td>↑</td>
<td>N</td>
<td>↑↑</td>
<td>N</td>
<td>AST N or ↑; ALT N or ↑</td>
</tr>
<tr>
<td>Primary biliary cirrhosis</td>
<td>↑↑</td>
<td>↑</td>
<td>↑</td>
<td>↑ globulin</td>
<td>↑↑</td>
<td>N or ↑</td>
<td>↑</td>
</tr>
</tbody>
</table>


**AST** = aspartate aminotransferase; **ALT** = alanine aminotransferase.
### TABLE 8-11. HYPERLIPIDEMIA: CHARACTERISTICS AND LABORATORY FINDINGS IN PRIMARY HYPERLIPIDEMIA.1

<table>
<thead>
<tr>
<th>Lipoprotein Disorder</th>
<th>Lipoprotein Abnormalities or Defect</th>
<th>Appearance of Serum²</th>
<th>Cholesterol (mg/dL)</th>
<th>Triglyceride (mg/dL)</th>
<th>Clinical Presentation</th>
<th>Comments</th>
<th>Risk of Atherosclerosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>None</td>
<td>Clear</td>
<td>&lt;200</td>
<td>&lt;165</td>
<td>Normal</td>
<td></td>
<td>Nil</td>
</tr>
<tr>
<td>Familial hyper-cholesterolemia</td>
<td>LDL elevated; decreased or lack of LDL receptors in liver</td>
<td>Clear</td>
<td>Usually 300–600 but may be higher; LDL cholesterol high</td>
<td>Normal</td>
<td>Xanthelasma, tendon and skin xanthomas, accelerated atherosclerosis. Detectable in childhood.</td>
<td>Onset at all ages. Consider hypothyroidism, nephrotic syndrome, hepatic obstruction.</td>
<td>↑↑</td>
</tr>
<tr>
<td>Familial combined hyperlipidemia</td>
<td>LDL or VLDL elevated</td>
<td>Turbid or clear</td>
<td>Usually 250–600, LDL cholesterol high</td>
<td>Usually 200–600</td>
<td>Accelerated atherosclerosis. Associated with obesity or diabetes.</td>
<td>Cholesterol or triglyceride or both may be elevated—at different times and in different members of the family.</td>
<td>↑↑</td>
</tr>
<tr>
<td>Familial hyper-triglyceridemia</td>
<td>VLDL elevated</td>
<td>Turbid</td>
<td>Typically normal</td>
<td>200–5000</td>
<td>Eruptive xanthomas. Triglycerides, if high enough, may cause pancreatitis.</td>
<td>Consider nephrotic syndrome, hypothyroidism, alcoholism, glycogen storage disease, oral contraceptives.</td>
<td>Nil</td>
</tr>
<tr>
<td>Lipoprotein Disorder</td>
<td>Lipoprotein Abnormalities or Defect</td>
<td>Appearance of Serum</td>
<td>Cholesterol (mg/dL)</td>
<td>Triglyceride (mg/dL)</td>
<td>Clinical Presentation</td>
<td>Comments</td>
<td>Risk of Atherosclerosis</td>
</tr>
<tr>
<td>----------------------</td>
<td>------------------------------------</td>
<td>--------------------</td>
<td>---------------------</td>
<td>---------------------</td>
<td>----------------------</td>
<td>----------</td>
<td>------------------------</td>
</tr>
<tr>
<td>Hyper-chylomicronemia</td>
<td>Chylomicrons elevated; deficiency of lipoprotein lipase or, less commonly, of C-II apolipoprotein</td>
<td>Creamy, separates into creamy supernate and clear infranate</td>
<td>Increased</td>
<td>Often 1000 – 10,000; chylomicrons</td>
<td>Eruptive xanthomas, lipemia retinalis, recurrent abdominal pain, hepatosplenomegaly, pancreatitis.</td>
<td>Onset in infancy or childhood. Aggravated by high fat intake, diabetes, alcohol.</td>
<td>Nil</td>
</tr>
<tr>
<td>Mixed hyper-triglyceridemia</td>
<td>VLDL and chylomicrons elevated</td>
<td>Creamy, separates into creamy supernate and turbid infranate</td>
<td>300 – 1000</td>
<td>Usually 500 – &gt;10,000; chylomicrons high</td>
<td>Recurrent abdominal pain, hepatosplenomegaly, eruptive xanthomas, glucose intolerance</td>
<td>Symptoms begin in adult life. Sensitive to dietary fat. Alcohol and diabetes aggravate.</td>
<td>Nil to ↑</td>
</tr>
<tr>
<td>Dysbetalipoproteinemia (type III)</td>
<td>VLDL, IDL elevated; apolipoprotein E dysfunction</td>
<td>Turbid</td>
<td>200 – 500</td>
<td>200 – 500</td>
<td>Palmar xanthoma typical; other xanthomas common.</td>
<td>Aggravated by alcohol, estrogen.</td>
<td>↑↑</td>
</tr>
</tbody>
</table>


2 Refrigerated serum overnight at 4°C.

**Key:** LDL = low-density lipoprotein, calculated as: Total cholesterol — HDL cholesterol — [Triglycerides/5]; VLDL = very low density lipoprotein; IDL = intermediate density lipoprotein.
The osmolal gap ($\Delta \text{osm}$) is determined by subtracting the calculated serum osmolality from the measured serum osmolality.

$$\text{Calculated osmolality (osm)} = 2(\text{Na}^+ \text{[meq/L]} + \frac{\text{Glucose (mg/dL)}}{18} + \frac{\text{BUN (mg/dL)}}{2.8})$$

$$\text{Osmolal gap (}\Delta \text{osm}) = \text{Measured osmolality} - \text{Calculated osmolality}$$

Serum osmolality may be increased by contributions of circulating alcohols and other low-molecular-weight substances. Since these substances are not included in the calculated osmolality, there will be an osmolal gap directly proportionate to their serum concentration and inversely proportionate to their molecular weight:

$$\text{Serum concentration (mg/dL)} = \Delta \text{osm} \times \frac{\text{Molecular weight of toxin}}{10}$$

For ethanol (the most common cause of $\Delta \text{osm}$), a gap of 30 mosm/kg $\text{H}_2\text{O}$ indicates an ethanol level of:

$$30 \times \frac{46}{10} = 138 \text{ mg/dL}$$

See the following for lethal concentrations of alcohols and their corresponding osmolal gaps.

**LEthal concentrations of alcohols and their corresponding osmolal gaps**

<table>
<thead>
<tr>
<th>Alcohol</th>
<th>Molecular Weight</th>
<th>Lethal Concentration (mg/dL)</th>
<th>Corresponding Osmolal Gap (mosm/kg $\text{H}_2\text{O}$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ethanol</td>
<td>46</td>
<td>350</td>
<td>75</td>
</tr>
<tr>
<td>Methanol</td>
<td>32</td>
<td>80</td>
<td>25</td>
</tr>
<tr>
<td>Ethylene glycol</td>
<td>62</td>
<td>200</td>
<td>35</td>
</tr>
<tr>
<td>Isopropanol</td>
<td>60</td>
<td>350</td>
<td>60</td>
</tr>
</tbody>
</table>

**Note:** Most laboratories use the freezing point method for calculating osmolality. If the vaporization point method is used, alcohols are driven off and their contribution to osmolality is lost.


$\text{Na}^+$ = sodium; BUN = blood urea nitrogen.
### TABLE 8–13. PLEURAL FLUID: PLEURAL FLUID PROFILES IN VARIOUS DISEASE STATES.¹

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Gross Appearance</th>
<th>Protein (g/dL)</th>
<th>Glucose² (mg/dL)</th>
<th>WBC and Differential (per µL)</th>
<th>RBC (per µL)</th>
<th>Microscopic Exam</th>
<th>Culture</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Clear</td>
<td>1–1.5</td>
<td>Equal to serum</td>
<td>≤1000, mostly MN</td>
<td>0 or Few</td>
<td>Neg</td>
<td>Neg</td>
<td></td>
</tr>
<tr>
<td><strong>TRANSUDATES³</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Serous</td>
<td>&lt;3: sometimes ≥3</td>
<td>Equal to serum</td>
<td>&lt;1000</td>
<td>&lt;10,000</td>
<td>Neg</td>
<td>Neg</td>
<td>Most common cause of pleural effusion. Effusion right-sided in 55–70% of patients.</td>
</tr>
<tr>
<td>Nephrotic syndrome</td>
<td>Serous</td>
<td>&lt;3</td>
<td>Equal to serum</td>
<td>&lt;1000</td>
<td>&lt;1000</td>
<td>Neg</td>
<td>Neg</td>
<td>Occurs in 20% of patients. Cause is low protein osmotic pressure.</td>
</tr>
<tr>
<td>Hepatic cirrhosis</td>
<td>Serous</td>
<td>&lt;3</td>
<td>Equal to serum</td>
<td>&lt;1000</td>
<td>&lt;1000</td>
<td>Neg</td>
<td>Neg</td>
<td>From movement of ascites across diaphragm. Treatment of underlying ascites usually sufficient.</td>
</tr>
<tr>
<td><strong>EXUDATES³</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tuberculosis</td>
<td>Usually serous; can be bloody</td>
<td>90% ≥3; may exceed 5 g/dL</td>
<td>Equal to serum; Occ &lt;60</td>
<td>500–10,000, mostly MN</td>
<td>&lt;10,000</td>
<td>Concentrate Pos for AFB in &lt;50%</td>
<td>May yield MTb</td>
<td>PPD usually positive; pleural biopsy positive; eosinophils (&gt;10%) or mesothelial cells (&gt;5%) make diagnosis unlikely.</td>
</tr>
<tr>
<td>Malignancy</td>
<td>Usually turbid, bloody; Occ serous</td>
<td>90% ≥3</td>
<td>Equal to serum; &lt;60 in 15% of cases</td>
<td>1000–10,000, mostly MN</td>
<td>&gt;100,000</td>
<td>Pos cytology in 50%</td>
<td>Neg</td>
<td>Eosinophils uncommon; fluid tends to reaccumulate after removal.</td>
</tr>
<tr>
<td>Condition</td>
<td>Pleural Fluid Characteristics</td>
<td>Range</td>
<td>Protein/Serum Protein Ratio</td>
<td>Enzyme Activity</td>
<td>Drainage Necessary</td>
<td>Notes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------------------</td>
<td>-------------------------------</td>
<td>-------</td>
<td>-----------------------------</td>
<td>-----------------</td>
<td>-------------------</td>
<td>---------------------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Empyema</td>
<td>Turbid to purulent</td>
<td>≥3</td>
<td>Less than serum, often &lt;20</td>
<td>25,000–100,000, mostly PMN</td>
<td>&lt;5,000 Pos Pos</td>
<td>Drainage necessary; putrid odor suggests anaerobic infection.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parapneumonic effusion, uncon-</td>
<td>Clear to turbid</td>
<td>≥3</td>
<td>Equal to serum</td>
<td>5000–25,000, mostly PMN</td>
<td>&lt;5,000 Neg Neg</td>
<td>Tube thoracostomy unnecessary; associated infiltrate on chest x-ray; fluid pH ≥7.2.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>complicated</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Variable findings; 25% are transudates.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pulmonary embolism, infarction</td>
<td>Serous to grossly bloody</td>
<td>≥3</td>
<td>Equal to serum</td>
<td>1000–50,000, MN or PMN</td>
<td>100–&gt;100,000 Neg Neg</td>
<td>Rapid clotting time; secondary empyema common.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rheumatoid arthritis or other</td>
<td>Turbid or yellow-green</td>
<td>≥3</td>
<td>Very low (&lt;40 in most); in RA, 5–20 mg/dL</td>
<td>1000–20,000, mostly MN</td>
<td>&lt;1000 Neg Neg</td>
<td>Effusion usually left-sided; high amylase level.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>collagen-vascular disease</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pancreatitis</td>
<td>Turbid to serosanguineous</td>
<td>≥3</td>
<td>Equal to serum</td>
<td>1000–50,000, mostly PMN</td>
<td>1000–10,000 Neg Neg</td>
<td>Effusion usually left-sided; high amylase level (salivary); pneumothorax in 25% of cases; pH &lt;6.0 strongly suggests diagnosis.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Esophageal rupture</td>
<td>Turbid to purulent; red-brown</td>
<td>≥3</td>
<td>Usually low</td>
<td>&lt;5000–over 50,000, mostly PMN</td>
<td>&lt;5000 Pos Pos</td>
<td>Effusion usually left-sided; high fluid amylase level (salivary); pneumothorax in 25% of cases; pH &lt;6.0 strongly suggests diagnosis.</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


2 Glucose of pleural fluid in comparison to serum glucose.

3 Exudative pleural effusions meet at least one of the following criteria: (1) pleural fluid protein/serum protein ratio >0.5; (2) pleural fluid LDH/serum LDH ratio >0.6; and (3) pleural fluid LDH >1/3 upper normal limit for serum LDH. Transudative pleural effusions meet none of these criteria. Transudative effusions also occur in myxedema and sarcoidosis. MN = mononuclear cells (lymphocytes or monocytes); PMN = polymorphonuclear cells; AFB = acid-fast bacilli; MTb = Mycobacterium tuberculosis.
### TABLE 8–14. PRENATAL DIAGNOSTIC METHODS: AMNIOCENTESIS AND CHORIONIC VILLUS SAMPLING.

<table>
<thead>
<tr>
<th>Method</th>
<th>Procedure</th>
<th>Laboratory Analysis</th>
<th>Waiting Time for Results</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
</table>
| Aminocentesis                 | Between the 12th and 16th weeks, and by the transabdominal approach, 10–30 mL of amniotic fluid is removed for cytologic and biochemical analysis. Preceding ultrasound locates the placenta and identifies twinning and missed abortion. | 1. **Amniotic fluid:**  
  - Alpha-fetoprotein  
  - Limited biochemical analysis  
  - Virus isolation studies  
  2. **Amniotic cell culture:**  
  - Chromosomal analysis | 3–4 weeks                  | Over 35 years of experience.                                                       | Therapeutic abortion, if indicated, must be done in the second trimester. (RhoGam should be given to Rh-negative mothers to prevent sensitization.) |
| Chorionic villus sampling     | Between the 8th and 12th week, and with constant ultrasound guidance, the trophoblastic cells of the chorionic villi are obtained by transcervical or transabdominal endoscopic needle biopsy or aspiration. | 1. **Direct cell analysis**  
  - Chromosomal studies  
  2. **Cell culture:**  
  - Limited biochemical analysis | 1–10 days                  | Over 15 years of experience. Therapeutic abortion, if indicated, can be done in the first trimester. | Risks (approximately 3%):  
  - Fetal: abortion.  
  - Maternal: bleeding and infection (uncommon). |

### TABLE 8–15. PULMONARY FUNCTION TESTS: INTERPRETATION IN OBSTRUCTIVE AND RESTRICTIVE PULMONARY DISEASE.\(^1\)

<table>
<thead>
<tr>
<th>Tests</th>
<th>Units</th>
<th>Definition</th>
<th>Obstructive Disease</th>
<th>Restrictive Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SPIROMETRY</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Forced vital capacity (FVC)</td>
<td>L</td>
<td>The volume that can be forcefully expelled from the lungs after maximal inspiration.</td>
<td>N or ↓</td>
<td>↓</td>
</tr>
<tr>
<td>Forced expiratory volume in one second (FEV(_1))</td>
<td>L</td>
<td>The volume expelled in the first second of the FVC maneuver.</td>
<td>↓</td>
<td>N or ↓</td>
</tr>
<tr>
<td>FEV(_1)/FVC</td>
<td>%</td>
<td></td>
<td>↓</td>
<td>N or ↑</td>
</tr>
<tr>
<td>Forced expiratory flow from 25% to 75% of the forced vital capacity (FEF 25–75%)</td>
<td>L/sec</td>
<td>The maximal midexpiratory airflow rate.</td>
<td>↓</td>
<td>N or ↓</td>
</tr>
<tr>
<td>Peak expiratory flow rate (PEFR)</td>
<td>L/sec</td>
<td>The maximal airflow rate achieved in the FVC maneuver.</td>
<td>↓</td>
<td>N or ↑</td>
</tr>
<tr>
<td>Maximum voluntary ventilation (MVV)</td>
<td>L/min</td>
<td>The maximum volume that can be breathed in 1 minute (usually measured for 15 seconds and multiplied by 4).</td>
<td>↓</td>
<td>N or ↓</td>
</tr>
<tr>
<td><strong>LUNG VOLUMES</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slow vital capacity (SVC)</td>
<td>L</td>
<td>The volume that can be slowly exhaled after maximal inspiration.</td>
<td>N or ↓</td>
<td>↓</td>
</tr>
<tr>
<td>Total lung capacity (TLC)</td>
<td>L</td>
<td>The volume in the lungs after a maximal inspiration.</td>
<td>N or ↑</td>
<td>↓</td>
</tr>
<tr>
<td>Functional residual capacity (FRC)</td>
<td>L</td>
<td>The volume in the lungs at the end of a normal tidal expiration.</td>
<td>↑</td>
<td>N or ↑</td>
</tr>
<tr>
<td>Expiratory reserve volume (ERV)</td>
<td>L</td>
<td>The volume representing the difference between functional residual capacity and residual volume.</td>
<td>N or ↓</td>
<td>N or ↓</td>
</tr>
<tr>
<td>Residual volume (RV)</td>
<td>L</td>
<td>The volume remaining in the lungs after maximal expiration.</td>
<td>↑</td>
<td>N or ↑</td>
</tr>
<tr>
<td>RV/TLC ratio</td>
<td>. . .</td>
<td></td>
<td>↑</td>
<td>N or ↑</td>
</tr>
</tbody>
</table>

\(N\) = normal; \(\downarrow\) = less than predicted; \(\uparrow\) = greater than predicted. Normal values vary according to subject sex, age, body size, and ethnicity.
### TABLE 8–16. RANSON’S CRITERIA FOR SEVERITY OF ACUTE PANCREATITIS.¹

#### Criteria present at diagnosis or admission
- Age over 55 years
- White blood cell count >16,000/µL
- Blood glucose >200 mg/dL
- Serum LDH >350 IU/L (laboratory-specific)
- AST (SGOT) >250 IU/L (laboratory-specific)

#### Criteria developing during first 48 hours
- Hematocrit fall >10%
- BUN rise >5 mg/dL
- Serum calcium <8 mg/dL
- Arterial PO₂ <60 mm Hg
- Base deficit >4 meq/L
- Estimated fluid sequestration >6 L

#### Mortality Rates Correlate with the Number of Criteria Present:

<table>
<thead>
<tr>
<th>Number of Criteria</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–2</td>
<td>1%</td>
</tr>
<tr>
<td>3–4</td>
<td>16%</td>
</tr>
<tr>
<td>5–6</td>
<td>40%</td>
</tr>
<tr>
<td>7–8</td>
<td>100%</td>
</tr>
</tbody>
</table>


**LDH** = lactic dehydrogenase; **AST** = aspartate dehydrogenase; **BUN** = blood urea nitrogen.
### TABLE 8-17. CLASSIFICATION AND DIFFERENTIAL DIAGNOSIS OF RENAL FAILURE.

<table>
<thead>
<tr>
<th>Classification</th>
<th>Prerenal Azotemia</th>
<th>Postrenal Azotemia</th>
<th>Intrinsic Renal Disease</th>
<th>Acute Tubular Necrosis (Oliguric or Polyuric)</th>
<th>Acute Glomerulonephritis</th>
<th>Acute Interstitial Nephritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Etiology</td>
<td>Poor renal perfusion</td>
<td>Obstruction of the urinary tract</td>
<td>Ischemia, nephrotoxins</td>
<td>Poststreptococcal; collagen-vascular disease</td>
<td>Allergic reaction; drug reaction</td>
<td></td>
</tr>
<tr>
<td>Urinary indices</td>
<td>Serum BUN: Cr ratio</td>
<td>&gt;20 : 1</td>
<td>&lt;20 : 1</td>
<td>&gt;20 : 1</td>
<td>&lt;20 : 1</td>
<td></td>
</tr>
<tr>
<td>U[Na] (meq/L)</td>
<td>&lt;20</td>
<td>Variable</td>
<td>&gt;20</td>
<td>&lt;20</td>
<td>Variable</td>
<td></td>
</tr>
<tr>
<td>FE[Na]+ (%)</td>
<td>&lt;1</td>
<td>Variable</td>
<td>&gt;1</td>
<td>&lt;1</td>
<td>&lt;1; &gt;1</td>
<td></td>
</tr>
<tr>
<td>Urine osmolality (mosm/kg)</td>
<td>&gt;500</td>
<td>&lt;400</td>
<td>250–300</td>
<td>Variable</td>
<td>Variable</td>
<td></td>
</tr>
<tr>
<td>Urinary sediment</td>
<td>Benign, or hyaline casts</td>
<td>Normal or red cells, white cells, or crystals</td>
<td>Granular casts, renal tubular cells</td>
<td>Dysmorphic red cells and red cell casts</td>
<td>White cells, white cell casts, with or without eosinophils</td>
<td></td>
</tr>
</tbody>
</table>


\[
FE_{Na^+} = \left(\frac{\text{Urine Na}^+}{\text{Plasma Na}^+}\right) \times \left(\frac{\text{Urine creatinine}}{\text{Plasma creatinine}}\right) \times 100
\]

\[U_{Na} = \text{urine sodium.}\]
<table>
<thead>
<tr>
<th>Clinical Condition</th>
<th>Renal Defect</th>
<th>GFR</th>
<th>Serum $\text{[HCO}_3\text{]}^-$ (meq/L)</th>
<th>Serum $[\text{K}^+]$ (meq/L)</th>
<th>Minimal Urine pH</th>
<th>Associated Disease States</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>None</td>
<td>N</td>
<td>24–28</td>
<td>3.5–5</td>
<td>4.8–5.2</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Proximal RTA (type II)</td>
<td>Proximal H⁺ secretion</td>
<td>N</td>
<td>15–18</td>
<td>↓</td>
<td>&lt;5.5</td>
<td>Drugs, Fanconi’s syndrome, various genetic disorders, dysproteinemic states, secondary hyperparathyroidism, toxins (heavy metals), tubulointerstitial diseases, nephrotic syndrome, paroxysmal nocturnal hemoglobinuria.</td>
<td>NaHCO₃ or KHCO₃ (10–15 meq/kg/d), thiazides.</td>
</tr>
<tr>
<td>Classic distal RTA (type I)</td>
<td>Distal H⁺ secretion</td>
<td>N</td>
<td>20–23</td>
<td>↓</td>
<td>&gt;5.5</td>
<td>Various genetic disorders, autoimmune diseases, nephrocalcinosis, drugs, toxins, tubulointerstitial diseases, hepatic cirrhosis, empty sella syndrome.</td>
<td>NaHCO₃ (1–3 meq/kg/d).</td>
</tr>
<tr>
<td>Buffer deficiency distal RTA (type III)</td>
<td>Distal NH₃ delivery</td>
<td>↓</td>
<td>15–18</td>
<td>N</td>
<td>&lt;5.5</td>
<td>Chronic renal insufficiency, renal osteodystrophy, severe hypophosphatemia.</td>
<td>NaHCO₃ (1–3 meq/kg/d).</td>
</tr>
<tr>
<td>Generalized distal RTA (type IV)</td>
<td>Distal Na⁺ re-absorption, K⁺ secretion, and H⁺ secretion</td>
<td>↓</td>
<td>24–28</td>
<td>↑</td>
<td>&lt;5.5</td>
<td>Primary mineralocorticoid deficiency (eg, Addison’s disease), hyporeninemic hypoaldosteronism (diabetes mellitus, tubulointerstitial diseases, nephrosclerosis, drugs), salt-wasting mineralocorticoid-resistant hyperkalemia.</td>
<td>Fludrocortisone (0.1–0.5 mg/d), dietary K⁺ restriction, furosemide (40–160 mg/d), NaHCO₃ (1–3 meq/kg/d).</td>
</tr>
</tbody>
</table>


$GFR = \text{glomerular filtration rate.}$
### TABLE 8–19. SYNOVIAL FLUID: CLASSIFICATION OF SYNOVIAL (JOINT) FLUID.¹

<table>
<thead>
<tr>
<th>Type of Joint Fluid</th>
<th>Volume (mL)</th>
<th>Viscosity</th>
<th>Appearance</th>
<th>WBC (per µL)</th>
<th>PMNs</th>
<th>Gram’s Stain &amp; Culture</th>
<th>Glucose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;3.5</td>
<td>High</td>
<td>Clear, light yellow</td>
<td>&lt;200</td>
<td>&lt;25%</td>
<td>Neg</td>
<td>Equal to serum</td>
<td>Protein 2–3.5 g/dL. Degenerative joint disease, trauma, avascular necrosis, osteochondritis dissecans; osteochondromatosis, neuropathic arthritis, subsiding or early inflammation, hypertrophic osteoarthritis, pigmented villonodular synovitis.</td>
</tr>
<tr>
<td>Non-inflamatory (Class I)</td>
<td>Often &gt;3.5</td>
<td>High</td>
<td>Clear, light yellow</td>
<td>200–2000</td>
<td>&lt;25%</td>
<td>Neg</td>
<td>Equal to serum</td>
<td></td>
</tr>
<tr>
<td>Inflammatory (Class II)</td>
<td>Often &gt;3.5</td>
<td>Low</td>
<td>Cloudy to opaque, dark yellow</td>
<td>3000–100,000</td>
<td>≥50%</td>
<td>Neg</td>
<td>&gt;25, but lower than serum</td>
<td>Protein &gt;3 g/dL. Rheumatoid arthritis, acute crystal-induced synovitis (gout, pseudogout), Reiter’s syndrome, ankylosing spondylitis, psoriatic arthritis, sarcoidosis, arthritis accompanying ulcerative colitis and Crohn’s, rheumatic fever, SLE, scleroderma; tuberculous, viral, or mycotic infections. Crystals diagnostic of gout or pseudogout: gout (urate) crystals show negative birefringence, pseudogout (calcium pyrophosphate) show positive birefringence when red compensator filter is used with polarized light microscopy.</td>
</tr>
<tr>
<td>Type of Joint Fluid</td>
<td>Volume (mL)</td>
<td>Viscosity</td>
<td>Appearance</td>
<td>WBC (per µL)</td>
<td>PMNs</td>
<td>Gram Stain &amp; Culture</td>
<td>Glucose</td>
<td>Comments</td>
</tr>
<tr>
<td>---------------------</td>
<td>------------</td>
<td>-----------</td>
<td>------------</td>
<td>--------------</td>
<td>------</td>
<td>----------------------</td>
<td>---------</td>
<td>----------</td>
</tr>
<tr>
<td>Inflammatory (Class II) (Continued)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Phagocytic inclusions in PMNs suggest rheumatoid arthritis (RA cells). Phagocytosis of leukocytes by macrophages seen in Reiter’s syndrome.</td>
</tr>
<tr>
<td>Purulent (Class III)</td>
<td>Often &gt;3.5</td>
<td>Low</td>
<td>Cloudy to opaque, dark yellow to green</td>
<td>Usually &gt;40,000, often &gt;100,000</td>
<td>≥75%</td>
<td>Usually positive</td>
<td>&lt;25, much lower than serum</td>
<td>Pyogenic bacterial infection (e.g., <em>N. gonorrhoeae, S. aureus</em>). Bacteria on culture or Gram-stained smear. Commonest exception: gonococci seen in only about 25% of cases. WBC count and % PMN lower with infections caused by organisms of low virulence or if antibiotic therapy already started.</td>
</tr>
<tr>
<td>Hemorrhagic (Class IV)</td>
<td>Often &gt;3.5</td>
<td>Variable</td>
<td>Cloudy, pink to red</td>
<td>Usually &gt;2000</td>
<td>30%</td>
<td>Neg</td>
<td>Equal to serum</td>
<td>Trauma with or without fracture, hemophilia or other hemorrhagic diathesis, neuropathic arthropathy, pigmented villonodular synovitis, synovioma, hemangioma and other benign neoplasms. Many RBCs found also. Fat globules strongly suggest intra-articular fracture.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Stage</th>
<th>Onset After Exposure</th>
<th>Persistence</th>
<th>Clinical Findings</th>
<th>Sensitivity of VDRL or RPR (%)</th>
<th>Sensitivity of FTA-ABS (%)</th>
<th>Sensitivity of MHA-TP (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary</td>
<td>21 days (range 10–90)</td>
<td>2–12 wk</td>
<td>Chancre</td>
<td>72</td>
<td>91</td>
<td>50–60</td>
</tr>
<tr>
<td>Secondary</td>
<td>6 wk–6 mo</td>
<td>1–3 mo</td>
<td>Rash, condylomata lata, mucous patches, fever, lymphadenopathy, patchy alopecia</td>
<td>100</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Early latent</td>
<td>&lt;1 yr</td>
<td>Up to 1 yr</td>
<td>Relapses of secondary syphilis</td>
<td>73</td>
<td>97</td>
<td>98</td>
</tr>
<tr>
<td>Late latent</td>
<td>&gt;1 yr</td>
<td>Lifelong unless tertiary syphilis appears</td>
<td>Clinically silent</td>
<td>73</td>
<td>97</td>
<td>98</td>
</tr>
<tr>
<td>Tertiary</td>
<td>1 yr until death</td>
<td>Until death</td>
<td>Dementia, tabes dorsalis, aortitis, aortic aneurysm, gummas</td>
<td>77</td>
<td>99</td>
<td>98</td>
</tr>
</tbody>
</table>

1 Modified, with permission, from Harvey AM et al (editors): The Principles and Practice of Medicine, 22nd ed. Originally published by Appleton & Lange. Copyright © 1988 by The McGraw-Hill Companies, Inc.

2 VDRL is a slide flocculation test for nonspecific (anticardiolipin) antibodies, used for screening, quantitation of titer, and monitoring response to treatment; RPR is an agglutination test for nonspecific antibodies, used primarily for screening.

3 FTA-ABS is an immunofluorescence test for treponemal antibodies utilizing serum absorbed for nonpathogenic treponemes, used for confirmation of infection, not routine screening.

4 MHA-TP is a microhemagglutination test similar to the FTA-ABS, but one which can be quantitated and automated.

**VDRL** = Venereal Disease Research Laboratories test; **RPR** = rapid plasma reagin test; **FTA-ABS** = fluorescent treponemal antibody absorption test; **MHA-TP** = microhemagglutination assay for *T. pallidum*. 
### TABLE 8–21. ALPHA-THALASSEMIA SYNDROMES.¹ ²

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Alpha Globin Genes</th>
<th>Hematocrit</th>
<th>MCV (fL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>4</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Silent carrier</td>
<td>3</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>Thalassemia minor</td>
<td>2</td>
<td>32–40%</td>
<td>60–75</td>
</tr>
<tr>
<td>Hemoglobin H disease</td>
<td>1</td>
<td>22–32%</td>
<td>60–75</td>
</tr>
<tr>
<td>Hydrops fetalis</td>
<td>0</td>
<td>Fetal death occurs in utero</td>
<td></td>
</tr>
</tbody>
</table>


² Alpha thalassemias are due primarily to deletion in the alpha globin gene on chromosome 16.

### TABLE 8–22. BETA-THALASSEMIA SYNDROMES: FINDINGS ON HEMOGLOBIN ELECTROPHORESIS.¹ ²

<table>
<thead>
<tr>
<th>Syndrome</th>
<th>Beta Globin Genes</th>
<th>Hb A³</th>
<th>Hb A₂⁴</th>
<th>Hb F₅</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Homozygous beta</td>
<td>97–99%</td>
<td>1–3%</td>
<td>&lt;1%</td>
</tr>
<tr>
<td>Thalassemia minor</td>
<td>Heterozygous beta°⁶</td>
<td>80–95%</td>
<td>4–8%</td>
<td>1–5%</td>
</tr>
<tr>
<td></td>
<td>Heterozygous beta⁺⁷</td>
<td>80–95%</td>
<td>4–8%</td>
<td>1–5%</td>
</tr>
<tr>
<td>Thalassemia intermedia</td>
<td>Homozygous beta⁺ (mild)</td>
<td>0–30%</td>
<td>0–10%</td>
<td>6–100%</td>
</tr>
<tr>
<td>Thalassemia major</td>
<td>Homozygous beta⁰</td>
<td>0</td>
<td>4–10%</td>
<td>90–96%</td>
</tr>
<tr>
<td></td>
<td>Homozygous beta⁺</td>
<td>4–10%</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


² Beta thalassemias are usually caused by point mutations in the beta globin gene on chromosome 11 that result in premature chain terminations or defective RNA transcription, leading to reduced or absent beta globin chain synthesis.

³ Hb A is composed of two alpha chains and two beta chains: $\alpha_2\beta_2$.

⁴ Hb A₂ is composed of two alpha chains and two delta chains: $\alpha_2\delta_2$.

⁵ Hb F is composed of two alpha chains and two gamma chains: $\alpha_2\gamma_2$.

⁶ Beta⁺ refers to defects that result in absent globin chain synthesis.

⁷ Beta⁺ refers to defects that cause reduced globin chain synthesis.
### TABLE 8-23. THYROID FUNCTION TESTS.¹

<table>
<thead>
<tr>
<th></th>
<th>Total T₄ (µg/dL)</th>
<th>Free T₄ (ng/dL)</th>
<th>Total T₃ (ng/dL)</th>
<th>Sensitive Serum TSH (RIA) (µU/mL)</th>
<th>RAI (¹²³I) Uptake (at 24 hours)</th>
<th>Comments and Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal²</td>
<td>5–12</td>
<td>Varies with method</td>
<td>95–190</td>
<td>0.3–5</td>
<td>10–30%</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroidism</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td>↑</td>
<td>In TRH stimulation test, TSH shows no response. Thyroid scan shows increased diffuse activity (Graves' disease) versus &quot;hot&quot; areas (hyperfunctioning nodules). Thyroperoxidase (TPO) and thyroid-stimulating hormone receptor antibodies (TSH-R Ab [stim]) elevated in Graves' disease.</td>
</tr>
<tr>
<td>Hypothyroidism</td>
<td>↓</td>
<td>↓</td>
<td></td>
<td>Usually ↑ (primary³ hypothyroidism, rarely ↓ (secondary⁴ hypothyroidism))</td>
<td>N or ↓</td>
<td>TRH stimulation test shows exaggerated response in primary hypothyroidism. In secondary hypothyroidism, TRH test helps to differentiate pituitary from hypothalamic disorders. In pituitary lesions, TSH fails to rise after TRH; in hypothalamic lesion, TSH rises but response is delayed. Antithyroglobulin and thyroperoxidase (TPO) antibodies elevated in Hashimoto's thyroiditis.</td>
</tr>
<tr>
<td><strong>HYPOTHYROIDISM ON REPLACEMENT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T₄ replacement</td>
<td>N</td>
<td>N</td>
<td>V</td>
<td>N or ↓</td>
<td>↓</td>
<td>TSH ↓ with 0.1–0.2 mg T₄ daily.</td>
</tr>
<tr>
<td>T₃ replacement</td>
<td>↓</td>
<td>↓</td>
<td>V</td>
<td>N or ↓</td>
<td>↓</td>
<td>TSH ↓ with 50 µg T₃ daily.</td>
</tr>
<tr>
<td>Euthyroid following injection of radiocontrast dye</td>
<td>N</td>
<td>N or ↑</td>
<td>N</td>
<td>N</td>
<td>↓</td>
<td>Effects may persist for 2 weeks or longer.</td>
</tr>
</tbody>
</table>
### TABLE 8–23 (CONTINUED).

<table>
<thead>
<tr>
<th></th>
<th>Total T₄ (µg/dL)</th>
<th>Free T₄ (ng/dL)</th>
<th>Total T₃ (ng/dL)</th>
<th>Sensitive Serum TSH (RIA) (µU/mL)</th>
<th>RAI (¹²³I) Uptake (at 24 hours)</th>
<th>Comments and Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PREGNANCY</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperthyroid</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↓</td>
<td></td>
<td>Effects may persist for 6–10 weeks post-partum. RAI uptake contraindicated in pregnancy.</td>
</tr>
<tr>
<td>Euthyroid</td>
<td>↑</td>
<td>N</td>
<td>↑</td>
<td>N</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypothyroid</td>
<td>N or ↓</td>
<td>↓</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oral contraceptives, estrogens, methadone, heroin</td>
<td>↑</td>
<td>N</td>
<td>↑</td>
<td>N</td>
<td>N</td>
<td>Increased serum thyroid-binding globulin.</td>
</tr>
<tr>
<td>Glucocorticoids, androgens, phenytoin, asparaginase, salicylates (high-dose)</td>
<td>↓</td>
<td>N</td>
<td>N or ↓</td>
<td>N</td>
<td>N</td>
<td>Decreased serum thyroid-binding globulin.</td>
</tr>
<tr>
<td>Nephrotic syndrome</td>
<td>↓</td>
<td>N</td>
<td>N or ↓</td>
<td>N</td>
<td>N</td>
<td>Loss of thyroid-binding globulin accounts for serum T₄ decrease.</td>
</tr>
<tr>
<td>Iodine deficiency</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>↑</td>
<td>Extremely rare in USA.</td>
</tr>
<tr>
<td>Iodine ingestion</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>↓</td>
<td>Excess iodine may cause hypothyroidism or hyperthyroidism is susceptible individuals.</td>
</tr>
</tbody>
</table>

2 Normal values vary with laboratory.
3 Thyroid (end-organ) failure.
4 Pituitary or hypothalamic lesions.

**N** = normal; **V** = variable.
### TABLE 8–24. URINE COMPOSITION: IN COMMON DISEASE STATES.¹

<table>
<thead>
<tr>
<th>Disease</th>
<th>Daily Volume</th>
<th>Specific Gravity</th>
<th>Protein² (mg/dL)</th>
<th>Esterase</th>
<th>Nitrite</th>
<th>RBC</th>
<th>WBC</th>
<th>Casts</th>
<th>Other Microscopic Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>600–2500 mL</td>
<td>1.003–1.030</td>
<td>0–trace (0–30)</td>
<td>Neg</td>
<td>Neg</td>
<td>0 or Occ</td>
<td>0 or Occ</td>
<td>0 or Occ</td>
<td>Hyaline casts</td>
</tr>
<tr>
<td>Fever</td>
<td>↓</td>
<td>↑</td>
<td>Trace or 1+ (&lt;30)</td>
<td>Neg</td>
<td>Neg</td>
<td>0</td>
<td>Occ</td>
<td>0 or Occ</td>
<td>Hyaline casts, tubular cells</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>↓</td>
<td>↑ (varies)</td>
<td>1–2+ (30–100)</td>
<td>Neg</td>
<td>Neg</td>
<td>None or 1+</td>
<td>0</td>
<td>1+</td>
<td>Hyaline and granular casts</td>
</tr>
<tr>
<td>Eclampsia</td>
<td>↓</td>
<td>↑</td>
<td>3–4+ (30–2000)</td>
<td>Neg</td>
<td>Neg</td>
<td>None or 1+</td>
<td>0</td>
<td>3–4+</td>
<td>Hyaline casts</td>
</tr>
<tr>
<td>Diabetic coma</td>
<td>↑ or ↓</td>
<td>↑</td>
<td>1+ (30)</td>
<td>Neg</td>
<td>Neg</td>
<td>0</td>
<td>0</td>
<td>0 or 1+</td>
<td>Hyaline casts</td>
</tr>
<tr>
<td>Acute glomerulonephritis</td>
<td>↓</td>
<td>↑</td>
<td>2–4+ (100–2000)</td>
<td>Pos</td>
<td>Neg</td>
<td>1–4+</td>
<td>1–4+</td>
<td>2–4+</td>
<td>Blood; RBC, cellular, granular, and hyaline casts; renal tubular epithelium</td>
</tr>
<tr>
<td>Nephrotic syndrome</td>
<td>N or ↓</td>
<td>N or ↑</td>
<td>4+ (&gt;2000)</td>
<td>Neg</td>
<td>Neg</td>
<td>1–2+</td>
<td>0</td>
<td>4+</td>
<td>Granular, waxy, hyaline, and fatty casts; fatty tubular cells</td>
</tr>
<tr>
<td>Chronic renal failure</td>
<td>↑ or ↓</td>
<td>Low; invariable</td>
<td>1–2+ (30–100)</td>
<td>Neg</td>
<td>Neg</td>
<td>Occ or 1+</td>
<td>0</td>
<td>1–3+</td>
<td>Granular, hyaline, fatty, and broad casts</td>
</tr>
<tr>
<td>Disease</td>
<td>Daily Volume</td>
<td>Specific Gravity</td>
<td>Protein$^2$ (mg/dL)</td>
<td>Esterase</td>
<td>Nitrite</td>
<td>RBC</td>
<td>WBC</td>
<td>Casts</td>
<td>Other Microscopic Findings</td>
</tr>
<tr>
<td>------------------------</td>
<td>--------------</td>
<td>------------------</td>
<td>---------------------</td>
<td>----------</td>
<td>---------</td>
<td>-----</td>
<td>-----</td>
<td>-------</td>
<td>---------------------------</td>
</tr>
<tr>
<td>Collagen-vascular disease</td>
<td>N, ↑ or ↓</td>
<td>N or ↓</td>
<td>1–4+ (30–2000)</td>
<td>Neg</td>
<td>Neg</td>
<td>1–4+</td>
<td>0 or Occ</td>
<td>1–4+</td>
<td>Blood, cellular, granular, hyaline, waxy, fatty, and broad casts; fatty tubular cells; telescoped sediment</td>
</tr>
<tr>
<td>Pyelonephritis</td>
<td>N or ↓</td>
<td>N or ↓</td>
<td>1–2+ (30–100)</td>
<td>Pos</td>
<td>Pos</td>
<td>0 or 1+</td>
<td>4+</td>
<td>0 or 1+</td>
<td>WBC casts and hyaline casts; many pus cells; bacteria</td>
</tr>
<tr>
<td>Hypertension</td>
<td>N of ↑</td>
<td>N or ↓</td>
<td>None or 1+ (&lt;30)</td>
<td>Neg</td>
<td>Neg</td>
<td>0 or Occ</td>
<td>0 or Occ</td>
<td>0 or 1+</td>
<td>Hyaline and granular casts</td>
</tr>
</tbody>
</table>


$^2$ Protein concentration in mg/dL is listed in parentheses.
TABLE 8–25. VAGINAL DISCHARGE: LABORATORY EVALUATION.\(^1\)

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>pH</th>
<th>Odor With KOH (Positive “Whiff” Test)</th>
<th>Epithelial Cells</th>
<th>WBCs</th>
<th>Organisms</th>
<th>KOH Prep</th>
<th>Gram Stain</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;4.5</td>
<td>No</td>
<td>N</td>
<td>Occ</td>
<td>Variable, large rods not adherent to epithelial cells</td>
<td>Neg</td>
<td>Gram-positive rods</td>
<td></td>
</tr>
<tr>
<td><em>Trichomonas vaginalis</em> vaginitis</td>
<td>&gt;4.5</td>
<td>Yes</td>
<td>N</td>
<td>↑</td>
<td>Motile, flagellated organisms</td>
<td>Neg</td>
<td>Flagellated organisms</td>
<td></td>
</tr>
<tr>
<td>Bacterial vaginosis (Gardnerella vaginalis)</td>
<td>&gt;4.5</td>
<td>Yes</td>
<td>Clue cells(^2)</td>
<td>Occ</td>
<td>Coccobacilli adherent to epithelial cells</td>
<td>Neg</td>
<td>Gram-negative coccobacilli</td>
<td></td>
</tr>
<tr>
<td><em>Candida albicans</em> vaginitis</td>
<td>&lt;4.5</td>
<td>No</td>
<td>N</td>
<td>Occ</td>
<td>Budding yeast or hyphae</td>
<td>Budding yeast or hyphae</td>
<td>Budding yeast or hyphae</td>
<td>Usually white “cottage cheese” curd</td>
</tr>
<tr>
<td>Mucopurulent cervicitis (N gonorrhoeae)</td>
<td>Variable, usually &gt;4.5</td>
<td>No</td>
<td>N</td>
<td>↑</td>
<td>Variable</td>
<td>Neg</td>
<td>Intracellular gram-negative diplococci</td>
<td></td>
</tr>
</tbody>
</table>


\(^2\) Epithelial cells covered with bacteria to the extent that cell nuclear borders are obscured.
<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Chest X-ray</th>
<th>ECG</th>
<th>Echocardiography</th>
<th>Comments</th>
</tr>
</thead>
</table>
| **MITRAL STENOSIS**  
(MS)  
Rheumatic disease | Straight left heart border. Large LA sharply indenting esophagus. Elevation of left main bronchus. Calcification occ seen in MV. | Broad negative phase of biphasic P in V1. Tall peaked P waves, right axis deviation, or RVH appear if pulmonary hypertension is present. | **M-Mode:** Thickened, immobile MV with anterior and posterior leaflets moving together. Slow early diastolic filling slope. LA enlargement. Normal to small LV.  
**2D:** Maximum diastolic orifice size reduced. Reduced subvalvular apparatus. Foreshortened, variable thickening of other valves.  
**Doppler:** Prolonged pressure half-time across MV. Indirect evidence of pulmonary hypertension. | “Critical” MS is usually defined as a valve area <1.0 cm². Balloon valvuloplasty has high initial success rates and higher patency rates than for AS. Open commissurotomy can be effective. Valve replacement is indicated when severe regurgitation is present. Catheterization can confirm echo results. |
| **MITRAL REGURGITATION**  
(MR)  
Myxomatous degeneration  
(MV prolapse)  
Infective endocarditis  
Subvalvular dysfunction  
Rheumatic disease | Enlarged LV and LA. | Left axis deviation or frank LVH. P waves broad, tall, or notched, with broad negative phase in V1. | **M-Mode and 2D:** Thickened MV in rheumatic disease. MV prolapse; flail leaflet or vegetations may be seen. Enlarged LV.  
**Doppler:** Regurgitant flow mapped into LA. Indirect evidence of pulmonary hypertension. | In nonrheumatic MR, valvuloplasty without valve replacement is increasingly successful. Acute MR (endocarditis, ruptured chordae) requires emergent valve replacement. Catheterization is the best assessment of regurgitation. |
| **AORTIC STENOSIS**  
(AS)  
Calcific (especially in congenitally bicuspid valve)  
Rheumatic disease | Concentric LVH. Prominent ascending aorta, small knob. Calcified valve common. | LVH. | **M-Mode:** Dense persistent echoes of the AoV with poor leaflet excursion. LVH with preserved contractile function.  
**2D:** Poststenotic dilatation of the aorta with restricted opening of the leaflets. Bicuspid AoV in about 30%. | “Critical” AS is usually defined as a valve area <0.7 cm² or a peak systolic gradient of >50 mm Hg. Catheterization is definitive diagnostic test. |
<table>
<thead>
<tr>
<th>Condition</th>
<th>Etiology</th>
<th>Diagnosis and Imaging</th>
<th>Prognosis and Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AORTIC REGURGITATION (AR)</strong></td>
<td>Bicuspid valves, Infective endocarditis, Hypertension, Rheumatic disease, Aorta/aortic root disease</td>
<td><strong>Doppler:</strong> Increased transvalvular flow velocity, yielding calculated gradient.</td>
<td>Prognosis without surgery is less than 50% survival at 3 yr when CHF, syncope, or angina occur. Balloon valvuloplasty has a high restenosis rate.</td>
</tr>
<tr>
<td></td>
<td></td>
<td><strong>M-Mode:</strong> Diastolic vibrations of the anterior leaflet of the MV and septum. Early closure of the valve when severe. Dilated LV with normal or decreased contractility. <strong>2D:</strong> May show vegetations in endocarditis, bicuspid valve, or root dilatation. <strong>Doppler:</strong> Demonstrates regurgitation. Estimates severity.</td>
<td>Aortography at catheterization can demonstrate AR. Acute incompetence leads to LV failure and requires AoV replacement.</td>
</tr>
<tr>
<td><strong>TRICUSPID STENOSIS (TS)</strong></td>
<td>Rheumatic disease</td>
<td><strong>M-Mode and 2D:</strong> TV thickening. Decreased early diastolic filling slope of the TV. MV also usually abnormal. <strong>Doppler:</strong> Prolonged pressure half-time across TV.</td>
<td>Right heart catheterization is diagnostic. Valvulotomy may lead to success, but TV replacement is usually needed.</td>
</tr>
<tr>
<td><strong>TRICUSPID REGURGITATION (TR)</strong></td>
<td>RV overload (pulmonary hypertension), Inferior infarction, Infective endocarditis</td>
<td><strong>M-Mode and 2D:</strong> Enlarged RV. MV often abnormal and may prolapse. <strong>Doppler:</strong> Regurgitant flow mapped into RA and venae cavae. RV systolic pressure estimated.</td>
<td>RA and jugular pressure tracings show a prominent V wave and rapid Y descent. Replacement of TV is rarely done. Valvuloplasty is often preferred.</td>
</tr>
</tbody>
</table>

1 Modified, with permission, from Tierney LM Jr, McPhee SJ, Papadakis MA (editors): Current Medical Diagnosis & Treatment 2000. McGraw Hill, 2000. RA = right atrium; RV = right ventricle; LA = left atrium; LV = left ventricle; AoV = aortic valve; MV = mitral valve; TV = tricuspid valve; LVH = left ventricular hypertrophy; RVH = right ventricular hypertrophy; CHF = congestive heart failure.
# Table 8-27. White Blood Cells: White Blood Cell Count and Differential

<table>
<thead>
<tr>
<th>Cells</th>
<th>Range ((10^3/\mu L))</th>
<th>Increased in</th>
<th>Decreased in</th>
</tr>
</thead>
<tbody>
<tr>
<td>WBC count (total)</td>
<td>3.4–10.0</td>
<td>Infection, hematologic malignancy.</td>
<td>Decreased production (aplastic anemia, folate or (B_{12}) deficiency, drugs [eg, ethanol, chloramphenicol]), decreased survival (sepsis, hypersplenism, drugs).</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>1.8–6.8</td>
<td>Infection (bacterial or early viral), acute stress, acute and chronic inflammation, tumors, drugs, diabetic ketoacidosis, leukemia (rare).</td>
<td>Aplastic anemia, drug-induced neutropenia (eg, chloramphenicol, phenothiazines, antithyroid drugs, sulfonamide), folate or (B_{12}) deficiency, Chédiak-Higashi syndrome, malignant lymphoproliferative disease, physiologic (in children up to age 4 years).</td>
</tr>
<tr>
<td>Lymphocytes</td>
<td>0.9–2.9</td>
<td>Viral infection (especially infectious mononucleosis, pertussis), thyrotoxicosis, adrenal insufficiency, ALL and CLL, chronic infection, drug and allergic reactions, autoimmune diseases.</td>
<td>Immune deficiency syndromes.</td>
</tr>
<tr>
<td>Monocytes</td>
<td>0.1–0.6</td>
<td>Inflammation, infection, malignancy, tuberculosis, myeloproliferative disorders.</td>
<td>Depleted in overwhelming bacterial infection.</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0–0.4</td>
<td>Allergic states, drug sensitivity reactions, skin disorders, tissue invasion by parasites, polyarteritis nodosa, hypersensitivity response to malignancy (eg, Hodgkin’s disease), pulmonary infiltrative disease, disseminated eosinophilic hypersensitivity disease.</td>
<td>Acute and chronic inflammation, stress, drugs (corticosteroids).</td>
</tr>
<tr>
<td>Basophils</td>
<td>0–0.1</td>
<td>Hypersensitivity reactions, drugs, myeloproliferative disorders (eg, CML), myelofibrosis.</td>
<td></td>
</tr>
</tbody>
</table>

1 In the automated differential, 10,000 WBCs are classified on the basis of size and peroxidase staining as neutrophils, monocytes, or eosinophils (peroxidase-positive) and as lymphocytes or large unstained cells (LUC), which are peroxidase-negative. LUCs, larger than normal lymphocytes, may be atypical lymphocyte or peroxidase-negative blasts. Basophils are identified using two-angle light scattering, based on their singular resistance to lysis. The reproducibility of 100-cell manual differentials is notoriously poor. Review of blood smears is useful to visually identify rare abnormal cells, blasts, nucleated RBCs, morphologic abnormalities (eg, hypersegmentation, toxic granulation, sickle cells, target cells, spherocytes, basophilic stippling) and to look for rouleaux (stacking of red cells due to increased globulins) and clumped platelets. WBC differential is unlikely to be abnormal with a normal WBC count or to be changed if the total WBC count is unchanged.

ALL = Acute lymphocytic leukemia; CLL = Chronic lymphocytic leukemia; CML = Chronic myelocytic leukemia.
<table>
<thead>
<tr>
<th>Component</th>
<th>Major Indications</th>
<th>Action</th>
<th>Not Indicated For—</th>
<th>Special Precautions</th>
<th>Hazards²</th>
<th>Rate of Infusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Whole blood</td>
<td>Symptomatic anemia with large volume deficit</td>
<td>Restoration of oxygen-carrying capacity, restoration of blood volume</td>
<td>Condition responsive to specific component</td>
<td>Must be ABO-identical Labile coagulation factors deteriorate within 24 hours after collection</td>
<td>Infectious diseases; septic/toxic, allergic, febrile reactions; circulatory overload; GVHD</td>
<td>For massive loss, as fast as patient can tolerate</td>
</tr>
<tr>
<td>Red blood cells; red blood cells with adenine-saline added³</td>
<td>Symptomatic anemia</td>
<td>Restoration of oxygen-carrying capacity</td>
<td>Pharmacologically treatable anemia Coagulation deficiency</td>
<td>Must be ABO-compatible</td>
<td>Infectious diseases; septic/toxic, allergic, febrile reactions; GVHD</td>
<td>As patient can tolerate, but less than 4 hours</td>
</tr>
<tr>
<td>Red blood cells, leukocyte-reduced</td>
<td>Symptomatic anemia, febrile reactions from leukocyte antibodies or cytokines, prevention of platelet refractoriness due to allo-immunization, decrease in infections and cancer recurrence (controversial)</td>
<td>Restoration of oxygen-carrying capacity</td>
<td>Pharmacologically treatable anemia Coagulation deficiency</td>
<td>Must be ABO-compatible</td>
<td>Infectious diseases; septic/toxic, allergic reactions (unless plasma also removed, eg, by washing); GVHD</td>
<td>As patient can tolerate, but less than 4 hours</td>
</tr>
<tr>
<td>Fresh-frozen plasma³</td>
<td>Deficit of labile and stable plasma coagulation factors and TTP</td>
<td>Source of labile and nonlabile plasma factors</td>
<td>Condition responsive to volume replacement</td>
<td>Must be ABO-compatible</td>
<td>Infectious diseases, allergic reactions, circulatory overload</td>
<td>Less than 4 hours</td>
</tr>
<tr>
<td>Component</td>
<td>Major Indications</td>
<td>Action</td>
<td>Not Indicated For—</td>
<td>Special Precautions</td>
<td>Hazards</td>
<td>Rate of Infusion</td>
</tr>
<tr>
<td>-----------</td>
<td>------------------</td>
<td>--------</td>
<td>-------------------</td>
<td>-------------------</td>
<td>---------</td>
<td>-----------------</td>
</tr>
<tr>
<td>Liquid plasma; plasma; and thawed plasma</td>
<td>Deficit of stable coagulation factors</td>
<td>Source of nonlabile plasma factors</td>
<td>Deficit of labile coagulation factors or volume replacement</td>
<td>Must be ABO-compatible</td>
<td>Infectious diseases; allergic reactions</td>
<td>Less than 4 hours</td>
</tr>
<tr>
<td>Cryoprecipitate AHF</td>
<td>Hemophilia A, von Willebrand’s disease, hypofibrinogenemia, factor XIII deficiency</td>
<td>Provides factor VIII, fibrinogen, von Willebrand factor, factor XIII</td>
<td>Deficit of any plasma protein other than those enriched in cryoprecipitated AHF</td>
<td>Frequent repeat doses may be necessary for factor VIII</td>
<td>Infectious diseases; allergic reactions</td>
<td>Less than 4 hours</td>
</tr>
<tr>
<td>Platelets; platelets from pheresis</td>
<td>Bleeding from thrombocytopenia or platelet function abnormality</td>
<td>Improves hemostasis</td>
<td>Plasma coagulation deficits and some conditions with rapid platelet destruction (eg, ITP)</td>
<td>Should not use some microaggregate filters (check manufacturer’s instructions)</td>
<td>Infectious diseases; septic/toxic, allergic, febrile reactions; GVHD</td>
<td>Less than 4 hours</td>
</tr>
<tr>
<td>Granulocytes from pheresis</td>
<td>Neutropenia with infection</td>
<td>Provides granulocytes</td>
<td>Infection responsive to antibiotics</td>
<td>Must be ABO-compatible; do not use depth-type microaggregate filters or leukodepletion filters</td>
<td>Infectious diseases; allergic, febrile reactions; GVHD</td>
<td>One unit over 2–4 hours. Observe closely for reactions.</td>
</tr>
</tbody>
</table>

2 For all cellular components, there is a risk the recipient may become alloimmunized.
3 Solvent detergent pooled plasma is an alternative in which some viruses are inactivated, but clotting factor composition is changed.
4 When virus-inactivated concentrates are not available.
5 Red blood cells and platelets may be processed in a manner that yields leukocyte-reduced components. The main indications for leukocyte-reduced components are prevention of febrile, nonhemolytic transfusion reactions and prevention of leukocyte alloimmunization. Risks are the same as for standard components except for reduced risk of febrile reactions.

**AHF** = antihemophilic factor; **GVHD** = graft-versus-host disease; **ITP** = idiopathic thrombocytopenic purpura; **TTP** = thrombotic thrombocytopenic purpura.
Index

Page numbers followed by t denote tables. Those followed by i denote illustrations.

A
Abdomen
abscess of
  computed tomography in, 259
  ultrasound in, 258
computed tomography of, 259
imaging test selection and interpretation
  in evaluation of, 258
infection of, leukocyte scan in, 281
magnetic resonance imaging of, 260
mass in, barium enema in, 263
mesenteric angiography of, 261
pain in, in hyperlipidemia, 380
trauma to, evaluation of
  angiography in, 279
  computed tomography in, 259
  mesenteric angiography in, 261
  tumors of, angiography in, 279
ultrasound of, 258
  in cholangitis/cholecystitis, 229
  in diverticulitis, 228
  in tuberculous peritonitis/enterocolitis, 227
  x-ray of (plain radiograph), 258
  in diverticulitis, 228
Abdominal aortic aneurysm, evaluation of
  angiography in, 279
  preoperative, magnetic resonance
    angiography in, 280
  spiral computed tomography angiography in, 259
  ultrasound in, 258
Aberrant ventricular conduction, 286
Abetalipoproteinemia
  cholesterol levels in, 71
  triglyceride levels in, 172
ABO grouping, 44, 401–402
  in type and cross-match, 44, 54, 175
  in type and screen, 44, 53, 176
Abortion
  therapeutic, prenatal diagnosis and, 384
  threatened, chorionic gonadotropin
    levels in, 72
  thrombophlebitis after, test selection in, 221
Abscess
  abdominal
    computed tomography in, 259
    ultrasound in, 258
  amebic, Entamoeba histolytica antibodies in, 50
  brain
    neighborhood meningeal reaction in, 371
    test selection in, 197
  197
  liver
    computed tomography in, 228, 268
    test selection in, 228
  lung
    computed tomography in, 252
    test selection in, 213
  neck, computed tomography in, 249
  in osteomyelitis, test selection in, 237
  in perinephric, test selection in, 232
  retropharyngeal, magnetic resonance
    imaging in, 248
Absolute neutrophil count, in bacteremia
  of unknown source, 241
ACA (centromere antibody), serum levels
  of, 69, 367
Acanthamoeba, test selection for, in
  keratitis, 205
Acanthocytes, 29
Acarbose, testosterone levels affected by, 165
Accelerated cardiac rhythm, 285
  of, 287, 289, 289
Accessory pathway
  left lateral, 330
  posteroseptal, 330
Accuracy of tests, 4, 4t, 5i
ACE. See Angiotensin-converting enzyme
ACE inhibitors. See Angiotensin-
  converting enzyme inhibitors
Acetaminophen
  lactate dehydrogenase levels affected
    by, 117
  serum levels of, 44
    and hepatotoxicity, 336
Acetaminophen (cont.)
  toxicity of, 44, 336
  nomogram for, 336
Acetazolamide
  ammonia levels affected by, 51
  carbon dioxide levels affected by, 66
  chloride levels affected by, 70
  phosphorus levels affected by, 138
  urinary calcium levels affected by, 65
Acetoacetate, serum or urine levels of, 45
Acetohexamide, urine osmolality affected by, 133
Acetone
  nitroprusside test for, 45
  serum osmolality affected by, 132
Acetylcholine receptor antibody, serum levels of, 45
Acetylcysteine, for acetaminophen toxicity, 44
N-Acetylprocainamide, electrocardiography affected by, 326
Achlorhydria
  gastrin levels in, 95
  in vitamin B_{12} deficiency, 363
Acid(s)
  increased intake of, pH in, 137
Acid-base disorders. See also specific disorders
  ionized calcium levels in, 64
  laboratory characteristics and clinical features of, 362
  lactate levels in, 118
  nomogram for, 337
  pH in, 137, 362
  phosphorus levels in, 138
  platelet count in, 140
  potassium levels in, 143
Acid-base status
  chloride levels and, 70
  PCO_{2} in, 66, 137, 362
  pH in, 137
  total carbon dioxide levels and, 66
Acid citrate, in specimen tubes, 25, 42
Acid elution test, for fetal hemoglobin, 103
Acid-fast bacilli. See also specific bacillus
  ascitic fluid, in tuberculous peritonitis/enterocolitis, 227
  brain abscess aspirate for, in brain abscess, 197
  bronchoalveolar brushings for, in community-acquired pneumonia, 212
  cerebrospinal fluid for, in tuberculous meningitis, 203, 369
  gastric washings for, in mycobacterial pneumonia, 215
  on Gram-stained smear, 28
  pericardial fluid for, in tuberculous pericarditis, 217
  pleural fluid for, 382–383
  sputum for
    in AIDS-related pneumonia, 214
    in mycobacterial pneumonia, 215
Acid-fast stain
  in HIV-associated diarrhea, 225
  in mycobacterial pneumonia, 215
  in tuberculous peritonitis/enterocolitis, 227
\alpha_{1}-Acid glycoprotein, electrophoresis in detection of, 146
Acidemia, ammonia levels in, 51
Acidosis. See also Diabetic ketoacidosis; Metabolic acidosis; Renal tubular acidosis; Respiratory acidosis
  lactic
    lactate levels in, 118
    pH in, 137
    phosphorus levels in, 138
    potassium levels in, 143
  Acinetobacter, test selection for, in hospital-acquired pneumonia, 213
Acquired immunodeficiency syndrome. See AIDS/HIV infection
Acromegaly
  creatinine clearance in, 81
  growth hormone levels in, 99
  insulin levels in, 115
  phosphorus levels in, 138
  somatomedin C levels in, 162
ACT. See Activated clotting time
ACTH. See Adrenocorticotropic hormone
Actinobacillus, test selection for, in infective endocarditis, 219
Actinomyces, test selection for
  in brain abscess, 197
  in liver abscess, 228
Activated clotting time, 73
Activated partial thromboplastin time, 136, 377
  inhibitor screen in evaluation of, 114
Acute abdominal series, 258
Acute intermittent porphyria
  chloride levels in, 70
  cholesterol levels in, 71
  urinary porphobilinogen levels in, 142
Acute lymphocytic leukemia
  bcr/abl translocation in, 57
  CD4/CD8 ratio in, 68
  leukocyte count in, 400
Acute myelocytic leukemia, vitamin B12 levels in, 400
Acute phase protein pattern, 146
Acute phase reactants
  and erythrocyte sedimentation rate, 86
  and factor VIII assay, 88
  and functional fibrinogen levels in, 92
  and haptoglobin levels, 99
  and von Willebrand’s factor protein levels, 184
Addison’s disease
  calcium levels in, 63
  cortisol levels in, 76
  glucose tolerance test in, 96
  potassium levels in, 143
  renal tubular acidosis in, 388
  thyroperoxidase antibody in, 167
Adenoma
  benign nonhyperfunctioning adrenal, magnetic resonance imaging in, 260
  parathyroid, evaluation of magnetic resonance imaging in, 248
  parathyroid scan in, 251
  pituitary, amenorrhea caused by, 339
  thyroid, thyroglobulin antibody in, 166
Adenosine deaminase, pericardial fluid levels of, in tuberculous pericarditis, 217
Adenosine diphosphate, platelet aggregation by, 139
Adenovirus, test selection for in conjunctivitis, 204
  in infectious myocarditis, 218
  in laryngitis, 209
  in laryngotracheobronchitis, 210
  in pharyngitis, 209
  in sinusitis, 208
  in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230
ADH. See Antidiuretic hormone
Adrenal/adrenocortical tumors/cancer
  Cushing’s syndrome caused by, 340
  glucose levels in, 95
  hirsutism caused by, 346
  in hypertension associated with hypokalemia, 348
  magnetic resonance imaging in, 260
  pheochromocytoma evaluation, 355
  testosterone levels in, 165
Adrenal gland
  computed tomography of, in hypertension, 348
  imaging test selection and interpretation in evaluation of, 272
Adrenal hyperplasia
  hirsutism caused by, 346
  in hypertension associated with hypokalemia, 348
  testosterone levels in, 165
Adrenal insufficiency (adrenocortical insufficiency)
  ACTH levels in, 46, 338
  chloride levels in, 70
  cortisol levels in, 77, 127, 338
  cosyntropin stimulation test in, 77, 338
  diagnostic algorithm for, 338
  glucose levels in, 95
  glucose tolerance test in, 96
  hypoglycemia and, 349
  laboratory evaluation of, 338
  leukocyte count in, 400
  metyrapone test in, 127, 338
  phosphorus levels in, 138
  plasma renin activity in, 152
  primary, 338
  secondary, 338
  serum osmolality in, 132
  serum sodium levels in, 161
Adrenergic agents, potassium levels affected by, 143
Adrenocorticotropic hormone in adrenocortical insufficiency, 46, 338
  ectopic production of, Cushing’s syndrome caused by, 340
  plasma levels of, 46
  in Cushing’s syndrome, 46, 340
  in hypoglycemia, 349
Adrenogenital syndrome, ACTH levels in, 46
Aerodigestive tract, upper, evaluation of computed tomography in, 249
  magnetic resonance imaging in, 248
Aeromonas, test selection for, in infectious colitis/dysentery, 223
AFB. See Acid-fast bacilli
Afibrinogenemia
  activated clotting time in, 73
  fibrinogen levels in, 92
  reptilase clotting time in, 153
Russell’s viper venom clotting time in, 157
AFP. See Alpha-fetoprotein
Agammaglobulinemia, protein levels in, 147
Agglutination, latex. See Latex agglutination test
Agglutinins
Brucella antibody, 60
cold, 74
Q fever antibody, 149
tularemia, 175
Aging
25-hydroxy vitamin D₃ levels affected by, 182
erthrocyte sedimentation rate affected by, 86
nuclear antibody levels affected by, 131
rapid plasma reagin test affected by, 150
rheumatoid factor levels affected by, 155, 368
testosterone levels affected by, 165
uric acid levels affected by, 177
AIDS/HIV infection
troponin-I levels in, 174
CD4/CD8 ratio in, 68
cholesterol levels in, 71
cosyntropin stimulation test in, 77
enteropathy, D-xylose absorption test in, 185
ferritin levels in, 90
fluorescent treponemal antibody-absorbed test reactivity affected by, 92
heterophile agglutination (Monospot/ Paul-Bunnell) test in, 107
Histoplasma capsulatum antigen levels in, 108
HIV antibody levels in, 110
lactate dehydrogenase levels in, 117
methylmalonic acid levels in, 126
β₂-microglobulin levels in, 128
rapid plasma reagin test in, 150
smooth muscle antibody levels in, 160
test selection for infections associated with
aseptic meningitis, 199
brain abscess, 197
cholangitis, 229, 266
cholecystitis, 229
cryptococcal meningitis, 82
diarrhea, 225
encephalitis, 198
fungal meningitis, 201
infectious colitis/dysentery, 223
infectious esophagitis, 222
infectious myocarditis, 218
keratitis, 205
laryngitis, 209
otitis externa, 207
pericarditis, 217
pneumonia, 214–215
sinusitis, 208
spirochetal meningitis/neurosyphilis, 202
tuberculosis, 215
Toxoplasma antibody test in, 171
vitamin B₁₂ levels in, 180
AIDS-related complex, fluorescent treponemal antibody-absorbed test reactivity affected by, 92
Airway disease, chronic obstructive, test selection in, 210
Airway management, in epiglottis, 211
Airway obstruction, partial pressure of oxygen in, 133
Alanine aminotransferase, serum levels of, 46, 378
in hepatitis, 46, 343i–345i, 378
Albumin. See also Hypoalbuminemia
electrophoresis in detection of, 146
serum levels of, 47, 146, 378
and operative death rate after portacaval shunt (Child’s criteria), 372
ratio to ascites, 227, 365–366
in tuberculous peritonitis/enterocolitis, 227
testosterone binding to, 165
Albuterol, magnesium levels affected by, 123
Alcohol. See Ethanol
Alcoholic ketoacidosis, acetoacetate levels in, 45
Alcoholic liver disease. See also Cirrhosis; Hepatitis
gamma-glutamyl transpeptidase levels in, 94
Alcoholism
bacterial meningitis in, test selection in, 200
cortisol levels in, 76
creatine kinase levels in, 78
ferritin levels in, 90
folic acid deficiency in, 363
gamma-glutamyl transpeptidase levels in, 94
hyperlipidemia in, 379
magnesium levels in, 123
phosphorus levels in, 138
triglyceride levels in, 172, 379
Aldosterone
plasma, 48
plasma renin activity and, 48
salt-depleted, 48
salt-loaded, 48
urinary, 49
in hypertension associated with
hypokalemia, 348
i
plasma renin activity and, 49
salt-depleted, 49
salt-loaded, 49
Aldosteronism. See Hyperaldosteronism
Algorithms, clinical, 20
Alkali administration, pH affected by, 137
Alkaline body
fluids, increased loss of, pH
affected by, 137
Alkaline phosphatase
leukocyte, blood levels of, 120
serum levels of, 50, 378
in liver abscess, 50, 228
Alkaline picate method, for serum
creatinine, 80
Alkalosis
carbon dioxide levels in, 66
chloride levels in, 70
laboratory characteristics and clinical
features of, 362
nomogram for, 337
i
pH in, 137, 362
i
phosphorus levels in, 138
platelet count in, 140
potassium levels in, 143
Alkaptonuria, urine color affected by, 30
Allergic bronchopulmonary aspergillosis,
test selection in, 210
Allergic reactions
in intravenous contrast studies, 244
leukocyte count in, 400
i
Alloantibody, detection of
antibody screen for, 53
indirect antiglobulin test for, 54
Alloimmune reactions, direct antiglobulin
test for, 54
Alloimmune thrombocytopenia, platelet-
associated IgG in, 141
Allopurinol, uric acid levels affected by, 177
Alpha-fetoprotein
in amniotic fluid, 91, 384
i
serum levels of, 91
Alpha-globin gene, mutation in, in
α-thalassemia, 375
i, 392
i
Alpha-hydroxylase deficiency
1,25-dihydroxy vitamin D₃ levels in,
183
hypertension associated with
hypokalemia and, 348
i
Alpha-thalassemia, 392. See also Thalas-
semia syndromes
molecular diagnostic techniques for,
375
i
Alpha toxin, Clostridium perfringens
producing, 239
Alpha₁-antiprotease (α₁-antitrypsin)
electrophoresis in detection of, 146
serum levels of, 55
Alpha₁-antiprotease deficiency
congenital, 55
protein electrophoresis in, 146
Alpha₁-serum protein, 146
Alpha₂-macroglobulin, electrophoresis in
detection of, 146
Alpha₂-serum protein, 146
ALT. See Alanine aminotransferase
Alitude, red cell volume affected by, 151
Alveolar hypoventilation, partial pressure
of oxygen in, 133
Alveolar ventilation, pH affected by, 137
Alzheimer’s disease
positron emission tomography in, 247
vitamin B₁₂ levels in, 180
Amebic abscess, Entamoeba histolytica
antibodies in, 50
Amebic dysentery
Entamoeba histolytica antibodies in, 50
test selection in, 223
Amebic serology, 50
Amenorrhea
diagnostic algorithm for, 339
follicle-stimulating hormone levels in,
93, 339
i
luteinizing hormone levels in, 339
i
prolactin levels in, 144, 339
i
thyroid-stimulating hormone levels in,
339
i
Amikacin
therapeutic monitoring of, 191
i
tobramycin levels affected by, 194
i
Aminopyrine, leukocyte count affected by, 121
Aminotransferases. See Alanine aminotrans-
ferase: Aspartate aminotransferase
Amiodarone
digoxin levels affected by, 192
i
electrocardiography affected by, 326
free thyroxine index affected by, 170
free thyroxine levels affected by, 170
total thyroxine levels affected by, 169
total triiodothyronine affected by, 173
Amitriptyline
therapeutic monitoring of, 191
urine color affected by, 30
Ammonia, plasma levels of, 51
Ammonium chloride, pH affected by, 137
Amniocentesis, 384
Amniotic cell culture, 384
in fragile X syndrome, 374
in hemophilia A, 374
in thalassemia syndromes, 375–376
Amniotic fluid
contamination of, lecithin/sphingomyelin ratio in, 119
α-fetoprotein in, 91
laboratory analysis of, 384
in chorioamnionitis/endometritis, 236
lecithin/sphingomyelin ratio in, 119
Amphotericin B, magnesium levels affected by, 123
Amylase, serum levels of, 52
Amyloidosis
angiotensin-converting enzyme levels in, 52
complement C3 levels in, 75
and low-voltage QRS complex in ECG, 308
β2-microglobulin levels in, 128
thrombin time in, 165
Anabolic steroids
phosphorus levels affected by, 138
protein levels affected by, 147
Anaeorobes, test selection for
in anaerobic pneumonia or lung abscess, 213
in aspiration pneumonia, 212
in brain abscess, 197
in cellulitis, 240
in chorioamnionitis/endometritis, 236
in community-acquired pneumonia, 212
in empyema, 216
in gas gangrene, 239
in hospital-acquired pneumonia, 213
in impetigo, 239
in laryngotracheobronchitis, 210
in necrotizing fasciitis, 240
in neutropenic pneumonia, 214
in otitis media, 206
in peritonitis, 226
in salpingitis/pelvic inflammatory disease, 236
in sinusitis, 208
Anaerobic pneumonia/lung abscess, test selection in, 213
Analbuminemia, congenital, albumin levels in, 47
Anasarca, and low-voltage QRS complex in ECG, 308
Anastomotic leak, evaluation of
Hypaque enema in, 264
upper GI study in, 262
ANCA. See Neutrophil cytoplasmic antibodies
Androgen resistance, partial, in infertility, 352
Androgens
chloride levels affected by, 70
protein levels affected by, 147
thyroid function tests affected by, 394
thyroxine levels affected by, 169
Anemia
basophilic stippling in, 363–364
calcitonin levels in, 62
causes of, 363
of chronic disease. See Chronic disease, anemia of
cold agglutinin levels in, 74
cryoglobulin levels in, 82
diagnosis of, based on red blood cell indices, 363–364
direct antiglobulin test for, 54
erthrocyte count in, 85
erthrocyte sedimentation rate in, 86
erthropoietin levels in, 86
ferritin levels in, 90, 364
fetal hemoglobin levels in, 103
free erythrocyte protoporphyrin in, 94, 363–364
gastrin levels in, 95
glucose-6-phosphate dehydrogenase deficiency and, 97
glycohemoglobin levels in, 98
haptoglobin levels in, 99
hematocrit in, 101
hemoglobin levels in, 103
hemosiderin levels in, 104
laboratory and clinical findings in, 363–364
lactate dehydrogenase isoenzyme levels in, 117
lactate dehydrogenase levels in, 117
mean corpuscular hemoglobin in, 123–124, 363
mean corpuscular volume in, 124,
363–364
methylmalonic acid levels in, 126
molecular diagnostic techniques for, 375–376
protein electrophoresis in, 146
reticulocyte count in, 153, 363
serum iron levels in, 115, 363–364
thyroglobulin antibody in, 166
total iron-binding capacity in, 116, 363–364
transferrin saturation with iron in, 116, 364
transfusion in, 401
vitamin B12 absorption test (Schilling’s test) in, 181
vitamin B12 deficiency in, 93
vitamin B12 levels in, 180
Anephric patients, 1,25-dihydroxy vitamin D3 levels in, 183
Anesthetics
methemoglobin levels affected by, 126
urine osmolality affected by, 133
Anesthetics, methemoglobin levels affected by, 126
Aneurysm
abdominal. See Abdominal aortic aneurysm
aortic, evaluation of computed tomography in, 252
magnetic resonance imaging in, 253
intracranial, magnetic resonance angiography in, 246
splanchnic artery, mesenteric angiography in evaluation of, 261
splenic artery, mesenteric angiography in evaluation of, 261
ventricular, ST segment elevation in, 320
Angina
cardiac troponin-I levels in, 174
intestinal, mesenteric angiography in evaluation of, 261
Angioedema, hereditary
C1 esterase inhibitor levels in, 61
complement C4 levels in, 75
Angiography
in aorta evaluation, 279
hepatic, 271
magnetic resonance in aorta evaluation, 280
in brain evaluation, 246
in neck evaluation, 248
mesenteric, 261
in pheochromocytoma evaluation, 355
pulmonary, 255
in pulmonary embolism, 255, 357
spiral computed tomography, 259
Angiotensin-converting enzyme, serum levels of, 52
Angiotensin-converting enzyme inhibitors
plasma renin activity affected by, 152
potassium levels affected by, 143
for renal scan enhancement, 274
sodium levels affected by, 161
Angiotensinogen, angiotensin I generated from, 152
Aniline dyes, methemoglobin levels affected by, 126
Animals, birthing, pneumonia associated with exposure to, test selection in, 212
Anion gap, in acid-base disorders, 362
lactate levels and, 118
serum chloride levels and, 70
Anisocytosis, erythrocyte sedimentation rate in, 86
Ankylosing spondylitis
HLA-B27 typing in, 111
synovial fluid sampling in, 389
Anorexia nervosa
cholesterol levels in, 71
follicle-stimulating hormone levels in, 93
luteinizing hormone levels in, 122
triglyceride levels in, 172
Anosmia, gonadotropin deficiency associated with, luteinizing hormone levels in, 122
Anovulation, amenorrhea caused by, 339
Antacids
gastrin levels affected by, 95
pH affected by, 137
phosphorus levels affected by, 138
serum calcium levels affected by, 63
Anterior cutaneous nerve of neck, 342
Anterior femoral cutaneous nerve, 341–342
Antibiotics, electrocardiography affected by, 326
Anti-double-stranded-DNA antibody (anti-ds-DNA) test, 84, 367
Anti-EA antibody (early antigen antibodies), 85
Anti-EB nuclear antigen antibody (anti-EBNA), 85
Anti-HAV antibody, serum levels of, 104, 343
Anti-HBc antibody, serum levels of, 105, 344
Anti-HBs antibody, serum levels of, 105, 344
Anti-HCV antibody, serum levels of, 106, 345
Anti-HDV antibody, serum levels of, 107
Anti-insulin antibodies, serum levels of, 114
  in hypoglycemia, 349
Anti-neutrophil cytoplasmic antibody, 130, 368
Anti-nuclear antibody test, 131, 367
Anti-Rh antibodies, Rh grouping for detection of, 154
Anti-ribonucleoprotein antibody (RNP) test, 155, 367
Anti-Scl-70 antibody, serum levels of, 158, 368
Anti-Smith (anti-Sm) antibody, serum levels of, 159, 367
Anti-SS-A/Ro antibody, serum levels of, 163, 368
Anti-SS-B/La antibody, serum levels of, 163
Antiarrhythmic drugs, electrocardiography affected by, 324, 325, 326
  ST segment depression or T wave inversion with, 322
Antibiotic(s), electrocardiography affected by, 326
Antibiotic-associated colitis
  Clostridium difficile enterotoxin levels in, 73, 224
test selection in, 224
Antibiotic-associated diarrhea
  Clostridium difficile enterotoxin levels in, 73
test selection in, 224
Antibiotic-associated peritonitis, test selection in, 226
Antibiotic-associated pseudomembranous colitis, test selection in, 224
Antibody screen, 53
  for platelet-associated IgG, 141
  in type and cross-match, 54, 175
  in type and screen, 53, 176
Anticardiolipin antibody, inhibitor screen in detection of, 114
Anticoagulants
  activated clotting time in monitoring of, 73
  antithrombin III levels affected by, 56
  circulating
    activated clotting time affected by, 73
    inhibitor screen in detection of, 114
    partial thromboplastin time in detection of, 136
    prothrombin time in detection of, 148
    Russell’s viper venom clotting time in detection of, 157
    thrombin time in screening for, 165
hemostatic function tests affected by, 377
  lupus
    inhibitor screen in detection of, 114
    partial thromboplastin time in, 136
    in Q fever, 149
    Russell’s viper venom clotting time in, 157
    and thrombosis, 157
protein C levels affected by, 145
Anticonvulsants
cyclosporine levels affected by, 191
phosphorus levels affected by, 138
  testosterone levels affected by, 165
Antidepressants
electrocardiography affected by, 326
sodium levels affected by, 161
Antidiuretic hormone (vasopressin). See also Syndrome of inappropriate antidiuretic hormone
plasma levels of, 53
Antifungals, electrocardiography affected by, 326
Antiglobulin test
direct (direct Coombs test), 54
indirect (indirect Coombs test), 54
Antihemophilic factor (factor VIII)
  assay for, 77, 388
  cryoprecipitated, transfusion of, 402
  disorders of
    activated clotting time in, 73
    bleeding time in, 59
    cryoprecipitated antihemophilic factor transfusion for, 402
    factor VIII assay in, 88, 377
  hemostatic function in, 377
Antihistamines, electrocardiography affected by, 326
Antimalarial drugs, glucose-6-phosphate dehydrogenase deficiency and, 97
Antimetabolites, uric acid levels affected by, 177
Antimyosin antibody scintigraphy, in infectious myocarditis, 218
Antiphospholipid antibody. See also Lupus anticoagulant
  inhibitor screen in detection of, 114
  in Q fever, 149
Russell’s viper venom clotting time in detection of, 157
Antiplatelet antibody test, 141
  α1-Antiprotease (α1-antitrypsin) deficiency of
    congenital, 55
    protein electrophoresis in, 146
electrophoresis in detection of, 146
serum levels of, 55
Antipsychotics, sodium levels affected by, 161
Antistreptolysin O titer, 55
Antithrombin III
deficiency of, 56
plasma levels of, 56
Antithyroid drugs, leukocyte count affected by, 400t
Antithyroid peroxidase antibody test, 166–167
\( \alpha_1 \)-Antitrypsin. See \( \alpha_1 \)-Antiprotease
Antiviral capsid antibodies (anti-VCA), Epstein-Barr virus, 85
Antral G cell hyperplasia, gastrin levels in, 95
Antrectomy, gastric levels affected by, 95
Anxiety
cortisol levels in, 76
pH in, 137
vanillylmandelic acid levels in, 178
Aorta, evaluation of angiography in, 279
imaging test selection and interpretation in evaluation of, 279–280
magnetic resonance angiography in, 280
spiral computed tomography angiography in, 259
Aorta/aorta root disease, diagnostic evaluation of, 399t
Aortic aneurysm
abdominal. See Abdominal aortic aneurysm
evaluation of computed tomography in, 252
magnetic resonance imaging in, 253
Aortic dissection, evaluation of angiography in, 279
computed tomography in, 252
magnetic resonance imaging in, 253
Aortic regurgitation, diagnostic evaluation of, 399t
Aortic rupture, chest x-ray in evaluation of, 252
Aortic stenosis, diagnostic evaluation of, 398t
Aortoenteric fistula, computed tomography in, 259
Aortofemoral bypass reconstructive surgery, angiography in preoperative evaluation for, 279
Aplastic anemia
erthropoietin levels in, 86
fetal hemoglobin levels in, 103
iron levels in, 115
leukocyte count in, 121, 400t
reticulocyte count in, 153
transferrin saturation with iron in, 116
Apolipoprotein C-II deficiency, in hyperchylomiconemina, 380t
Apolipoprotein E dysfunction, in dysbeta-lipoproteinemia, 380t
Appendicitis
evaluation of computed tomography in, 259
ultrasound in, 258
leukocyte count in, 121
Arachidonic acid, platelet aggregation by, 139
Arboviruses, test selection for, in encephalitis, 198
ARC. See AIDS-related complex
Arcancobacterium hemolyticum, test selection for, in pharyngitis, 209
Arm cables, reversal of, in ECG, 327
Arrhenoblastoma, testosterone levels in, 165
Arrhythmia, 284–299. See also specific types
Arterial blood sampling, specimen handling for, 26t
Arterial dissection. See also Aortic dissection cerviocranial, magnetic resonance angiography in evaluation of, 248
Arterial portography, computed tomography, 268
Arterial thrombosis. See Thrombosis
Arteries, patency of, ultrasound in evaluation of, 249
Arteriography. See Angiography
Arteriovenous malformations
angiography in, 279
computed tomography in, 252
magnetic resonance imaging in, 253
Arteritis, temporal, erythrocyte sedimentation rate in, 86
Arthritis
bacterial/septic
synovial fluid sampling in, 238, 390t
test selection in, 238
bone scan in, 276
cryoglobulin causing, 82
rheumatoid. See Rheumatoid arthritis
synovial fluid sampling in, 389t
Arthropathy, neuropathic, synovial fluid profile in, 389r–390t
Artificial ventilation, pH affected by, 137
Ascaris lumbricoides, test selection for, in cholangitis/cholecystitis, 229
Ascites
ascitic fluid profiles in various disease states, 365t–366t
chylous, ascitic fluid profile in, 366t
and operative death rate after portocaval shunt (Child’s criteria), 372t
ratio of albumin to serum albumin, 227, 365t–366t
ultrasound in evaluation of, 267
Ascitic fluid sampling
normal values for, 365t
in peritonitis, 226
specimen handling for, 26t
in tuberculous peritonitis/enterocolitis, 227, 365t
Ascorbic acid, triglyceride levels affected by, 172
Aseptic meningitis
cerebrospinal fluid profile in, 199, 369t
test selection in, 199
Aseptic necrosis, magnetic resonance imaging in, 278
Asparaginase
ammonia levels affected by, 51
thyroid function tests affected by, 394t
Aspartate aminotransferase, serum levels of, 56, 378t
in hepatitis, 56, 343i–344i, 378t
Aspergillus, test selection for
in fungal meningitis, 201
in infectious esophagitis, 222
in keratitis, 205
in laryngotracheobronchitis, 210
in neutropenic pneumonia, 214
in otitis externa, 207
in peritonitis, 226
in sinusitis, 208
in transplant-related pneumonia, 214
Aspiration, of gastrointestinal contents and lung abscess, 213
pH affected by, 137
pneumonia caused by esophageal reflux study in, 264
test selection in, 212–213
Aspirin
bleeding time affected by, 59
magnesium levels affected by, 123
overdose of. See also Salicylate, poisoning/toxicity
nomogram for determining severity of, 360i
plasma renin activity affected by, 152
platelet aggregation affected by, 139
serum levels of, 158
urinary calcium levels affected by, 65
Asplenia, liver/spleen scan in, 271
AST. See Aspartate aminotransferase
Astemizole, electrocardiography affected by, 326
Asthma, partial pressure of oxygen in, 133
AT III. See Antithrombin III
Ataxia-telangiectasia
CD4/CD8 ratio in, 68
IgA levels in, 113
Atelectasis, partial pressure of oxygen in, 133
Atherosclerosis
carotid bifurcation, magnetic resonance angiography in evaluation of, 248
hyperlipidemia and, 379t
Atherosclerotic disease, angiography in, 279
Asthombia, essential, platelet aggregation in, 139
Atopic dermatitis, CD4/CD8 ratio in, 68
Atrial abnormalities, electrocardiographic findings of, 300–301
Atrial arrhythmia, focal, 287
Atrial bradycardia, ectopic, 288t
QRS duration in, 285t
Atrial enlargement, 300–301, 307
Atrial fibrillation, 288
anterograde conduction of, in Wolff-Parkinson-White syndrome, 285t
irregularly irregular QRS rhythm in, 286
QRS duration in, 285t
Atrial flutter, 285t, 286, 288, 299
Atrial pause
causes of, 286
definition of, 286
Atrial rhythms, 287, 288t
ectopic, 288t
QRS duration in, 285t
Atrial tachycardia, 288t, 299
with atrioventricular block
irregularly irregular QRS rhythm in, 286–287
QRS duration in, 285t
multifocal, 287–288
irregularly irregular QRS rhythm in, 286
QRS duration in, 285t
QRS duration in, 285t
Atrioventricular block, 297
atrial tachycardia with
irregularly irregular QRS rhythm in, 286–287
QRS duration in, 285
definitions of, 297
first-degree, 297
new, in left anterior descending artery occlusion, 316
in inferior myocardial infarction, 316
Mobitz type II, 287, 297
multiform atrial rhythm, QRS duration in, 285
second-degree, 297
prolonged QT interval in, 327
type I, 297
type II, 297
third-degree, 297–298
prolonged QT interval in, 327
Wenckebach, 287, 297
Atrioventricular conduction, variable, QRS duration in, 285
Atrioventricular dissociation, 298
complete, 298
incomplete, 298
in wide QRS complex tachycardia with regular rhythm (WCT-RR), 290, 295
Atrioventricular reentry tachycardia, 299
Atypical gastritis, gastrin levels in, 95
Atypical pneumonia, test selection in, 212
Atypical transient ischemic attack, carotid Doppler in, 278
Auditory canal, infection of. See Otitis
Auto manufacturers, lead poisoning in, 119
Autoagglutination
erythrocyte count affected by, 85
mean corpuscular hemoglobin concentration in, 124
mean corpuscular volume in, 124
Autoantibodies. See also specific autoantibodies
antibody screen for, 53
in connective tissue disease, 367–368
indirect antiglobulin test for, 54
neutrophil cytoplasmic, serum levels of, 130, 368
Autoimmune disease
IgG levels in, 113
leukocyte count in, 400
β2-microglobulin levels in, 128
protein electrophoresis in, 146
rapid plasma reagin test in, 150
renal tubular acidosis in, 388
Autoimmune hemolytic anemia
cold agglutinin levels in, 74
complement C3 levels in, 75
direct antiglobulin test for, 54
haptoglobin levels in, 99
Autoimmune thrombocytopenia, platelet-associated IgG in, 141
Autoimmune thyroid disease
antithyroid peroxidase antibody test in, 166
thyroglobulin antibody in, 166
thyroperoxidase antibody in, 167
Automobile exhaust, carboxyhemoglobin blood levels affected by, 66
AV block. See Atrioventricular block
AV dissociation. See Atrioventricular dissociation
AV nodal reentry tachycardia, 299
Avascular necrosis
bone scan in, 276
synovial fluid sampling in, 389
AVNRT (AV nodal reentry tachycardia), 299
Avocado, 5-hydroxyindoleacetic acid levels affected by, 111
AVRT (atrioventricular reentry tachycardia), 299
Axillary nerve, 341–342
Axis, electrocardiographic deviations in
left (LAD), 305
and presumption of disease, 304
right (RAD), 305–306
right superior, 306
mean
determination of, 304–306
in frontal plane (limb leads), 304–305
normal range, in adults, 304
Azathioprine, and amylase levels, 52
Azotemia
postrenal, 387
prerenal, 387
B
B cell immunoglobulin heavy chain gene rearrangement, 57
B cell transplants, C-peptide levels in, 62
Babesia, test selection for, in bacteremia of unknown source, 241
Bacillus cereus, test selection for in endophthalmitis, 205
in infectious colitis/dysentery, 223
Bacteremia
gram-negative, complement C3 levels in, 75
Bacteremia (cont.)
perinephric abscess associated with, test
selection in, 232

Bacterial meningitis
cerebrospinal fluid profile in, 200, 369t
test selection in, 200

Bacterial overgrowth
intestinal
vitamin B_{12} absorption test
(Schilling’s test) in, 181
vitamin B_{12} levels in, 180
D-xylose absorption test in, 185
in vaginitis/vaginosis, 234

Bacterial pericarditis, test selection in, 217

Bacterial peritonitis, ascitic fluid profile in,
365t

Bacterial tracheitis, endoscopy in, 210

Bacteriuria
in prostatitis, 231
untreated, and pyelonephritis, 232
in urinary tract infection/cystitis/
pyuria-dysuria syndrome, 230

Bacteroides
on Gram-stain smear, 27
test selection for,
in anaerobic pneumonia or lung
abscess, 213
in aspiration pneumonia, 212
in brain abscess, 197
in cellulitis, 240
in cholangitis/cholecystitis, 229
in community-acquired pneumonia, 212
in conjunctivitis, 204
in empyema, 216
in endophthalmitis, 205
in epididymitis/orchitis, 233
in epiglottitis, 211
in gas gangrene, 239
in hospital-acquired pneumonia, 213
in immunocompromise-related pneu-
monia, 214
in infectious myocarditis, 218
in infectious thrombophlebitis, 221
in infective endocarditis, 219
in keratitis, 205
in liver abscess, 228
in mucopurulent cervicitis, 235
in mycobacterial pneumonia, 215
in osteomyelitis, 237
in otitis externa, 207
in otitis media, 206
in perinephric abscess, 232
in pharyngitis, 209
in prosthetic valve infective endo-
carditis, 220
in pyelonephritis, 232
in salpingitis/pelvic inflammatory
disease, 236
in sinusitis, 208
in tuberculous pericarditis, 217
in tuberculous peritonitis, 227
in urethritis, 233
in vaginitis/vaginosis, 234

Bacterial cultures, in peritonitis, 226

Bacterial endophthalmitis, test selection
in, 205

Bacterial growth products, antistreptolysin
O titer affected by, 55

Bacterial keratitis, test selection in, 205

Bacterial meningitis
cerebrospinal fluid profile in, 200, 369t
test selection in, 200

Bacterial overgrowth
intestinal
vitamin B_{12} absorption test
(Schilling’s test) in, 181
vitamin B_{12} levels in, 180
D-xylose absorption test in, 185
in vaginitis/vaginosis, 234

Bacterial pericarditis, test selection in, 217

Bacterial peritonitis, ascitic fluid profile in,
365t

Bacterial tracheitis, endoscopy in, 210

Bacteriuria
in prostatitis, 231
untreated, and pyelonephritis, 232
in urinary tract infection/cystitis/
pyuria-dysuria syndrome, 230

Bacteroides
on Gram-stain smear, 27
test selection for,
in anaerobic pneumonia or lung
abscess, 213
in aspiration pneumonia, 212
in brain abscess, 197
in cellulitis, 240
in cholangitis/cholecystitis, 229
in community-acquired pneumonia, 212
in conjunctivitis, 204
in empyema, 216
in endophthalmitis, 205
in epididymitis/orchitis, 233
in epiglottitis, 211
in gas gangrene, 239
in hospital-acquired pneumonia, 213
in immunocompromise-related pneu-
monia, 214
in infectious myocarditis, 218
in infectious thrombophlebitis, 221
in infective endocarditis, 219
in keratitis, 205
in liver abscess, 228
in mucopurulent cervicitis, 235
in mycobacterial pneumonia, 215
in osteomyelitis, 237
in otitis externa, 207
in otitis media, 206
in perinephric abscess, 232
in pharyngitis, 209
in prosthetic valve infective endo-
carditis, 220
in pyelonephritis, 232
in salpingitis/pelvic inflammatory
disease, 236
in sinusitis, 208
in tuberculous pericarditis, 217
in tuberculous peritonitis, 227
in urethritis, 233
in vaginitis/vaginosis, 234

Bacterial cultures, in peritonitis, 226

Bacterial endophthalmitis, test selection
in, 205

Bacterial growth products, antistreptolysin
O titer affected by, 55

Bacterial keratitis, test selection in, 205
Barium enema, 263
   in diverticulitis, 228
Barium esophagram, in infectious esophagitis, 222
Barium fluoroscopy
   enteroclysis, 262
   peroral pneumocolon, 263
Barium swallow (upper GI study), 262
Bartlett tube, for bronchoalveolar sampling, in anaerobic pneumonia or lung abscess, 213
Bartonella, test selection for
   in bacteremia of unknown source, 241
   in conjunctivitis, 204
Bartonella henselae, test selection for
   in encephalitis, 198
   in osteomyelitis, 237
Bartter’s syndrome, plasma renin activity in, 152
Basophil, 29
   Basophil count, 400t
   Basophilic stippling, 29
   in anemias, 363t–364t
Bazett’s formula, 324
BCL/1 mutation, in hemophilia A, 374t
   bcr/abl translocation, 57
Beets, urine color affected by, 30
Behçet’s disease, smooth muscle antibody levels in, 160
Bence Jones proteins, 82, 146
Benign prostatic hypertrophy, prostate-specific antigen levels in, 144
Benzocaine, methemoglobin levels affected by, 126
Bernard–Soulier syndrome, bleeding time in, 59
Beta-adrenergic agonists, phosphorus levels affected by, 138
Beta-blockers
   free thyroxine index affected by, 170
   free thyroxine levels affected by, 170
   plasma renin activity affected by, 152
   potassium levels affected by, 143
   total thyroxine levels affected by, 169
   triglyceride levels affected by, 172
Beta-globin gene, mutation in, in β-thalassemia, 376t, 392t
Beta-hemolytic group A Streptococcus. See also Streptococcus, Group A
   antistreptolysin O titer in infection caused by, 55
Beta-hydroxylase deficiency, hypertension associated with hypokalemia and, 348i
Beta-thalassemia. See also Thalassemia syndromes
   hemoglobin electrophoresis in, 392t
   molecular diagnostic techniques for, 376t
Beta2-microglobulin, serum levels of, 128
Beta2-serum protein, 146
Bias, spectrum, 8
Bicarbonate
   in acid-base disorders, 362t
   chloride levels affected by, 70
   pH affected by, 137
   serum levels of, in renal tubular acidosis, 388t
   serum osmolality affected by, 132
   total carbon dioxide serum levels and, 66
   urinary calcium levels affected by, 65
Bicarbonate-carbonic acid buffer, in pH maintenance, 66
Bicuspid valves, diagnostic evaluation of, 398t–399t
Bile duct obstruction
   hepatic function tests in, 378t
   hepatic inminodiacetic acid scan in, 266
Bile leaks, hepatic inminodiacetic acid scan in, 266
Biliary cirrhosis
   25-hydroxy levels vitamin D3 levels in, 182
   angiotensin-converting enzyme levels in, 52
   CD4/CD8 ratio in, 68
   ceruloplasmin levels in, 69
   hepatic function tests in, 378t
   mitochondrial antibody levels in, 129
   nuclear antibody levels in, 131
   smooth muscle antibodies in, 160
Biliary enteric bypass patency, hepatic inminodiacetic acid scan in, 266
Biliary tract
   atresia of, hepatic inminodiacetic acid scan in, 266
   dilation of, ultrasound in, 258, 266
   drainage
      percutaneous transhepatic, 270
      pH affected by, 137
   imaging test selection and interpretation in evaluation of, 267, 270
   lipase levels in disorders of, 121
   obstruction of
      alanine aminotransferase levels in, 46
      alkaline phosphatase levels in, 50
      aspartate aminotransferase levels in, 56
Biliary tract (cont.)
obstruction of (cont.)
bilirubin levels in, 58
in cholangitis/cholecystitis, test selection in, 229
cholesterol levels in, 71
computed tomography in, 259, 268
endoscopic retrograde cholangiopancreato- 
graphy in, 267
gamma-glutamyl transpeptidase 
levels in, 94
haptoglobin levels in, 99
mitochondrial antibody levels in, 129
percutaneous transhepatic cholangiogram in, 270
triglyceride levels in, 172
ultrasound in, 272

Bilirubin
direct, 58, 378t
indirect, 58, 378t
serum creatinine levels affected by, 80
serum levels of, 58, 378t
hemolysis and, 363t
and operative death rate after porto-
caval shunt (Child’s criteria), 372t
urine, 378t
dipstick testing of, 30, 31t

Bilirubinemia
in hemolysis, 383t
urine color affected by, 30

Bioavailability, and therapeutic drug moni-
toring, 189

Birds, pneumonia associated with expo-
sure to, test selection in, 212

Birth control pills. See Oral contraceptives

Birthing animals, pneumonia associated 
with exposure to, test selection in, 212

Bite cells, 29t

Bladder, x-ray of (KUB plain radiograph), 258

Bladder cancer
magnetic resonance imaging in, 273, 275
urine calcium levels in, 65

Blastomyces spp., test selection for, in
infectious esophagitis, 222

Blastomyces dermatitidis, test selection 
for, in community-acquired pneumonia, 212

Blastomycosis
Histoplasma capsulatum antigen levels 
in, 108
Histoplasma capsulatum complement 
fixation antibody test in, 109

Histoplasma capsulatum precipitin 
levels in, 109

Bleeding. See Hemorrhage

Bleeding time, 59, 377t

Blood
amniotic fluid contaminated by, and
lecithin/sphingomyelin ratio, 119
fecal occult, 89
in anemia caused by blood loss, 363t
imaging test selection and interpretation 
in evaluation of, 281
in urine, dipstick testing of, 30, 32t

Blood agar, for bacterial culture, in 
pharyngitis, 209

Blood components, 401t–402t

Blood culture
in anaerobic pneumonia or lung abscess,
213
in aspiration pneumonia, 213
in bacteremia of unknown source, 241
in bacterial meningitis, 200
in bacterial/septic arthritis, 238
in cholangitis/cholecystitis, 229
in chorioamnionitis/endometritis, 236
in community-acquired pneumonia, 212
in empyema, 216
in epiglottitis, 211
in fungal meningitis, 201
HIV-associated pneumonia, 214
in hospital-acquired pneumonia, 213
in immunocompromise-related pneu-
monia, 214
in infectious myocarditis, 218
in infectious thrombophlebitis, 221
in infective endocarditis, 219
in osteomyelitis, 237
in otitis media, 206
in pericarditis, 217
in perinephric abscess, 232
in peritonitis, 226
in prosthetic valve infective endo-
carditis, 220
in pyelonephritis, 232

Blood donors, hepatitis C antibody levels 
in, 106

Blood dyscrasias, uric acid levels in, 177

Blood loss. See also Hemorrhage
glycohemoglobin levels in, 98
imaging test selection and interpretation 
in evaluation of, 281
platelet count in, 140
reticulocyte count in, 153
Blood smear, peripheral
common findings on, 29
Wright stain of, 27–28
Blood transfusion. See Transfusion
Blood urea nitrogen
ratio of, to serum creatinine (BUN/Cr), 60, 387
serum levels of, 60, 377
Blue-top tubes, 25, 42
Body fluids. See also specific fluid
obtaining and processing, 24–25
Bone
biopsy of, in osteomyelitis, 237
broken. See Fractures
imaging test selection and interpretation
in evaluation of, 276
infections of, magnetic resonance
imaging in, 278
metastases to
alkaline phosphatase levels affected by, 50
calcium levels affected by, 65
osteoblastic, phosphorus levels in, 138
osteolytic, phosphorus levels in, 138
physiologic growth of, alkaline phosphatase levels affected by, 50
retropulsed fragments of, after trauma, computed tomography in, 277
tumor of, magnetic resonance imaging in, 278
x-ray of, in osteomyelitis, 237
Bone disease
alkaline phosphatase levels in, 50
calcium levels in
serum, 63
urine, 65
erthrocyte sedimentation rate in, 86
phosphorus levels in, 138
temporal, computed tomography in evaluation of, 245
Bone marrow
culture, in immunocompromise-related pneumonia, 214
depression/suppression
hemostatic function tests in, 377
platelet count in, 140
reticulocyte count in, 153
hyporesponsiveness, erythropoietin levels in, 86
iron in, in iron deficiency, 363–364
sampling
absence of stainable iron and, 115
in fungal meningitis, 201
sideroblasts, in sideroblastic anemia, 363
transplantation, recovery from,
CD4/CD8 ratio in, 68
Bone scan
in hypercalcemia, 347
in osteomyelitis, 237, 276
whole body, 276
Bordetella pertussis, test selection for, in laryngotracheobronchitis, 210
Borrelia burgdorferi
Lyme disease antibody test for, 122
test selection for
in bacterial/septic arthritis, 238
in infectious myocarditis, 218
in spirochetal meningitis, 202
Borrelia hermsii infection, Lyme disease
antibody test in, 122
Bowel disease, inflammatory
barium enema in evaluation of, 263
fecal occult blood in, 89
leukocyte scan in, 281
Bowel gas patterns, evaluation of, abdominal x-ray in, 258
Bowel infarct
alkaline phosphatase levels in, 50
amylase levels in, 52
Bowel ischemia, lactate levels in, 118
Bowel metastases, enteroclysis in, 262
Bowel obstruction
amylase levels in, 52
barium enema in, 263
computed tomography of, 259
enteroclysis in, 262
Hypaque enema in, 264
Bowel perforation
Hypaque enema in, 264
peritonitis associated with, test selection in, 226
upper GI study in, 262
Bowel resection, vitamin B12 levels after, 180
Bowel wall thickening, computed tomography of, 259
Brachial plexopathy, magnetic resonance
imaging in evaluation of, 248
Bradycardia
ectopic atrial, 288
QRS duration in, 285
sinus, 287
with junctional escape rhythm, 298
QRS duration in, 285
U waves in, 324
Brain, imaging test selection and interpretation in evaluation of, 246–247
Brain abscess
neighboring meningeal reaction in, 371

test selection in, 197

Brain death, confirmation of, brain scan in, 246

Brain scan, 246
for brain abscess, 197

Brain tumor, neighboring meningeal reaction in, 371

Breast, imaging test selection and interpretation in evaluation of, 256

Breast cancer
calcitonin levels in, 62
carcinoembryonic antigen levels in, 67
mammography in, 256
parathyroid hormone-related protein levels in, 135
urinary calcium levels in, 65

Breastfeeding (lactation, nursing)
1,25-dihydroxy vitamin D3 levels in, 183
prolactin levels in, 144

Bretylium, electrocardiography affected by, 326
Bronchial carcinoids, 5-hydroxyindoleacetic acid levels in, 111
Bronchial epithelial cells, on Gram-stained smear, 28i

Bronchiolitis, test selection in, 210

Bronchitis, test selection in, 210

Bronchoalveolar sampling
in anaerobic pneumonia or lung abscess, 213
in aspiration pneumonia, 213
in community-acquired pneumonia, 212
in hospital-acquired pneumonia, 213
in immunocompromise-related pneumonia, 214
in mycobacterial pneumonia, 215

Bronchoscopy
in anaerobic pneumonia or lung abscess, 213
in community-acquired pneumonia, 212
in empyema, 216
in Pneumocystis carinii pneumonia, 214

Broviac catheter
bacteremia of unknown source associated with, test selection in, 241
infectious thrombophlebitis associated with, test selection in, 221

Brucella spp.

antibody test, 60
cross-reactivity with tularemia antibody test, 175

infection (brucellosis)
Brucella antibody in, 60
heterophile agglutination
(Monospot/Paul-Bunnell) test in, 107
tularemia agglutinin levels in, 175
test selection for
in bacterial/septic arthritis, 238
in infective endocarditis, 219

Brucella canis infection, Brucella antibody in, 60

Brucellergin skin test, Brucella antibody test affected by, 60

Brugada algorithm, for diagnosis of ventricular tachycardia, 293–295

Budd-Chiari syndrome, hepatic angiography in, 271

Buerger’s disease, angiography in, 279

Bullets, retained, lead poisoning from, 119

BUN. See Blood urea nitrogen

Bundle branch block, 301–302
incomplete, 302–303
left, 302
diagnostic criteria for, 302
incomplete, 302
as mimic of myocardial infarction, 320t

morphology of, in wide QRS complex, 291–292

poor R wave progression in, 309

QRS complex in, 290–292, 295–296, 302

ST segment depression or T wave inversion in, 322t

ST segment elevation in, 321t

ST-T changes in, 302

right, 301
diagnostic criteria for, 301
incomplete, 302–303
as mimic of myocardial infarction, 320t

morphology of, in wide QRS complex, 291–292

new, in left anterior descending artery occlusion, 316
in pulmonary embolism, 356i–357i

QRS complex in, 290–292, 295–296, 301

ST segment depression or T wave inversion in, 322t

ST-T changes in, 301

Burkholderia cepacia, test selection for, in community-acquired pneumonia, 212
Burkitt’s lymphoma, Epstein-Barr virus antibody levels in, 85
Burn injury
  albumin levels in, 47
  blood urea nitrogen levels in, 60
  CD4/CD8 ratio in, 68
  cholesterol levels in, 71
  complement C3 levels in, 75
  complement C4 levels in, 75
  hematocrit in, 101
  hemoglobin levels in, 103
  inhalation, ventilation-perfusion scan in, 253
  phosphorus levels in, 138
  protein levels in, 147
  serum osmolality in, 132

C
  C-peptide, serum levels of, 62
    in factitious/surreptitious insulin use, 62, 115, 349i
    in hypoglycemia, 349i
    in insulinoma, 62, 115, 349i
  C-reactive protein, serum levels of, 60
  C. urealyticum, test selection for, in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230
  C1 esterase inhibitor (C1 INH)
    deficiency of, 61
    serum levels of, 61
  C3 (complement)
    electrophoresis in detection of, 146
    serum levels of, 75
  C4 (complement), serum levels of, 75
  Ca. See Calcium
  CA 125, serum levels of, in tuberculous peritonitis/enterocolitis, 227
  Cables, ECG
    right-left arm, reversal of, versus mirror image dextrocardia, 327
    right leg, misplacement of, 327–328
  Cachexia. See also Malnutrition; Starvation
    blood urea nitrogen in, 60
    phosphorus levels in, 138
  Caffeine, theophylline levels affected by, 194i
  Calcaneal nerve, 341i
  Calcitonin, plasma levels of, 62
  Calcium. See also Hypercalcemia; Hypocalcemia
    ionized, serum levels of, 64
    serum levels of, 63
    parathyroid hormone and, 60, 63, 134, 347i, 354i
    parathyroid hormone-related protein and, 63, 135
    vitamin D affecting, 63, 347i
    urine, 65
    ratio of, with urine creatinine, 65
  Calcium channel blockers, cyclosporine levels affected by, 191i
  Calcium/creatinine clearance ratio, in hypercalcemia, 347i
  Calcium pyrophosphate crystals, in synovial fluid, 36, 36i, 389i
  Calcium salts
calium levels affected by serum, 63
  urine, 65
  magnesium levels affected by, 123
  Calculi
    genitourinary, intravenous pyelogram in evaluation of, 273
    renal (kidney stones)
      computed tomography in, 259
      intravenous pyelogram in, 273
      urinary calcium levels in, 65
  Caliciviruses, test selection for, in infectious colitis/dysentery, 223
  California encephalitis, test selection in, 198
  Campylobacter jejuni, test selection for in HIV-associated diarrhea, 225
    in infectious colitis/dysentery, 223
  Campylobacter serotypes, Legionella antibody cross-reaction with, 120
  Cancer. See also specific type or structure or organ affected and Malignancy
    complement C3 levels in, 75
    laryngitis in, test selection in, 209
    total iron-binding capacity in, 116
  Candida
    KOH preparation in identification of, 33–35, 35i, 397i
test selection for
    in bacteremia of unknown source, 241
    in endophthalmitis, 205
    in epididymitis/orchitis, 233
    in epiglottitis, 211
    in fungal meningitis, 201
    in infectious esophagitis, 222
    in infectious thrombophlebitis, 221
    in keratitis, 205
    in laryngitis, 209
    in liver abscess, 228
    in neutropenic pneumonia, 214
    in pericarditis, 217
Candida (cont.)
test selection for (cont.)
in perinephric abscess, 232
in peritonitis, 226
in prosthetic valve infective endocarditis, 220
in urinary tract
  infection/cystitis/pyuria-dysuria syndrome, 230
in vaginitis/vaginosis, 234
  laboratory evaluation of vaginal discharge, 397
Capnocytophaga, test selection for, in infective endocarditis, 219
Capture complex, 298
Carbamazepine
gamma-glutamyl transpeptidase levels affected by, 94
sodium levels affected by, 161
therapeutic monitoring of, 191
thyroxine levels affected by, 169
urine osmolality affected by, 133
vitamin B12 levels affected by, 180
Carbenicillin, gentamicin levels affected by, 192
Carbohydrates
phosphorus levels affected by, 138
restriction of intake of, acetoacetate levels affected by, 45
Carbon dioxide, total serum levels of, 66
Carbon monoxide poisoning, carboxyhemoglobin blood levels in, 66
Carboxyhemoglobin, whole blood levels of, 66
Carboxyhemoglobinemia, red cell volume in, 151
Carcinoembryonic antigen, serum levels of, 67
Carcinoids
5-hydroxy-indoleacetic acid levels in, 111
MBG (metaiodobenzyl-guanidine) in evaluation of, 272
Carcinoma. See also specific type and Malignancy
iron levels in, 115
lactate dehydrogenase levels in, 117
Carcinomatosis, computed tomography in, 259
Carcinomatous meningitis, cerebrospinal fluid profile in, 370
Cardiac muscle injury, lactate dehydrogenase levels in, 117
Cardiac output, and creatinine clearance, 81
Cardiac rhythms. See also specific rhythms
electrocardiographic diagnosis of, 283–299, 285t
  sustained irregular, 285t
  sustained regular, 285t
Cardiac surgery
cardiomyopathy levels in, 174
ionized calcium levels affected by, 64
Cardiac trauma
cardiomyopathy levels in, 174
creatine kinase MB isoenzyme levels in, 79
Cardiac troponin-I, 174
  in myocardial infarction, 174, 353i
Cardiac valves
diagnostic evaluation of disorders of, 398t–399t
prosthetic, infective endocarditis with, test selection in, 220
Cardiac waveforms, morphological diagnosis of, electrocardiography in, 284, 299–330
Cardiobacterium, test selection for, in infective endocarditis, 219
Cardiogenic pulmonary edema, chest x-ray in, 252
Cardiolipin antibody, in Q fever, 149
Cardiomyopathies
cardiomyopathy levels in, 174
electrocardiographic findings of, 301, 303
  mimicking myocardial infarction, 320t
radionuclide ventriculography in, 257
Cardiopulmonary bypass, platelet aggregation after, 139
Cardiotoxic drugs, radionuclide ventriculography in evaluation of, 257
Cardiovascular risk, uric acid levels as marker of, 177
Carotid bifurcation atherosclerosis, magnetic resonance angiography in evaluation of, 248
Carotid bruit, carotid Doppler in, 278
Carotid Doppler ultrasound, 278
Castration, follicle-stimulating hormone levels in, 93
Castration, follicle-stimulating hormone levels in, 93
Cat scratch disease
  heterophile agglutination (Monospot/Paul-Bunnell) test in, 107
test selection in, in encephalitis, 198
Catecholamines
electrocardiography affected by, 322t
phosphorus levels affected by, 138
plasma, in pheochromocytoma, 355i
urinary metanephrines, 125
urinary vanillylmandelic acid, 178
Catheters
hepatic arterial, liver/spleen scan for
evaluation of, 271
peritonitis associated with, test selection
in, 226
urinary
pyelonephritis associated with, test selection
in, 232
urinary tract infection associated
with, test selection in, 230
venous
bacteremia of unknown source associated
with, test selection in, 241
thrombophlebitis associated with, test
selection in, 221
Cavernous hemangioma, magnetic resonance
imaging in evaluation of, 260, 269
Cavitary Coccidioides infection, Coccidioides antibody test in, 74
CBG (cortisol-binding globulin), cortisol
levels in disorders of, 76
CD4/CD8 ratio, 68
CD4 cells, 68
CD8 cells, 68
Cecal volvulus, Hypaque enema in evaluation of, 264
Celiac disease
fecal fat levels in, 88
glucose tolerance test in, 96
urinary calcium levels in, 65
vitamin B12 levels in, 180
Cellular dehydration syndromes, mean
corpuscular hemoglobin concentration in, 124
Cellulitis
predisposing factors for, 240
test selection in, 240
Central nervous system disorders
cerebrospinal fluid profiles in, 369t–371t
ceruloplasmin levels in, 69
IgG index in, 112
lead poisoning and, 119, 363t
lidocaine causing, 192t
oligoclonal bands in, 131
troponin-I levels in, 174
Centromere antibody, serum levels of, 69, 367t
Cephalosporins
creatinine levels affected by, 80
direct antiglobulin test affected by, 54
platelet count affected by, 140
Cerebellar hemangioblastomas, erythropoietin levels with, 86
Cerebral infarction, creatine kinase levels
in, 78
Cerebral lupus erythematosus, cerebrospinal fluid profile in, 370t
Cerebrospinal fluid
leaks
cisternography in evaluation of, 247
with recurrent bacterial meningitides, 200
otorrhea, cisternography in evaluation of, 247
rhinorrhea, cisternography in evaluation of, 247
sampling
in aseptic meningitis, 199, 369t
in bacterial meningitis, 200, 369t
in brain abscess, 197
in carcinomatous meningitis, 370t
in cerebral lupus erythematosus, 370t
in diabetic coma, 371t
in encephalitis, 198
in fungal meningitis, 201, 369t
in hepatic encephalopathy, 371t
in leptospirosis, 202
in neighborhood meningeal reaction, 371t
in neuroborreliosis, 202
in neurosyphilis, 202, 371t
normal values for, 369t
in otitis media, 206
in parasitic
meningoencephalitis/meningitis, 203, 370t
specimen handling for, 26t
in spirochetal meningitis, 202, 371t
in subarachnoid hemorrhage, 370t
in syphilitic meningitis, 202, 371t
in tuberculous meningitis, 203, 369t
in uremia, 371t
Cerebrospinal fluid profiles, in central nervous system disease, 369t–371t
Cerebrovascular accident, prolonged QT
interval in, 327
Ceruloplasmin
electrophoresis in detection of, 146
serum levels of, 69
Cervical cancer
- α-fetoprotein levels in, 91
- magnetic resonance imaging in, 273, 275

Cervical swab specimen, in mucopurulent cervicitis, 235

Cervicitis, mucopurulent
- laboratory evaluation of vaginal discharge in, 235, 397
- test selection in, 235

Cervicocranial arterial dissection, magnetic resonance angiography in evaluation of, 248

CF test. See Complement fixation test

CFTR (cystic fibrosis transmembrane regulator gene), 373

CH50 (complement), plasma or serum levels of, 76

Chagas’ disease, myocardiitis in, test selection in, 218

Chédiak-Higashi syndrome, leukocyte count in, 400

Chemotherapy
- electrocardiography affected by, 326
- platelet count affected by, 140
- uric acid levels affected by, 177

Chest, imaging test selection and interpretation in evaluation of, 252–253

Chest deformity, as electrocardiographic mimic of myocardial infarction, 320

Chile
- platelet count affected by, 121

CHF (congestive heart failure). See Heart failure

Children
- 1,25-dihydroxy vitamin D₃ levels in, 183
- growth hormone deficiency in, 99
- haptoglobin levels in, 99
- lead levels in, 119
- test selection in disorders in
  - bacteremia of unknown source, 241
  - bacterial meningitis, 200
- bacterial/septic arthritis, 238
- brain abscess, 197
- community-acquired pneumonia, 212
- conjunctivitis, 204
- empyema, 216
- epiglottitis, 211
- HIV-associated pneumonia, 214
- infectious colitis/dysentery, 223
- laryngotracheobronchitis, 210
- osteomyelitis, 237
- otitis media, 206
- urinary tract
  - infection/cystitis/pyuria-dysuria syndrome, 230

Child’s criteria, 372

Chlamydia spp.
- antigens, in prostatitis, 231
- test selection for, in laryngotracheobronchitis, 210

Chlamydia pneumoniae, test selection for
- in community-acquired pneumonia, 212
- in infectious myocardiitis, 218
- in infective endocarditis, 219
- in laryngotracheobronchitis, 210
- in otitis media, 206

Chlamydia psittaci, test selection for
- in community-acquired pneumonia, 212

Chlamydia trachomatis, test selection for
- in chorioamnionitis/endometritis, 236
- in community-acquired pneumonia, 212
- in conjunctivitis, 204
- in epididymitis/orchitis, 233
- in mucopurulent cervicitis, 235
- in salpingitis/pelvic inflammatory disease, 236
- in urethritis, 233
- in urinary tract
  - infection/cystitis/pyuria-dysuria syndrome, 230

Chloral hydrate, electrocardiography affected by, 326

Chloramphenicol
- and leukocyte count, 121
- leukocyte count affected by, 400

Chloride, serum levels of, 70

Chloridorrhea, congenital, carbon dioxide levels in, 66

Chloroquine, glucose-6-phosphate dehydrogenase deficiency and, 97

Chlorpropamide
- antidiuretic hormone levels affected by, 53
- sodium levels affected by, 161
- urine osmolality affected by, 133
Chlorthalidone, urinary calcium levels affected by, 65
Chocolate agar, for bacterial culture, in pharyngitis, 209
Cholangiocarcinomas, endoscopic retrograde cholangiopancreatography in, 267
Cholangiogram
magnetic resonance, in cholangitis/cholecystitis, 229
percutaneous transhepatic, 270
Cholangitis
alanine aminotransferase levels in, 46
aspartate aminotransferase levels in, 56
endoscopic retrograde cholangiopancreatography in, 267
test selection in, 229
Cholecystitis
amylase levels in, 52
hepatic iminodiacetic acid scan in, 266
test selection in, 229
Choledocholithiasis
alanine aminotransferase levels in, 46
amylase levels in, 52
aspartate aminotransferase levels in, 56
endoscopic retrograde cholangiopancreatography in, 267
Cholelithiasis (gallstones)
cholecystitis and, test selection in, 229
endoscopic retrograde cholangiopancreatography in, 267
Cholelithiasis (gallstones) ultrasound in, 258, 266
Cholera vaccination, Brucella antibody affected by, 60
Cholestasis, drugs causing alanine aminotransferase levels affected by, 46
aspartate aminotransferase levels affected by, 56
hepatic function tests in, 378t
Cholesterol, serum levels of, 71, 379t–380t in hyperlipidemia, 71, 379t–380t
Cholesteryramine triglyceride levels affected by, 172
urinary calcium levels affected by, 65
Chorioamnionitis, test selection in, 236
Choriocarcinoma, chorionic gonadotropin levels in, 72
Choriomeningitis, lymphocytic, test selection in, 198–199
Chorionic gonadotropin β-subunit, quantitative measurement of serum levels of, 72
cross-reactivity with luteinizing hormone, 122
Chorionic villus sampling, 384t
cell culture, 384t
direct cell analysis, 384t
in thalassemia syndromes, 375t–376t
Chromosomal analysis in amniocentesis, 384t
in chorionic villus sampling, 384t
Chronic ambulatory peritoneal dialysis, peritonitis associated with, test selection in, 226
Chronic bronchitis, test selection in, 210
Chronic cavitary Coccidioides infection, Coccidioides antibody test for, 74
Chronic cold agglutinin disease, cold agglutinin levels in, 74
Chronic disease
anemia of bone marrow iron stores in, 364t
erthropoietin levels in, 86
ferritin levels in, 90
free erythrocyte protoporphyrin in, 364t
hematocrit in, 101
hemoglobin levels in, 103
laboratory and clinical findings in, 363t–364t
mean corpuscular hemoglobin in, 123–124, 363t
mean corpuscular volume in, 124, 363t–364t
reticulocyte count in, 153
serum iron levels in, 115, 363t–364t
total iron-binding capacity in, 116, 363t–364t
transferrin saturation in, 364t
cholesterol levels in, 71
dexamethasone suppression test in, 83
somatomedin C levels in, 162
Chronic fatigue syndrome, 85
Chronic lymphocytic leukemia cryoglobulin levels in, 82
early antigen antibodies in, 85
leukocyte count in, 400t
vitamin B12 levels in, 180
Chronic myelogenous leukemia bcr/abl translocation in, 57
leukocyte alkaline phosphatase levels in, 120
leukocyte count in, 400t
platelet count in, 140
vitamin B12 levels in, 180
Chronic obstructive airway disease, test selection in, 210
Chronic obstructive pulmonary disease
\( \alpha_1 \)-antiprotease levels in, 55
electrocardiographic findings in, 305, 308–309, 330
as electrocardiographic mimic of myocardial infarction, 320
erythropoietin levels in, 86
partial pressure of oxygen in, 133
pH in, 137
preoperative evaluation of, ventilation-perfusion scan in, 253
Chylomicrons
in hyperchylomicronemia, 380
in mixed hypertriglyceridemia, 380
Chylous ascites, ascitic fluid profile in, 366
Chyluria, urine color affected by, 30
CIE test, in immunocompromise-related pneumonia, 214
Cimetidine
gastrin levels affected by, 95
lidocaine levels affected by, 192
prolactin levels affected by, 144
theophylline levels affected by, 194
don't lists
Circadian rhythms, 3, 76
Circulating anticoagulants. See also Lupus anticoagulant
activated clotting time affected by, 73
inhibitor screen in detection of, 114
partial thromboplastin time in detection of, 136
prothrombin time in detection of, 148
Russell’s viper venom clotting time in detection of, 157
thrombin time in screening for, 165
don't lists
Cirrhosis
alanine aminotransferase levels in, 46
ammonia levels in, 51
angiotensin-converting enzyme levels in, 52
\( \alpha_1 \)-antiprotease levels in, 55
ascitic fluid profile in, 365
aspartate aminotransferase levels in, 56
bilirubin levels in, 58, 378
cardiac troponin-I levels in, 174
CD4/CD8 ratio in, 68
ceruloplasmin levels in, 69
cholesterol levels in, 71
creatinine levels in, 80
\( \alpha \)-fetoprotein levels in, 91
gamma-glutamyl transpeptidase levels in, 94
hepatic function tests in, 378
25-hydroxy vitamin D\(_3\) levels in, 182
lactate dehydrogenase levels in, 117
lidocaine levels affected in, 192
mitochondrial antibody levels in, 129
nuclear antibody levels in, 131
peritonitis associated with, test selection in, 226
phenobarbital levels affected in, 193
phosphorus levels in, 138
plasma renin activity in, 152
pleural fluid profile in, 382
protein C levels in, 145
protein electrophoresis in, 146
renal tubular acidosis in, 388
serum osmolality in, 132
smooth muscle antibody levels in, 160
sodium levels in, 161
somatomedin C levels in, 162
testosterone levels in, 165
theophylline levels affected in, 194
triglyceride levels in, 172
valproic acid levels affected in, 194
don't lists
Cisapride, electrocardiography affected by, 326
Cisplatin, magnesium levels affected by, 123
don't lists
Cisternography, 247
don't lists
Citrate
ionized calcium levels affected by, 64
magnesium levels affected by, 123
in specimen tubes, 25, 42
CK. See Creatine kinase
CKMB. See Creatine kinase, MB isoenzyme of
Cl. See Chloride
don't lists
Class Ia drugs, electrocardiography affected by, 325, 326
don't lists
Class Ic agents, electrocardiography affected by, 326
don't lists
Class III agents, electrocardiography affected by, 326
don't lists
Cl\(_{\text{Cr}}\). See Clearance, creatinine
Clearance
creatinine, 80–81
calculation of, 81
estimation of, from serum creatinine, 81
in hypercalcemia, 347
nomogram and procedure for rapid evaluation of, 359
therapeutic drug monitoring and, 189
vitamin B\(_{12}\) absorption test
(Schilling’s test) affected by, 181
estimation of, from serum creatinine, 81, 359
nomogram and procedure for rapid evaluation of, 359
in renal failure, 81, 359
therapeutic drug monitoring and, 189 impairment affecting, 187 vitamin B₁₂ absorption test (Schilling’s test) affected by, 181 Clinical algorithms, 20 Clinical practice guidelines, 20 Clofibrate antiuretic hormone levels affected by, 53 creatine kinase levels affected by, 78 lactate dehydrogenase levels affected by, 117 triglyceride levels affected by, 172 Clonidine phosphorus levels affected by, 138 plasma renin activity affected by, 152 Clonorchis sinensis, test selection for, in cholangitis/cholecystitis, 229 Clostridium spp., test selection for in cholangitis/cholecystitis, 229 in gas gangrene, 239 in infectious thrombophlebitis, 221 Clostridium difficile enterotoxin in infection caused by, 73, 225 test selection for in antibiotic-associated pseudomembranous colitis, 224 in HIV-associated diarrhea, 225 in infectious colitis/dysentery, 223 Clostridium perfringens, test selection for in antibiotic-associated pseudomembranous colitis, 224 in cellulitis, 240 in gas gangrene, 239 Clostridium tetani, test selection for, in cellulitis, 240 Clotting cascade, factor VIII in, 88 Clotting factor deficiency. See also specific type under Factor activated clotting time in, 73 hemostatic function tests in, 377 inhibitors screen in, 114 partial thromboplastin time in, 136 prothrombin time in, 148 Russell’s viper venom clotting time in, 157 Clotting time activated, 73 reptilase, 153 Russell’s viper venom, 157 Clue cells, Gardnerella vaginalis-associated vaginos, 33, 35t, 234 CMV. See Cytomegalovirus antibody; Cytomegalovirus infection CNS. See Central nervous system Coagulation. See also Hemostasis disseminated intravascular. See Disseminated intravascular coagulation intravascular, prothrombin time in, 148 Coagulation factors. See specific types under Factor and Clotting factor deficiency Coagulation pathway extrinsic, prothrombin time in evaluation of, 148 intrinsic, partial thromboplastin time in evaluation of, 136 Cobalamin deficiency. See Vitamin B₁₂. deficiency Coccidioides antibody test, 74 in meningitis, 74, 201 Coccidioides immitis Coccidioides antibody test in infection caused by, 74, 201 test selection for in community-acquired pneumonia, 212 in fungal meningitis, 201 in HIV-associated pneumonia, 214 Coccidioidin skin test, Coccidioides antibody test affected by, 74 Coccidioidomycosis Histoplasma capsulatum antigen levels in, 108 Histoplasma capsulatum complement fixation antibody test in, 109 Histoplasma capsulatum precipitin levels in, 109 Cold agglutinin disease, chronic, cold agglutinin levels in, 74 Cold agglutinins, plasma levels of, 74 Colitis antibiotic-associated/pseudomembranous Coliostium difficile enterotoxin levels in, 73, 224 test selection in, 224 carcinoembryonic antigen levels in, 67 infectious, test selection in, 223 ulcerative arthritis associated with, synovial fluid sampling in, 389t erythrocyte sedimentation rate in, 86
Colitis (cont.)
  ulcerative (cont.)
    haptoglobin levels in, 99
    neutrophil cytoplasmic antibody levels in, 130
Collagen, platelet aggregation by, 139
Collagen-vascular disease
  antistreptolysin O titers in, 55
  cold agglutinin levels in, 74
  cryoglobulin levels in, 82
  fluorescent treponemal antibody-absorbed test in, 92
  heterophile agglutination (Monospot/Paul-Bunnell) test in, 107
  microhemagglutination-Treponema pallidum (M-TP) test in, 129
  pleural fluid profile in, 383t
  urine characteristics in, 396t
Venereal Disease Research Laboratory Test in, 178
Colon cancer
  barium enema in, 263
  carcinoembryonic antigen levels in, 67
  fecal occult blood screening for, 89
  α-fetoprotein levels in, 91
Colonic mucosa, barium enema in evaluation of, 263
Colonic obstruction, Hypaque enema in, 264
Colonic polyps, fecal occult blood in, 89
Colonoscopy, in antibiotic-associated pseudomembranous colitis, 224
Coma
  diabetic
    cerebrospinal fluid profile in, 371t
    urine characteristics in, 395t
  nonketotic hyperosmolar hyperglycemia, serum osmolality in, 132
  serum sodium levels in, 161
Combined immunodeficiency disorders, IgG levels in, 113
Common bile duct obstruction
  hepatic function tests in, 378t
  hepatic iminodiacetic acid scan in, 266
Community-acquired pneumonia, test selection in, 212
Complement
  deficiency of components of, complement CH50 test in, 76
  direct antiglobulin test for red cell coating by, 53
Complement C3
  electrophoresis in detection of, 146
  serum levels of, 75
Complement C4, serum levels of, 75
Complement CH50, plasma or serum levels of, 76
Complement fixation test
  for Coccidioides antibody, 74
  in community-acquired pneumonia, 212
  for Histoplasma capsulatum antibody, 109
  in meningitis, 201
  for immunocompromise-related pneumonia, 214
  for Q fever antibody, 149
  for rubella antibody, 156
Complement pathway, C1 esterase inhibitor in, 61
Compound S (11-deoxycortisol), in metyrapone test, 127, 338t
Computed tomography
  in abdomen evaluation, 259
  adrenal, in hypertension, 348t
  in amenorrhea, 339t
  in antibiotic-associated pseudomembranous colitis, 224
  in brain abscess, 197
  in chest evaluation, 252
  in cholangitis/cholecystitis, 229
  in Cushing’s syndrome, 340t
  in diverticulitis, 228
  in empyema, 216
  in encephalitis, 198
  in head evaluation, 245
  in infectious thrombophlebitis, 221
  in liver abscess, 228, 268
  in liver evaluation, 268
  in mycobacterial pneumonia, 215
  in neck evaluation, 249
  in osteomyelitis, 237
  in otitis externa, 207
  in pancreas evaluation, 272
  pelvic, in hirsutism, 346t
  in pericarditis, 217
  in perinephric abscess, 232
  in pheochromocytoma evaluation, 355t
  in sinusitis, 208
  in spine evaluation, 277
  spiral, in lung evaluation, 254
  in tuberculous peritonitis/enterocolitis, 227
Computed tomography angiography, spiral, in aortic evaluation, 259
Computed tomography arterial portography (CTAP), 268
Computer access, to medical information, 20
Conducting system disease, idiopathic, left
posterior fascicular block in, 303
Conduction
aberrant ventricular, 286
variable atrioventricular, QRS duration
in, 285
Conduction defects, 297–298
intraventricular, 303
Congenital heart disease
brain abscess with, 197
chest x-ray in, 252
magnetic resonance imaging in, 253
partial pressure of oxygen in, 133
red cell volume in, 151
Congestive heart failure. See Heart failure
Conjunctival Gram stain, in conjunctivitis,
204
Conjunctival scrapings/smears in conjunc-
tivitis, 204
Conjunctivitis, test selection in, 204
Connective tissue disease
autoantibodies in, 367t–368t
centromere antibody test in, 69, 367t
mixed
autoantibodies in, 367t
nuclear antibody in, 131
ribonucleoprotein antibody in, 155,
367t
SS-A/Ro antibody in, 163
Contact lens keratitis, test selection in, 205
Contrast studies, intravenous, risks of, 244
Convulsions. See Seizures
Coombs test
direct, 54
indirect, 54
COPD. See Chronic obstructive pulmonary
disease
Copper deficiency, ceruloplasmin levels
in, 69
Coproporphyrin, urinary porphobilinogen
levels in, 142
Cor pulmonale
as electrocardiographic mimic of
myocardial infarction, 320t
poor R wave progression in, 309
ST segment elevation in, 321t
Corneal scrapings, in keratitis, 205
Cornell voltage, 306
Coronary arteries
culprits, in myocardial injury or infarc-
tion, 310–316
electrocardiographic identification of
lesions within, 315–316
Coronary artery disease
electrocardiographic findings of, 301
triglyceride levels in, 172
Coronary stenosis, myocardial perfusion
scan in, 257
Coronavirus, test selection for
in laryngotracheobronchitis, 210
in pharyngitis, 209
Corticosteroids
chloride levels affected by, 70
cholesterol levels affected by, 71
erthrocyte sedimentation rate affected
by, 86
follicle-stimulating hormone levels
affected by, 93
glucose levels affected by, 95
and laryngitis, test selection in, 209
leukocyte count affected by, 121, 400t
protein levels affected by, 147
serum osmolality affected by, 132
thyroid-stimulating hormone levels
affected by, 168
triglyceride levels affected by, 172
urinary calcium levels affected by, 65
Corticosterone, in hypertension associated
with hypokalemia, 348i
Corticotropin-releasing hormone stimula-
tion test, 84
Cortisol
circadian fluctuations in levels of, 76
in Cushing’s syndrome, 76–77, 340i
plasma/serum levels of, 76
in adrenocortical insufficiency, 77,
127, 338i
in dexamethasone suppression test,
83–84, 348
in hypertension associated with
hypokalemia, 348i
in metyrapone test, 127, 338i
response to cosyntropin stimulation test,
77, 338i
urinary free, 77
Cortisol-binding globulin, cortisol levels
in disorders of, 76
Corynebacteria, test selection for, in
sinusitis, 208
Corynebacterium diphtheriae, test selec-
tion for
in infectious myocarditis, 218
in pharyngitis, 209
Corynebacterium glucuronolyticum, test
selection for, in urinary tract
infection/cystitis/pyuria-dysuria
syndrome, 230
**Index**

*Corynebacterium jeikeium*, test selection for, in bacteremia of unknown source, 241

*Corynebacterium pseudodiphtheriticum*, test selection for
- in community-acquired pneumonia, 212
- in HIV-associated pneumonia, 214
- in pharyngitis, 209

Cosyntropin stimulation test, 77, 338

Countershock, creatine kinase levels affected by, 78

*Coxiella burnetii*
- antibodies to, serum levels of, 149
- test selection for
  - in community-acquired pneumonia, 212
  - in infectious myocarditis, 218
  - in infective endocarditis, 219

*Coxsackie viruses*, test selection for
- in aseptic meningitis, 199
- in conjunctivitis, 204
- in encephalitis, 217
- in infectious myocarditis, 218
- in pericarditis, 217

Cr. See Creatinine

Craniofacial trauma, computed tomography in evaluation of, 245

Craniopharyngioma, in growth-hormone deficient patients, somatomedin C levels in, 162

Creatine kinase
- MB isoenzyme of
  - in myocardial infarction, 79, 353
  - serum levels of, 79
  - serum levels of, 78
  - in infectious myocarditis, 218
  - in myocardial infarction, 78

Creatinine
- serum levels of, 80, 377
- ratio of, to blood urea nitrogen (BUN/Cr), 60, 387
- urine, ratio of, with urine calcium, 65

Creatinine clearance, 80–81
- estimation of, from serum creatinine, 81, 359
- nomogram and procedure for rapid evaluation of, 359
- in renal failure, 81, 359
- vitamin B₁₂ absorption test (Schilling’s test) affected by, 181

Creatinine clearance/calcium ratio, in hypercalcemia, 347

Crescentic glomerulonephritis, neutrophil cytoplasmic antibody levels in, 130

CREST syndrome
- autoantibodies in, 367
- centromere antibody test in, 69, 367

Creutzfeldt-Jakob disease, encephalitis in, test selection in, 198

CRH test (corticotropin-releasing hormone stimulation test), 84

Crigler-Najjar syndrome, bilirubin levels in, 58

Crohn’s disease (regional enteritis/ileitis)
- arthritis associated with, synovial fluid sampling in, 389
- enterolysis in evaluation of, 262
- erythrocyte sedimentation rate in, 86
- fecal fat levels in, 88
- α-fetoprotein levels in, 91
- synovial fluid sampling in, 389
- vitamin B₁₂ absorption test (Schilling’s test) in, 181
- vitamin B₁₂ levels in, 180

Cross-match, 175
- ABO grouping for, 44, 54, 175
- antibody screen for, 53, 175
- indirect antiglobulin test for, 54
- Rh grouping for, 54, 154, 175

Croup, epiglottitis differentiated from, 211

Cryoglobulin, serum levels of, 82

Cryoglobulinemia, cryoglobulin levels in, 82

Cryoprecipitated antihemophilic factor, 402

Cryptococcal antigen test, 82
- in meningitis, 82, 201

Cryptococcal infection
- cryptococcal antigen test in, 82
- test selection in
  - in laryngitis, 209
  - in meningitis, 201

*Cryptococcus* spp., test selection for, in peritonitis, 226

*Cryptococcus neoformans*
- antigen, in serum or cerebrospinal fluid, 82
- test selection for
  - in brain abscess, 197
  - in fungal meningitis, 201
  - in HIV-associated pneumonia, 214

Cryptogenic cirrhosis
- mitochondrial antibody levels in, 129
- smooth muscle antibody levels in, 160

Cryptorchidism, semen analysis in, 159

Cryptosporidiosis, D-xylose absorption test in, 185
Cryptosporidium spp., test selection for
in HIV-associated cholangitis/cholecystitis, 229
in HIV-associated diarrhea, 225
in infectious colitis/dysentery, 223
in infectious esophagitis, 222
Cryptosporidium parvum, test selection
for, in sinusitis, 208

Crystals
synovial fluid examination for, 35–36, 37i, 389t
and urinary color, 30
and urinary turbidity, 30
CT. See Computed tomography
CTAP (computed tomography arterial portography), 268
cTnI. See Cardiac-troponin I
Culprit artery, in myocardial infarction electrocardiographic identification of, 310–315
electrocardiographic identification of lesions within, 315–316
Culture. See specific culture (e.g., Bacterial culture, Blood culture)
[13C]urea breath test, in gastritis, 222
Curschmann’s spirals, on Gram-stained smear, 28i
Cushing’s syndrome
ACTH levels in, 46, 340i
cholesterol levels in, 71
corticotropin-releasing hormone stimulation test in, 84
cortisol levels in serum or plasma, 76, 340i
urinary free, 77
dexamethasone suppression test in, 83–84, 340i
diagnostic algorithm for, 340i
glucose levels in, 95
glucose tolerance test in, 96
hirsutism in, 346i
hypertension associated with hypokalemia in, 348i
metyrapone test in, 127
potassium levels in, 143
Cutaneous innervation, 341i–342i
Cyanosis, methemoglobin levels in, 126
Cyclooxygenase deficiency, platelet aggregation in, 139
Cyclophosphamide
antidiuretic hormone levels affected by, 53
serum osmolality affected by, 132
urine osmolality affected by, 133

Cyclospora spp., test selection for, in infectious colitis/dysentery, 223
Cyclosporine
magnesium levels affected by, 123
therapeutic monitoring of, 191t
Cystic adventitial disease, angiography in, 279
Cystic fibrosis
amylase levels in, 52
community-acquired pneumonia in, test selection in, 212
molecular diagnostic techniques for, 373t
sinusitis in, test selection in, 208
vitamin B12 absorption test (Schilling’s test) in, 181
Cystic fibrosis transmembrane regulator gene, 373t
Cysticercosis, test selection in
in brain abscess, 197
in parasitic meningoencephalitis, 203
Cystitis, test selection in, 230
Cystoscopy, in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230
Cytomegalovirus antibody, serum levels of, 83
Cytomegalovirus infection
CD4/CD8 ratio in, 68
cold agglutinin levels in, 74
cytomegalovirus antibody levels in, 83
heterophile agglutination (Monospot/Paul-Bunnell) test in, 107
β2-microglobulin levels in, 128
test selection for
in cholangitis/cholecystitis, 229
in encephalitis, 198
in epididymitis/orchitis, 233
in HIV-associated cholangitis/cholecystitis, 229
in HIV-associated diarrhea, 225
in HIV-associated pneumonia, 214
in infectious esophagitis, 222
in laryngitis, 209
in pericarditis, 217
in prostatitis, 231
in sinusitis, 208
in transplant-related pneumonia, 214
Cytotoxic T cells (CD8 cells), 68
Cytotoxic therapy-related malabsorption,
D-xylose absorption test in, 185
D
D antigen, Rh, testing for, 154
D bilirubin (delta bilirubin), serum levels of, 58
D-dimer fibrin assay, 91, 377t
Dapsone
  glucose-6-phosphate dehydrogenase deficiency and, 97
  hemosiderin levels affected by, 104
  heterophile agglutination (Monospot/Paul-Bunnell) test affected by, 107
  methemoglobin levels affected by, 126
DAT. See Direct antiglobulin test (direct Coombs test)
DC countershock, creatine kinase levels affected by, 78
Debilitated patients, barium enema in evaluation of, 263
Decision analysis, 17–20, 19i
Decision making, threshold approach to, 16–17, 17i–18i
Decision trees, 19i, 19–20
Decubitus ulcers, cellulitis associated with, test selection for, 240
Deep peroneal nerve, 342i
Deep venous thrombosis. See Thrombosis
Deer mice, pneumonia associated with exposure to, test selection in, 212
Defibrillation
  cardiac troponin-I levels after, 174
  countershock, creatinine levels affected by, 78
Dehydration
  albumin levels in, 47
  blood urea nitrogen levels in, 60
  cellular syndromes, mean corpuscular hemoglobin concentration in, 124
  chloride levels in, 70
  creatinine clearance in, 81
  hematocrit in, 101
  hemoglobin levels in, 103
  hypernatremia secondary to, serum osmolality in, 132
  magnesium levels in, 123
  plasma renin activity in, 152
  potassium levels in, 143
  protein levels in, 147
  serum osmolality in, 132
  serum sodium levels in, 161
Degroepiandrosterone
  in hirsutism, 346i
  as testosterone precursor, 165
Delayed puberty, follicle-stimulating hormone levels in, 93
Delta agent, hepatitis D antibody in infection caused by, 107
Delta bilirubin (D bilirubin), serum levels of, 58
Delta wave, in Wolff-Parkinson-White pattern, 305
Demeclocycline, urine osmolality affected by, 133
Dementia, evaluation of, positron emission tomography in, 247
Demyelinating disease, magnetic resonance imaging in evaluation of, 245
11-Deoxycortisol, in metyrapone test, 127, 338i
Depression
cortisol levels in, 76
dexamethasone suppression test affected by, 83
Derangements, traumatic, magnetic resonance imaging in, 278
Dermatitis, atopic, CD4/CD8 ratio in, 68
Dermatome chart, 341i–342i
Dermatomyositis
  nuclear antibody levels in, 131
  rheumatoid factor levels in, 155
Desipramine, therapeutic monitoring of, 191t
Dexamethasone, angiotensin-converting enzyme levels affected by, 52
Dexamethasone suppression test in Cushing’s syndrome, 83–84, 340i
  high-dose, overnight, 84
  in hypertension with hypokalemia, 348i
  single low-dose, overnight, 83
Dextrocardia, mirror image, versus right-left arm cable reversal in ECG, 327
DFA. See Direct fluorescent antigen
DHEA. See Dehydroepiandrosterone
Diabetes insipidus
  antidiuretic hormone levels in, 53
  chloride levels in, 70
  serum osmolality in, 132
  sodium levels in, 161
  urine osmolality in, 133
Diabetes mellitus
  angiotensin-converting enzyme levels in, 52
  C-peptide levels in, 62
  CD4/CD8 ratio in, 68
  cellulitis in, test selection for, 240
cholesterol levels in, 71
complement C3 levels in, 75
creatinine clearance in, 81
ferritin levels in, 90
fructosamine levels in, 94
gastroparesis in, gastric emptying study in, 265
gestational, glucose tolerance test in, 96
glucose levels in, 95
insulin levels, serum, in, 115
laryngitis in, test selection in, 209
lecithin/sphingomyelin ratio affected in offspring of mothers with, 119
lipase levels in, 121
otitis externa in, test selection in, 207
pH in, 137
phosphorus levels in, 138
serum osmolality in, 132
sodium levels in, 161
test selection in
in antibiotic-associated pseudomembranous colitis, 224
in HIV infection, 225
in infectious colitis/dysentery, 223
Diazepam, triglyceride levels affected by, 172
DIC. See Disseminated intravascular coagulation
Dietary excess, triglyceride levels in, 172
decision analysis in, 17–20, 19i
for diagnosis, 2, 10–11
interfering factors and, 6–7
for management, 2, 10–11
odds-likelihood ratios and, 11–16, 13t, 14i–15i
patient preparation for, 3
performance of, 3–4
precision of, 4t, 4–5, 5i
reference range for, 5–6, 6t, 6i
risks of, 1–3
for screening, 1–2, 2t
sensitivity and specificity of, 7–9, 8i–10i
sequential, 16
threshold approach to decision making in, 16–17, 18i
use and interpretation of, basic principles of, 1–21
Dialysis
chronic ambulatory peritoneal, peritonitis associated with, test selection in, 226
creatine kinase MB levels affected by, 79
hepatitis C antibody levels affected by, 106
phosphorus levels affected by, 138
test selection in
in antibiotic-associated pseudomembranous colitis, 224
in HIV infection, 225
in infectious colitis/dysentery, 223
Diabetic acidosis, glucose-6-phosphate dehydrogenase deficiency and, 97
Diabetic coma
cerebrospinal fluid profile in, 371t
urine characteristics in, 395t
Diabetic ketoacidosis
acetoacetate levels in, 45
amylase levels in, 52
chloride levels in, 70
creatinine levels in, 80
lactate levels in, 118
leukocyte count in, 400t
lipase levels in, 121
magnesium levels in, 123
pH in, 137
phosphorus levels in, 138
serum osmolality in, 132
Diabetic mother, infants of
glucose levels in, 95
lecithin/sphingomyelin ratio in, 119
Diagnostic imaging, 243–281. See also specific anatomic location and type of study
Diagnostic/laboratory tests
accuracy of, 4, 4t, 5i
benefits of, 1–3
characteristics of, 4t, 4–9
costs of, 1–3
Digibind, digoxin monitoring affected by, 192r

Digitalis
ECG parameters affected by
  QT interval, 325r, 326–327
  ST segment depression or T wave inversion with, 322r–323r
  ST-T-U abnormalities, 325r
  U waves, 324, 325r
toxicity, 192r
electrocardiographic findings of, 325t

Digoxin
luteinizing hormone affected by, 122
testosterone levels affected by, 165
therapeutic monitoring of, 192r

Digoxin-specific antibody, digoxin monitoring affected by, 192r

Dihydrotachysterol, urinary calcium levels affected by, 65

1,25-Dihydroxy vitamin D₃
  serum or plasma levels of, 183, 347r
  in vitamin D deficiency, 183
  in vitamin D toxicity, 183

Dilated cardiomyopathy, electrocardiographic findings of, 303

Diphtheroids, test selection for
  in infectious thrombophlebitis, 221
  in prosthetic valve infective endocarditis, 220

Diphyllobothrium latum infestation, vitamin B₁₂ levels in, 180

Dipstick (reagent strip) testing of urine, 30
  components of, 31r–32r
  in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230

Direct antiglobulin test (direct Coombs test), 54

Direct fluorescent antigen (DFA)
in community-acquired pneumonia, 212
in immunocompromise-related pneumonia, 214
in impetigo, 239
in laryngotracheobronchitis, 210

Discoid lupus
  nuclear antibody levels in, 367t
  ribonucleoprotein antibody levels in, 155, 367t

Disopyramide, electrocardiography affected by, 325r, 326

Disseminated eosinophilic hypersensitivity disease, leukocyte count in, 400t
Disseminated gonococcal infection, bacterial/septic arthritis in, test selection in, 238

Disseminated intravascular coagulation
  antithrombin III levels in, 56
  complement C₃ levels in, 75
  factor VIII assay in, 88
  fibrin D-dimer levels in, 91, 377t
  fibrinogen assay in, 377t
  hemostatic function tests in, 377t
  partial thromboplastin time in, 136
  platelet count in, 140, 377t
  protein C levels in, 145
  protein S levels in, 147
  prothrombin time in, 148
  reptilase clotting time in, 153
  thrombin time in, 165

Distribution, volume of, and therapeutic drug monitoring, 189

Distribution phases, and therapeutic drug monitoring, 189

Diuretics
  ammonia levels affected by, 51
  calcium levels affected by
    serum, 63
    urine, 65
  carbon dioxide levels affected by, 66
  chloride levels affected by, 70
  glucose levels affected by, 95
  glucose tolerance test affected by, 96
  lithium levels affected by, 192r
  magnesium levels affected by, 123
  pH affected by, 137
  phosphorus levels affected by, 138
  potassium levels affected by, 143
  serum osmolality affected by, 132
  sodium levels affected by, 161
  triglyceride levels affected by, 172
  uric acid levels affected by, 177

Diverticulitis
  barium enema in, 263
  fecal occult blood in, 89
  test selection in, 228

DNA
  antibody to (nuclear), serum levels of, 131, 367t
  double-stranded, antibody to, serum levels of, 84, 367t

DNA probes, in tuberculous meningitis, 203

Dogs, exposure to, tularemia associated with, test selection in, 175

Dopamine
  growth hormone affected by, 99
  prolactin secretion and, 144
  thyroid-stimulating hormone levels affected by, 168
Dopamine antagonists, prolactin levels affected by, 144
Doppler echocardiography, in valvular heart disease, 398t–399t
Doppler ultrasound
carotid, 278
in epididymitis/orchitis, 233
in pyelonephritis, 232
Dot blot assay, reverse
for cystic fibrosis mutation, 373t
for factor V mutation (Leiden mutation), 87, 373t
for thalassemia syndromes, 376t
Double-stranded DNA antibody, serum levels of, 84, 367t
Drug abusers
bacteremia of unknown source in, test selection in, 241
endophthalmitis in, test selection in, 205
hepatitis C antibody levels in, 106
osteomyelitis in, test selection in, 237
rapid plasma reagin test in, 150
Venereal Disease Research Laboratory Test in, 178
Drug interactions, therapeutic monitoring and, 190
Drug monitoring, 187–190, 191t–194t
effective, information required for, 188–190
indications for, 187–188
pharmacokinetic parameters and, 189–190
reliability of analytic method for, 188–189
situations not useful in, 188
specimen collection for, 190
underlying assumptions of, 187
Drug sensitivity
hemostatic function tests in, 377t
leukocyte count in, 400t
ds-DNA antibody (double-stranded DNA antibody), serum levels of, 84, 367t
Dubin-Johnson syndrome, bilirubin levels in, 58
Duchenne’s muscular dystrophy, tall R waves in right precordial leads with, 310
Dumping syndrome, gastric emptying study in, 265
Duodenal mucosa, upper GI study in evaluation of, 262
Duodenal ulcers, *Helicobacter pylori* infection in, 100
Dural sinus thrombosis, magnetic resonance venography in, 246
Dwarfism
Laron
growth hormone levels in, 99
somatomedin C levels in, 162
pituitary
growth hormone levels in, 99
somatomedin C levels in, 162
Dysbeta-lipoproteinemia
characteristics and laboratory findings in, 380t
cholesterol levels in, 71, 380t
triglyceride levels in, 172, 380t
Dysentery
amebic, *Entamoeba histolytica* antibodies in, 50
test selection in, 223
Dysfibrinogenemia
functional fibrinogen levels in, 92
reptilase clotting time in, 153
Russell’s viper venom clotting time in, 157
thrombin time in, 165
Dysglobulinemia, microhemagglutination-*Treponema pallidum* (M-TP) test in, 129
Dysmyelinating disease, magnetic resonance imaging in evaluation of, 245
Dyspepsia, chronic, *Helicobacter pylori* antibody levels in, 100
Dysproteinemic states, renal tubular acidosis in, 388t
Dystrophy, cardiac troponin-I levels in, 174
Dysuria
in prostatitis, 231
test selection in, 230

E
*E. coli*. See *Escherichia coli*
Ear drainage, in otitis externa, 207
Ear infection. See Otitis externa; Otitis media
Early antigen antibodies, 85
EBV antibodies. See Epstein-Barr virus antibodies
ECG. See Electrocardiography
*Echinococcus granulosus*, test selection for, in cholangitis/cholecystitis, 229
Echinococcus multilocularis, test selection for, in cholangitis/cholecystitis, 229
Echinocytes, 29
Echocardiography
of left ventricular hypertrophy, 306
transesophageal
in infective endocarditis, 219
in prosthetic valve infective endocarditis, 220
in valvular heart disease, 398–399
Echoviruses, test selection for
in encephalitis, 198
in pericarditis, 217
Eclampsia, urine characteristics in, 395
Ectopic ACTH syndrome, 340
Ectopic atrial bradycardia, 288
QRS duration in, 285
Ectopic atrial rhythm, 288
QRS duration in, 285
Ectopic pregnancy
amylase levels in, 52
chorionic gonadotropin levels in, 72
ultrasound in, 275
Eczema, otitis externa in, test selection in, 207
Edema
plasma renin activity in, 152
pulmonary, chest x-ray in, 252
scrotal, in epididymitis/orchitis, 233
EDTA
ionized calcium levels affected by, 64
in specimen tubes, 42
Edwardsiella spp., test selection for, in infective colitis/dysentery, 223
Effective renal plasma flow, renal scan in evaluation of, 274
Efficacy, drug, therapeutic monitoring and, 188
Eggplant, 5-hydroxy-indoleacetic acid levels affected by, 111
Ehrlichia spp., test selection for, in bac-
teremia of unknown source, 241
Ehrlichia chaffeensis, test selection for, in parasitic meningoencephalitis, 203
Eikenella spp., test selection for, in infective endocarditis, 219
Ejaculate, analysis of, 159, 352
Ejection fraction, 257
Elderly. See also Aging
lidocaine toxicity in, 192
nuclear antibody levels in, 131
rheumatoid factor in, 368

Electrocardiography (ECG), 283–330
atrial abnormalities in, 300–301
atrioventricular block in, 297
atrioventricular dissociation in, 298
bundle branch block in, 290–292, 295–296, 301–302
diagnosis of cardiac rhythm in, 283–299, 285

Edwardsiella spp., test selection for, in infective colitis/dysentery, 223

Effective renal plasma flow, renal scan in evaluation of, 274

Electrocardiography (ECG), 283–330
atrial abnormalities in, 300–301
atrioventricular block in, 297
atrioventricular dissociation in, 298
bundle branch block in, 290–292, 295–296, 301–302
diagnosis of cardiac rhythm in, 283–299, 285

early repolarization normal variant versus ST-T abnormality in, 328
fascicular blocks in, 303
hypothermia and, 327–328
incomplete bundle branch block in, 302–303
mean QRS axis in, determination of, 304–306
miscellaneous abnormalities in, 327–330
morphological diagnosis of cardiac waveforms in, 284, 299–330
myocardial injury, ischemia and infarction findings in, 310–320
normal, QRST patterns in, 299–300
paroxysmal supraventricular tachycardia in, 298–299
QRS complex, 284–285. See also QRS complex
low voltage of, 308
wide, with regular rhythm (WCT-RR) in, 290–296
QT interval in, 324–326
R wave progression in precordial leads in, 309
right-left arm cable reversal versus mirror image dextrocardia in, 327
right leg cable misplacement in, 327–328
ST segment in, 320, 321–323
stepwise interpretation of, 283–284
torsade de pointes in, 296, 326
U waves in, 324
in valvular heart disease, 398–399
ventricular hypertrophy in, 306–308
ventricular tachycardia diagnosis in, 290–296
Wolff-Parkinson-White patterns in, 305–306, 310, 320, 329–330

Electrophoresis
hemoglobin, 102
in thalassemia syndromes, 102, 392

Index
oligoclonal bands detected in, 131
protein, 112, 146
ELISA. See Enzyme-linked immuno-
sorbent assay
Elliptocytes, 29i
Emboli, pulmonary. See Pulmonary emboli
Emphysema
panacinar, α1-antitrypsin levels in, 55
subcutaneous, and low-voltage QRS complex in ECG, 308
Empty sella syndrome, 339i
renal tubular acidosis in, 388t
Empyema
computed tomography in, 252
pleural fluid profile in, 216, 383t
test selection in, 216
Endocarditis, test selection in, 198
Encephalopathy, hepatic
ammonia levels in, 51
cerebrospinal fluid profile in, 371t
glutamine levels in, 97
and operative death rate after portocaval shunt (Child’s criteria), 372t
Endarterectomy, monitoring after, carotid Doppler in, 278
Endocervical sampling
in mucopurulent cervicitis, 235
in salpingitis/pelvic inflammatory disease, 236
Endocrine disorders, cardiac troponin-I levels in, 174
Endometrial cancer
α-fetoprotein levels in, 91
magnetic resonance imaging in, 275
Endometriosis, smooth muscle antibody levels in, 160
Endometritis, test selection in, 236
Endomyocardial biopsy, in infectious myocarditis, 218
Endophthalmitis, test selection in, 205
Endoscopic retrograde cholangiopancre-
atography (ERCP), 267
Endoscopy
in bacterial tracheitis, 210
in infectious esophagitis, 222
in tuberculous peritonitis/enterocolitis, 227
Endothelial damage/disorders, von Wille-
brand’s factor protein levels in, 184
Endotracheal sampling, in aspiration pneu-
monia, 213
Enema
barium. See Barium enema
Hypaque, 264
phosphorus levels affected by, 138
Entamoeba histolytica
antibodies, serologic tests for, 50
test selection for
in brain abscess, 197
in HIV-associated diarrhea, 225
in infectious colitis/dysentery, 223
in liver abscess, 228
in parasitic meningoencephalitis, 203
Entero fistula, magnesium levels affected by, 123
Enteritis, regional. See Crohn’s disease
Enterobacteriaceae, test selection for
in aspiration pneumonia, 212
in bacteremia of unknown source, 241
in neutropenic pneumonia, 214
Enterobacter spp.
gas formation caused by, gas gangrene differentiated from, 239
test selection for
in bacteremia of unknown source, 241
in bacteremia of unknown source, 241
in bacterial meningitis, 200
in bacterial/septic arthritis, 238
in brain abscess, 197
in cellulitis, 240
in cholangitis/cholecystitis, 229
in community-acquired pneumonia, 212
in diverticulitis, 228
in empyema, 216
in epididymitis/orchitis, 233
in HIV-associated pneumonia, 214
in hospital-acquired pneumonia, 213
in infectious thrombophlebitis, 221
in keratitis, 205
in laryngotracheobronchitis, 210
in liver abscess, 228
in necrotizing fasciitis, 240
in osteomyelitis, 237
in otitis externa, 207
Enterobacteriaceae, test selection for (cont.)
in otitis media, 206
in pericarditis, 217
in perinephric abscess, 232
in peritonitis, 226
in prostatitis, 231
in prosthetic valve infective endocarditis, 220
in pyelonephritis, 232
in salpingitis/pelvic inflammatory disease, 236
in sinusitis, 208
in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230

Enteroclysis, 262
*Enterococcus* spp.
test selection for
in bacterial/septic arthritis, 238
in cellulitis, 240
in cholangitis/cholecystitis, 229
in chorioamnionitis/endometritis, 236
in diverticulitis, 228
in infective endocarditis, 219
in liver abscess, 228
in peritonitis, 226
in prostatitis, 231
in prosthetic valve infective endocarditis, 220
in pyelonephritis, 232
in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230
urinalysis in identification of infection caused by, 30

Enterocolitis
necrotizing, test selection in, 223
tuberculous, test selection in, 227
*Enterocytozoon bieneusi*, test selection for
in cholangitis/cholecystitis, 229
in HIV-associated diarrhea, 225
Enterohemorrhagic *E. coli*, test selection for
in infectious colitis/dysentery, 223
Enteroinvasive *E. coli*, test selection for,
in infectious colitis/dysentery, 223

Enteropathies
albumin levels in, 47
HIV, D-xylose absorption test in, 185
protein-losing complement C4 levels in, 75
protein levels affected by, 147

Enteropathogenic *E. coli*, test selection for,
in infectious colitis/dysentery, 223
Enterotoxigenic *E. coli*, test selection for,
in infectious colitis/dysentery, 223
Enterotoxin, in *Clostridium difficile* infection, 73
Enteroviruses, test selection for
in aseptic meningitis, 199
in conjunctivitis, 204
in encephalitis, 198
in infectious myocarditis, 218
in pericarditis, 217
Enterocyte, 29
Eosinophil, 29
Eosinophil count, 400

Epicardial injury, 310
*Epidermophyton* spp., KOH preparation in identification of, 33–35

Epididymitis
test selection in, 233
tuberculous, test selection in, 233
Epiglottitis, test selection in, 211
Epinephrine
platelet aggregation by, 139
protein levels affected by, 147

Epithelial tumors, carcinoembryonic antigen levels with, 67

EPO. See Erythropoietin

Epstein-Barr virus
infection
Epstein-Barr antibodies in, 85
heterophile agglutination (Monospot/Paul-Bunnell) test in, 107
test selection for
in laryngitis, 209
in pericarditis, 217
in pharyngitis, 209
Epstein-Barr virus antibodies, serum levels of, 85
ERCP (endoscopic retrograde cholangiopancreatography), 267
Ergocalciferol, phosphorus levels affected by, 138
ERPF (effective renal plasma flow), renal scan in evaluation of, 274
ERV (expiratory reserve volume), 385
Erysipelas, antistreptolysin O titer in, 55
Erysipelothrix rhusiopathiae, test selection for, in infective endocarditis, 219
Erythrocyte(s) (red blood cells)
antibody screen in detection of antibody to antigen of, 53
casts of, in urine, 32, 387
direct antiglobulin test for immunoglobulin or complement coating of, 54
folic acid levels in, 93
free protoporphyrin in, 94
in anemias, 94, 363r–364t
in iron deficiency, 94, 364t
in lead poisoning, 94, 363t
on Gram-stain smear, 28t
hemoglobin in, mean corpuscular, 123–124, 363t
hemosiderin levels affected by mechanical destruction of, 104
lactate dehydrogenase levels affected by hemolysis of, 117
for transfusion, 401t
adenine-saline added, 401t
leukocyte reduced, 401t
urinary color affected by, 30
urinary turbidity affected by, 30
urinary turbidity caused by, 30
urine, 395t–396t
volume of, 151
mean corpuscular, 124, 363t
on Wright-stained peripheral blood smear, 29t
young, glucose-6-phosphate dehydrogenase levels in, 97
Erythrocyte count (red blood cell count), 85
ascitic fluid, 365t–366t
in tuberculous peritonitis/enterocolitis, 227
cerebrospinal fluid, 369t–371t
in encephalitis, 198
in meningeal reaction, 371t
in subarachnoid hemorrhage, 370t
in mean corpuscular hemoglobin calculation, 123
pleural fluid, 382t–383t
in cirrhosis, 382t
in collagen vascular disease, 383t
in empyema, 383t
in esophageal rupture, 383t
in heart failure, 382t
in malignancy, 382t
in nephrotic syndrome, 382t
in pancreatitis, 383t
in parapneumonic effusion, 383t
in pulmonary embolism/infarction, 383t
in rheumatoid arthritis, 383t
in tuberculosis, 382t
Erythrocyte scan, labeled, in gastrointestinal bleeding, 265
Erythrocyte sedimentation rate, 86
Erythromycin
cyclosporine levels affected by, 191t
electrocardiography affected by, 326
Erythropoietic protoporphyria, free erythrocyte protoporphyrin levels in, 94
Erythropoietin
serum levels of, 86
tumors producing, erythropoietin levels in, 86
Escape complex, definition of, 288–289
Escape rhythms
definition of, 288–289
junctional, 288–289, 289t
ventricular, 289t
QRS duration in, 285t
Escherichia coli
gas formation caused by, gas gangrene differentiated from, 239
test selection for
in bacteremia of unknown source, 241
in bacterial meningitis, 200
in chorioamnionitis/endometritis, 236
in community-acquired pneumonia, 212
in empyema, 216
in epididymitis/orchitis, 233
in infectious colitis/dysentery, 223
in pyelonephritis, 232
in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230
urinalysis in identification of infection caused by, 30
Esophageal dysmotility, centromere antibody levels in, 69
Esophageal reflux study, 264
Esophageal varices
  fecal occult blood in, 89
  upper GI study in evaluation of, 262
Esophagitis, infectious
  predisposing factors for, 222
  test selection in, 222
Esophagram, barium, in infectious esophagitis, 222
Esophagus
  cancer of
    fecal occult blood in, 89
    upper GI study in evaluation of, 262
  rupture of, pleural fluid in, 383
    upper GI study in evaluation of, 262
ESR. See Erythrocyte sedimentation rate
Essential mixed cryoglobulinemia, cryoglobulin levels in, 82
Essential thrombocythemia
  lactate dehydrogenase isoenzyme levels in, 117
  platelet count in, 140
Esterase, urine, 32t, 395t–396t
Estrogens
  albumin levels affected by, 47
  cortisol levels affected by, 76
  glucose levels affected by, 95
  hyperlipidemia aggravated by, 380t
  iron levels affected by, 115
  phosphorus levels affected by, 138
  plasma renin activity affected by, 152
  prolactin levels affected by, 144
  testosterone levels affected by, 165
  thyroid function tests affected by, 394t
  triglyceride levels affected by, 172
  urinary calcium levels affected by, 65
Ethacrynic acid, magnesium levels affected by, 123
Ethanol
  antidiuretic hormone levels affected by, 53
  gamma-glutamyl transpeptidase levels affected by, 94
  glucose levels affected by, 95
  hyperlipidemia aggravated by, 380t
  intoxication
    definition of, 87
    osmolal gap in, 132, 381t
    serum osmolality in, 87, 132
  iron levels affected by, 115
  lactate levels in, 118
  lethal concentrations of, 381t
  leukocyte count affected by, 400t
  pH affected by, 137
platelet count affected by, 140
serum levels of, 87
serum osmolality affected by, 87, 132
testosterone levels affected by, 165
triglyceride levels affected by, 172
uric acid levels affected by, 177
Ethosuximide, therapeutic monitoring of, 192t
Ethyl ether, serum osmolality affected by, 132
Ethylene glycol
  osmolal gap in lethal concentrations of, 381t
  pH affected by, 137
  serum osmolality affected by, 132
Evidence-based medicine, 20
Exercise
  cardiac troponin-I levels affected by, 174
  CD4/CD8 ratio in, 68
  creatinine clearance in, 81
  hematocrit in, 101
  hemoglobin levels in, 103
  lactate levels in, 118
  potassium levels in, 143
  prolactin levels in, 144
  uric acid levels affected by, 177
Expiratory flow-volume curve, 358i
Expiratory reserve volume (ERV), 385t
Extracellular fluid volume
  assessment of, in hyponatremia, 350i
  expansion of, and chloride levels, 70
Extremity grafts, ultrasound in evaluation of, 278
Extrinsic coagulation pathway, prothrombin time in evaluation of, 148
Exudates
  ascitic fluid, characteristics of, 365t–366t
  pleural fluid, characteristics of, 382t–383t

F
Factor I deficiency
  in aseptic meningitis, 199
  hemostatic function tests in, 377t
  liver function tests in, 377t
Factor II deficiency
  hemostatic function tests in, 377t
  liver function tests in, 377t
  prothrombin time in, 148
Factor V deficiency
  hemostatic function tests in, 377t
  liver function tests in, 377t
  prothrombin time in, 148
Russell’s viper venom clotting time in, 157
Factor V mutation (Leiden mutation), 87
molecular diagnostic techniques for, 373
Factor VII deficiency
activated clotting time in, 73
hemostatic function tests in, 377t
prothrombin time in, 148
Factor VII inhibitor, hemostatic function
tests affected by, 377t
Factor VIII antibodies, acquired, factor
VIII assay in, 88, 377t
Factor VIII assay, 88, 377t
Factor VIII disorders. See also Hemophilia
activated clotting time in, 73
bleeding time in, 59
cryoprecipitated anti-hemophilic factor
transfusion for, 402t
factor VIII assay in, 88, 377t
hemostatic function in, 377t
Factor VIII inhibitor
hemostatic function tests affected by,
377t
inhibitor screen in detection of, 114
Factor IX deficiency
hemostatic function tests in, 377t
prothrombin time in, 148
Factor X deficiency
hemostatic function tests in, 377t
liver function tests in, 377t
prothrombin time in, 148
Russell’s viper venom clotting time in,
157
Factor XI deficiency, hemostatic function
tests in, 377t
Factor XII, hemostatic function tests in,
377t
Factor XIII assay, in factor XIII de-
ciency, 377t
Factor XIII deficiency
cryoprecipitated anti-hemophilic factor
for, 402t
factor XIII assay in, 377t
hemostatic function tests in, 377t
urea stabilizing test in, 377t
Factor-specific antibodies, inhibitor screen
detection of, 114
Familial abetalipoproteinemia, cholesterol
levels in, 71
Familial combined hyperlipidemia
characteristics and laboratory findings
in, 379t
cholesterol levels in, 71, 379t
triglyceride levels in, 172, 379t
Familial dysbetalipoproteinemia
cholesterol levels in, 71
triglyceride levels in, 172
Familial hypercholesterolemia
characteristics and laboratory findings
in, 379t
cholesterol levels in, 71, 379t
Familial hypertriglyceridemia, characteristics
and laboratory findings in, 379t
Familial hypocalciuria, calcium levels in,
63, 347t
Familial lipoprotein lipase deficiency,
triglyceride levels in, 172
Familial mental retardation-1 gene, mutation
in, fragile X syndrome
caused by, 374t
Familial periodic paralysis, potassium
levels in, 143
Fanconi’s syndrome
carbon dioxide levels in, 66
pH in, 137
renal tubular acidosis in, 388t
uric acid levels in, 177
urine calcium levels in, 65
Fascicular blocks (hemiblocks), 303
left anterior, 305
diagnostic criteria for, 303
as mimic of myocardial infarction,
320t
new, in left anterior descending
artery occlusion, 316
poor R wave progression in, 309
left posterior, 306
diagnostic criteria for, 303
Fasciola hepatica, test selection for, in
cholangitis/cholecystitis, 229
Fasting
acetoacetate levels affected by, 45
bilirubin levels in, 58
somatomedin C levels in, 162
Fasting hypoglycemia. See also Hypo-
glycemia
diagnostic algorithm for, 349i
Fat, fecal, 88
Fatigue, chronic, 85
Fatty infiltration, liver, magnetic reso-
nance imaging in, 269
Fatty liver
magnetic resonance imaging in, 269
triglyceride levels in, 172
Fava beans, glucose-6-phosphate dehydro-
genase deficiency and, 97
Fecal contamination, urine color affected by, 30
Fecal fat, 88
Fecal leukocyte
in antibiotic-associated pseudomembranous colitis, 224
in HIV-associated diarrhea, 225
Fecal occult blood
in anemia caused by blood loss, 363\textit{t}
screening for, 89
FEF 25\%–75\% (forced expiratory flow rate from 25\% to 75\% of forced vital capacity), 385\textit{t}
Felty’s syndrome, nuclear antibody levels in, 131
Feminization, testicular, testosterone levels in, 165
Ferritin, serum levels of, 90
in anemias, 90, 364\textit{t}
Fetal hemoglobin (hemoglobin F)
blood levels of, 103
glycohemoglobin levels affected by, 98
hereditary persistence of, 103
in thalassemia, 103, 392\textit{t}
Fetal lung maturity, lecithin/sphingomyelin ratio in estimation of, 119
Fetal methemoglobin, 126
Fetal neural tube defects, \(\alpha\)-fetoprotein screening for, 91
\(\alpha\)-Fetoprotein
in amniotic fluid, 91, 384\textit{t}
serum levels of, 91
FEV\textsubscript{1} (forced expiratory volume in one second), 385\textit{t}
FEV\textsubscript{1}/FVC (forced expiratory volume in one second/forced vital capacity ratio), 385\textit{t}
Fever
in bacteremia of unknown source, test selection in, 241
pH in, 137
serum osmolality in, 132
urine characteristics in, 395\textit{t}
Fever of unknown origin, leukocyte scan in evaluation of, 281
Fibrillation, atrial, 288
QRS duration in, 285\textit{t}
Fibrin D-dimers, plasma levels of, 91, 377\textit{t}
Fibrinogen
deficiency
activated clotting time in, 73
fibrinogen levels in, 92
reptilase clotting time in, 153
Russell’s viper venom clotting time in, 157
thrombin time in, 165
and erythrocyte sedimentation rate, 86
functional, plasma levels of, 92, 377\textit{t}
Fibrinolytic therapy, thrombin time for monitoring of, 165
Fibromuscular disease, angiography in, 279
Fibrosarcoma, glucose levels in, 95
Fine-needle aspiration
of abdominal lesions, ultrasound-guided, 258
of neck lesions
computed tomography-guided, 249
ultrasound-guided, 249
of thyroid nodule, 361\textit{t}
transthoracic
in community-acquired pneumonia, 212
in mycobacterial pneumonia, 215
First-degree atrioventricular block, 297
new, in left anterior descending artery occlusion, 316
First-pass effect, of drugs, 189
Fish tapeworm infestation, vitamin B\textsubscript{12} levels in, 180
Fistulas
albumin levels in, 47
aortoenteric, computed tomography in, 259
barium enema in, 263
enteric, magnesium levels affected by, 123
pH affected by, 137
vitamin B\textsubscript{12} levels in, 180
Flocculation test, rapid plasma reagin, for syphilis, 150, 391\textit{t}
Flow cytometry
for platelet count, 140
for reticulocyte count, 153
Fluorescein-conjugated polyvalent anti-human immunoglobulin, for nuclear antibody test, 131
Fluorescent antibody test
in community-acquired pneumonia, 212
in immunocompromise-related pneumonia, 214
in impetigo, 239
in laryngotracheobronchitis, 210
Fluorescent treponemal antibody-absorbed test (FTA-ABS), 92, 391\textit{t}
in spirochetal meningitis/neurosyphilis, 202
Fluoride
and lactate dehydrogenase levels, 117
in specimen tubes, 25
Fluoroscopy
enteroclysis, 262
Hypaque enema, 264
intravenous pyelogram, 273
peroral pneumocolon, 263
videofluoroscopy, 213
Fluorouracil, and ammonia levels, 51
FMRI mutation, fragile X syndrome
caused by, 374
Focal atrial arrhythmia, 287
Folate. See Folic acid
Folic acid (folate)
deficiency
hematocrit in, 101
hemoglobin levels in, 103
laboratory and clinical findings in,
363
lactate dehydrogenase levels in, 117
leukocyte count in, 121, 400
mean corpuscular volume in, 124
red cell folic acid levels in, 93, 363
t reticulocyte count in, 153
red cell levels of, 93
in folic acid deficiency, 93, 363
Follicle-stimulating hormone
in infertility evaluation, 93, 352
serum levels of, 93
in amenorrhea, 93, 339
in hirsutism, 346
Follicular cancer, thyroglobulin levels in,
166
Follicular development, ultrasound in
evaluation of, 275
Forced expiratory flow rate from 25% to
75% of forced vital capacity
(FEF 25%–75%), 385
Forced expiratory volume in one second
(FEV₁), 385
Forced expiratory volume in one second/forced vital capacity
ratio (FEV₁/FVC), 385
Forced vital capacity (FVC), 385
Foregut carcinoids, 5-hydroxy-
indoleacetic acid levels in, 111
Foreign body, airway obstruction caused
by, partial pressure of oxygen in,
133
Forensic testing, HLA typing in, 110
Fractures
healing, phosphorus levels with, 138
radiographically occult, bone scan in,
276
synovial fluid sampling in, 390
Fragile X syndrome, molecular diagnostic
techniques for, 374
Francisella tularensis
agglutinating antibodies to, 175
infection
Brucella antibody in, 60
tularemia agglutinin levels in, 175
Legionella antibody cross-reaction with,
120
test selection for, in community-
acquired pneumonia, 212
Franklin’s disease, immuno-electrophoresis
in, 112
FRC (functional residual capacity), 385
Free erythrocyte protoporphyrin, 94
in anemias, 94, 363–364
in iron deficiency, 94, 364
in lead poisoning, 94, 363
Free thyroxine, serum levels of, 169–170
Free thyroxine index, 169–170
in hyperthyroidism, 169–170
in hypothyroidism, 169–170, 351
Fresh-frozen plasma, 401
Fructosamine, serum levels of, 94
Fructose, semen, in infertility, 352
Fructose 1,6-diphosphatase deficiency,
lactate levels in, 118
FSH. See Follicle-stimulating hormone
FTA-ABS. See Fluorescent treponemal
antibody-absorbed test
Functional residual capacity (FRC), 385
Fungal keratitis, test selection in, 205
Fungal meningitis
cerebrospinal fluid profile in, 201, 369
test selection in, 201
Fungi
on Gram-stained smear, 27
test selection for
in brain abscess, 197
in community-acquired pneumonia, 212
in endophthalmitis, 205
in hospital-acquired pneumonia, 213
in immunocompromise-related pneu-
monia, 214
in keratitis, 205
in laryngitis, 209
in otitis externa, 207
in pericarditis, 217
in sinusitis, 208
Furosemide  
carbon dioxide levels affected by, 66  
free thyroxine levels affected by, 170  
parathyroid hormone levels affected by, 134  
phosphorus levels affected by, 138  
urinary calcium levels affected by, 65  
Furuncle, external canal, test selection in, 207  
Fusarium spp., test selection for, in keratitis, 205  
Fusion complex, 298  
Fusobacterium spp., test selection for  
in brain abscess, 197  
in empyema, 216  
in sinusitis, 208  
FVC (forced vital capacity), 385t  

G  
G cell hyperplasia, gastrin levels in, 95  
G6PD screen. See Glucose-6-phosphate dehydrogenase screen  
Galactorrhea, prolactin levels in, 144  
Galactosemia, glucose levels in, 95  
Gallbladder  
cancer of, computed tomography in, 268  
imaging test selection and interpretation in evaluation of, 266  
wall thickness, ultrasound in evaluation of, 258, 266  
Gallium scanning  
in infectious myocarditis, 218  
in osteomyelitis, 237  
Gallstones. See Cholelithiasis  
Gamma-glutamyl transpeptidase levels and, 50  
Gamma-serum protein, 146  
Gammopathies  
immunoelectrophoresis in, 112  
monoclonal  
protein electrophoresis in, 146  
protein levels in, 147  
of undetermined significance, immunoelectrophoresis in, 112  
polyclonal  
protein electrophoresis in, 146  
protein levels in, 147  
Ganglioneuroma  
metanephrine levels with, 125  
vanillylmandelic acid levels with, 178  
Gangrene  
gas, test selection in, 239  
hemolytic streptococcal, test selection in, 240  
Gardnerella vaginalis  
chorioamnionitis/endometritis caused by, test selection for, 236  
salpingitis/pelvic inflammatory disease caused by, test selection in, 236  
vaginosis caused by  
laboratory evaluation of vaginal discharge in, 234, 397t  
test selection in, 234  
vaginal fluid KOH preparation in, 33, 234, 397t  
vaginal fluid wet preparation in, 33, 35t, 234  
Gas gangrene, test selection in, 239  
Gasoline refinery workers, lead poisoning in, 119  
Gastrectomy  
glucose tolerance test in, 96  
vitamin B12 absorption test (Schilling’s test) after, 181  
vitamin B12 levels after, 180  
Gastric cancer. See Stomach cancer  
Gastric emptying study, 265  
Gastric mucosa, evaluation of biopsy, in gastritis, 222  
upper GI study in, 262  
Gastric outlet obstruction, evaluation of  
gastric emptying study in, 265  
upper GI study in, 262  
Gastric perforation, lipase levels in, 121  
Gastric washings, in mycobacterial pneumonia, 215  
Gastrin, serum levels of, 95  
Gastrinoma (Zollinger-Ellison syndrome)  
calcitonin levels in, 62  
gastrin levels in, 95  
Gastritis  
fecal occult blood in, 89  
gastrin levels in, 95  
_Helicobacter pylori_ antibody levels in, 100  
test selection in, 222  
Gastroenteropathies, complement C3 levels in, 75  
Gastroenterostomy, glucose tolerance test affected by, 96  
Gastroesophageal reflux, upper GI study in evaluation of, 262  
Gastrograin, for upper GI study, 262  
Gastrointestinal bleeding  
angiography in, 279  
blood urea nitrogen levels in, 60
enteroclysis in, 262
fecal occult blood in, 89
GI bleeding scan in, 265
mesenteric angiography in, 261
Gastrointestinal disease. See also under Bowel
alkaline phosphatase levels in, 50
carcinoembryonic antigen levels in, 67
enteroclysis in evaluation of, 262
erthrocyte sedimentation rate in, 86
fecal occult blood in, 89
glucose tolerance test in, 96
lipase levels in, 121
Gastrointestinal obstruction, computed tomography in, 259
Gastrointestinal perforation, upper GI study in evaluation of, 262
Gastrointestinal suction, and chloride levels, 70
Gastrointestinal tract. See also specific structure or organ
aspiration of contents of. See Aspiration cancer of
barium enema in evaluation of, 263
computed tomography in staging of, 259
imaging test selection and interpretation in evaluation of, 262–265
infection of, IgA levels in, 113
upper, imaging of, 262
Gastroparesis, gastric emptying study in, 265
Gaucher’s disease
angiotensin-converting enzyme levels in, 52
cholesterol levels in, 71
Gemfibrozil, triglyceride levels affected by, 172
Genetic diseases, molecular diagnosis of, 373t–376t
Genitourinary culture, in bacterial/septic arthritis, 238
Genitourinary tract
congenital anomalies of, magnetic resonance imaging in, 275
imaging test selection and interpretation in evaluation of, 273–274
Gentamicin
blood urea nitrogen levels affected by, 60
therapeutic monitoring of, 187, 192t
Germ cell tumors
chorionic gonadotropin levels with, 72
α-fetoprotein levels in, 91
German measles, rubella antibody titer in, 156
Germinal compartment failure, isolated, infertility caused by, 352t
Gershorn catheter, infectious thrombophlebitis associated with, test selection in, 221
Gestational diabetes, glucose tolerance test in, 96
GFR. See Glomerular filtration rate
GGT. See Gamma-glutamyl transpeptidase
GI bleeding scan, 265
Giardia intestinalis, test selection for, in HIV-associated diarrhea, 225
Giardia lamblia, test selection for, in infectious colitis/dysentery, 223
Giardiasis, vitamin B12 absorption test (Schilling’s test) in, 181
Giemsa stain
of corneal scrapings/smear, in keratitis, 205
of Pneumocystis carinii, 28t
of sputum or bronchiolar samples, in immunocompromise-related pneumonia, 214
Gilbert’s syndrome
bilirubin levels in, 58
hepatic function tests in, 378t
Glanzmann’s thrombasthenia, platelet aggregation in, 139
α-Globin gene, in α-thalassemia, 375t, 392t
β-Globin gene, in β-thalassemia, 376t, 392t
Globulins
electrophoresis in detection of, 146
serum levels of, calculation of from total protein, 147
Glomerular filtration rate
creatinine clearance as measure of, 80–81
renal scan in estimation of, 274
in renal tubular acidosis, 388t
Glomerulonephritis
albumin levels in, 47
complement C3 levels in, 75
complement C4 levels in, 75
double-stranded DNA antibody levels in, 84, 367t
magnesium levels in, 123
neutrophil cytoplasmic antibody in, 368t
poststreptococcal, antistreptolysin O titer in, 55
proliferative, complement C4 levels in, 75
Glomerulonephritis (cont.)

- urinary calcium levels in, 65
- urine characteristics in, 395
- urine indices in, 387

Glossitis, in vitamin B12 deficiency, 363

Gloves, handling and disposing of, 24

Glucocorticoids

- glucose tolerance test affected by, 96
- serum insulin levels affected by, 115
- thyroid function tests affected by, 394

Glucose

- amniotic fluid, in chorioamnionitis/endometritis, 236
- ascitic fluid, 365t–366t
- cerebrospinal fluid, 369t–371t
  - in aseptic meningitis, 199, 369t
  - in bacterial meningitis, 200, 369t
  - in carcinomatous meningitis, 370t
  - in cerebral lupus erythematosus, 370t
  - in diabetic coma, 371t
  - in encephalitis, 198
  - in fungal meningitis, 201, 369t
  - in hepatic encephalopathy, 371t
  - in leptocephalitis, 202
  - in neighborhood meningeal reaction, 371t
  - in neuroborreliosis, 202
  - in neurosyphilis, 202, 371t
  - in parasitic meningoencephalitis/meningitis, 203, 370t
  - in spirochetal meningitis, 202, 371t
  - in subarachnoid hemorrhage, 370t
  - in syphilitic meningitis, 202, 371t
  - in tuberculous meningitis, 203, 369t
  - in uremia, 371t

- excess intake of, glucose tolerance test in, 96
- peritoneal fluid, in peritonitis, 226
- plasma/serum levels of, 95
  - in hypoglycemia, 349t
  - in hyponatremia, 350t
- pleural fluid, 382t–383t
  - in cirrhosis, 382t
  - in collagen vascular disease, 383t
  - in empyema, 216, 383t
  - in esophageal rupture, 383t
  - in heart failure, 382t
  - in malignancy, 382t
  - in nephrotic syndrome, 382t
  - in pancreatitis, 383t
  - in parapneumonic effusion, 383t
  - in pulmonary embolism/infarction, 383t
  - in rheumatoid arthritis, 383t
  - in tuberculosis, 382t
  - synovial fluid, 389t–390t
  - urine, dipstick testing of, 30, 31t
- Glucose-6-phosphate dehydrogenase
deficiency, 97
- oxidant drugs and, 97
- hemosiderin levels affected by, 104
- Glucose-6-phosphate dehydrogenase screen, 97
- Glucose infusion, phosphorus levels affected by, 138
- Glucose tolerance
  - in hyperlipidemia, 380t
  - impaired, glucose tolerance test in, 96
- Glucose tolerance test, 96
- Glutamine, cerebrospinal fluid levels of, 97
- Glyburide, urine osmolality affected by, 133
- Glycated (glycosylated) hemoglobin, serum levels of, 98
- Glycerin, serum osmolality affected by, 132
- Glycogen storage disease
  - hyperlipidemia in, 379t
  - lactate levels in, 118
  - triglyceride levels in, 172, 379t
  - uric acid levels in, 177
- Glycohemoglobin, serum levels of, 98
- Gold standard, 7–8
- Gonadotropin deficiency, luteinizing hormone levels in, 122
- Gonadotropin-releasing hormone, luteinizing hormone stimulated by, 122
- Gonococcal conjunctivitis, 204
- Gonococcal pharyngitis, 209
- Gonococcal urethritis, test selection in, 233
- GOT. See Aspartate aminotransferase
- Gout
  - phosphorus levels in, 138
  - synovial fluid sampling in, 36, 389t
  - triglyceride levels in, 172
  - uric acid levels in, 177
- Graft-versus-host disease
  - CD4/CD8 ratio in, 68
  - transfusion and, 401t–402t
- Grafts
  - extremity, ultrasound in evaluation of, 278
  - infections, leukocyte scan in, 281
  - stenosis of, angiography in post-operative assessment of, 279
vein, cellulitis at donor site, test selection for, 240
Gram-negative bacteremia, complement C3 levels in, 75
Gram-negative bacteria, 27. See also specific organisms
Gram stain, 25–27
of amniotic fluid, in chorioamnionitis/ endometritis, 236
of ascitic fluid, 365t–366t
in bacteremia of unknown source, 241
of brain abscess aspirate, 197
of bronchoalveolar brushings, in anaerobic pneumonia or lung abscess, 213
in cellulitis, 240
of cerebrospinal fluid, in bacterial meningitis, 200, 369t
conjunctival, in conjunctivitis, 204
of corneal scrapings, in keratitis, 205
of ear drainage, in otitis externa, 207
in gas gangrene, 239
microscopic examination of, 27, 28i
of pericardial fluid, in pericarditis, 217
of peritoneal fluid, in peritonitis, 226
preparation of smear, 25
of prostatic secretions, in epididymitis/orchitis, 233
of sputum
in anaerobic pneumonia or lung abscess, 213
in community-acquired pneumonia, 212
in empyema, 216
in hospital-acquired pneumonia, 213
in immunocompromise-related pneumonia, 214
in laryngotracheobronchitis, 210
of synovial fluid, 389t–390t
in bacterial/sepptic arthritis, 238, 390t
technique for, 26–27
of tympanocentesis sampling, in otitis media, 206
of urethral discharge, in urethritis, 233
of urine, in urinary tract infection/
cystitis/pyuria-dysuria syndrome, 230
of vaginal fluid, 397t
of vesicle scrapings, in impetigo, 239
Granulocyte transfusion, 402t
Granulomatosis, Wegener's, neutrophil cytoplasmic antibody levels in, 130, 368t
Granulomatous disease
angiotensin-converting enzyme levels in, 52
calcium levels in, 63, 347i
Graves’ disease. See also Hyperthyroidism
thyroglobulin antibody in, 166
thyroid function tests in, 393t
thyroid-stimulating hormone receptor antibody in, 169, 393t
thyroperoxidase antibody in, 167
Gray-top tubes, 25, 42
Great occipital nerve, 341i
Greater auricular nerve, 341i
Green-top tubes, 25, 42
Griffith method, for diagnosis of ventricular tachycardia, 295–296
Group beating (regular irregular QRS rhythm), 287, 297
Growth, normal, 1,25-dihydroxy vitamin D3 levels in, 183
Growth hormone deficiency of, 99
in hypoglycemia, 349i
phosphorus levels in, 138
somatomedin C levels in, 162
serum levels of, 99
in hypoglycemia, 349i
Growth hormone receptor, defective (Laron dwarfism), growth hormone levels in, 99
Guaiac test, for fecal occult blood, 89
GUSTO study, 310–312, 311t, 312
Gynecologic disease. See also specific type
α-fetoprotein levels in, 91
ultrasound in, 275

H
H bands, Histoplasma capsulatum precipitin, 109
H2 blockers. See also specific types
gastrin levels affected by, 95
Haemophilus spp., on Gram-stained smear, 27
Haemophilus aphrophilus, test selection for, in infective endocarditis, 219
Haemophilus influenzae, test selection for in bacteremia of unknown source, 241
in bacterial meningitis, 200
in bacterial/sepptic arthritis, 238
in brain abscess, 197
in community-acquired pneumonia, 212
in conjunctivitis, 204
Haemophilus influenzae, test selection for
(cont.)
in empyema, 216
in endophthalmitis, 205
in epididymitis/orchitis, 233
in epiglottitis, 211
in HIV-associated pneumonia, 214
in hospital-acquired pneumonia, 213
in laryngotracheobronchitis, 210
in osteomyelitis, 237
in otitis media, 206
in sinusitis, 208
Haemophilus parainfluenzae, test selection for
in infective endocarditis, 219
Hal-life, of drugs, therapeutic drug monitoring and, 189
Haloperidol
electrocardiography affected by, 326
prolactin levels affected by, 144
Hantavirus, test selection for, in community-acquired pneumonia, 212
Haptoglobin
electrophoresis in detection of, 146
serum levels of, 99, 146
hemolysis and, 99, 363t
Hashimoto’s thyroiditis
thyroglobulin antibody in, 166, 393t
thyroxoperoxidase antibody in, 167, 393t
Hb. See Hemoglobin
HbA1c. See Glycated hemoglobin
HbA2. See Hemoglobin A2
HBcAb. See Hepatitis B core antibody
HBCo. See Carboxyhemoglobin
HBeAg/Ab. See Hepatitis B antigen/antibody
HBsAb. See Hepatitis B surface antibody
HBsAg. See Hepatitis B surface antigen
HCAb. See Hepatitis C antibody
β-hCG. See Chorionic gonadotropin
HCO3–. See Bicarbonate
Hct. See Hematocrit
Head
imaging test selection and interpretation in evaluation of, 245
injury, partial pressure of oxygen in, 133
Head and neck radiation
thyroid uptake and scan in patients with history of, 250
ultrasound screening of patients with history of, 249
Head and neck tumors, magnetic resonance imaging in evaluation of, 248
Heart
imaging test selection and interpretation in evaluation of, 257
normal, as electrocardiographic mimic of myocardial infarction, 320t
Heart block
atrioventricular. See Atrioventricular block
bundle branch. See Bundle branch block congenital, SS-A/Ro antibody in, 163
Heart disease
C-reactive protein levels in, 60
chest x-ray in, 252
congenital brain abscess with, 197
chest x-ray in, 252
magnetic resonance imaging in, 253
partial pressure of oxygen in, 133
red cell volume in, 151
ischemic, radionuclide ventriculography in, 257
magnetic resonance imaging in, 253
pharmacological therapy for, radionuclide ventriculography in evaluation of, 257
valvular, diagnostic evaluation of, 398t–399t
Heart failure
alanine aminotransferase levels in, 46
albumin levels in, 47
ascitic fluid profile in, 365t
aspartate aminotransferase levels in, 56
blood urea nitrogen levels in, 60
creatinine clearance in, 81
digoxin levels affected in, 192t
erythrocyte sedimentation rate in, 86
lactate dehydrogenase isoenzyme levels in, 117
lactate dehydrogenase levels in, 117
lidocaine levels affected in, 192t
plasma renin activity in, 152
pleural fluid profile in, 382t
quinidine levels affected in, 194t
serum osmolality in, 132
sodium levels in, 161
theophylline levels affected in, 194t
urine characteristics in, 395t
Heart rate, and QT interval, 324
Heart valve hemolysis, hemosiderin levels in, 104
Heartburn, esophageal reflux study in, 264
Heavy chain disease, immunoelectrophoresis in, 112
Heavy metals, and renal tubular acidosis, 388t
Helicobacter pylori
antibody, serum levels of, 100
in gastritis, 222
test selection for
in gastritis, 222
in infectious esophagitis, 222
Hemagglutination inhibition, for rubella
antibody detection, 156
Hemangioblastomas, cerebellar, erythropoietin levels with, 86
Hemangioma
cavernous, magnetic resonance imaging
in evaluation of, 260, 269
synovial fluid sampling in, 390
Hematocrit, 101
conversion from hemoglobin to, 101
spun, 101
in thalassemia, 101
Hematologic malignancy. See also specific malignancy
leukocyte count in, 121, 400
Hematopoiesis, active, iron levels in, 115
Hemiblocks. See Fascicular blocks
Hemoccult test, 89
Hemochromatosis
ferritin levels in, 90
hemosiderin levels in, 104
iron levels in, 115
and low-voltage QRS complex in ECG, 308
magnetic resonance imaging in, 269
transferrin saturation with iron in, 116
Hemoconcentration
albumin levels in, 47
erthrocyte count in, 85
hematocrit in, 101
hemoglobin levels in, 103
red cell volume in, 151
Hemodialysis patients
creatine kinase MB levels in, 79
hepatitis C antibody levels in, 106
phosphorus levels in, 138
testosterone levels in, 165
vitamin B12 levels in, 180
Hemodilution
albumin levels affected by, 47
magnesium levels in, 123
Hemoglobin. See also specific hemoglobin
conversion to hematocrit, 101
electrophoresis of, 102
in thalassemia syndromes, 102, 392
glycated (glycosylated), serum levels of, 98
mean corpuscular, 123–124, 363
percent saturation of, 133
total blood levels of, 103
variants of, methemoglobin levels affected by, 126
Hemoglobin A2
blood levels of, 101
in thalassemia, 392
Hemoglobin C, elevated levels of, 102
Hemoglobin C disease, hemoglobin electrophoresis in, 102
Hemoglobin C trait, hemoglobin electrophoresis in, 102
Hemoglobin F (fetal hemoglobin)
blood levels of, 103
glycohemoglobin levels affected by, 98
hereditary persistence of, 103
in thalassemia, 103, 392
Hemoglobin H
elevated levels of, 102
thalassemia mutation and, 375
Hemoglobin H disease, hemoglobin A2 levels in, 101
Hemoglobinemia, protein electrophoresis
in, 146
Hemoglobinopathies
glycohemoglobin levels in, 98
hemoglobin electrophoresis in, 102
prenatal diagnosis of, fetal hemoglobin levels in, 103
Hemoglobinuria
complement C3 levels in, 75
in hemolysis, 363
paroxysmal nocturnal
complement C3 levels in, 73
hemosiderin levels in, 104
leukocyte alkaline phosphatase levels in, 120
renal tubular acidosis in, 388
urine color affected by, 30
Hemolysis
bilirubin levels in, 58, 363
direct antiglobulin test for, 54
haptoglobin levels in, 99, 363
hemosiderin levels in, 104
hepatic function tests in, 378
lactate dehydrogenase isoenzyme levels in, 117
lactate dehydrogenase levels in, 117
mean corpuscular hemoglobin concentration in, 124
normocytic anemia caused by, 363
potassium levels in, 143
protein electrophoresis in, 146
urine color affected by, 30
Hemolytic anemia
  cold agglutinin levels in, 74
  complement C3 levels in, 75
  cryoglobulin levels in, 82
  glucose-6-phosphate dehydrogenase deficiency and, 97
  glycohemoglobin levels in, 98
  haptoglobin levels in, 99
  hematocrit in, 101
  hemoglobin levels in, 103
  hemosiderin levels in, 104
  iron levels in, 115
  lactate dehydrogenase isoenzyme levels in, 117
  lactate dehydrogenase levels in, 117
  reticulocyte count in, 153
  transferrin saturation with iron in, 116
Hemolytic disease of newborn
  direct antiglobulin test in, 54
  fetal hemoglobin levels in, 103
  glucose-6-phosphate dehydrogenase deficiency and, 97
  Rh grouping in prevention of, 154
Hemolytic streptococcal gangrene, test selection in, 240
Hemopexin, electrophoresis in detection of, 146
Hemophilia
  hemostatic function tests in, 377
  hepatitis C antibody levels in, 106
  synovial fluid sampling in, 390
  type A
    cryoprecipitated anti-hemophilic factor for, 402
    factor VIII assay in, 88
    factor VIII inhibitor in, inhibitor screen in detection of, 114
    molecular diagnostic techniques for, 374
    partial thromboplastin time in, 136
    type B, partial thromboplastin time in, 136
Hemorrhage
  albumin levels in, 47
  creatinine clearance in, 81
  gastrointestinal. See Gastrointestinal bleeding
glycohemoglobin levels in, 98
  hematocrit in, 101
  hemoglobin levels in, 103
  intracranial/subarachnoid
    cerebrospinal fluid profile in, 370
    computed tomography in evaluation of, 245
  prolonged QT interval in, 327
  ST segment depression or T wave inversion in, 322
  normocytic anemia caused by, 363
  platelet count after, 140
  reticulocyte count in, 153
  retroperitoneal, computed tomography in, 259
  Hemorrhagic diathesis, synovial fluid sampling in, 390
Hemorrhoids, fecal occult blood in, 89
Hemosiderin, urine levels of, 104
Hemosiderosis
  iron levels in, 115
  magnetic resonance imaging in, 269
Hemostasis
  factor VIII assay in evaluation of, 88
  laboratory evaluation of, 377
  Hemostatic function tests, 377
  Henderson-Hasselbalch equation, 137
Heparin
  activated clotting time affected by, 73
  antithrombin III levels affected by, 56
  free thyroxine levels affected by, 170
  ionized calcium levels affected by, 64
  partial thromboplastin time for monitoring therapy with, 136
  reptilase clotting time unaffected by, 153
  Russell’s viper venom clotting time affected by, 157
  in specimen tubes, 42
  thrombin time affected by, 165
Hepatic abscess. See Liver, abscess of
Hepatic angiography, 271
Hepatic arterial perfusion catheters, liver/spleen scan for evaluation of, 271
Hepatic arteries, ultrasound in evaluation of, 267
Hepatic cirrhosis. See Cirrhosis
Hepatic clearance, 189
Hepatic encephalopathy
  ammonia levels in, 51
  cerebrospinal fluid profile in, 371
  glutamine levels in, 97
  and operative death rate after portocaval shunt (Child’s criteria), 372
Hepatic failure
  ammonia levels in, 51
  blood urea nitrogen in, 60
Hepatic function tests, 377–378
Hepatic iminodiacetic acid scan (HIDA), 266
Hepatic insufficiency, testosterone levels in, 165
Hepatic mitochondria, antibodies against, 129
Hepatic necrosis, α-fetoprotein levels in, 91
Hepatic obstruction, hyperlipidemia in, 379
Hepatic synthesis, decreased, functional fibrinogen levels in, 92
Hepatic veins, ultrasound in evaluation of, 267, 278
Hepatitis
  alanine aminotransferase levels in, 46, 343i–345i, 378t
  angiotensin-converting enzyme levels in, 52
  aspartate aminotransferase levels in, 56, 343i–344i, 378t
  bilirubin levels in, 58, 378t
  CD4/CD8 ratio in, 68
  cholesterol levels in, 71
  complement C3 levels in, 75
  cryoglobulin levels in, 82
  α-fetoprotein levels in, 91
  gamma-glutamyl transpeptidase levels in, 94
  hepatic function tests in, 378t
  heterophile agglutination (Monospot/Paul-Bunnell) test in, 107
  IgM levels in, 113
  iron levels in, 115
  lactate dehydrogenase levels in, 117
  mitochondrial antibody levels in, 129
  non-A/non-B, hepatitis C antibody levels in, 106
  nuclear antibody levels in, 131
  posttransfusion, hepatitis C antibody screening in prevention of, 106
  rheumatoid factor levels in, 155
  serologic changes in, 343i–344i
  smooth muscle antibody levels in, 160
  total iron-binding capacity in, 116
  triglyceride levels in, 172
  type A
    hepatitis A antibody levels in, 104, 343i
    serologic changes in, 343i
  type B
    in cholangitis/cholecystitis, test selection in, 229
    hepatitis B core antibody levels in, 105, 344i
Hepatitis B surface antibody levels in, 105, 344i
Hepatitis B surface antigen levels in, 105, 344i
Hepatitis B e antigen/antibody levels in, 106
  immunity to hepatitis B surface antibody levels in, 105
  serologic changes in, 344i
  type C
    hepatitis C antibody levels in, 106, 345i
    typical course of, 345i
  type D, hepatitis D antibody levels in, 107
Hepatitis A antibody, serum levels of, 104, 343i
Hepatitis B core antibody, total serum levels of, 105, 344i
Hepatitis B surface antibody, serum levels of, 105, 344i
Hepatitis B surface antigen, serum levels of, 105, 344i
Hepatitis B e antigen/antibody, serum levels of, 106
Hepatitis C antibody, serum levels of, 106, 345i
Hepatitis D antibody, serum levels of, 107
Hepatobiliary disease, alkaline phosphatase levels in, 50
Hepatobiliary scintigraphy, in cholangitis/cholecystitis, 229
Hepatocellular carcinoma. See also Liver, cancer of
  α-fetoprotein levels in, 91
  magnetic resonance imaging in, 269
Hepatocellular jaundice, hepatic function tests in, 378t
Hepatolenticular degeneration
ceruloplasmin levels in, 69
Kayser-Fleischer rings in, 69
urine calcium levels in, 65
Hepatoma
  cholesterol levels in, 71
  red cell volume in, 151
Hepatosplenomegaly
  in hyperlipidemia, 380r
  liver/spleen scan in evaluation of, 271
Hepatotoxic drugs
  acetaminophen, 44, 336i
  alanine aminotransferase levels affected by, 46
Hepatotoxic drugs (cont.)
alkaline phosphatase levels affected by, 50
aspartate aminotransferase levels affected by, 56
bilirubin levels affected by, 58
lactate dehydrogenase levels affected by, 117
Hereditary angioedema
C1 esterase inhibitor levels in, 61
complement C4 levels in, 75
Hernia, hiatal, upper GI study in evaluation of, 262
Heroin, thyroid function tests affected by, 394
Herpes infection, CD4/CD8 ratio in, 68
Herpes simplex virus, test selection for in aseptic meningitis, 199
in conjunctivitis, 204
in encephalitis, 198
in HIV-associated diarrhea, 225
in impetigo, 239
in infectious esophagitis, 222
in keratitis, 205
in laryngitis, 209
in urethritis, 233
in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230
in vaginitis/vaginosis, 234
Herpes zoster virus, test selection for, in pericarditis, 217
Heterocyclic antidepressants, electrocardiography affected by, 326
Heterophile agglutination test, 107
Hexaxial reference system, 304
Hiatal hernia, upper GI study in evaluation of, 262
Hickman catheter
bacteremia of unknown source associated with, test selection in, 241
infectious thrombophlebitis associated with, test selection in, 221
HIDA (hepatic iminodiacetic acid scan), 266
High density lipoprotein, serum levels of, 71
High performance liquid chromatography for cyclosporine monitoring, 191t
for fetal hemoglobin measurement, 103
for hemoglobin A2 measurement, 101
Hirsutism
diagnostic algorithm for, 346i
testosterone levels in, 165, 346i
Histone proteins, antibodies to (nuclear antibody), serum levels of, 131
Histoplasma spp., test selection for, in infectious esophagitis, 222
Histoplasma capsulatum
antigen, 108
complement fixation (CF) antibody test, 109
in meningitis, 201
precipitins, serum levels of, 109
test selection for in community-acquired pneumonia, 212
in fungal meningitis, 201
in HIV-associated pneumonia, 214
Histoplasmin skin test
Histoplasma capsulatum complement fixation antibody test affected by, 109
Histoplasma capsulatum precipitin levels affected by, 109
Histoplasmosis
angiotensin-converting enzyme levels in, 52
Histoplasma capsulatum antigen levels in, 108
Histoplasma capsulatum complement fixation antibody test in, 109
Histoplasma capsulatum precipitin levels in, 109
HIV antibody, serum levels of, 110
HLA-B27 typing, 111
HLA typing, 110
Hodges correction (QT nomogram), 324–326
Hodgkin’s disease
ferritin levels in, 90
leukocyte count in, 400t
Homosexual men
epididymitis/orchitis in, test selection in, 233
hepatitis C antibody levels in, 106
Hospital-acquired peritonitis, test selection in, 226
Hospital-acquired pneumonia, test selection in, 213
Hospitalized patients, dexamethasone suppression test in, 83
Howell-Jolly bodies, 29i
HPLC. See High performance liquid chromatography
Human chorionic gonadotropin. See Chorionic gonadotropin
Human granulocytic ehrlichiosis, test selection for, in parasitic meningoencephalitis, 203
Human herpes 6 virus infection, encephalitis after, test selection for, 198
Human herpes virus 8, test selection for, in bacteremia of unknown source, 241
Human immunodeficiency virus. See AIDS/HIV infection
Human leukocyte antigen typing, 110–111
Human monocytic ehrlichiosis, test selection for, in parasitic meningoencephalitis, 203
Humoral hypercalcemia of malignancy, parathyroid hormone-related protein levels in, 135
Huntington’s disease, molecular diagnostic techniques for, 375t
Hürthle cell cancer, thyroglobulin levels in, 166
Hyaline casts, in urine, 387t, 395t–396t
Hyaline membrane disease, lecithin/sphingomyelin ratio in, 119
Hydatidiform mole, chorionic gonadotropin levels in, 72
Hydralazine, plasma renin activity affected by, 152
Hydrocephalus, cisternography in evaluation of, 247
Hydrochlorothiazide amylase levels affected by, 52
chloride levels affected by, 70
phosphorus levels affected by, 138
Hydrogen ion excretion, decreased, pH affected by, 137
Hydronephrosis, ultrasound in evaluation of, 258, 273
Hydrops fetalis, thalassemia mutation and, 375t
5-Hydroxy-indoleacetic acid, urine levels of, 111
25-Hydroxy vitamin D3, serum or plasma levels of, 182, 347i
in hypercalciuria, 182, 347i
in vitamin D deficiency, 182
in vitamin D intoxication, 182
in vitamin D overdose, 182
in vitamin D toxicity, 182
1α-Hydroxylase deficiency, 1,25-dihydroxy vitamin D3 levels in, 183
11β-Hydroxylase deficiency, hypertension associated with hypokalemia and, 348i
17α-Hydroxylase deficiency, hypertension associated with hypokalemia and, 348i
Hypaque enema, 264
Hyperadrenocorticism, chloride levels in, 70
Hyperaldosteronism aldosterone levels in plasma, 48
urine, 49
chloride levels in, 70
in hypertension associated with hypokalemia, 348i
magnesium levels in, 123
plasma renin activity in, 152
potassium levels in, 143
sodium levels in, 161
Hyperalimentation with inadequate phosphate repletion, phosphorus levels in, 138
infectious thrombophlebitis associated with, test selection in, 221
Hypercalcemia calcium levels in serum, 63
urine, 65
chloride levels in, 70
diagnostic algorithm for, 347i
electrocardiographic findings in, 325t, 327
familial hypocalciuric, calcium levels in, 63, 347i
humoral, of malignancy, parathyroid hormone-related protein levels in, 135
25-hydroxy vitamin D3 levels in, 182, 347i
malignancy with, parathyroid hormone levels in, 134
non-parathyroid, parathyroid hormone levels in, 134, 354i
1,25-OH vitamin D levels in, 347i
phosphorus levels in, 138
serum osmolality in, 132
Hypercalciuria 1,25-dihydroxy vitamin D3 levels in, 183
urine calcium levels in, 65
urine calcium/urine creatinine ratio in, 65
Hypercholesterolemia characteristics and laboratory findings in, 379t
cholesterol levels in, 71, 379t
Hyperchylomicronemia, characteristics and laboratory findings in, 380t
Hypercoagulability. See also Thrombosis

factor V (Leiden) mutation in, 87, 373t

partial thromboplastin time in, 136

protein C deficiency and, 145

Hyperemesis gravidarum, chorionic
gonadotropin levels in, 72

Hyperglobulinemia, microhemagglutination-
Treponema pallidum (MHA-TP) test in, 129

Hyperglycemia, potassium levels in, 143

Hyperkalemia
electrocardiographic findings in
mimicking myocardial infarction,
320t

ST segment elevation, 321t

renal tubular acidosis in, 388t

Hyperkalemic familial periodic paralysis,
potassium levels in, 143

Hyperlipidemia
characteristics and laboratory findings
in, 379t–380t

cholesterol levels in, 71, 379t–380t

lipoprotein levels in, 71, 379t–380t

protein electrophoresis in, 146

triglyceride levels in, 172, 379t–380t

Hypernatremia
with normal hydration, serum osmolality
in, 132

with overhydration, serum osmolality
in, 132

secondary to dehydration, serum osmolality in, 132

Hyperparathyroidism
alkaline phosphatase levels in, 50

calcium levels in, 347i, 354i

serum, 63

urine, 65

chloride levels in, 70

1,25-dihydroxy vitamin D₃ levels in,
183

parathyroid hormone levels in, 134,
347i

phosphorus levels in, 138

renal tubular acidosis in, 388t

Hyperphosphatemia
benign familial, alkaline phosphatase
levels in, 50

calcium levels in, 63

25-hydroxy vitamin D₃ levels in, 182

Hyperprolactinemia
in growth-hormone deficient patients,
somatedin C levels in, 162

prolactin levels in, 144

Hyperpyrexia, malignant, phosphorus
levels in, 138

Hypersegmented neutrophil, 29i

Hypersensitivity reaction, leukocyte count
in, 400t

Hypersplenism, leukocyte count in, 400t

Hypertension
aldosterone levels in, 48

with hypokalemia, diagnostic algorithm
for, 348i

pheochromocytoma causing, diagnostic
algorithm for, 355i

plasma renin activity in, 152, 348i

of pregnancy, uric acid levels in, 177

renal vascular, renal scan in, 274

urine characteristics in, 396t

valvular heart disease in, diagnostic
evaluation of, 399t

Hyperthermia, malignancy, creatine
kinase levels in, 78

Hyperthyroidism
angiotensin-converting enzyme levels
in, 52

calcium levels in, 347i

serum, 63

urine, 65

ch孔sterol levels in, 71

digoxin levels affected in, 192t

electrocardiographic findings of, 327

factitious, thyroglobulin levels in, 166

ferritin levels in, 90

free thyroxine index in, 169–170

free thyroxine levels in, 169–170

glucose tolerance test in, 96

leukocyte count in, 400t

neonatal, thyroid-stimulating hormone
receptor antibody in, 169

parathyroid hormone levels in, 134

in pregnancy, thyroid function tests in,
394t

protein electrophoresis in, 146

radionuclide thyroid therapy for, 251

relapse, thyroid-stimulating hormone
receptor antibody in prediction
of, 169

testosterone levels in, 165

thyroglobulin antibody in, 166

thyrogblobulin levels in, 166

thyroid function tests in, 393t

thyroid-stimulating hormone levels in,
168, 393t

thyroid uptake and scan in, 250, 393t

total iron-binding capacity in, 116

total thyroxine levels in, 169

total triiodothyronine in, 173

triglyceride levels in, 172

triiodothyronine levels in, 173
Index 453

Hypertonic hyponatremia, 350
Hypertriglyceridemia
characteristics and laboratory findings in, 379
hemoglobin levels affected by, 103
mixed, characteristics and laboratory findings in, 380
triglyceride levels in, 172, 379–380
Hypertrophic cardiomyopathy, electrographic findings of, mimicking myocardial infarction, 320
Hypertrophic osteoarthropathy, synovial fluid sampling in, 389
Hyperuricemia, uric acid levels in, 177
Hyperventilation
pH in, 137
serum osmolality in, 132
Hypervitaminosis D
25-hydroxy vitamin D₃ levels in, 182
phosphorus levels in, 138
Hypervolemia
serum osmolality in, 132
sodium levels in, 132, 161
Hypervolemic hypotonic hyponatremia, 350
Hypoadrenocorticism, magnesium levels in, 123
Hypoalbuminemia
calcium levels in, 63
with hypoproteinemia, 147
phenytoin levels affected in, 193
Hypoaldosteronism
aldosterone levels in plasma, 48
urine, 49
hyporeninemic plasma renin activity in, 152
potassium levels in, 143
renal tubular acidosis in, 388
Hypocalcemia
electrocardiographic findings in, 325, 326
25-hydroxy vitamin D₃ levels in, 182
neonatal, ionized calcium levels in, 64
urinary calcium levels in, 65
Hypocalciuria, familial, calcium levels in, 63, 347
Hypocalciuric hypercalcemia, urinary calcium levels in, 65
Hypochlorhydria, gastrin levels in, 95
Hypochromia, mean corpuscular hemoglobin in, 123
Hypochromic anemia
diagnosis of, based on red blood cell indices, 363
laboratory and clinical findings in, 363–364
mean corpuscular hemoglobin concentration in, 124
molecular diagnostic techniques for, 375
Hypochromic microcytic erythrocytes, 29
Hypofibrinogenemia
cryoprecipitated anti-hemophilic factor for, 402
erythrocyte sedimentation rate in, 86
functional fibrinogen levels in, 92
reptilase clotting time in, 153
Russell’s viper venom clotting time in, 157
Hypogammaglobulinemia, erythrocyte sedimentation rate in, 86
Hypoglycemia
C-peptide levels in, 62
diagnostic algorithm for, 349
glucose levels in, 95
prolactin levels in, 144
serum insulin levels in, 115, 349
Hypoglycemic drugs, oral
C-peptide levels affected by, 62
glucose levels affected by, 95
serum insulin levels affected by, 115
surreptitious use of, 349
Hypogonadism
infertility caused by, 352
luteinizing hormone levels in, 122
testosterone levels in, 165
Hypogonadotropic hypogonadism, 352
Hypokalemia
and digoxin monitoring, 192
electrocardiographic findings in prolonged QT interval, 296, 325
ST segment depression or T wave inversion, 322–323, 325
ST-T-U abnormalities, 325
torsade de pointes, 296
U waves, 324, 325
hypertension with, diagnostic algorithm for, 348
phosphorus levels in, 138
plasma renin activity in, 152
Hypolipoproteinemia, triglyceride levels in, 172
Hypomagnesemia
magnesium levels in, 123
parathyroid hormone levels in, 134
Hyponatremia
diagnostic algorithm for, 350
hypertonic, 350
Hypovolemia (cont.)
  hypervolemic, 132, 350
  hypovolemic, 132
  isotonic, 350
  isovolemic, 350
  normovolemic, 132, 350
  serum osmolality in, 132, 350
  sodium levels in, 161
  urine sodium levels in, 350
Hyponatremic hypealdosteronism, 388t
Hypoparathyroidism
  calcium levels in, 354
    serum, 63
    urine, 65
  1,25-dihydroxy vitamin D$_3$ levels in, 183
  magnesium levels in, 123
  parathyroid hormone levels in, 134
  phosphorus levels in, 138
Hypoperfusion, lactate levels in, 118
Hypophosphatasia, alkaline phosphatase levels in, 50
Hypophosphatemia, renal tubular acidosis in, 388t
Hypopituitarism
  glucose levels in, 95
  glucose tolerance test in, 96
  growth hormone affected by, 99
  serum insulin levels in, 115
  sodium levels in, 161
  somatomedin C levels in, 162
Hypoplastic anemia, iron levels in, 115
Hypoproteinemia
  protein levels in, 147
  total iron-binding capacity in, 116
Hyporeninemic hypealdosteronism
  aldosterone levels in, 48
  plasma renin activity in, 152
  potassium levels in, 143
Hypothalamic disorders/failure
  follicle-stimulating hormone levels in, 93
  hirsutism with, 346
  in hypothyroidism, 351, 393
  luteinizing hormone levels in, 122
  serum osmolality in, 132
  sodium levels in, 161
Hypothalamic/pituitary stalk lesions, prolactin levels with, 144
Hypothermia, electrocardiographic findings in, 328
  Osborn wave, 328
  prolonged QT interval, 327–328

Hypothyroidism
  amenorrhea in, 339
  angiotensin-converting enzyme levels in, 52
  cholesterol levels in, 71
  creatine kinase levels in, 78
  creatinine clearance in, 81
  creatinine levels in, 80
  diagnostic algorithm for, 351
  digoxin levels affected in, 192
  free thyroxine index in, 169–170
  free thyroxine levels in, 169–170, 351
  hematocrit in, 101
  hemoglobin levels in, 103
  hyperlipidemia in, 379
  iron levels in, 115
  magnesium levels in, 123
  phosphorus levels in, 138
  in pregnancy, thyroid function tests in, 394
    primary, 351
  prolactin levels in, 144
  radionuclide thyroid therapy and, 251
  replacement therapy for, thyroid function tests in, 393
    secondary, 351
  somatomedin C levels in, 162
  thyroid function tests in, 393
  thyroid-releasing hormone test in, 351
  thyroid-stimulating hormone levels in, 168, 351, 393
  thyroid uptake and scan in, 250, 393–394
  total thyroxine levels in, 169
  total triiodothyronine in, 173
  triglyceride levels in, 172
  urinary calcium levels in, 65
Hypoventilation, partial pressure of oxygen in, 133
Hypovitaminosis D, phosphorus levels in, 138
Hypovolemic hypotonic hyponatremia, 350
Hypoxanthine-guanine phosphoribosyltransferase deficiency, X-linked, uric acid levels in, 177
Hypoxemia
  in Pneumocystis carinii pneumonia, 214
  red cell volume in, 151
Hypoxia
alanine aminotransferase level in, 46
aspartate aminotransferase levels in, 56
erthropoietin levels in, 86
lactate levels in, 118

I
I, RAI uptake of (iodine, radioactive uptake of), 250, 393–394
Idiopathic thrombocytopenic purpura
hemostatic function tests in, 377
platelet-associated IgG in, 141
platelet count in, 140, 377
IFA. See Immunoﬂuorescent antibody test
IgA
electrophoresis in detection of, 146
inherited deﬁciency of, IgA levels in, 113
Q fever antibody, 149
serum levels of, 146
quantitative, 113
IgA myeloma, 113
IgC, serum levels of, quantitative, 113
IgD, electrophoresis in detection of, 146
IgE, electrophoresis in detection of, 146
IgG
deficiency of, 113
direct antiglobulin test for red cell coating by, 53
electrophoresis in detection of, 146
hepatitis A, 104, 343
hepatitis B core antibody, 105
Lyme disease, 122
platelet-associated, 141
Q fever antibody, 149
serum levels of, 113
cerebrospinal fluid levels and (IgG index), 112
quantitative, 113
Toxoplasma, 171
IgG index, 112
IgG myeloma
IgG serum levels in, 113
immunoelectrophoresis in, 112
IgM
electrophoresis in detection of, 146
hepatitis A, 104, 343
hepatitis B core antibody, 105
Lyme disease, 122
Q fever antibody, 149
serum levels of, quantitative, 113
Toxoplasma, 171
IHA. See Indirect hemagglutination
Ileal disease/resection
vitamin B12 absorption test (Schilling’s test) after, 181
vitamin B12 absorption test (Schilling’s test) in, 181
Ileitis, regional. See Crohn’s disease
Ileum, peroral pneumocolon in evaluation of, 263
Iliac stenosis, angiography in evaluation of, 279
Iliohypogastric nerve, 341–342
Imaging, 243–281. See also speciﬁc anatomic location and type of study
Imipramine, therapeutic monitoring of, 192
Immobiliation, calcium levels in, 65, 134
Immune complex disease
complement C3 levels in, 75
cryoglobulin causing, 82
Immune responses, rheumatoid factor levels affected by, 155
Immunoassay
for C1 esterase inhibitor, 61
for cardiac troponin-I, 174
for chlamydial antigen, in salpingitis/pelvic inﬂammatory disease, 236
for complement C3, 75
for complement C4, 75
for cyclosporine monitoring, 191
for free thyroxine measurement, 170
for group A Streptococcus antigen, in pharyngitis, 209
for hepatitis A antibody, 104
for HIV antibody, 110
for luteinizing hormone, 122
for parathyroid hormone, 134
for somatomedin C, 162
Immunoblot assay, recombinant, for hepatitis C antibody, 106
Immunocompromised host
bacteremia of unknown source in, test selection in, 241
pneumonia in, test selection in, 214
Immunodeﬁciency. See also AIDS/HIV infection
IgG levels in, 113
leukocyte count in, 400
protein electrophoresis in, 146
quantitative immunoglobulin levels in, 113
Immunodiffusion
in fungal meningitis, 201
in immunocompromise-related pneumonia, 214

Immunoelectrophoresis, 112
Immunofixation, 112
Immunofluorescent antibody test in *Chlamydia trachomatis* conjunctivitis, 204
for HIV antibody, 110
for *Legionella* antibody, 120
for Q fever antibody, 149

Immunoglobulins. See also specific types under Ig
electrophoresis in detection of, 146
immunoelectrophoresis in identification of classes of, 112
serum levels of, 113, 146

Immunoproliferative disorders, cryoglobulin levels in, 82

Induction therapy
epididymitis/orchitis associated with, test selection in, 233

Impetigo neonatorum, test selection in, 239
test selection in, 239

Impotence
follicle-stimulating hormone levels in, 93
prolactin levels in, 144

Inanition, hypoglycemia and, 349

Incomplete bundle branch block, 302–303
India ink preparation, for *Coccidioides*, in fungal meningitis, 201

Indirect antiglobulin test (indirect Coombs test), 54

Indirect hemagglutination for *Entamoeba histolytica* antibodies, 50
for rubella antibody, 156

Indium-111 antimyosin antibody imaging, in infectious myocarditis, 218

Indium scanning
leukocyte, 281
in osteomyelitis, 237
utility in critically ill patients, 281

Indomethacin
and calcium levels in urine, 65
plasma renin activity affected by, 152

Inducer T cells (CD4 cells), 68

Infants of diabetic mother
- glucose levels in, 95
- lecithin/sphingomyelin ratio in, 119
test selection in disorders in
- bacterial/septic arthritis, 238
- community-acquired pneumonia, 212
- empyema, 216
- impetigo, 239
- infectious colitis/dysentery, 223
- laryngotracheobronchitis, 210
- osteomyelitis, 237
- otitis media, 206

total iron-binding capacity in, 116

Infection
- α1-antiprotease levels in, 55
- creatinine clearance in, 81
- erythrocyte sedimentation rate in, 86
- free erythrocyte protoporphyrin levels in, 94
- glucose tolerance test in, 96
- haptoglobin levels in, 99
- IgA levels in, 113
- IgG levels in, 113
- IgM levels in, 113
- iron levels in, 115
- leukocyte alkaline phosphatase levels in, 120
- leukocyte count in, 121, 400t
- leukocyte scan in, 281
- protein electrophoresis in, 146
- Venereal Disease Research Laboratory Test in, 178

Infectious mononucleosis. See Mononucleosis

Infertility
male, diagnostic algorithm for, 352i
semen analysis in, 159, 351i
ultrasound in, 275

Infertility
follicle-stimulating hormone levels in, 93, 352t
male, diagnostic algorithm for, 352i
semen analysis in, 159, 351i
ultrasound in, 275

Inflammation
- anti-nuclear antibody in, 367t
- α1-antiprotease levels in, 55
- C-reactive protein levels in, 60
- ceruloplasmin levels in, 69
- complement C3 levels in, 75
- erythrocyte sedimentation rate in, 86
- erythropoietin levels in, 86
- factor VIII assay in, 88
- ferritin levels in, 90
-
Index

functional fibrinogen levels in, 92
IgG index in, 112
iron-binding capacity in, 116
leukocyte count in, 121, 400
β₂-microglobulin levels in, 128
platelet count in, 140
protein electrophoresis in, 146
pulmonary angiography in, 255
synovial fluid sampling in, 389
von Willebrand’s factor protein levels in, 184
Inflammatory bowel disease
barium enema in evaluation of, 263
fecal occult blood in, 89
leukocyte scan in, 281
Influenza, encephalitis after, test selection in, 198
Influenza virus, test selection for
in community-acquired pneumonia, 212
in hospital-acquired pneumonia, 213
in infectious myocarditis, 218
in laryngitis, 209
in laryngotracheobronchitis, 210
in pericarditis, 217
in sinusitis, 208
in transplant-related pneumonia, 214
Inhalation burn injury, ventilation-perfusion scan in, 253
Inhibitor screen, 114
Injections
    intraarticular, bacterial/septic arthritis associated with, test selection in, 238
    intramuscular, creatine kinase levels with, 78
Innervation, dermatome chart of, 341–342
Insecticides, electrocardiography affected by, 326
Insulin
    factitious/surreptitious use of
        C-peptide levels in, 62, 115, 349
        glucose levels in, 95
        phosphorus levels affected by, 138
        serum levels of
            in hypoglycemia, 115, 349
            immunoreactive, 115
    Insulin antibody test, 114
        in hypoglycemia, 349
    Insulin resistance
        insulin antibody levels in, 114
        serum insulin levels in, 115
    Insulin therapy
        insulin antibody levels and, 114
        phosphorus levels affected by, 138
Insulinoma
    C-peptide levels in, 62, 115, 349
    glucose levels in, 95
    hypoglycemia caused by, 349
    serum insulin levels in, 115
Interfering factors, 6–7
Intermediate density lipoprotein (cholesterol), serum levels of, in hyperlipidemia, 380
International Normalized Ratio, for prothrombin time, 148, 188
Interstitial lung disease
    computed tomography in, 252
    partial pressure of oxygen in, 133
Intestinal absorption, phosphorus levels affected by, 138
Intestinal angina, mesenteric angiography in evaluation of, 261
Intestinal disease. See Gastrointestinal disease
Intestinal lymphangiectasia
    cholesterol levels in, 71
    triglyceride levels in, 172
Intoxication, ethanol
definition of, 87
osmolal gap in, 132, 381
serum osmolality in, 87, 132
Intraarticular injection, bacterial/septic arthritis associated with, test selection in, 238
Intracranial aneurysm, magnetic resonance angiography in, 246
Intracranial disease, magnetic resonance imaging in evaluation of, 245
Intracranial hemorrhage. See also Subarachnoid hemorrhage
    computed tomography in evaluation of, 245
Intracranial masses, computed tomography in evaluation of, 245
Intrahepatic cholestasis, hepatic function tests in, 378
Intramuscular injections, creatine kinase levels with, 78
Intraperitoneal disorders, computed tomography in, 259
Intrathecal drug therapy, neighborhood meningeal reaction in, 371
Intrauterine device, localization of, ultrasound in, 275
Intravascular hemolysis
  hemosiderin levels in, 104
  posttransfusion, haptoglobin levels in, 99
  urine color affected by, 30
Intravenous contrast studies, risks of, 244
Intravenous drug abusers. See Drug abusers
Intravenous pyelogram, 273
  in pyelonephritis, 232
  in urinary tract infection/cystitis/pyuria-dysuria syndrome, 230
Intraventricular conduction delay or defect, 303
Intrinsic coagulation pathway, partial thromboplastin time in evaluation of, 136
Iodine
  ingestion of, thyroid function tests affected by deficiency or ingestion of, 394
  radioactive uptake of, in thyroid evaluation, 250, 393–394
Iodochlorhydroxyquin, urine color affected by, 30
Ion-exchange resins, pH affected by, 137
Ion-selective electrode, of serum sodium measurement, 161
Ionized calcium, serum levels of, 64
IRMA. See Immunoradiometric assay
Iron
deficiency, 363–364
  bone marrow iron stores in, 363–364
  erythropoietin levels in, 86
  ferritin levels in, 90
  free erythrocyte protoporphyrin levels in, 94, 364
  hematocrit in, 101
  hemoglobin A2 levels in, 101
  hemoglobin levels in, 103
  laboratory and clinical findings in, 363–364
  mean corpuscular hemoglobin in, 123–124, 363
  mean corpuscular volume in, 124, 363–364
  platelet count in, 140
  protein electrophoresis in, 146
  recovery from, reticulocyte count in, 153
  reticulocyte count in, 153
  serum iron levels in, 115, 363–364
  total iron-binding capacity in, 116, 363–364
  transferrin saturation with iron in, 116, 364
  excess administration of, serum iron levels in, 115
  overload
    ferritin levels in, 90
    transferrin saturation with iron in, 116
  plasma/signal levels of, 115
    in anemias, 115, 363–364
  poisoning, transferrin saturation with iron in, 116
  total body stores of, serum ferritin and, 90
Iron-binding capacity, total, 116
  in anemias, 116, 363–364
Ischemia
  alanine aminotransferase level in, 46
  aspartate aminotransferase levels in, 56
  at distance, 316
  mesenteric, angiography in, 261
  myocardial
    definition of, 310
    electrocardiographic findings in, 310–320, 323
  mesenteric angiography in, 261
  serum insulin levels in, 115
  total body stores of, serum ferritin and, 90
Ischemic attack, atypical transient, carotid Doppler in, 278
Islet cell antibodies, serum levels of, 114
Islet cell disease/tumors. See also Insulinoma; Pancreas
  glucose levels in, 95
  glucose tolerance test in, 96
  mesenteric angiography in, 261
  serum insulin levels in, 115
Isolated germinal compartment failure, infertility caused by, 352
Isoniazid, phosphorus levels affected by, 138
Isoniazid toxicity, and lactate levels, 118
Isopropanol, osmolal gap in lethal concentrations of, 132, 381
Isopropyl alcohol, serum osmolality affected by, 132
Isoproterenol, potassium levels affected by, 143
Isospora belli, test selection for, in HIV-associated diarrhea, 225
Isotonic hyponatremia, 350
Isovolemic hypotonic hyponatremia, 350

Index
ITP. See Idiopathic thrombocytopenic purpura

Itraconazole, electrocardiography affected by, 326

IUD (intrauterine device), ultrasound in localization of, 275

IVCD (intraventricular conduction delay or defect), 303

IVP. See Intravenous pyelogram

J

J point

in atrioventricular reentry tachycardia, 299
depression at, catecholamines and, 322t
in myocardial infarction, inferior, 314 normal, 300

Jaundice

bilirubin levels in, 58, 378t
in hepatitis C, 345t
hepatocellular, hepatic function tests in, 378t
obstructive, lactate dehydrogenase levels in, 117

Jejunal biopsy

in HIV-associated diarrhea, 225
in infectious colitis/dysentery, 223

Joint(s), magnetic resonance imaging in evaluation of, 278

Joint aspiration fluid. See Synovial fluid sampling

Joint disease, synovial fluid sampling in, 389t

Joint prosthesis

bacterial/septic arthritis associated with, test selection in, 238
bone scan in evaluation of, 276
osteomyelitis associated with, test selection in, 237

Jugular venous pressure, in pulmonary embolism, 356i–357i

Junctional complexes, definition of, 288

Junctional rhythms, 288–289
accelerated, 289, 289t, 298
classification of, 289, 289t
escape, 288–289, 289t
sinus bradycardia with, 298

K

Kallman’s syndrome, luteinizing hormone levels in, 122

Kanamycin

amikacin levels affected by, 191t
ammonia levels affected by, 51
tobramycin levels affected by, 194t

Kaposi’s sarcoma, of lung, HIV-associated pneumonia and, 214

Kayser-Fleischer rings, in Wilson’s disease, 69

Keratitis, test selection in, 205

Ketoacidosis

alcoholic, acetoacetate levels in, 45
diabetic
acetoacetate levels in, 45
amylase levels in, 52
chloride levels in, 70
creatine levels in, 80
lactate levels in, 118
leukocyte count in, 400t
lipase levels in, 121
magnesium levels in, 123
pH in, 137
phosphorus levels in, 138
serum osmolality in, 132

Ketoconazole
cyclosporine levels affected by, 191t
electrocardiography affected by, 326

Ketones, urine, dipstick testing of, 30, 31t

Ketosis
pH in, 137
phosphorus levels in, 138

Kidney(s). See also under Renal cancer of

calcium levels in, 63
computed tomography in, 259
magnetic resonance imaging in, 260, 273
parathyroid hormone-related protein levels in, 135
red cell volume in, 151
imaging test selection and interpretation in evaluation of, 273–274
injury, lactate dehydrogenase levels in, 117
medullary sponge, intravenous pyelogram in, 273
transplantation of
pyelonephritis associated with, test selection in, 232
renal scan in evaluation of, 274
x-ray of (KUB plain radiograph), 258

Kidney disease
albumin levels in, 47
angiotensin-converting enzyme levels in, 52
Kidney disease (cont.)
- α1-antiprotease levels in, 55
- antithrombin III levels in, 56
- blood urea nitrogen levels in, 60
- C-peptide levels in, 62
- calcitonin levels in, 62
- calcium levels in
  - ionized, serum, 64
  - serum, 63
  - urine, 65
- ceruloplasmin levels in, 69
- chloride levels in, 70
- cholesterol levels in, 71
- complement C3 levels in, 75
- complement C4 levels in, 75
- cortisol levels in, 76
- creatinine clearance in, 81
- creatinine levels in, 80
- cryoglobulin causing, 82
- double-stranded DNA antibody levels in, 84, 367
- erythrocyte sedimentation rate in, 86
- erythropoietin levels in, 86
- 5-hydroxy-indoleacetic acid levels in, 111
- hypoglycemia and, 349
- imaging test selection and interpretation in, 273–274
- intrinsic, urine indices in, 387
- iron levels in, 115
- lactate dehydrogenase isoenzyme levels in, 117
- lactate dehydrogenase levels in, 117
- magnesium levels in, 123
- methylmalonic acid levels in, 126
- β2-microglobulin levels in, 128
- neutrophil cytoplasmic antibody levels in, 130
- nuclear antibody levels in, 131
- parathyroid hormone levels in, 134
- pH in, 137
- phosphorus levels in, 138
- plasma renin activity in, 152
- potassium levels in, 143
- prolactin levels in, 144
- serum osmolality in, 132
- sodium levels in, 161
- total iron-binding capacity in, 116
- ultrasound in, 258
Kidney stones (renal calculi)
- computed tomography in, 259
- intravenous pyelogram in, 273
- urinary calcium levels in, 65

Kingella spp., test selection for, in infectious endocarditis, 219

Kingella kingae, test selection for, in bacterial/septic arthritis, 238

Klebsiella spp.
- test selection for
  - in anaerobic pneumonia or lung abscess, 213
  - in bacteremia of unknown source, 241
  - in hospital-acquired pneumonia, 213
  - in laryngotracheobronchitis, 210
  - in liver abscess, 228
  - in neutropenic pneumonia, 214
  - in otitis media, 206
  - urinalysis in identification of infection caused by, 30

Klebsiella oxytoca, test selection for, in antibiotic-associated pseudomembranous colitis, 224

Klebsiella pneumoniae, test selection for
- in aspiration pneumonia, 212
- in community-acquired pneumonia, 212
- in empyema, 216

Klinefelter’s syndrome
- follicle-stimulating hormone levels in, 93
- testosterone levels in, 165

KOH preparation
- in keratitis, 205
- in vaginitis/vaginosis, 33–35, 35i–36i, 234, 397

KUB (kidneys, ureters, bladder) x-ray, 258

Kwashiorkor, iron levels in, 115

Kyphoscoliosis, partial pressure of oxygen in, 133

L
- L/S ratio (lecithin/sphingomyelin ratio), in amniotic fluid, 119

LA. See Latex agglutination test

Labeled red cell scan, in gastrointestinal bleeding, 265

Labeled white blood cell scan, 281

Laboratory procedures, in clinical setting, 23–39

Laboratory tests. See Diagnostic/ laboratory tests

LAC. See Lupus anticoagulant

Lactate, blood levels of, 118

Lactate dehydrogenase
- ascitic fluid, in tuberculous peritonitis/enterocolitis, 227
- isoenzymes of, serum levels of, 117
peritoneal fluid, in peritonitis, 226
  serum levels of, 117
  in Pneumocystis carinii pneumonia, 214
Lactation (breastfeeding, nursing)
  1,25-dihydroxy vitamin D₃ levels in, 183
  prolactin levels in, 144
Lactic acidosis
  lactate levels in, 118
  pH in, 137
  phosphorus levels in, 138
Lactulose, ammonia levels affected by, 51
LAD. See Left anterior descending artery
  occlusion of, electrocardiographic signs of, 316
Left anterior fascicular block, 305
  diagnostic criteria for, 303
  as mimic of myocardial infarction, 320
  new, in left anterior descending artery occlusion, 316
  poor R wave progression in, 309
Left atrial enlargement
  clinical correlations of, 300
  electrocardiographic findings of, 300–301
Left axis deviation, 305
Left bundle branch block, 302
  diagnostic criteria for, 302
  incomplete, 302
  as mimic of myocardial infarction, 320
  morphology of, in wide QRS complex, 291–292
  poor R wave progression in, 309
  QRS complex in, 290–292, 295–296, 302
  ST segment depression or T wave inversion in, 322
  ST segment elevation in, 321
  ST-T changes in, 302
Left circumflex coronary artery, as culprit in posterior myocardial injury or infarction, 315
Left posterior fascicular block, 306
  diagnostic criteria for, 303
Left ventricular hypertrophy
  echocardiography of, 306
  electrocardiographic findings in
  inverted U waves, 324
  prolonged QT interval, 327
  ST segment depression or T wave inversion in, 322
  ST segment elevation, 307, 321, 323
  electrocardiographic findings of, 306–307
  Cornell voltage in, 306
  incomplete left bundle branch block, 302
Left ventricular hypertrophy (cont.)
electrocardiographic findings of (cont.)
left atrial enlargement, 301
mimicking myocardial infarction, 320t
poor R wave progression, 309
repolarization abnormalities, 307, 321r–322t
Romhilt-Estes criteria for, 306
Sokolow-Lyon criteria for, 306
plasma renin activity in, 152
Leg cable, right, misplacement in ECG, 327–328
Legionella spp.
antibody, serum levels of, 120
infection (legionellosis), Legionella antibody levels in, 120
test selection for
in community-acquired pneumonia, 212
in empyema, 216
in hospital-acquired pneumonia, 213
in neutropenic pneumonia, 214
in transplant-related pneumonia, 214
Legionnaire’s disease, Legionella antibody levels in, 120
Leiden mutation (factor V mutation), 87
molecular diagnostic techniques for, 87, 373t
Leiomyomas, uterine, red cell volume in, 151
Leishmaniasis, Histoplasma capsulatum complement fixation antibody test in, 109
Leprosy
rapid plasma reagin test in, 150
rheumatoid factor levels in, 155
Venereal Disease Research Laboratory Test in, 178
Leptospira interrogans, Legionella antibody cross-reaction with, 120
Leptospirosis
cerebrospinal fluid profile in, 202
test selection in, in spirochetal meningitis, 202
Lesch-Nyhan syndrome, uric acid levels in, 177
Lesser occipital nerve, 341t
Leucovorin rescue therapy, 193t
Leukemia
bcr/abl translocation in, 57
CD4/CD8 ratio in, 68
cryoglobulin levels in, 82
ferritin levels in, 90
fetal hemoglobin levels in, 103
immunoelectrophoresis in, 112
leukocyte alkaline phosphatase levels in, 120
leukocyte count in, 121, 400t
nuclear antibody levels in, 131
phosphorus levels in, 138
platelet count in, 140
T cell receptor gene rearrangement in, 164
uric acid levels in, 177
vitamin B12 levels in, 180
Leukemoid reaction, leukocyte alkaline phosphatase levels in, 120
Leukocyte(s) (white blood cells)
ascitic fluid, in tuberculous peritonitis/enterocolitis, 227
fecal
in antibiotic-associated pseudomembranous colitis, 224
in HIV-associated diarrhea, 225
on Gram-stain smear, 28i
increase, and erythrocyte count, 85
peritoneal fluid, in peritonitis, 226
urethral, in urethritis, 233
urine, 395r–396t
cast of, 32, 387t
dipstick testing of, 30, 32t
in pyelonephritis, 232
on Wright-stained peripheral blood smear, 29i
Leukocyte alkaline phosphatase, blood levels of, 120
Leukocyte count, 400t
ascitic fluid, 365t–366t
in bacteremia of unknown source, 241
cerebrospinal fluid, 369r–371t
in aseptic meningitis, 199, 369t
in bacterial meningitis, 200, 369t
in carcinomatous meningitis, 370t
in cerebrospinal fluid, 370t–371t
in diabetic coma, 371t
in encephalitis, 198
in fungal meningitis, 201, 369t
in hepatic encephalopathy, 371t
in leptospirosis, 202
in neighborhood meningeal reaction, 371t
in neuroborreliosis, 202
in neurosyphilis, 202, 371t
in parasitic meningoencephalitis/meningitis, 203, 370t
in spirochetal meningitis, 202, 371t
in subarachnoid hemorrhage, 370t

in syphilitic meningitis, 202, 371
in tuberculous meningitis, 203, 369
in uremia, 371
hemoglobin levels affected by, 103
high
and mean corpuscular hemoglobin concentration, 124
and mean corpuscular volume, 124
in mucopurulent cervicitis, 235
pleural fluid, 382t–383t
in cirrhosis, 382t
in collagen vascular disease, 383t
in empyema, 216, 383t
in esophageal rupture, 383t
in heart failure, 382t
in malignancy, 382t
in nephrotic syndrome, 382t
in parapneumonic effusion, 383t
in pulmonary embolism/infarction, 383t
in rheumatoid arthritis, 383t
in tuberculosis, 371
and mean corpuscular hemoglobin concentration, 124
antistreptolysin O titer affected by, 55
beta, electrophoresis in detection of, 146
disorders of, laboratory and clinical characteristics of, 379t–380t
electrophoresis in detection of, 146
serum levels of, 71
in hyperlipidemia, 71, 379t–380t
α-Lipoprotein deficiency, triglyceride levels in, 172
Lipoprotein lipase deficiency
familial, triglyceride levels in, 172
in hyperchylomicronemia, 380t
Lipoproteinemia, protein electrophoresis in, 146
Liquid chromatography
for cyclosporine monitoring, 191t
for fetal hemoglobin measurement, 103
for hemoglobin A2 measurement, 101
Liquid plasma, 401t
Listeria spp., on Gram-stained smear, 27
Listeria monocytogenes, test selection for
in bacterial meningitis, 200
in chorioamnionitis/endometritis, 236
Listeriosis, heterophile agglutination (Monospot/Paul-Bunnell) test in, 107
Lithium
calcium levels affected by
serum, 63
urine, 65
magnesium levels affected by, 123
parathyroid hormone levels affected by, 134
therapeutic monitoring of, 187, 192t
urine osmolality affected by, 133
Liver. See also under Hepatic
abscess of
alanine aminotransferase (ALT) levels in, 46
aspartate aminotransferase levels in, 56
computed tomography in, 228, 268
test selection in, 228
cancer of
alanine aminotransferase (ALT) levels in, 46
aspartate aminotransferase levels in, 56
cholesterol levels in, 71
chorionic gonadotropin levels in, 72
computed tomography arterial portography in, 268
Liver (cont.)
cancer of (cont.)
computed tomography in, 268
α-fetoprotein levels in, 91
gamma-glutamyl transpeptidase in, 94
hepatic angiography in, 271
magnetic resonance imaging in, 260, 269
red cell volume in, 151
transcatheter embolitherapy for,
hepatic angiography in evaluation of, 271
ultrasound in, 258, 267
failure of
ammonia levels in, 51
blood urea nitrogen in, 60
fatty
magnetic resonance imaging in, 269
triglyceride levels in, 172
focal fatty infiltration, magnetic resonance imaging in, 269
focal nodular hyperplasia of
liver/spleen scan in, 271
magnetic resonance imaging in, 269
function of
in clotting factor deficiencies, 377t
laboratory tests evaluating, 378t
normal, 378t
and operative death rate after porto-
caval shunt (Child’s criteria), 372t
plasma protein concentration affected by, 147, 378t
imaging test selection and interpretation
in evaluation of, 267–271
injury of, lactate dehydrogenase levels
in, 117
liver/spleen scan in evaluation of, 271
transplantation of
hepatic angiography in preoperative
evaluation for, 271
ionized calcium levels affected by, 64
trauma to, hepatic angiography in, 271
Liver disease
alanine aminotransferase levels in, 46
albumin levels in, 47
alcoholic. See also Cirrhosis
gamma-glutamyl transpeptidase levels
in, 94
alkaline phosphatase levels in, 50, 228
ammonia levels in, 51
anemia in, 363t
α1-antiprotease levels in, 55
antithrombin III levels in, 56
aspartate aminotransferase levels in, 56
bilirubin levels in, 58, 378t
blood urea nitrogen in, 60
carcinoid embryonic antigen levels in, 67
ceruloplasmin levels in, 69
cholesterol levels in, 71
chorionic gonadotropin levels in, 72
complement C3 levels in, 75
computed tomography in, 259
cortisol levels in, 76
cryoglobulin levels in, 82
ferritin levels in, 90
α-fetoprotein levels in, 91
functional fibrinogen levels in, 92
gamma-glutamyl transpeptidase levels
in, 94
glucose levels in, 95
glucose tolerance test in, 96
glutamine levels in, 97
haptoglobin levels in, 99
hematocrit in, 101
hemoglobin levels in, 103
hemostatic function tests in, 377t
hypoglycemia and, 349i
IgA levels in, 113
IgG levels in, 113
insulin levels in, 115
lactate dehydrogenase isoenzyme levels
in, 117
lactate dehydrogenase levels in, 117
lactate levels in, 118
lidocaine levels affected in, 192t
lipase levels in, 121
mean corpuscular volume in, 124, 363t
metastatic
computed tomography in, 268
magnetic resonance imaging in, 269
mitochondrial antibody levels in, 129
nuclear antibody levels in, 131
pH in, 137
phenobarbital levels affected in, 193t
protein C levels in, 145
protein electrophoresis in, 146
protein levels in, 147, 378t
protein S antigen levels in, 147
prothrombin time in, 148
quinidine levels affected in, 194t
testosterone levels in, 165
total iron-binding capacity in, 116
triglyceride levels in, 172
ultrasound in, 258, 267
uric acid levels in, 177
Liver enzyme abnormalities, congenital, bilirubin levels in, 58
Liver function tests, 378t
Liver/spleen scan, 271
Long QT syndrome, 296, 327
Loop diuretics, lithium levels affected by, 192t
Low density lipoprotein (cholesterol), serum levels of, 71 in hyperlipidemia, 71, 379t
LPFB. See Left posterior fascicular block
LR. See Likelihood ratio
Lumbar puncture
for positron emission tomography, 247 traumatic, cerebrospinal fluid profile in, 370t
Lung. See also Pulmonary abcess of computed tomography in, 252 test selection in, 213 imaging test selection and interpretation in evaluation of, 253–255 Kaposi’s sarcoma of, HIV-associated pneumonia and, 214 Lung biopsy, in community-acquired pneumonia, 212 Lung cancer calcitonin levels in, 62 calcium levels in, 63 carcinoembryonic antigen levels in, 67 chorionic gonadotropin levels in, 72 computed tomography in, 252 parathyroid hormone-related protein levels in, 135 Lung capacity, total, 385t Lung disease cardiac troponin-I levels in, 174 interstitial computed tomography in, 252 partial pressure of oxygen in, 133 Lung maturity, fetal, lecithin/sphingomyelin ratio in estimation of, 119 Lung volumes, 385t
Lupus anticoagulant detection of inhibitor screen in, 114 partial thromboplastin time in, 136 Russell’s viper venom clotting time in, 157 in Q fever, 149 and thrombosis, 157 Lupus erythematosus cerebral, cerebrospinal fluid profile in, 370t discoid nuclear antibody levels in, 367t ribonucleoprotein antibody levels in, 155, 367t drug-induced, nuclear antibody levels in, 131, 367t heterophile agglutination (Monospot/Paul-Bunnell) test in, 107 systemic CD4/CD8 ratio in, 68 complement C3 levels in, 75 complement C4 levels in, 75 cryoglobulin levels in, 82 double-stranded DNA antibody levels in, 84, 367t IgG levels in, 113 neonatal, SS-A/Ro antibody in, 163 nuclear antibody levels in, 131, 367t rapid plasma reagin test in, 150 rheumatoid factor levels in, 155 ribonucleoprotein antibody levels in, 367t Smith antibody in, 159, 367t SS-A/Ro antibody in, 163, 368t SS-B/La antibody in, 163 synovial fluid sampling in, 389t Toxoplasma antibody test in, 171 Venereal Disease Research Laboratory Test in, 178 Luteinizing hormone, serum levels of, 122 in amenorrhea, 339i in hirsutism, 346i in infertility evaluation, 352i Luteoma, virilizing, testosterone levels in, 165 LVH. See Left ventricular hypertrophy Lyme disease bacterial/septic arthritis in, test selection in, 238 heterophile agglutination (Monospot/Paul-Bunnell) test in, 107 Lyme disease antibody test in, 122 myocarditis in, test selection in, 218 Lyme disease antibody test, 122 Lymphadenopathy cervical, magnetic resonance imaging in evaluation of, 248 hilar computed tomography in evaluation of, 252 magnetic resonance imaging in evaluation of, 253
Lymphadenopathy (cont.)
mediastinal, computed tomography in
evaluation of, 252
mesenteric, computed tomography in, 259
pelvic, magnetic resonance imaging in, 275
retroperitoneal
computed tomography in, 259
magnetic resonance imaging in, 260
Lymphangiectasia, intestinal
cholesterol levels in, 71
triglyceride levels in, 172
Lymphocyte count, 400t
Lymphocytes, 29i
CD4/CD8 ratio, 68
Lymphocytic choriomeningitis virus, test
selection in
in aseptic meningitis, 199
in encephalitis, 198
Lymphocytic myocarditis, test selection
in, 218
Lymphoid disorders
methylmalonic acid levels in, 126
β2-microglobulin levels in, 128
Lymphoid interstitial pneumonia, in
AIDS/HIV infection, test selection in, 214
Lymphoid malignancies. See also
Lymphoma
protein electrophoresis in, 146
Lymphoma
B cell immunoglobulin heavy chain
gene rearrangement in, 57
Burkitt’s, Epstein-Barr virus antibody
levels in, 85
calcium levels in, 347i
cryoglobulin levels in, 82
1,25-dihydroxy vitamin D3 levels in, 183
IgG levels in, 113
IgM levels in, 113
immunoelectrophoresis in, 112
lactate dehydrogenase levels in, 117
leukocyte count in, 121
T cell receptor gene rearrangement in,
164
uric acid levels in, 177
Lymphoproliferative disorders
cold agglutinin levels in, 74
leukocyte count in, 400t

M bands, Histoplasma capsulatum precip-
itin, 109
M-mode echocardiography, in valvular
heart disease, 398t–399t
Macroamylasemia, amylase levels in, 52
Macrocytes, 29i
Macrocytic anemia
diagnosis of, based on red blood cell
indices, 363t
hematocrit in, 101
hemoglobin levels in, 103
laboratory and clinical findings in, 363t
Macrocytosis, mean corpuscular hemoglo-in in, 123
α2-Macroglobulin, electrophoresis in
detection of, 146
Macroglobulinemia, Waldenström’s.
See Waldenström’s
Macroglobulinemia
Magnesium, serum levels of, 123
Magnesium deficiency
calcium levels in, 63
magnesium levels in, 123
Magnesium salts, magnesium levels
affected by, 123
Magnetic resonance angiography
in aorta evaluation, 280
in brain evaluation, 246
in neck evaluation, 248
Magnetic resonance cholangiography, in
cholangitis/cholecystitis, 229
Magnetic resonance imaging
in abdomen evaluation, 260
in amenorrhea, 339t
in bacterial/septic arthritis, 238
in brain abscess, 197
in brain evaluation, 246
in cellulitis, 240
in chest evaluation, 253
in Cushing’s syndrome, 340t
in encephalitis, 198
in genitourinary tract evaluation, 273
in head evaluation, 245
in infectious thrombophlebitis, 221
in liver evaluation, 269
in musculoskeletal system evaluation,
278
in neck evaluation, 248
in osteomyelitis, 237
in otitis externa, 207
in pelvis evaluation, 275
in pericarditis, 217
in salpingitis/pelvic inflammatory dis-
ease, 236
in sinusitis, 208
in spine evaluation, 277
Magnetic resonance venography, 246
Malabsorption
albumin levels in, 47
ceruloplasmin levels in, 69
cholesterol levels in, 71
fecal fat levels in, 88
25-hydroxy levels vitamin D₃ levels in, 182
phosphorus levels in, 138
protein levels in, 147
somatomedin C levels in, 162
triglyceride levels in, 172
vitamin B₁₂ levels in, 180
d-xylene absorption test in, 185
Malaria
rapid plasma reagin test in, 150
Venereal Disease Research Laboratory Test in, 178
Malassezia furfur
KOH preparation in identification of, 33–35
test selection for
in bacteremia of unknown source, 241
in infectious thrombophlebitis, 221
Malignancy. See also specific type or structure or organ affected
albumin levels in, 47
α₁-antiprotease levels in, 55
ascitic fluid profile in, 366t
B cell immunoglobulin heavy chain gene rearrangement in, 57
calcitonin levels in, 62
calcium levels in, 347i
serum, 63
urine, 65
carcinoembryonic antigen levels in, 67
cholesterol levels in, 71
chorionic gonadotropin levels in, 72
complement C4 levels in, 75
early antigen antibodies in, 85
erthrocyte sedimentation rate in, 86
ferritin levels in, 90
α-fetoprotein levels in, 91
glucose levels in, 95
haptoglobin levels in, 99
head and neck, magnetic resonance imaging in evaluation of, 248
hematologic, leukocyte count in, 121
with hypercalcemia, parathyroid hormone levels in, 134
IgG levels in, 113
iron-binding capacity in, 116
iron levels in, 115
lactate dehydrogenase levels in, 117
leukocyte count in, 400t
β₂-microglobulin levels in, 128
parathyroid hormone levels in, 134
parathyroid hormone-related protein levels in, 135
phosphorus levels in, 138
platelet count in, 140
pleural fluid profile in, 382t
protein electrophoresis in, 146
red cell volume in, 151
rheumatoid factor in, 368t
T cell receptor gene rearrangement in, 164
Malignant hyperthermia, creatine kinase levels in, 78
Malnutrition
albumin levels in, 47
ceruloplasmin levels in, 69
cholesterol levels in, 71
complement C3 levels in, 75
folic acid deficiency in, 363t
luteinizing hormone levels in, 122
phosphorus levels in, 138
somatomedin C levels in, 162
total iron-binding capacity in, 116
triglyceride levels in, 172
Mammography, 256
Mannitol, serum osmolality affected by, 132
Marbled-top tubes, 24, 42
Marrow space disease, magnetic resonance imaging in, 278
Mastoiditis, neighborhood meningeal reaction in, 371t
Maxillary sinus sampling, in sinusitis, 208
Maximum voluntary ventilation, 385t
MB isoenzyme, of creatine kinase in myocardial infarction, 79, 353i
serum levels of, 79
MCH. See Mean corpuscular hemoglobin
MCHC. See Mean corpuscular hemoglobin concentration
MCV. See Mean corpuscular volume
Mean corpuscular hemoglobin concentration, 123–124, 363t
Mean corpuscular volume, 124
in anemias, 124, 363r–364t
Measles
CD4/CD8 ratio in, 68
encephalitis after, test selection in, 198
Meconium, amniotic fluid contaminated by, lecithin/sphingomyelin ratio in, 119
Medial cutaneous nerve of arm, 342
Medial cutaneous nerve of forearm, 341–342
Medial femoral cutaneous nerve, 341–342
Medial plantar nerve, 341–342
Median nerve, 341–342
Mediastinal disease/mass
chest x-ray in, 252
computed tomography in, 252
magnetic resonance imaging in, 253
thyroid uptake and scan in, 250
Medullary sponge kidney, intravenous
pyelogram in, 273
Medullary thyroid carcinoma
calcitonin levels in, 62
MIBG (metaiodobenzyl-guanidine) in
evaluation of, 272
Megaloblastic anemia
fetal hemoglobin levels in, 103
lactate dehydrogenase isoenzyme levels in, 117
mean corpuscular volume in, 124
reticulocyte count in, 153
vitamin B₁₂ deficiency in, 93
Melanoma, urine color affected by, 30
Membranoproliferative glomerulonephritis, complement C3 levels in, 75
Mendelson’s syndrome, test selection in, 213
Meningeal Coccidioides infection, Coc-cidioides antibody test for, 74
Meningeal reaction, neighborhood, cerebrospinal fluid profile in, 371
Meningitis
aseptic
cerebrospinal fluid profile in, 199, 369
 test selection in, 199
bacterial
cerebrospinal fluid profile in, 200, 369
 test selection in, 200
carcinomatous, cerebrospinal fluid profile in, 370
cryptococcal antigen test in, 82, 201
fungal
cerebrospinal fluid profile in, 201, 369
 test selection in, 201
parasitic
cerebrospinal fluid profile in, 203, 370
 test selection in, 203
spirochetal
cerebrospinal fluid profile in, 202, 371
 test selection in, 202
syphilitic
cerebrospinal fluid profile in, 202, 371
 test selection in, 202
tuberculous
cerebrospinal fluid profile in, 203, 369
 test selection in, 203
Meningococcus. See Neisseria meningitidis
Meningoencephalitis, parasitic, test selection in, 203
Menkes’ disease, ceruloplasmin levels in, 69
Menopause
atrophic vaginitis after, 234
follicle-stimulating hormone levels in, 93
luteinizing hormone levels after, 122
osteoporosis after, 1,25-dihydroxy vitamin D₃ levels in, 183
uric acid levels in, 177
Mesenteric angiography, 261
Mesenteric disorders, computed tomography in, 259
Mesenteric ischemia, angiography in, 261
Meta-analysis, 20
Metabolic acidosis
carbon dioxide levels in, 66
chloride levels in, 70
laboratory characteristics and clinical features of, 362
lactate levels in, 118
nomogram for, 337
pH in, 137, 362
Metabolic alkalosis
carbon dioxide levels in, 66
chloride levels in, 70
laboratory characteristics and clinical features of, 362
nomogram for, 337
pH in, 137, 362
Metabolic disorders, bone scan in, 276
Metaiodobenzyl-guanidine (MIBG), 272
in pheochromocytoma evaluation, 272, 355
Metal smelters, lead poisoning in, 119
Metanephrines, urine, 125
in pheochromocytoma, 125, 355
Metformin
lactate levels affected by, 118
vitamin B₁₂ levels affected by, 180
Methadone
direct antiglobulin test affected by, 54
thyroid function tests affected by, 394t
Methanol
blood levels of, 125
pH affected by, 137
Methanol intoxication
methanol levels in, 125
osmolal gap in, 138, 381t
serum osmolality in, 132
Methemoglobin
blood levels of, 126
oxidant drugs and, 126
Methemoglobin reductase deficiency,
methemoglobin levels in, 126
Methemoglobinemia, red cell volume in,
151
Methenamine stain, of sputum or
bronchiolar samples, in immunocompromise-related
pneumonia, 214
Methotrexate, therapeutic monitoring of,
193t
Methyldopa
direct antiglobulin test affected by, 54
indirect antiglobulin test affected by, 54
plasma renin activity affected by, 152
prolactin levels affected by, 144
rheumatoid factor levels affected by, 155
urine color affected by, 30
Methylenetetrahydrofolate reductase deficiency,
373t
Methylmalonic acid, serum levels of, 126
Methotroplaxone, urine osmolality affected by,
133
Metyrapone test, 127, 338i
MHA-TP test (microhemagglutination-
Treponema pallidum test), 129, 391t
in spirochetal meningitis/neurosyphilis,
202
Methylation toxicity test, for HLA
typing, 110
Microsporidium spp., test selection for
in cholangitis/cholecystitis, 229
in HIV-associated diarrhea, 225
in keratitis, 205
in sinusitis, 208
Microsporum spp., KOH preparation in
identification of, 33–35
Midgut carcinoids, 5-hydroxy-indoleacetic
acid levels in, 111
Milk-alkali syndrome
calcium levels in, 63, 347i
phosphorus levels in, 138
Milk consumption, 25-hydroxy levels vita-
mim D₃ levels affected by, 182
Mineralocorticoid deficiency, renal tubular
acidosis in, 388t
Mineralocorticoid excess
carbon dioxide levels in, 66
in hypertension associated with
hypokalemia, 348i
Miners, lead poisoning in, 119
Minoxidil, plasma renin activity affected
by, 152
Mirror image dextrocardia, versus right-
left arm cable reversal in ECG,
327
Mitochondrial antibody, serum levels of,
129
Mitrval valve
disease, electrocardiographic findings
of, 301
prolapse, diagnostic evaluation of, 398t
regurgitation, diagnostic evaluation of,
398t
stenosis, diagnostic evaluation of, 398t
Mixed connective tissue disease
autoantibodies in, 367
nuclear antibody in, 131
ribonucleoprotein antibody in, 155, 367
Mixed hypertriglyceridemia, characteristics and laboratory findings in, 380

*Mobiluncus* spp., test selection for, in vaginitis/vaginosis, 234
Mobitz type II atrioventricular block, 287, 297
Molar pregnancy, chorionic gonadotropin levels in, 72
Molecular diagnosis, of genetic diseases, 373–376
Mollaret’s syndrome, test selection in, 199
Monoamine oxidase inhibitors
5-hydroxy-indoleacetic acid levels affected by, 111
metanephrine levels affected by, 125
vanillylmandelic acid levels affected by, 178
Monoclonal fluorescence polarization immunoassay, in cyclosporine monitoring, 191
Monoclonal gammopathies
protein electrophoresis in, 146
protein levels in, 147
of undetermined significance, immunoelectrophoresis in, 112
Monoclonal IgA, serum levels of, 113
Monoclonal IgG, serum levels of, 113
Monoclonal IgM, serum levels of, 113
Monoclonal paraprotein, immunoelectrophoresis in identification of, 112
Monocyte count, 400
Monocytes, 29i
Monocytic leukemia, vitamin B12 levels in, 180
Mononucleosis
CD4/CD8 ratio in, 68
classic signs of, 107
cold agglutinin levels in, 74
cytomegalovirus antibodies in, 83
Epstein-Barr virus antibody levels in, 85
gamma-glutamyl transpeptidase levels in, 94
heterophile agglutination test (Monospot/ Paul-Bunnell) in, 107
IgM levels in, 113
leukocyte count in, 400
microhemagglutination-*Treponema pallidum* (MHA-TP) test in, 129
mitochondrial antibody levels in, 129
nuclear antibody levels in, 131
pharyngitis in, test selection in, 209
rapid plasma reagin test in, 150
rheumatoid factor levels in, 155
smooth muscle antibody levels in, 160
Venereal Disease Research Laboratory Test in, 178
Monospot test, 107
Moonshine whiskey, lead poisoning caused by ingestion of, 119
*Moraxella* spp., test selection for, in keratitis, 205
*Moraxella catarrhalis*, test selection for in community-acquired pneumonia, 212
in laryngitis, 209
in laryngotracheobronchitis, 210
in otitis media, 206
in sinusitis, 208
*Moraxella lacunata*, test selection for, in conjunctivitis, 204
Morphine, and antiidiuretic hormone levels, 53
MRA. See Magnetic resonance angiography
MRI. See Magnetic resonance imaging
Mucopurulent cervicitis, test selection in, 235
laboratory evaluation of vaginal discharge, 235, 397
*Mucor* spp., test selection for, in neutropenic pneumonia, 214
MUGA (multigated acquisition) radionuclide ventriculography, 257
Multifocal atrial tachycardia, 287–288
irregularly irregular QRS rhythm in, 286
QRS duration in, 285
Multiform atrial rhythm atrioventricular block, QRS duration in, 285
Multigated acquisition radionuclide ventriculography, 257
Multinodular goiter, thyroglobulin antibody in, 166
Multiple endocrine neoplasia type II, genetic testing for, 62
Multiple myeloma. See Myeloma
Multiple sclerosis
IgG index in, 112
oligoclonal bands in, 131
Mumps
amylase levels in, 52
cold agglutinin levels in, 74
Mumps virus, test selection for in aseptic meningitis, 199
in encephalitis, 198
in epididymitis/orchitis, 233
in pericarditis, 217
Muscle contraction, potassium levels affecting, 143
Muscle damage/disorders
  creatine kinase levels in, 78
  lactate dehydrogenase isoenzyme levels in, 117
  lactate dehydrogenase levels in, 117
  troponin-I levels in, 174
Muscle mass, reduced, creatinine levels in, 80
Muscular dystrophy
  creatine kinase levels in, 78
  creatine kinase MB isoenzyme levels in, 79
Duchenne’s, tall R waves in right precordial leads with, 310
Muscular exertion, severe, creatine kinase levels in, 78
Musculoskeletal system
  imaging test selection and interpretation in evaluation of, 278
  lactate dehydrogenase levels in disease of, 117
MVV (maximum voluntary ventilation), 385
Myasthenia gravis
  acetylcholine receptor antibodies in, 45
  nuclear antibody levels in, 131
  pH in, 137
Myelia, on Gram-stained smear, 27
Mycobacterial pneumonia, test selection for, 215
*Mycobacterium* spp., test selection for
  in brain abscess, 197
  in endophthalmitis, 205
*Mycobacterium avium*, test selection for
  in HIV-associated pneumonia, 214
*Mycobacterium avium-intracellulare*, test selection for
  in bacteremia of unknown source, 241
  in HIV-associated cholangitis/cholecystitis, 229
  in HIV-associated diarrhea, 225
  in pneumonia, 215
*Mycobacterium kansasii*, test selection for
  in pneumonia, 215
*Mycobacterium marinum*, test selection for
  in bacterial/septic arthritis, 238
*Mycobacterium tuberculosis*, test selection for
  in empyema, 216
  in epididymitis/orchitis, 233
  in HIV-associated pneumonia, 214
  in infectious esophagitis, 222
  in laryngitis, 209
  in mycobacterial pneumonia, 215
  in otitis media, 206
  in tuberculous meningitis, 203
  in tuberculous pericarditis, 217
  in tuberculous peritonitis/enterocolitis, 227
*Mycoplasma* spp., test selection for
  in pericarditis, 217
*Mycoplasma genitalium*, test selection for
  in urethritis, 233
*Mycoplasma hominis*, test selection for
  in chorioamnionitis/endometritis, 236
  in salpingitis/pelvic inflammatory disease, 236
  in vaginitis/vaginosis, 234
*Mycoplasma pneumoniae*
  cold agglutinin levels in pneumonia caused by, 74
*Legionella* antibody cross-reaction with, 120
test selection for
  in community-acquired pneumonia, 212
  in infectious myocarditis, 218
  in laryngotracheobronchitis, 210
  in otitis media, 206
Mycotic infections, synovial fluid sampling in, 389
Myelodysplasia syndrome, CD4/CD8 ratio in, 68
Myelofibrosis
  leukocyte count in, 400
  with myeloid metaplasia, leukocyte alkaline phosphatase levels in, 120
  platelet count in, 140
Myelography, in osteomyelitis, 237
Myeloid metaplasia, myelofibrosis with, leukocyte alkaline phosphatase levels in, 120
Myeloma
  angiotensin-converting enzyme levels in, 52
  calcium levels in, 63, 347
  ionized, 64
cryoglobulin levels in, 82
IgA levels in, 113
Myeloma (cont.)
- IgG levels in, 113
- immunoelectrophoresis in, 112
- β₂-microglobulin levels in, 128
- protein electrophoresis in, 146
- uric acid levels in, 177

Myeloproliferative disorders
- leukocyte count in, 400
- platelet aggregation in, 139
- platelet count in, 140
- uric acid levels in, 177

Myocardial enzymes, after myocardial infarction, time course of, 353

Myocardial infarction
- anterior
  - acute injury in, 312–313
  - earliest findings in, 312–313
  - evolutionary changes in, 313
  - fully evolved pattern of, 313
  - hyperacute changes in, 312
  - pathologic Q waves in, 313
  - primary area, electrocardiography of, 312
  - primary process, electrocardiography of, 312–313
  - primary process, identification of lesion within artery, 316
  - QRS complex in, 318
  - ST segment elevation in, 312–313
- anterolateral (lateral), QRS complex in, 318
- apical
  - QRS complex in, 318
  - right axis deviation in, 306
- cardiac troponin-I levels in, 174, 353
- complement C3 levels in, 75
- creatine kinase levels in, 78
- creatine kinase MB isoenzyme levels in, 79, 353
- definition of, 310
- electrocardiography in, 310–320
- combination of all observations in, for final diagnosis, 311, 320
- determining age of infarction in, 311, 319–320
- identification of lesion within artery, 311, 315–316
- identification of presence and areas of injury in, 310–311, 311
- identification of primary area of involvement and culprit artery in, 310–315
- left posterior fascicular block in, 303
- low voltage of QRS complex in, 308
- poor R wave progression in, 309
- prolonged QT interval in, 327
- QRS complex in, 308, 311, 316–320, 318–319
- reciprocal changes in, 310, 316
- ST segment in, 310–316, 311, 319–320, 322–323
- steps for diagnosis in, 310–320
- test performance characteristics for, in diagnosis, 317–319, 318–319
- haptoglobin levels in, 99
- inferior
  - earliest findings in, 314
  - evolutionary changes in, 314
  - left axis deviation in, 305
  - primary area, electrocardiography of, 312
  - primary process, electrocardiography of, 314–315
  - primary process, identification of lesion within artery, 316
  - QRS complex in, 318
  - valvular heart disease in, diagnostic evaluation of, 399
- lactate dehydrogenase isoenzyme levels in, 117
- lactate dehydrogenase levels in, 117
- lateral, right axis deviation in, 306
- mimics of, 319, 320
- old or age-indeterminate, 319
- posterior
  - acute pattern of, 315
  - chronic pattern of, 315
  - electrocardiography of, 315
  - tall R waves in right precordial leads in, 309
- posterolateral, QRS complex in, 319
- right ventricular, 316
- electrocardiography of, 314–315
- risk stratification for electrocardiography in, 311, 315–316
- myocardial perfusion scan in, 257

Myocardial injury
- acute, in anterior myocardial infarction, 312–313
- anterior
  - ST segment depression or T wave inversion in, 322
  - ST segment elevation in, 321
- definition of, 310
- electrocardiography of, 310–320
- identifying presence and areas of injury in, 310–311, 311
- reciprocal changes in, 310
inferior
  ST segment depression or T wave inversion in, 322
ST segment elevation in, 321
lactate dehydrogenase levels in, 117
posterior, 311
  acute pattern of, 315
chronic pattern of, 315
electrocardiography of, 315
ST segment depression or T wave inversion in, 322
right ventricular, 311, 316
electrocardiography of, 314–315
Myocardial ischemia
  definition of, 310
electrocardiographic findings in, 310–320, 323, 324, 327
myocardial perfusion scan in, 257
radionuclide ventriculography in, 257
Myocardial perfusion scan, 257
Myocarditis
creatine kinase levels in, 78
infectious, test selection in, 218
Myoglobinuria, urine color affected by, 30
Myopathy, cardiac troponin-I levels in, 174
Myositis, cardiac troponin-I levels in, 174
Myxedema
  and low-voltage QRS complex in ECG, 308
sodium levels in, 161
thyroperoxidase antibody in, 167
Myxomatous degeneration, valvular heart disease in, diagnostic evaluation of, 398
N
N. gonorrhoeae. See Neisseria gonorrhoeae
N. meningitidis. See Neisseria meningitidis
Na. See Sodium
Naegleria spp., test selection in, 203
Naegleria fowleri, test selection for, in parasitic meningoencephalitis, 203
Nalidixic acid, glucose-6-phosphate dehydrogenase deficiency and, 97
Nasal sampling, in sinusitis, 208
Nasogastric suction, phosphorus levels affected by, 138
Nasopharyngeal carcinoma, Epstein-Barr virus antibody levels in, 85
Nasopharyngeal sampling
  in laryngotracheobronchitis, 210
  in otitis media, 206
Navy-top tubes, 42
Neck
  abscess, computed tomography in evaluation of, 249
  imaging test selection and interpretation in evaluation of, 248–249
  masses, staging of
    computed tomography in, 249
    magnetic resonance imaging in, 248
  x-ray of, in epiglottitis, 211
Necrosis, tissue, lactate dehydrogenase levels in, 117
Necrotizing enterocolitis, test selection in, 223
Necrotizing fasciitis, test selection in, 240
Needle aspiration
  of abdominal lesions, ultrasound-guided, 258
  of neck lesions
    computed tomography-guided, 249
    ultrasound-guided, 249
  pericardial, in tuberculous pericarditis, 217
  of perinephric abscess, 232
  of thyroid nodule, 361
  transthoracic
    in anaerobic pneumonia or lung abscess, 213
    in community-acquired pneumonia, 212
    in mycobacterial pneumonia, 215
Needle biopsy
  bone, in osteomyelitis, 237
  thyroid, in thyroid nodule evaluation, 361
Needle stick precautions, 24
Neighborhood meningeal reaction, cerebrospinal fluid profile in, 371
Neisseria spp., on Gram-stained smear, 27
Neisseria gonorrhoeae, test selection for in bacterial/septic arthritis, 238
  synovial fluid sampling in, 390
  in conjunctivitis, 204
  in epiddidymitis/orchitis, 233
  in mucopurulent cervicitis, 235
  laboratory evaluation of vaginal discharge, 397
  in pharyngitis, 209
  in salpingitis/pelvic inflammatory disease, 236
  in urethritis, 233
  in urinary tract infection/cystitis/pyruria-dysuria syndrome, 230
Neisseria meningitidis, test selection for  
in bacteremia of unknown source, 241  
in bacterial meningitis, 200  
in brain abscess, 197  
in community-acquired pneumonia, 212  
in pericarditis, 217

Neomycin  
ammonia levels affected by, 51  
urinary calcium levels affected by, 65

Neonatal isoimmune thrombocytopenia  
platelet-associated IgG in, 141  
platelet count in, 140

Neonatal jaundice, bilirubin levels in, 58

Neonatal purpura fulminans, protein C  
deficiency and, 145

Neonate. See Newborn

Neonatorum impetigo, test selection in, 239

Neoplasms. See also Malignancy  
mitochondrial antibody levels in, 129  
phosphorus levels in, 138  
red cell volume in, 151  
synovial fluid sampling in, 390  
T cell receptor gene rearrangement in, 164  
uric acid levels in, 177

Nephrectomy, renal function prior to, 274

Nephritis  
cryoglobulin causing, 82  
urine indices in, 387

Nephrocalcinosis, renal tubular acidosis in, 388

Nephrogenic diabetes insipidus  
antidiuretic hormone levels in, 53  
serum osmolality in, 132

Nephropathy  
lead, uric acid levels in, 177  
salt-losing  
chloride levels in, 70  
sodium levels in, 161

Nephrosclerosis, renal tubular acidosis in, 388

Nephrosis  
peritonitis associated with, test selection in, 226  
quiniidine levels affected in, 194

Nephrotic syndrome  
albumin levels in, 47  
α1-antiprotease levels in, 55  
ascitic fluid profile in, 365  
blood urea nitrogen in, 60  
calcium levels in  
ionized, 64  
urine, 65  
ceruloplasmin levels in, 69  
chloride levels in, 70  
cholesterol levels in, 71  
cortisol levels in, 76  
25-hydroxy vitamin D3 levels in, 182  
hyperlipidemia in, 379  
iron levels in, 115  
plasma renin activity in, 152  
pleural fluid profile in, 382  
protein electrophoresis in, 146  
protein levels in, 147  
protein S antigen levels in, 147  
renal tubular acidosis in, 388  
sodium levels in, 161  
thyroid function tests in, 394  
total iron-binding capacity in, 116  
triglyceride levels in, 172  
urine characteristics in, 395

Nephrotoxic drugs  
blood urea nitrogen levels affected by, 60  
creatinine clearance affected by, 81  
creatinine levels affected by, 80

Nerve root distribution, 341–342

Neural tube defects, α-fetoprotein screening for, 91

Neuroblastoma  
metanephrine levels with, 125  
MIBG (metaiodobenzyl-guanidine) in evaluation of, 272  
vanillylmandelic acid levels with, 178

Neuroborreliosis  
cerebrospinal fluid profile in, 202  
test selection in, 202

Neurologic dysfunction. See Central nervous system disorders

Neuromuscular disease  
partial pressure of oxygen in, 133  
pH in, 137

Neuromuscular irritability, potassium levels affecting, 143

Neuropathic arthropathy, synovial fluid profile in, 389–390

Neuropsychiatric disorders, vitamin B12 levels in, 180

Neurosurgery, meningitis after, test selection in, 200

Neurosyphilis  
cerebrospinal fluid profile in, 202, 371  
IgG index in, 112
Index

475

oligoclonal bands in, 131
test selection in, 202
Venereal Disease Research Laboratory
Test in, 179
Neutropenia
bacteremia of unknown source and, test
selection in, 241
granulocyte transfusion for, 402
leukocyte count in, 400
pneumonia in, test selection in, 214
Neutrophil, 29
band, 29
hypersegmented, 29
leukocyte alkaline phosphatase levels
in, 120
with toxic granulations, 29
Neutrophil count, 400
absolute, in bacteremia of unknown
source, 241
Neutrophil cytoplasmic antibodies, serum
levels of, 130, 368
Newborn
calcitonin levels in, 62
cytomegalovirus antibodies in, 83
haptoglobin levels in, 99
hemolytic disease of
direct antiglobulin test in, 54
fetal hemoglobin levels in, 103
glucose-6-phosphate dehydrogenase
deficiency and, 97
Rh grouping in prevention of, 154
mean corpuscular volume in, 124
premature, necrotizing enterocolitis in,
223
test selection in disorders in
bacteremia of unknown source, 241
bacterial meningitis, 200
community-acquired pneumonia, 212
conjunctivitis, 204
empyema, 216
otitis media, 206
Nicotine, and antidiuretic hormone levels,
53
Nicotinic acid
glucose tolerance test affected by, 96
triglyceride levels affected by, 172
uric acid levels affected by, 177
Nifedipine, plasma renin activity affected
by, 152
Nipple stimulation, prolactin levels in, 144
Nitrates, methemoglobin levels affected
by, 126
Nitrites
methemoglobin levels affected by, 126
urine, 395t–396t
in urine, dipstick testing of, 30, 32
Nitrofurantoin, glucose-6-phosphate dehy-
drogenase deficiency and, 97
Nitrogen, blood urea. See Blood urea
nitrogen
Nitroprusside test, for acetoacetate, 45
Nocardia spp., test selection for
in brain abscess, 197
in community-acquired pneumonia, 212
in transplant-related pneumonia, 214
Nomogram, 13, 14. See also specific
nomograms
Non-A/non-B hepatitis, hepatitis C anti-ody levels in, 106
Non-steroidal anti-inflammatory drugs
and aseptic meningitis, 199
potassium levels affected by, 143
Nongonococcal urethritis, test selection in,
233
Nonhistone proteins, antibodies to
(nuclear antibody), serum levels
of, 131
Nonketotic hyperosmolar hyperglycemia
coma, serum osmolality in, 132
Nontropical sprue, 5-hydroxy-indoleacetic
acid levels in, 111
Normal values
in ascitic fluid sampling, 365t
in cerebrospinal fluid sampling, 369t
for pleural fluid sampling, 382t
reference range for, 5–6, 6t, 6i
for synovial fluid sampling, 389t
in thyroid function tests, 393t
for urine composition, 395t
for vaginal discharge, 397t
Normochromic anemia
diagnosis of, based on red blood cell
indices, 363t
laboratory and clinical findings in, 363t
Normocytic anemia
diagnosis of, based on red blood cell
indices, 363t
hematocrit in, 101
hemoglobin levels in, 103
laboratory and clinical findings in, 363t
Normovolemia, hyponatremia with, serum
osmolality in, 132
Nortriptyline, therapeutic monitoring of,
193t
Norwalk agent, test selection for, in infec-
tious colitis/dysentery, 223
Nuclear antibody, serum levels of, 131,
367t
Nucleic acid assay
in mucopurulent cervicitis, 235
in urethritis, 233

Nursing (breastfeeding, lactation)
1,25-dihydroxy vitamin D₃ levels in,
prolactin levels in, 144
Nutrition, and operative death rate after
portocaval shunt (Child’s criteria), 372

Nutrition, inadequate. See Malnutrition

O

Obesity
dexamethasone suppression test in, 83
insulin levels in, 115
and low-voltage QRS complex in ECG, 308
triglyceride levels in, 172
Obstipation therapy, Hypaque enema in, 264

Obstructive pulmonary disease. See also
Chronic obstructive pulmonary
disease
angiotensin-converting enzyme levels
in, 52
pulmonary function tests in, 385

Obturator nerve, 341–342

Occupational exposures, and lead poisoning, 119

Ocular swabs, in conjunctivitis, 204

Odd(s)
converting to probability, 15, 15
posttest, 15, 15
Odds-likelihood ratios, 11–16, 13r, 14i–15i

Oligoclonal bands, serum and cerebrospinal fluid levels of, 131

Omeprazole
gastrin levels affected by, 95
vitamin B₁₂ levels affected by, 180
Onchocerca volvulus, test selection for, in
keratitis, 205

Ophthalmia neonatorum, test selection in, 204

Opiates/opioids
partial pressure of oxygen affected by, 133
prolactin levels affected by, 144
thyroid function tests affected by, 394

Opisthorchis felineus, test selection for, in
cholangitis/cholecystitis, 229

Opisthorchis viverrini, test selection for,
in cholangitis/cholecystitis, 229

Oral contraceptives
antithrombin III levels affected by, 56
ceruloplasmin levels affected by, 69
factor VIII assay affected by, 88
follicle-stimulating hormone levels
affected by, 93
functional fibrinogen levels affected by, 92
glucose tolerance test affected by, 96
hyperlipidemia with, 379
iron levels affected by, 115
luteinizing hormone levels affected by, 122
phosphorus levels affected by, 138
plasma renin activity affected by, 152
protein electrophoresis affected by, 146
sodium levels affected by, 161
testosterone levels affected by, 165
thyroid function tests affected by, 394
total iron-binding capacity affected by, 116
triglyceride levels affected by, 172
triglycerides affected by, 379

Orchidectomy, testosterone levels in, 165

Orchitis
mumps, cold agglutinin levels in, 74
test selection in, 233

Organophosphate insecticides, electrocardiography affected by, 326

ORT (orthodromic reciprocating tachycardia), 299

Orthodromic reciprocating tachycardia
(ORT), 299

Osborn wave, in hypothermia, 328

Osler-Weber-Rendu syndrome, brain
abscess with, 197

Osmal gap, 132, 381

Osmolality
serum, 132
ethanol and, 87, 132
in hyponatremia, 132, 350
urine, 133
normal random, with average fluid
intake, 133
in renal failure/disease, 387

Osmostat
defective, serum osmolality in, 132
reset, sodium levels in, 161

Osmotic diuresis
potassium levels in, 143
serum osmolality in, 132

Osteitis deformans, urine calcium levels
in, 65

Osteitis deformans, urine calcium levels
in, 65
Osteoarthritis
  angiotensin-converting enzyme levels
  in, 52
  25-hydroxy vitamin D$_3$ levels in, 182
  synovial fluid sampling in, 389–390
Osteoarthropathy, synovial fluid sampling in, 389
Osteoblastic metastases
  phosphorus levels in, 138
  urinary calcium levels in, 65
Osteochondritis dissecans, synovial fluid sampling in, 389
Osteochondromatosis, synovial fluid sampling in, 389
Osteodystrophy, renal, renal tubular acidosis in, 388
Osteogenic sarcoma, alkaline phosphatase levels in, 50
Osteolytic malignancy
  calcium levels in, 65, 347
  phosphorus levels in, 138
Osteomalacia
  alkaline phosphatase levels in, 50
  calcium levels in, urine, 65
  25-hydroxy levels vitamin D$_3$ levels in, 182
  phosphorus levels in, 138
Osteomyelitis
  bone scan in, 276
  erythrocyte sedimentation rate in, 86
  leukocyte scan in, 281
  test selection in, 237
Osteophytic spurring, computed tomography in evaluation of, 277
Osteoporosis
  postmenopausal, 1,25-dihydroxy vitamin D$_3$ levels in, 183
  urinary calcium levels in, 65
Otitis externa, test selection in, 207
Otitis media
  brain abscess with, 197
  test selection in, 206
Otorrhea, cerebrospinal fluid, cisternography in evaluation of, 247
Outflow tract defect, amenorrhea in, 339
Outlet obstruction, evaluation of gastric emptying study in, 265
  upper GI study in, 262
Ovalocytes, 29
Ovarian agenesis, follicle-stimulating hormone levels in, 93
Ovarian failure
  amenorrhea in, 339
  follicle-stimulating hormone levels in, 93
Ovarian tumors/mass
  chorionic gonadotropin levels with, 72
  α-fetoprotein levels in, 91
  hirsutism with, 346
  palpable, ultrasound in evaluation of, 275
  virilizing, testosterone levels in, 165
Overhydration, hypernatremia with, serum osmolality in, 132
Ovine corticotropin-releasing hormone stimulation test, 84
Oxidant drugs
  glucose-6-phosphate dehydrogenase deficiency and, 97
  hemosiderin levels affected by, 104
  methemoglobin levels affected by, 126
Oximetry, pulse, 36–38
  approach to patient for, 37
  contraindications to, 37
  indications for, 36
  technique for, 37–38
Oxygen, partial pressure of (oxygen tension), 133
Oxygen saturation, pulse oximetry for measurement of, 36–38
Oxygen therapy, partial pressure of oxygen affected by, 133

P
P. marneffei, test selection for, in HIV-associated pneumonia, 214
P wave
  in atrial rhythms, 287, 288
  in atroventricular block, 297
  in atroventricular dissociation, 298
  in atroventricular reentry tachycardia, 299
  in chronic obstructive pulmonary disease, 330
  in junctional rhythms, 288
  in multifocal atrial tachycardia, 287–288
  in right atrial enlargement, 300
  in sinus rhythms, 287, 287
  p24 antigen test, HIV antibody test and, 110
Pacemaker
  primary, slowing of, 298
  subsidiary, acceleration of, 298
  ventricular, ST segment elevation in, 321
Paget’s disease
  alkaline phosphatase levels in, 50
Paget’s disease (cont.)
calcium levels in
  serum, 63
  urine, 65
Paint, lead poisoning caused by ingestion of, 119
Paint manufacturers, lead poisoning in, 119
Paired sera. See also Complement fixation test
  in aseptic meningitis, 199
  in community-acquired pneumonia, 212
  in encephalitis, 198
  in infectious myocarditis, 218
  in laryngotracheobronchitis, 210
  in pericarditis, 217
Pancreas. See also Islet cell disease;
  Pancreatitis
  amylase levels in disorders of, 52
  cancer of
    amylase levels in, 52
    calcitonin levels in, 62
    carcinoembryonic antigen levels in, 67
    chorionic gonadotropin levels in, 72
    computed tomography in, 259, 272
    α-fetoprotein levels in, 91
    lipase levels in, 121
    fecal fat levels in disorders of, 88
    glucose levels in disorders of, 95
    glucose tolerance test in disorders of, 96
    imaging test selection and interpretation in evaluation of, 267, 272
    lipase levels in disorders of, 121
    D-xylose absorption test in disorders of, 185
  Pancreatectomy, C-peptide levels in, 62
  Pancreatic B cell destruction, insulin antibody levels and, 114
  Pancreatic duct obstruction, amylase levels in, 52
  Pancreatic ductal dilation, ultrasound in evaluation of, 272
  Pancreatic pseudocyst
    amylase levels in, 52
    lipase levels in, 121
    ultrasound in evaluation of, 258, 272
Pancreatitis
  amylase levels in, 52
  ascitic fluid profile in, 366t
  carcinoembryonic antigen levels in, 67
  computed tomography in, 259, 272
  endoscopic retrograde cholangiopancreatography in, 267
  glucose levels in, 95
  cerebrospinal fluid, 383t
  in hyperlipidemia, 379t–380t
  lipase levels in, 121
  magnesium levels in, 123
  pleural fluid profile in, 383t
  Ranson’s criteria for severity of, 386t
  serum osmolality in, 132
  triglyceride levels in, 172, 379t
  vitamin B₁₂ absorption test (Schilling’s test) in, 181
  Panencephalitis, subacute sclerosing
    IgG index in, 112
    oligoclonal bands in, 131
  Panhypogonadism, infertility caused by, 352t
  Papillary necrosis, genitourinary tract, intravenous pyelogram in evaluation of, 273
  Papovavirus, test selection for, in encephalitis, 198
  Paradrenal tumor, 355t
  Parainfluenza virus, test selection for in laryngitis, 209
    in laryngotracheobronchitis, 210
    in sinusitis, 208
  Paraldehyde, serum osmolality affected by, 132
  Analysis, hyperkalemic familial periodic, potassium levels in, 143
  Paraneoplastic vasculitis, neutrophil cytoplasmic antibody levels in, 130
  Parapneumonic effusion, pleural fluid profile in, 383t
  Paraprotein
    monoclonal, immunoelectrophoresis in identification of, 112
    quantitation of, 113
  Paraproteinemia
    erythrocyte sedimentation rate in, 86
    platelet aggregation in, 139
  Parasitic infection
    IgG levels in, 113
    IgM levels in, 113
    leukocyte count in, 400t
    test selection for, in bacteremia of unknown source, 241
  Parasitic keratitis, test selection in, 205
  Parasitic meningoencephalitis/meningitis cerebrospinal fluid profile in, 203, 370t
    test selection in, 203
  Parathyroid adenoma, evaluation of magnetic resonance imaging in, 248
    parathyroid scan in, 251
  Parathyroid gland
    imaging test selection and interpretation in evaluation of, 251
in phosphorus regulation, 138
ultrasound in evaluation of, 249
Parathyroid hormone
calcium levels affected by, 63, 65, 134, 347i, 354i
serum levels of, 134, 347i
in hypercalcemia, 347i
serum calcium levels and, 63, 134, 347i
Parathyroid hormone-related protein
calcium levels affected by, 63, 135
plasma levels of, 135
Parathyroid scan, 251
Parathyroid surgery, magnesium levels after, 123
Parinaud's oculoglandular syndrome, test selection in, 204
Parotitis, amylase levels in, 52
Paroxysmal nocturnal hemoglobinuria
complement C3 levels in, 75
hemosiderin levels in, 104
leukocyte alkaline phosphatase levels in, 120
renal tubular acidosis in, 388t
Paroxysmal supraventricular tachycardia (PSVT), 298–299
Partial thromboplastin time, activated, 136, 377t
inhibitor screen in evaluation of, 114
Paternity testing, HLA typing in, 110
Patient preparation, for testing, 3
Paul-Bunnell test, 107
PCO2, and acid-base status, 66, 137, 362t
PCP. See Pneumocystis carinii, pneumonia
PCR. See Polymerase chain reaction
Peak expiratory flow rate (PEFR), 385t
PEFR (peak expiratory flow rate), 385t
Pelvic inflammatory disease
erthrocyte sedimentation rate in, 86
test selection in, 236
Pelvic pain, ultrasound in evaluation of, 275
Pelvic thrombophlebitis
postpartum or post-abortion, test selection in, 221
puerperal sepsis, test selection in, 221
Pelvis
computed tomography of, in hirsutism, 346i
imaging test selection and interpretation in evaluation of, 275
Penicillin, direct antiglobulin test affected by, 54
Pentagastrin, in medullary thyroid carcinoma, 62
Pentamidine, electrocardiography affected by, 326
Peptic ulcer disease
alkaline phosphatase levels in, 50
amylase levels in, 52
carcinoembryonic antigen levels in, 67
fecal occult blood in, 89
gastrin levels in evaluation of, 95
Helicobacter pylori antibody levels in, 100
Peptostreptococcus spp., test selection for in anaerobic pneumonia or lung abscess, 213
in bacterial/septic arthritis, 238
in osteomyelitis, 237
in salpingitis/pelvic inflammatory disease, 236
in sinusitis, 208
in vaginitis/vaginosis, 234
Percutaneous transhepatic biliary drainage, 270
Percutaneous transhepatic cholangiogram, 270
Percutaneous transthoracic needle aspiration, in anaerobic pneumonia or lung abscess, 213
Perforation
bowel
Hypaque enema in, 264
peritonitis associated with, test selection in, 226
upper GI study in, 262
gastric, lipase levels in, 121
Pericardial biopsy
in pericarditis, 217
in tuberculous pericarditis, 217
Pericardial drainage, surgical, in pericarditis, 217
Pericardial effusion, and low-voltage QRS complex in ECG, 308
Pericardial fluid sampling
in pericarditis, 217
in tuberculous pericarditis, 217
Pericardial needle aspiration, in tuberculous pericarditis, 217
Pericarditis
electrocardiographic findings in, 329
versus early repolarization, 329
ST segment elevation, 321t, 329
tall R waves in right precordial leads, 310
test selection in, 217
tuberculous, test selection in, 217
Pericholecystic fluid, ultrasound in evaluation of, 258, 266
Perihepatic infection, leukocyte scan in, 281
Perinephric abscess, test selection in, 232
Peripancreatic fluid, ultrasound in evaluation of, 258, 272
Peripheral blood smear
common findings on, 29
Wright stain of, 27–28
Peripheral nerve distribution, 341–342
Peripheral vascular disease, angiography in, 279
Peristalsis, upper GI study in evaluation of, 262
Peritoneal dialysis, chronic ambulatory, peritonitis associated with, test selection in, 226
Peritoneal fluid sampling. See also Ascitic fluid sampling in peritonitis, 226
Peritonitis
amylase levels in, 52
ascitic fluid profile in, 365
computed tomography in, 259
lipase levels in, 121
serum osmolality in, 132
test selection in, 226
tuberculous
ascitic fluid profile in, 227, 365

test selection in, 227
Pernicious anemia
calcitonin levels in, 62
gastrin levels in, 95
iron levels in, 115
methylmalonic acid levels in, 126
remission of, iron levels in, 115
thyroglobulin antibody in, 166
vitamin B₁₂ absorption test (Schilling’s test) in, 181
vitamin B₁₂ levels in, 180
Peroral pneumocolon, 263
Pertussis
leukocyte count in, 400

test selection in, 210
Pertussis vaccine, encephalitis after, test selection for, 198
PET. See Positron emission tomography pH
blood, 137
in acid-base disorders, 137
chloride levels and, 70
ionized calcium levels affected by, 64
total carbon dioxide levels and, 66
peritoneal fluid, in peritonitis, 226
pleural fluid, in empyema, 216
urine
dipstick testing of, 30, 31
in renal tubular acidosis, 31, 388
vaginal, in vaginitis/vaginosis, 234
vaginal fluid, 397
Pharmacokinetic parameters, and therapeutic drug monitoring, 189–190
Pharyngitis
streptococcal, antistreptolysin O titer in, 55
test selection in, 209
Phenacetin
direct antiglobulin test affected by, 54
glucose-6-phosphate dehydrogenase deficiency and, 97
methemoglobin levels affected by, 126
Phenazopyridine, urine color affected by, 30
Phenformin, and lactate levels, 118
Phenobarbital
25-hydroxy vitamin D₃ levels affected by, 182
primidone levels affected by, 193
therapeutic monitoring of, 193
Phenothiazines
electrocardiography affected by, 326
5-hydroxy-indoleacetic acid levels affected by, 111
leukocyte count affected by, 121, 400
luteinizing hormone affected by, 122
prolactin levels affected by, 144
Phenytoin
antidiuretic hormone levels affected by, 53
ceruloplasmin levels affected by, 69
dexamethasone suppression test affected by, 83
free thyroxine index affected by, 170
free thyroxine levels affected by, 170
gamma-glutamyl transpeptidase levels affected by, 94
glucose levels affected by, 95
glucose tolerance test affected by, 96
heterophile agglutination (Monospot/Paul-Bunnell) test affected by, 107
25-hydroxy vitamin D₃ levels affected by, 182
mean corpuscular volume affected by, 124
therapeutic monitoring of, 193
thyroid function tests affected by, 394
total thyroxine levels affected by, 169
Pheochromocytoma
diagnostic algorithm for, 355i
erythropoietin levels with, 86
glucose tolerance test in, 96
MIBG (metaiodobenzyl-guanidine) in
evaluation of, 272, 355i
urinary metanephrine levels with, 125,
355i
urinary vanillylmandelic acid levels with,
178
Philadelphia chromosome, 57
Phosphate-binding antacids, phosphorus
levels affected by, 138
Phosphate infusions, phosphorus levels
affected by, 138
Phospholipase A2, in Russell’s viper
venom clotting time, 157
Phosphorus/phosphates
parathyroid hormone levels affecting,
134
parathyroid hormone-related protein
affecting, 135
serum levels of, 138
urinary color affected by, 30
PID. See Pelvic inflammatory disease
Pineapple, 5-hydroxy-indoleacetic acid
levels affected by, 111
Pituitary disorders/insufficiency/failure
amenorrhea in, 339i
cosyntropin stimulation test in, 77
follicle-stimulating hormone levels in,
93
glucose levels in, 95
glucose tolerance test in, 96
growth hormone affected by, 99
hypothyroidism in, 168, 351i, 393t
luteinizing hormone levels in, 122
serum insulin levels in, 115
serum sodium levels in, 161
somatomedin C levels in, 162
Pituitary dwarfism
growth hormone levels in, 99
somatomedin C levels in, 162
Pituitary tumors
amenorrhea with, 339i
prolactin levels with, 144
Plasma
fresh-frozen, 401t
liquid, 401t
thawed, 401t
Plasma renin activity, 152
in hypertension, 152, 348i
plasma aldosterone and, 48
urine aldosterone and, 49
Plasmodium spp., test selection for, in
bacteremia of unknown source, 241
Platelet(s), 29i
Platelet aggregation, 139, 377t
Platelet antibodies, platelet count affected
by, 140
Platelet-associated IgG, 141
Platelet count, 140, 377t
Platelet disorders
activated clotting time in, 73
bleeding time in, 59, 377t
blood urea nitrogen in, 377t
creatinine in, 377t
hemostatic function tests in, 377t
platelet aggregation in, 139, 377t
Platelet release reaction, defects in,
platelet aggregation in, 139
Platelet transfusion, 402t
Pleur tic disease, chest x-ray in, 252
Pleural fluid profiles, in various disease
states, 382r–383t
Pleural fluid sampling
in cirrhosis, 382t
in collagen vascular disease, 383t
in community-acquired pneumonia, 212
in empyema, 216, 383t
in esophageal rupture, 383t
in heart failure, 382t
in malignancy, 382t
in nephrotic syndrome, 382t
normal values in, 382t
in pancreatitis, 383t
in parapneumonic effusion, 383t
in pulmonary embolism, infarction, 383t
in rheumatoid arthritis, 383t
specimen handling for, 26t
in tuberculosis, 382t
Plums, 5-hydroxy-indoleacetic acid
levels affected by, 111
Pneumatoceles, in community-acquired
pneumonia, 212
Pneumococcus. See Streptococcus
pneumoniae
Pneumoconiosis, angiotensin-converting
enzyme levels in, 52
Pneumocystis carinii
Giemsa stain of, 28i
pneumonia
lactate dehydrogenase levels in, 117
test selection for, 214
in AIDS-related pneumonia, 214
sputum sampling in, 28i
in transplant-related pneumonia,
214
Pneumonectomy, evaluation of candidates for, ventilation-perfusion scan in, 253

Pneumonia
anaerobic, test selection in, 213
aspiration
esophageal reflux study in, 264
test selection in, 212–213
atypical, test selection in, 212
community-acquired, test selection in, 212
hospital-acquired, test selection in, 213
in immunocompromised host, test selection in, 214
Legionella, Legionella antibody levels in, 120
mycobacterial, test selection in, 215
Mycoplasma
cold agglutinin levels in, 74
Legionella antibody cross-reaction with, 120
test selection for, 206, 210, 212, 218
neutropenic, test selection in, 214
partial pressure of oxygen in, 133
Pneumocystis carinii
lactate dehydrogenase levels in, 117
test selection for, 28i, 214
transplant-related, test selection in, 215
Pneumoperitoneum, evaluation of, abdominal x-ray in, 258
Pneumothorax
chest x-ray in evaluation of, 252
left, as electrocardiographic mimic of myocardial infarction, 320t
Poisons
electrocardiography affected by, 326
and renal tubular acidosis, 388t
Polio virus, test selection for
in aseptic meningitis, 199
in encephalitis, 198
Polyarteritis nodosa
angiography in, 279
anti-neutrophil cytoplasmic antibody in, 368t
cryoglobulin levels in, 82
leukocyte count in, 400t
mesenteric angiography in, 261
Polyclonal gammopathies
protein electrophoresis in, 146
protein levels in, 147
Polyclonal IgA, serum levels of, 113
Polyclonal IgG, serum levels of, 113
Polyclonal IgM, serum levels of, 113
Polycystic kidney disease, erythropoietin levels in, 86
Polycystic ovary syndrome
hirsutism with, 346i
luteinizing hormone levels in, 122
Polycythemia
erthrocyte count in, 85
erthrocyte sedimentation rate in, 86
erythropoietin levels in, 86
hematocrit in, 101
hemoglobin levels in, 103
red cell volume in, 151
vera
erthrocyte count in, 85
erthrocyte sedimentation rate in, 86
lactate dehydrogenase levels in, 117
leukocyte alkaline phosphatase levels in, 120
platelet count in, 140
red cell volume in, 151
uric acid levels in, 177
vitamin B12 levels in, 180
Polydipsia
antidiuretic hormone levels in, 53
urine osmolality in, 133
Polymerase chain reaction
for chlamydial DNA, in conjunctivitis, 204
for cystic fibrosis mutation, 373t
for cytomegalovirus genome, in encephalitis, 198
in endomyocardial biopsy, in infectious myocarditis, 218
for enterovirus
in aseptic meningitis, 199
in infectious myocarditis, 218
for factor V mutation (Leiden mutation), 373t
for hantavirus, in community-acquired pneumonia, 212
for Helicobacter pylori, in gastritis, 222
for hepatitis C viral RNA, in hepatitis C, 345t
for herpes simplex virus
in aseptic meningitis, 199
in conjunctivitis, 204
in encephalitis, 198
in Huntington’s disease, 375t
in immunocompromise-related pneumonia, 214
for Mycobacterium, in mycobacterial pneumonia, 215
in pericarditis, 217
for pertussis, in laryngotracheobronchitis, 210
for thalassemia mutation, 375t–376t
for Toxoplasma DNA, in brain abscess, 197
for varicella-zoster virus
in aseptic meningitis, 199
in conjunctivitis, 204
in encephalitis, 198
Polymorphonuclear leukocyte, 29i
Polymorphonuclear leukocytes, synovial fluid, 389–390
Polymyalgia rheumatica, erythrocyte sedimentation rate in, 86
Polymyositis
creatine kinase levels in, 78
creatine kinase MB isoenzyme levels in, 79
nuclear antibody levels in, 131
Polyuria, sodium levels in, 161
Pontiac fever, Legionella antibody levels in, 120
Poor R wave progression (PRWP), 309
Popliteal entrapment syndrome, angiography in, 279
Porphyrogen, urinary levels of, 142
Porphyria
acute intermittent
chloride levels in, 70
cholesterol levels in, 71
urinary porphobilinogen levels in, 142
urinary porphobilinogen levels in, 142
urine color affected by, 30
variegate, urinary porphobilinogen levels in, 142
Porphyromonas spp., test selection for, in empyema, 216
Portacaval shunt
ammonia levels with, 51
Child’s criteria in, 372
operative death rate after, relationship of hepatic function and nutrition or prothrombin time to, 372
Pugh modification in, 372
Portal cirrhosis, 25-hydroxy levels vitamin D3 levels in, 182
Portal vein patency, evaluation of hepatic angiography in, 271 ultrasound in, 267, 278
Portosystemic shunt procedure, transjugular intrahepatic hepatic angiography before, 271 ultrasound for evaluation of, 278 Positron emission tomography, brain scan, 247

Posterior cutaneous nerve of forearm, 341
Posterior femoral cutaneous nerve, 341
Posterior longitudinal ligament, ossification of, computed tomography in evaluation of, 277
Posterior rami of cervical nerves, 341
Postextrasystolic pause, 286
Postoperative state
iron levels in, 115
serum osmolality in, 132
Postpartum state, ferritin levels in, 90
Postpartum thrombophlebitis, test selection in, 221
Postrenal azotemia, urine indices in, 387
Posttest odds, 15, 15
Posttest probability, 9, 10–16, 11t, 12i, 14i
Potassium. See also Hyperkalemia; Hypokalemia
depletion of, pH in, 137
gastrointestinal losses of, plasma renin activity in, 152
low intake of, potassium levels affected by, 143
serum levels of, 143
in acid-base disorders, 362
in hypertension, 348
in renal tubular acidosis, 388
supplements, phosphorus levels affected by, 138
Potassium salts, potassium levels affected by, 143
Potassium-sparing diuretics, potassium levels affected by, 143
Pottery workers, lead poisoning in, 119
PPD skin testing, in tuberculous pericarditis, 217
PR segment abnormalities in pericarditis, 329
in WPW patterns, 329–330
PRA. See Plasma renin activity
Practice guidelines, clinical, 20
Prazosin, plasma renin activity affected by, 152
Precipitin test
for Coccidioides antibody, 74
for Entamoeba histolytica antibodies, 50
for Histoplasma capsulatum antibodies, 109
Precision
of analytic method for drug monitoring, 188
of tests, 4t, 4–5, 5i
Precordial leads, ECG, low-voltage QRS complex in, 308
R wave progression in, 309
right, tall R waves in, 309–310
Prednisone, electrocardiography affected by, 326
Preeclampsia, plasma renin activity in, 152
Pregnancy
albumin levels in, 47
alkaline phosphatase levels in, 50
anti-D antibody formation and, 154
α1-antiprotease levels in, 55
calcitonin levels in, 62
ceruloplasmin levels in, 69
chorionic gonadotropin levels in, 72
complement C3 levels in, 75
cortisol levels in, 76
1,25-dihydroxy vitamin D3 levels in, 183
ectopic
amylase levels in, 52
chorionic gonadotropin levels in, 72
ultrasound in, 275
erthrocyte sedimentation rate in, 86
erthropoietin levels in, 86
factor VIII assay in, 88
α-fetoprotein screening in, 91
follicle-stimulating hormone levels in, 93
functional fibrinogen levels in, 92
glucose tolerance test in, 96
hypertension of, uric acid levels in, 177
magnesium levels in, 123
molar, chorionic gonadotropin levels in, 72
mucopurulent cervicitis in, test selection in, 235
phosphorus levels in, 138
protein electrophoresis in, 146
radiation risks in, 245
rapid plasma reagin test in, 150
red cell volume in, 151
Rh testing in, 154
serum osmolality in, 132
thyroid function tests in, 394t
total iron-binding capacity in, 116
total thyroxine levels in, 169
triglyceride levels in, 172
trophoblastic disease during, testosterone levels in, 165
urinary tract infection/cystitis/pyruria-dysuria syndrome in, test selection in, 230
Venereal Disease Research Laboratory Test in, 178
vitamin B12 levels in, 180
Premature newborns, necrotizing enterocolitis in, 223
Prenatal diagnosis, 384t
of hemoglobinopathies, fetal hemoglobin, 103
maternal α-fetoprotein levels in, 91, 384t
Prerenal azotemia, urine indices in, 387t
Presyncope, with long QT syndrome, 327
Pretest probability, 10–16, 11r, 12i, 14i
Prevotella spp., test selection for
in chorioamnionitis/endometritis, 236
in empyema, 216
in sinusitis, 208
Primidone, therapeutic monitoring of, 193t
Printing workers, lead poisoning in, 119
Probability
converting to odds, 15, 15i
posttest, 9i, 10–16, 11r, 12i, 14i
pretest, 10–16, 11r, 12i, 14i
Probucol, electrocardiography affected by, 326
Procaainamide electrocardiography affected by, 193t, 325t, 326
therapeutic monitoring of, 193t
Proctosigmoidoscopy in HIV-associated diarrhea, 225
in infectious colitis/dysentery, 223
Progesterone, magnesium levels affected by, 123
Prolactin in infertility evaluation, 352i
serum levels of, 144
in amenorrhea, 144, 339i
Proliferative glomerulonephritis, complement C4 levels in, 75
Prpafenone, electrocardiography affected by, 326
Propionibacterium acnes, test selection for
in bacterial meningitis, 200
in bacterial/septic arthritis, 238
in osteomyelitis, 237
Propranolol and glucose levels, 95
thyroxine levels affected by, 169
Prostate cancer of
magnetic resonance imaging in, 273, 275
prostate-specific antigen levels in, 144
examination of, prostate-specific antigen levels affected by, 144
ultrasound in evaluation of, 273
Prostate-specific antigen, serum levels of, 144
Prostatectomy, prostate-specific antigen levels affected by, 144
Prostatic hypertrophy, benign, prostate-specific antigen levels in, 144
Prostatic massage, in prostatitis, 231
Prostatic secretion sampling, in epididymitis/orchitis, 233
Prostatitis, test selection in, 231
Prostatodynia, 231
Prosthetic joint
bacterial/septic arthritis associated with, test selection in, 238
bone scan in evaluation of, 276
osteomyelitis associated with, test selection in, 237
Prosthetic valve infective endocarditis (PVE), test selection in, 220
Protein
ascitic fluid, 365t–366t
in tuberculous peritonitis/enterocolitis, 227
cerebrospinal fluid, 369t–371t
in aseptic meningitis, 199, 369t
in bacterial meningitis, 200, 369t
in carcinomatous meningitis, 370t
in cerebral lupus erythematosus, 370t
in diabetic coma, 371t
in encephalitis, 198
in fungal meningitis, 201, 369t
in hepatic encephalopathy, 371t
in leptospirosis, 202
in neighborhood meningeal reaction, 371t
in neuroborreliosis, 202
in neurosyphilis, 202, 371t
in parasitic
meningoencephalitis/meningitis, 203, 370t
in spirochetal meningitis, 202, 371t
in subarachnoid hemorrhage, 370t
in syphilitic meningitis, 202, 371t
in tuberculous meningitis, 203, 369t
in uremia, 371t
deficiency, severe dietary, protein levels in, 147
peritoneal fluid, in peritonitis, 226
pleural fluid, 382t–383t
in cirrhosis, 382t
in collagen vascular disease, 383t
in empyema, 216, 383t
in esophageal rupture, 383t
in heart failure, 382t
in malignancy, 382t
in nephrotic syndrome, 382t
in pancreatitis, 383t
in parapneumonic effusion, 383t
in pulmonary embolism/infarction, 383t
in rheumatoid arthritis, 383t
in tuberculosis, 382t
serum, 329t
electrophoresis of, 112, 146
synovial fluid, 389t–390t
total, 147, 378t
in hyponatremia, 350t
urine, 395t–396t
dipstick testing of, 30, 31t
Protein binding of drugs
amitriptyline levels affected by, 191t
desipramine levels affected by, 191t
imipramine levels affected by, 192t
nortriptyline levels affected by, 193t
phenytoin levels affected by, 193t
therapeutic monitoring and, 190
Protein C
deficiency/resistance
factor V (Leiden) mutation in, 87, 373t
protein C levels in, 145
plasma levels of, 145
protein S as co-factor for, 147
Protein electrophoresis, 146
Protein-losing enteropathies
complement C4 levels in, 75
protein levels affected by, 147
Protein S
antigen, plasma levels of, 147
deficiency
congenital, 147
protein S antigen levels in, 147
Proteus spp.
OX-19 antigen, cross-reactivity with tularemia antibody test, 175
urinalysis in identification of infection caused by, 30
Prothrombin deficiency, Russell’s viper venom clotting time in, 157
Prothrombin time, 148, 377t–378t
dipstick testing of, 30, 31t
inhibitor screen in evaluation of, 114
for monitoring warfarin therapy, 148, 188
and operative death rate after portocaval shunt (Pugh modification), 372t
Proton pump inhibitors, and gastrin levels, 95
Protoporphyria, free erythrocyte protoporphyrin levels in, 94
Protoporphyrin, free erythrocyte, 94
in anemias, 94, 363–364
in iron deficiency, 94, 364
in lead poisoning, 94, 363
Prussian blue stain, for urine hemosiderin, 104
PRWP (poor R wave progression), 309
Pseudallescheria boydii, test selection for
in fungal meningitis, 201
in sinusitis, 208
in transplant-related pneumonia, 214
Pseudocyst, pancreatic
amylase levels in, 52
lipase levels in, 121
ultrasound in evaluation of, 258, 272
Pseudogout, synovial fluid sampling in,
389
Pseudohyphae, in KOH preparation,
33–35
Pseudohyponatremia, 350
Pseudohypoparathyroidism
calcium levels in
serum, 63
urine, 65
phosphorus levels in, 138
Pseudomembranous colitis
Clostridium difficile enterotoxin
levels in, 73, 224
test selection in, 224
Pseudomonas spp.
test selection for
in bacteremia of unknown source, 241
in bacterial meningitis, 200
in bacterial/septic arthritis, 238
in cellulitis, 240
in community-acquired pneumonia, 212
in empyema, 216
in epidermiditis/orchitis, 233
in hospital-acquired pneumonia, 213
in infectious thrombophlebitis, 221
in neutropenic pneumonia, 214
in otitis externa, 207
in otitis media, 206
in prostatitis, 231
in sinusitis, 208
urine color affected by, 30
Pseudomonas aeruginosa, test selection for
in anaerobic pneumonia or lung abscess, 213
in brain abscess, 197
in cholangitis/cholecystitis, 229
in community-acquired pneumonia, 212
in HIV-associated pneumonia, 214
in hospital-acquired pneumonia, 213
in keratitis, 205
in osteomyelitis, 237
in otitis externa, 207
in otitis media, 206
in peritonitis, 226
in pyelonephritis, 232
in sinusitis, 208
in transplant-related pneumonia, 214
Pseudomyxoma peritonei, ascitic fluid
profile in, 365
Pseudoparathyroidism, calcium levels in,
354
Psoriasis
CD4/CD8 ratio in, 68
uric acid levels in, 177
Psoriatic arthritis, synovial fluid sampling
in, 389
PSVT (paroxysmal supraventricular
tachycardia), 298–299
Psychiatric illness
free thyroxine index in, 170
free thyroxine levels in, 170
Psychotropic agents, electrocardiography
affected by, 326
PT. See Prothrombin time
PTBD (percutaneous transhepatic biliary
drainage), 270
PTC (percutaneous transhepatic cholangiogram), 270
PTCA, cardiac troponin-I levels after, 174
PHT. See Parathyroid hormone
PTT. See Partial thromboplastin time
Puberty, delayed, follicle-stimulating hor-
mone levels in, 93
Puerperal septic pelvic thrombophlebitis,
test selection in, 221
Pugh modification, 372
Pulmonary angiography, 255
in pulmonary embolism, 255, 357
Pulmonary disease
chest x-ray in, 252
infiltrative, leukocyte count in, 400
obstructive. See also Chronic obstruc-
tive pulmonary disease
angiotensin-converting enzyme levels
in, 52
pulmonary function tests in, 385
red cell volume in, 151
restrictive, pulmonary function tests in,
385
Pulmonary edema, chest x-ray in, 252
Pulmonary embolism
diagnostic algorithm for, 356i–357i
fibrin D-dimer assay in, 91
partial pressure of oxygen in, 133
pleural fluid profile in, 383t
protein C deficiency and, 145
pulmonary angiography in, 255, 357i
risk factors for, 356i–357i
spiral computed tomography in, 254
ST segment elevation in, 321r
ultrasound in, 357i
ventilation-perfusion scan in, 253,
356i–357i
Pulmonary fibrosis
erthropoietin levels in, 86
radionuclide thyroid therapy and, 251
Pulmonary function tests, 358t, 385
Pulmonary hypertension, valvular heart
disease in, diagnostic evaluation
of, 399t
Pulmonary infarction, pleural fluid profile
in, 383t
Pulmonary nodules, computed tomogra-
phy in evaluation of, 252
Pulmonary sequestration, pulmonary
angiography in, 255
Pulse oximetry, 36–38
approach to patient for, 37
contraindications to, 37
indications for, 36
technique for, 37–38
Purine, low dietary intake of, uric acid
levels in, 177
Purpura
cryoglobulins causing, 82
idiopathic thrombocytopenic
hemostatic function tests in, 377t
platelet-associated IgG in, 141
platelet count in, 140, 377t
neonatal fulminans, protein C de-
ciency and, 145
posttransfusion
platelet-associated IgG in, 141
platelet count in, 140
thrombotic thrombocytopenia
lactate dehydrogenase levels in, 117
plasma transfusion in, 401t
Pus
and urine color, 30
and urine turbidity, 30
Pyelogram, intravenous. See Intravenous
pyelogram
Pyelonephritis
leukocyte scan in, 281
perinephric abscess associated with, test
selection in, 232
test selection in, 232
urine characteristics in, 396t
Pyogenic infection, IgG deficiency in, 113
Pyomyositis, test selection in, 240
Pyridoxine deficiency. See Vitamin B₆
deficiency
Pyruvate dehydrogenase deficiency,
lactate levels in, 118
Pyuria
in epididymitis/orchitis, 233
in perinephric abscess, 232
in prostatitis, test selection in, 231
in pyelonephritis, 232
test selection in, 230
Pyuria-dysuria syndrome, test selection in,
230
Q
Q fever
myocarditis in, test selection in, 218
Q fever antibody levels in, 149
test selection in, in community-acquired
pneumonia, 212
vaccination, Q fever antibody levels
affected by, 149
Q wave
normal, 299–300
pathologic
in mimics of myocardial infarction,
319, 320t
in myocardial infarction, 313–314,
316–317, 318r–319r, 319
QRS axis
deviations in
left, 305
and presumption of disease, 304
right, 305–306
right superior, 306
mean
determination of, 304–306
in frontal plane (limb leads), 304–305
normal range, in adults, 304
QRS complex
in atroioventricular block, 297
in atroioventricular dissociation, 298
in bundle branch block, 290–292,
295–296, 301–302
electrical axis of. See QRS axis
in hyperkalemia, 321t
in incomplete bundle branch block,
302–303
QRS complex (cont.)
in intraventricular conduction delay or defect, 303
in junctional rhythms, 288
left bundle branch-type of, 291, 293, 295
low voltage of, 308
extracardiac causes of, 308
limb and precordial leads, 308
limb leads only, 308
myocardial causes of, 308
mean rate of, 284–285
in myocardial infarction, 308, 311, 316–320, 318–319
narrow, in paroxysmal supraventricular tachycardia, 298–299
premature activity, 286
rhythmicity of, 284, 286
right bundle branch-type of, 291, 294–295
torsade de pointes, 296
wide
  morphological type of, determination of, 291–292, 295
  in tachycardia with regular rhythm (WCT-RR), 290–296
width of, 284–285, 285t
in WPW patterns, 329
QRS rhythm irregularity
accelerating-decelerating, 287
categories of, 286–287
dominant regular rhythm with interruptions, 286
irregularly regular rhythm, 286–287
regularly irregular rhythm (group beating), 287, 297
QRST patterns, in normal electrocardiography, 299–300
QS complex, in right ventricular injury or infarction, 314
QT interval, 324–326
drugs affecting, 325t, 326–327
heart rate and, 324
in hypercalcemia, 325t, 327
measurement of, 324–326
normal, 325t
prolonged, 324–327, 325t
causes of, 326–327
clinical correlations of, 296
in congenital long QT syndrome, 327
drugs causing, 325t, 326
electrolyte abnormalities and, 326
in hypocalcemia, 325t, 326
in hypokalemia, 296, 325t
in hypothermia, 327–328
miscellaneous causes of, 327
torsade de pointes with, 296 short, 325t, 327
QT nomogram (Hodges correction), 324–326
Quinidine
digoxin levels affected by, 192
direct antiglobulin test affected by, 54
electrocardiography affected by, 325t, 326
platelet count affected by, 140
therapeutic monitoring of, 194t
R
R wave
in left ventricular hypertrophy, 306, 309 pathologic
  in mimics of myocardial infarction, 319, 320t
  in myocardial infarction, 309, 315–317, 318–319t
progression of
  poor, 309
  in precordial leads, 309 reversed, 309
in right bundle branch block, 309
in right ventricular hypertrophy, 309
tall, in right precordial leads, 309–310
etiology of, 309–310
rare or uncommon causes of, 310
in WPW pattern, 310
Rabbits, exposure to pneumonia associated with, test selection in, 212
tularemia associated with, test selection in, 175
Rabies
encephalitis after, test selection in, 198
vaccine, encephalitis after, test selection in, 198
RAD (right axis deviation), 305–306
Radial nerve, 341t–342t
Radiation risks, in pregnancy, 245
Radiation therapy, head and neck thyroid uptake and scan in patients with history of, 250
ultrasound screening of patients with history of, 249
Radiography. See X-ray
Radioimmunoassay, for thyroid peroxidase antibody, 167
Radionuclide scans/studies
  brain, 246–247
  in cholangitis/cholecystitis, 229
  in epididymitis/orchitis, 233
  esophageal reflux, 264
  gastric emptying, 265
  GI bleeding scan, 265
  leukocyte scan, 281
  liver/spleen scan, 271
  MIBG (metaiodobenzyl-guanidine), 272
  parathyroid, 251
  renal scan, 274
  thyroid uptake and scan, 250
  ventilation-perfusion, 253
Radionuclide therapy, thyroid, 251
  calculation of dosage, thyroid uptake and scan in, 250
Radionuclide ventriculography, 257
RAI uptake, in thyroid evaluation, 250, 393–394
Ranson’s criteria, for severity of pancreatitis, 386
Rapid ACTH stimulation test
  in adrenocortical insufficiency, 338i
  in hypoglycemia, 349i
Rapid plasma reagin test, 150, 391t
Raynaud’s disease/phenomenon
  centromere antibody test in, 69, 367t
  cryoglobulins causing, 82
  scleroderma-associated antibody in, 158
RBBB. See Right bundle branch block
RBC count. See Erythrocyte count
Reagent strip (dipstick) testing of urine, 30
  components of, 31t–32t
  in urinary tract
    infection/cystitis/pyuria-dysuria syndrome, 230
Receiver operator characteristic curves, 9, 10i
Reciprocal changes, with myocardial injury, ischemia and infarction, 310, 316
Recombinant immunoblot assay, for hepatitis C antibody, 106
Rectal biopsy
  in HIV-associated diarrhea, 225
  in infectious colitis/dysentery, 223
Rectal carcinoids, 5-hydroxy-indoleacetic acid levels in, 111
Rectal carcinoma, magnetic resonance imaging in, 275
Rectal culture, in bacterial/septic arthritis, 238
Red blood cells. See Erythrocyte(s)
Red-top tubes, 24, 42
Reentry tachycardia
  atrioventricular, 299
  AV nodal, 299
Reference range, for tests, 5–6, 6t, 6i
Reflex sympathetic dystrophy, bone scan in, 276
Reflex
  esophageal, esophageal study in evaluation of, 264
  gastroesophageal, upper GI study in evaluation of, 262
Regional enteritis. See Crohn’s disease
Regional ileitis. See Crohn’s disease
Regurgitation, esophageal reflux study in, 264
Reiter’s syndrome
  HLA-B27 typing in, 111
  synovial fluid sampling in, 389t–390t
Release abnormalities, congenital, platelet aggregation in, 139
Renal artery stenosis
  angiography in, 279
  magnetic resonance angiography in, 280
  plasma renin activity in, 152
Renal blood flow
  decreased, creatinine clearance in, 81
  renal scan in, evaluation of, 274
Renal cell carcinoma
  calcium levels in, 63
  computed tomography in staging of, 259
  red cell volume in, 151
  staging of, magnetic resonance imaging in, 260
Renal clearance, 189
Renal dysfunction/insufficiency
  amikacin levels affected in, 191t
  calcium levels in, 63
  digoxin levels affected in, 192t
  gentamicin levels affected in, 192t
  5-hydroxy-indoleacetic acid levels in, 111
  lithium levels affected in, 192t
  methotrexate levels affected in, 193t
  methylmalonic acid levels in, 126
  procainamide levels affected in, 193t
  renal tubular acidosis in, 388t
  tobramycin levels affected in, 194t
  vancomycin levels affected in, 194t
  D-xylose absorption test in, 185
Renal failure
  blood urea nitrogen levels in, 60
  C-peptide levels in, 62
Renal failure (cont.)
calcitonin levels in, 62
calcium levels in, ionized, 64
cardiac troponin-I levels in, 174
chloride levels in, 70
classification and differential diagnosis of, 387
contrast-induced, 244
cortisol levels in, 76
creatinine clearance in, 81, 359
creatinine levels in, 80
1,25-dihydroxy vitamin D₃ levels in, 183
erthrocyte sedimentation rate in, 86
erthropoietin levels in, 86
25-hydroxy vitamin D₃ levels in, 182
iron levels in, 115
magnesium levels in, 123
β₂-microglobulin levels in, 128
nuclear antibody levels in, 131
pH in, 137
phosphorus levels in, 138
plasma renin activity in, 152
postrenal azotemia in, 387
potassium levels in, 143
prerenal azotemia in, 387
prolactin levels in, 144
renal scan in evaluation of, 274
triglyceride levels in, 172
uric acid levels in, 177
urinary indices in, 387
urine characteristics in, 395
D-xylose absorption test in, 185
Renal function, renal scan for evaluation of, 274
Renal infarction, lactate dehydrogenase isoenzyme levels in, 117
Renal losses, serum osmolality with, 132
Renal osteodystrophy, renal tubular acidosis in, 388
Renal scan, 274
Renal stones/calculi (kidney stones)
computed tomography in, 259
intravenous pyelogram in, 273
urinary calcium levels in, 65
Renal tubular acidosis
chloride levels in, 70
laboratory diagnosis of, 388
pH in, 137, 388
phosphorus levels in, 138
potassium levels in, 143, 388
urinary calcium levels in, 65
Renal tubular concentrating ability, osmolality test for measurement of, 133
Renal tubular defects, phosphorus levels in, 138
Renal tubular necrosis, urine indices in, 387
Renal tumors, erythropoietin levels with, 86
Renal vascular hypertension, renal scan in, 274
Renal vein renin ratio, 152
Renin, plasma activity of, 152
in hypertension, 152, 348
plasma aldosterone and, 48
urine aldosterone and, 49
Renin-angiotensin system, aldosterone secretion controlled by, 49
Repolarization abnormalities of
in left ventricular hypertrophy, 307, 321–322
spectrum of, 307
in right ventricular hypertrophy, 308
ST segment depression or T wave inversion in, 322
early, normal variant
ST segment elevation in, 321
versus ST-T abnormality, 328
early, versus pericarditis, 329
mean QRS axis in, 304–306
Reptilase clotting time, 153
Reserpine
5-hydroxy-indoleacetic acid levels affected by, 111
plasma renin activity affected by, 152
prolactin levels affected by, 144
Residual urine volume, ultrasound in evaluation of, 273
Residual volume (pulmonary), 385
Residual volume overload, valvular heart disease in, diagnostic evaluation of, 399
Residual volume/total lung capacity ratio, 385
Respiratory acidosis
carbon dioxide levels in, 66
chloride levels in, 70
laboratory characteristics and clinical features of, 362
nomogram for, 337
pH in, 137, 362
phosphorus levels in, 138
Respiratory alkalosis
carbon dioxide levels in, 66
chloride levels in, 70
laboratory characteristics and clinical features of, 362
nomogram for, 337i
pH in, 137, 362t
phosphorus levels in, 138
Respiratory depressants, pH affected by, 137
Respiratory infection, IgA levels in, 113
Respiratory syncytial virus, test selection in
in hospital-acquired pneumonia, 213
in laryngotracheobronchitis, 210
in transplant-related pneumonia, 214
Restrictive pulmonary disease, pulmonary function tests in, 385t
Reticulocyte count, 153
in anemia, 153, 363t
Reticulocytosis labor and clinical findings in, 363t
mean corpuscular volume in, 124
Retroperitoneal disorders
computed tomography in, 259
magnetic resonance imaging in, 260
Retroperitoneal hemorrhage, computed tomography in, 259
Retropharyngeal abscess, magnetic resonance imaging in evaluation of, 248
Retropulsed bone fragments, after trauma, computed tomography in, 277
Reverse dot blot assay
for cystic fibrosis mutation, 373t
for factor V mutation (Leiden mutation), 87, 373t
for thalassemia syndromes, 376t
Reversed R wave progression (RRWP), 309
Reye’s syndrome
ammonia levels in, 51
creatine kinase levels in, 78
Rh grouping, 154
in type and cross-match, 54, 154, 175
in type and screen, 53, 176
Rh(D)-negative, 154
Rh(D)-positive, 154
Rh sensitization, fetal hemoglobin testing and, 103
Rhabdomyolysis
cardiac troponin-I levels in, 174
creatine kinase levels in, 78
potassium levels in, 143
Rheumatic disease
\( \alpha_1 \)-antiprotease levels in, 55
complement C3 levels in, 75
rheumatoid factor in, 155, 368t
valvular heart disease in, diagnostic evaluation of, 398t–399t
Rheumatic fever
antistreptolysin O titer in, 55
erthroyocyte sedimentation rate in, 86
synovial fluid sampling in, 389t
Rheumatoid arthritis
autoantibodies in, 368t
CD4/CD8 ratio in, 68
complement C3 levels in, 75
complement C4 levels in, 75
cryoglobulin levels in, 82
double-stranded DNA antibody in, 367t
ferritin levels in, 90
IgG levels in, 113
nuclear antibody levels in, 131, 367t
pleural fluid profile in, 383t
rapid plasma reagin test in, 150
rheumatoid factor levels in, 155, 368t
ribonucleoprotein antibody levels in, 155, 367t
SS-A/Ro antibody in, 163
synovial fluid sampling in, 389t–390t
Toxoplasma antibody test in, 171
Venereal Disease Research Laboratory Test in, 178
Rheumatoid factor
cryptococcal antigen test affected by, 82
cytomegalovirus antibody test affected by, 83
free thyroxine levels affected by, 170
rubella antibody titer affected by, 156
serum levels of, 155, 368t
Rhinorrhea, cerebrospinal fluid, cisternography in evaluation of, 247
Rhinovirus, test selection for
in laryngitis, 209
in laryngotraeheobronchitis, 210
in pharyngitis, 209
Rhizopus spp., test selection for, in sinusitis, 208
Rhodococcus equi, test selection for, in HIV-associated pneumonia, 214
Rhodotorula spp., test selection for, in keratitis, 205
RhoGam, fetal hemoglobin testing in dosage determination of, 103
Rhythms, cardiac. See also specific rhythms
electrocardiographic diagnosis of, 283–299, 285t
sustained irregular, 285t
sustained regular, 285t
Ribonucleoprotein antibody, serum levels of, 155, 367t
Rickets
1,25(OH)2-resistant, 1,25-dihydroxy vitamin D3 levels in, 183
alkaline phosphatase levels in, 50
calcium levels in, urine, 65
25-hydroxy vitamin D3 levels in, 182
phosphorus levels in, 138
*Rickettsia rickettsii*, test selection for, in infectious myocarditis, 218
Rickettsial infection, heterophile agglutination (Monospot/Paul-Bunnell) test in, 107
Riedel’s thyroiditis, thyroperoxidase antibody in, 167
Rifampin
cyclosporine levels affected by, 191t
urine color affected by, 30
Right atrial enlargement
clinical correlation of, 300
electrocardiographic findings of, 300, 307
Right axis deviation, 305–306
Right bundle branch block, 301
diagnostic criteria for, 301
incomplete, 302–303
as mimic of myocardial infarction, 320t
morphology of, in wide QRS complex, 291–292
new, in left anterior descending artery occlusion, 316
in pulmonary embolism, 356i–357i
QRS complex in, 290–292, 295–296, 301
ST segment depression or T wave inversion in, 322t
ST-T changes in, 301
tall R waves in right precordial leads in, 309
Right bundle branch-type of QRS, 291, 294–295
Right coronary artery, as culprit artery in myocardial infarction, 312, 314–315
Right heart failure. *See* Heart failure
Right-left arm cable reversal in ECG, versus mirror image dextrocardia, 327
Right leg cable, misplacement of, in ECG, 327–328
Right superior axis deviation, 306
Right-to-left shunt, partial pressure of oxygen in, 133
Right-to-left shunting, brain abscess with, 197
Right ventricular hypertrophy
diagnostic criteria for, 308
electrocardiographic findings in, 307–308
incomplete right bundle branch block, 302
mimicking myocardial infarction, 320t
repolarization abnormalities, 308
right atrial enlargement, 300, 307
right axis deviation, 305–306
tall R waves in right precordial leads, 309
ST segment depression or T wave inversion in, 322t
Right ventricular infarction, electrocardiography of, 314–315
Right ventricular injury, electrocardiography of, 311, 314–315
Ristocetin
platelet aggregation by, 139
in von Willebrand’s factor protein measurement, 184
River blindness, test selection in, 205
ROC. *See* Receiver operator characteristic curves
Rocky mountain spotted fever
*Brucella* antibody in, 60
myocarditis in, test selection in, 218
Romhilt-Estes criteria, 306
Rotavirus, test selection for, in infectious colitis/dysentery, 223
Rotor’s syndrome, bilirubin levels in, 58
Roux-en-Y hepaticojejunostomy, percutaneous transhepatic cholangiogram in evaluation of, 270
RPR. *See* Rapid plasma reagin test
RR (regular rhythm) intervals
in irregularly irregular QRS rhythm, 286
lengthening of, in tachycardia, 286
regularity of, in ventricular tachycardia, 290
RRWP (reversed R wave progression), 309
Rubella antibody, serum levels of, 156
Rubella infection
congenital, rubella antibody titer in, 156
rubella antibody titer in, 156
Rubella vaccination, rubella antibody titer affected by, 156
Russell’s viper venom clotting time, 157
RV (residual volume), 385t
RV/TLC (residual volume/total lung capacity ratio), 385
RVH. See Right ventricular hypertrophy

S
S. aureus. See Staphylococcus aureus
S. epidermidis. See Staphylococcus epidermidis
S. pneumoniae. See Streptococcus pneumoniae

S wave
in left ventricular hypertrophy, 306
normal, 300
SAAG. See Serum ascites albumin gradient
Safety precautions, in specimen collection/handling, 24
St. Louis encephalitis, test selection in, 198
Salicylate(s). See also Aspirin
chloride levels affected by, 70
hemostatic function tests affected by, 377t
pH affected by, 137
poisoning/toxicity
nomogram for, 360i
pH in, 137
phosphorus levels affected by, 138
salicylate serum levels in, 158
serum levels of, 158
therapeutic monitoring of, 194t
thyroid function tests affected by, 394t
uric acid levels affected by, 177
Saline, overtreatment with, chloride levels in, 70
Saliva culture, in rabies encephalitis, 198
Salmonella spp.
Brucella antibody in, 60
Reiter’s syndrome associated with, HLA-B27 typing in, 111
test selection for
in epididymitis/orchitis, 233
in HIV-associated diarrhea, 225
in infectious colitis/dysentery, 223
Salpingitis, test selection in, 236
Salt depletion, and aldosterone measurements
plasma, 48
urine, 49
Salt deprivation, and chloride levels, 70
Salt loading, and aldosterone measurements
plasma, 48
urine, 49
Salt-losing nephropathy
chloride levels in, 70
sodium levels in, 161
Salt-wasting mineralocorticoid-resistant hyperkalemia, renal tubular acidosis in, 388t
Saphenous nerve, 341i–342i
Sarcoidosis
angiotensin-converting enzyme levels in, 52
calcium levels in, 347i
serum, 63
urine, 65
complement C3 levels in, 75
1,25-dihydroxy vitamin D3 levels in, 183
IgG levels in, 113
and low-voltage QRS complex in ECG, 308
parathyroid hormone levels in, 134
phosphorus levels in, 138
rheumatoid factor levels in, 155
synovial fluid sampling in, 389t
Sarcoma, osteogenic, 50
Scarlet fever, antistreptolysin O titer in, 55
Schilling’s test (vitamin B12 absorption test), 180–181
Schistocytes, 29i
Schistosomiasis, urine calcium levels in, 65
Schizocytes, 29i
SCID. See Severe combined immunodeficiency
Scl-antibody. See Scleroderma-associated antibody
Scleroderma
autoantibodies in, 367t
centromere antibody test in, 69, 367t
nuclear antibody levels in, 131, 367t
rheumatoid factor levels in, 155
ribonucleoprotein antibody levels in, 155, 367t
scleroderma-associated antibody in, 158, 368t
synovial fluid sampling in, 389t
Scleroderma-associated antibody (Scl-antibody), serum levels of, 158, 368t
Sclerosing cholangitis, endoscopic retrograde cholangiopancreatography in, 267
Screening tests, 1–2, 2t
Scrotal edema, in epididymitis/orchitis, 233
Scrub typhus, myocarditis in, test selection in, 218
Sea water contaminated abrasion, cellulitis and, test selection in, 240
Seafood, raw, cellulitis associated with consumption of, test selection in, 240
Seborrhea, otitis externa in, test selection in, 207
Second-degree atrioventricular block, 297 prolonged QT interval in, 327
type I, 297
type II, 297
Seizures
generalized, creatine kinase levels in, 78 medically refractory, positron emission tomography in, 247
Selective serotonin reuptake inhibitors (SSRIs), sodium levels affected by, 161
Semen analysis, 159 in infertility, 159, 352i
Semenoma, chorionic gonadotropin levels with, 72
Senna, urine color affected by, 30
Sensitivity, of tests, 7–9, 8i–10i
Sepsis
cardiac troponin-I levels in, 174 lactate levels in, 118 leukocyte count in, 121, 400t pH in, 137 in transfusion reaction, 401t–402t
Septic arthritis
synovial fluid sampling in, 238, 390t test selection in, 238
Septic shock, cortisol levels in, 76
Septic thrombophlebitis, neighborhood meningeal reaction in, 371t
Sequential testing, 16
Serotonin, 5-hydroxy-indoleacetic acid as measure of, 111
Serum ascites albumin gradient, 227, 365t–366t
Serum osmolality, in hyponatremia, 132, 350t
Serum separator, in specimen tubes, 24, 42
Severe combined immunodeficiency, IgG levels in, 113
Sexual precocity,idiopathic, testosterone levels in, 165
Sézary syndrome, CD4/CD8 ratio in, 68 SGOT. See Aspartate aminotransferase Sheep, pneumonia associated with exposure to, test selection in, 212
Shigella spp.
Reiter’s syndrome associated with, HLA-B27 typing in, 111
test selection for in HIV-associated diarrhea, 225 in infectious colitis/dysentery, 223
Ship builders, lead poisoning in, 119
Shock albumin levels in, 47 blood urea nitrogen levels in, 60 creatinine clearance in, 81 lactate levels in, 118 partial pressure of oxygen in, 133 septic, cortisol levels in, 76
Shock liver alanine aminotransferase levels in, 46 aspartate aminotransferase levels in, 56
Shunt patency, cisternography in evaluation of, 247
SIADH. See Syndrome of inappropriate antidiuretic hormone
Sickle cell anemia
erthrocyte sedimentation rate in, 86 fetal hemoglobin levels in, 103 glycohemoglobin levels in, 98 hemoglobin electrophoresis in, 102 hemosiderin levels in, 104
Sickle cells, 29t
Sideroblastic anemia
laboratory and clinical findings in, 363t mean corpuscular hemoglobin in, 123–124, 363t mean corpuscular volume in, 363t reticulocyte count in, 153 serum iron levels in, 115, 363t transferrin saturation with iron in, 116
Sigmoid volvulus, Hypaque enema in, 264
Single photon emission computed tomography, brain scan, 247
Single photon emission computed tomography immunoscintigraphy, in infective endocarditis, 219
Sinoatrial exit block, accelerating-decelerating rhythm in, 287
Sinus arrhythmia, 287t accelerating-decelerating rhythm in, 287
Sinus bradycardia, 287t with junctional escape rhythm, 298 QRS duration in, 285t
Sinus disease, computed tomography in evaluation of, 245
Sinus rhythms, 287, 287t QRS duration in, 285t
Sinus tachycardia, 287t QRS complex in, 290 QRS duration in, 285t
Sinus thrombosis, dural, magnetic resonance venography in, 246
Sinusitis
  - brain abscess with, 197
  - neighborhood meningeal reaction in, 371
  - test selection in, 208
Sjögren’s syndrome
  - autoantibodies in, 367
  - complement C3 levels in, 75
  - nuclear antibody levels in, 131, 367
  - rheumatoid factor levels in, 155
  - ribonucleoprotein antibody levels in, 155, 367
  - SS-A/Ro antibody in, 163, 368
  - SS-B/La antibody in, 163
Skeletal muscle damage/disorders
  - cardiac troponin-I levels in, 174
  - lactate dehydrogenase isoenzyme levels in, 117
  - lactate dehydrogenase levels in, 117
Skin culture
  - in cellulitis, 240
  - in fungal meningitis, 201
  - in rabies encephalitis, 198
Skin damage/disorders
  - lactate dehydrogenase levels in, 117
  - leukocyte count in, 400
Skin test
  - brucellergin, Brucella antibody test affected by, 60
  - Coccidioidin, Coccidioides antibody test affected by, 74
  - histoplasmin
    - Histoplasma capsulatum complement fixation antibody test affected by, 109
    - Histoplasma capsulatum precipitin levels affected by, 109
  - PPD, in tuberculous pericarditis, 217
Skunks, exposure to, tularemia associated with, test selection in, 175
SLE. See Systemic lupus erythematosus
Sleep, prolactin levels in, 144
Slow vital capacity, 385
Small bowel
  - bacterial overgrowth in
    - vitamin B12 levels in, 180
  - D-xylene absorption test in, 185
  - metastases to, enteroclysis in, 262
  - obstruction of, enteroclysis in evaluation of, 262
Small bowel disease
  - enteroclysis in, 262
  - fecal fat levels in, 88
  - Smith antibody (anti-Sm), serum levels of, 159, 367
Smoking
  - carboxyhemoglobin blood levels affected by, 66
  - carcinoembryonic antigen levels affected by, 67
  - red cell volume affected by, 151
  - theophylline levels affected by, 194
Smooth muscle antibodies, serum levels of, 160
Sodium. See also Hypernatremia; Hyponatremia; Saline
dietary, plasma renin activity affected by, 152
  - gastrointestinal losses of, plasma renin activity in, 152
  - serum levels of, 161
  - in syndrome of inappropriate antidiuretic hormone, 161
  - serum osmolality affected by, 132
  - urine, in renal failure/disease, 387
  - urine levels of, in hyponatremia, 350
  - Sodium bicarbonate. See Bicarbonate
  - Sodium fluoride, in specimen tubes, 42
Soft tissue
calcification, 25-hydroxy vitamin D3 levels in, 182
  - infections of, magnetic resonance imaging in, 278
  - tumor of, magnetic resonance imaging in, 278
Sokolow-Lyon criteria, 306
Somatomedin C, plasma levels of, 162
Sore throat. See Pharyngitis
Sotalol, electrocardiography affected by, 326
Southern blot assay
  - for B cell immunoglobulin heavy chain rearrangement, 57
  - for bcr/abl translocation, 57
  - in hemophilia A, 374
  - in Huntington’s disease, 375
  - for T cell receptor gene rearrangement, 164
  - in thalassemia syndromes, 375–376
Specific gravity, urine, 395–396
  - dipstick testing of, 30, 31
Specificity
  - of analytic method for drug monitoring, 188
  - of tests, 7–9, 8i–10i
Specimen collection/handling, 3–4, 24–25, 26
  - for microbiology tests, 196
  - safety precautions in, 24
  - for therapeutic drug monitoring, 190
Specimen identiﬁcation, 3, 24
Specimen tubes, 24–25
color coding of tops, 24–25, 42
order of ﬁlling, 25
SPECT brain scan, 247
Spectrophotometry
for methemoglobin assay, 126
for total hemoglobin levels, 103
Spectrum bias, 8
Sperm count, 159, 352
Spherocytes, 29i
Spherocytosis
congenital, glycohemoglobin levels in, 98
erthrocyte sedimentation rate in, 86
mean corpuscular hemoglobin concentration in, 124
Spinal cord disease, magnetic resonance imaging in, 277
Spinal disease, magnetic resonance imaging in, 277
Spine, imaging test selection and interpretation in evaluation of, 277
Spiral computed tomography, in lung evaluation, 254
Spirochetal meningitis
cerebrospinal ﬂuid proﬁle in, 202, 371t
test selection in, 202
Spirometry, 358t
Spirolactone
potassium levels affected by, 143
testosterone levels affected by, 165
Splanchic artery aneurysm, mesenteric angiography in evaluation of, 261
Spleen
accessory, liver/spleen scan in evaluation of, 271
imaging test selection and interpretation in evaluation of, 271
infection of, leukocyte scan in, 281
Splenectomy
bacteremia of unknown source and, test selection in, 241
glycohemoglobin levels with, 98
platelet count after, 140
Splenic artery aneurysm, mesenteric angiography in evaluation of, 261
Splenic infarction, computed tomography in, 259
Splenomegaly
in hemolysis, 363t
platelet count in, 140
Spondyloarthritis, HLA-B27 typing in, 111
Sprue. See also Celiac disease
fecal fat levels in, 88
5-hydroxy-indoleacetic acid levels in, 111
vitamin B12 levels in, 180
Sputum sampling
in anaerobic pneumonia or lung abscess, 213
in community-acquired pneumonia, 212
in empyema, 216
in hospital-acquired pneumonia, 213
in immunocompromise-related pneumonia, 214
in laryngotracheobronchitis, 210
microscopic examination in, 27, 28i
in Pneumocystis carinii pneumonia, 28i, 214
Squamous cell carcinoma, calcium levels in, 63
SS-A/Ro antibody, serum levels of, 163, 368t
SS-B/La antibody, serum levels of, 163
ST segment, 320, 321t–323t. See also ST segment depression; ST segment elevation
abnormal, 323t
classes and morphologies of, 323t
in hypercalcemia, 325t
in hypocalcemia, 325t
in myocardial infarction, 310–316, 311t, 319–320, 322t–323t
normal, 299–300, 323t
ST segment depression
in anterior subendocardial injury or non-Q wave MI, 322t
catecholamines and, 322t
drugs causing, 322t–323t, 325t
in endocardial injury, 310
in hypokalemia, 322t–323t, 325t
in inferior subendocardial injury, 322t
in left bundle branch block, 322t
in left ventricular hypertrophy, 322t
major causes of, 322t
in myocardial infarction, 310, 322t
inferior, 316
posterior, 315
as reciprocal change, 316
in myocardial injury, 310, 315, 322t
in myocardial ischemia, 310
in posterior subepithelial injury, 322t
in right bundle branch block, 322t
in right ventricular hypertrophy, 322t
in subarachnoid hemorrhage, 322t
ST segment elevation
  in epicardial injury, 310
  in hyperkalemia, 321
  in left anterior descending artery occlusion, 316
  in left bundle branch block, 321
  in left ventricular hypertrophy, 307, 321, 323
  major causes of, 321
  in mimics of myocardial infarction, 319, 320
  in myocardial infarction, 319–320
    anterior, 312–313
    inferior, 314
    primary anterior area, 312
    primary inferior area, 312
  in myocardial injury, 310
    anterior, 321
    inferior, 321
  normal variant early repolarization and, 321, 328
  in pericarditis, 321, 329
  persistent, 320
  in pulmonary embolism, 321
  in right ventricular injury or infarction, 314
  in ventricular pacemaker, 321
ST-T segment
  classes and morphologies of, 323
  in left bundle branch block, 302
  in right bundle branch block, 301
ST-T-U abnormalities, classes and morphologies of, 325
Staining. See also specific types
  basic methods of, 25–28
Staphylococcus spp.
  on Gram-stained smear, 27
  test selection for
    in bacteremia of unknown source, 241
    in bacterial meningitis, 200
    in bacterial/septic arthritis, 238
    synovial fluid sampling in, 390
    in brain abscess, 197
    in cellulitis, 240
    in community-acquired pneumonia, 212
    in empyema, 216
    in endophthalmitis, 205
    in hospital-acquired pneumonia, 213
    in impetigo, 239
    in infectious colitis/dysentery, 223
    in infectious thrombophlebitis, 221
    in infective endocarditis, 219
    in laryngotracheobronchitis, 210
    in liver abscess, 228
    in osteomyelitis, 237
    in otitis externa, 207
    in otitis media, 206
    in pericarditis, 217
    in perinephric abscess, 232
    in peritonitis, 226
    in prosthetic valve infective endocarditis, 220
    in sinusitis, 208
    in transplant-related pneumonia, 214
Staphylococcus epidermidis, test selection for, in prosthetic valve infective endocarditis, 220
Staphylococcus saprophyticus
  test selection for
    in pyelonephritis, 232
    in urinary tract infection/cystitis/pyruria-dysuria syndrome, 230
    urinalysis in identification of infection caused by, 30
Starvation
  cortisol levels in, 76
  growth hormone levels in, 99
  magnesium levels in, 123
  pH in, 137
  phosphorus levels in, 138
  total iron-binding capacity in, 116
  urine osmolality in, 133
Steady state, of drug level, therapeutic drug monitoring and, 189
Steatorrhea
  phosphorus levels in, 138
  urinary calcium levels in, 65
  vitamin B₁₂ levels in, 180
Steroid hormone-binding globulin, testosterone binding to, 165
Steroids. See also Corticosteroids
  metyrapone test in monitoring treatment with, 127
Steroids (cont.)
phosphorus levels affected by, 138
protein electrophoresis affected by, 146
protein levels affected by, 147
serum sodium levels affected by, 161
Still’s disease, ferritin levels in, 90
Stomach, upper GI study in evaluation of, 262
Stomach cancer (gastric cancer)
carcinoembryonic antigen levels in, 67
chorionic gonadotropin levels in, 72
fecal occult blood in, 89
α-fetoprotein levels in, 91
glucose levels in, 95
lipase levels in, 121
vitamin B12 levels in, 180
Stomatocytes, 29

Stool
Clostridium difficile enterotoxin in, 73
fat levels in, 88
leukocytes in. See Leukocyte(s), fecal occult blood in, 89
in anemia caused by blood loss, 363
screening for, 89
sampling
in antibiotic-associated pseudo-membranous colitis, 224
in encephalitis, 198
in gastritis, 222
in HIV-associated diarrhea, 225
in infectious colitis/dysentery, 223
in infectious myocarditis, 218
in liver abscess, 228
in pericarditis, 217
Storage battery workers, lead poisoning in, 119
Storage pool disease, platelet aggregation in, 139
Strep throat. See Pharyngitis
Streptobacillus moniliformis, test selection for, in bacterial/septic arthritis, 238

Streptococcus spp.
antistreptolysin O titer in infection caused by, 55
on Gram-stained smear, 27
Group A
beta-hemolytic, antistreptolysin O titer in infection caused by, 55
rapid tests for, 209
test selection for
in bacterial/septic arthritis, 238
in cellulitis, 240
in chorioamnionitis/endometritis, 236
in community-acquired pneumonia, 212
in empyema, 216
in necrotizing fasciitis, 240
in osteomyelitis, 237
in peritonitis, 226
in pharyngitis, 209
in vaginitis/vaginosis, 234

Group B, test selection for
in bacteremia of unknown source, 241
in bacterial meningitis, 200
in cellulitis, 240
in chorioamnionitis/endometritis, 236
in community-acquired pneumonia, 212
in empyema, 216
in osteomyelitis, 237
in otitis media, 206
in vaginitis/vaginosis, 234

Group C, test selection for
in cellulitis, 240
in pharyngitis, 209

Group D, test selection for, in bacterial meningitis, 200

Group G, test selection for, in cellulitis, 240
test selection for
in anaerobic brain abscess, 197
in anaerobic sinusitis, 208
in bacterial meningitis, 200
in cellulitis, 240
in empyema, 216
in endophthalmitis, 205
in infectious thrombophlebitis, 221
in infective endocarditis, 219
in necrotizing fasciitis, 240
in osteomyelitis, 237
in peritonitis, 226
in salpingitis/pelvic inflammatory disease, 236

Streptococcus pneumoniae, test selection for
in anaerobic pneumonia or lung abscess, 213
in aspiration pneumonia, 212
in bacteremia of unknown source, 241
in bacterial meningitis, 200
in brain abscess, 197
in community-acquired pneumonia, 212
in conjunctivitis, 204
in empyema, 216
in endophthalmitis, 205
in HIV-associated pneumonia, 214
in hospital-acquired pneumonia, 213
in infective endocarditis, 219
in keratitis, 205
in laryngotracheobronchitis, 210
in otitis media, 206
in pericarditis, 217
in peritonitis, 226
in sinusitis, 208
in transplant-related pneumonia, 214
*Streptococcus pyogenes*, test selection for
in brain abscess, 197
in cellulitis, 240
in community-acquired pneumonia, 212
in conjunctivitis, 204
in epiglottitis, 211
in impetigo, 239
in laryngitis, 209
in otitis externa, 207
in otitis media, 206
in pericarditis, 217
in pharyngitis, 209
in sinusitis, 208
Streptokinase, thrombin time affected by, 165
Streptolysin O antigen, antistreptolysin O titer for detection of, 55
Stress
glucose tolerance test in, 96
leukocyte count in, 400
luteinizing hormone levels in, 122
triglyceride levels in, 172
urinary free cortisol levels in, 77
Stress fractures, bone scan in identification of, 276
Stroke, partial pressure of oxygen in, 133
Strongyloides spp., test selection for
in infectious colitis/dysentery, 223
in transplant-related pneumonia, 214
Subarachnoid hemorrhage
cerebrospinal fluid profile in, 370
computed tomography in evaluation of, 245
prolonged QT interval in, 327
ST segment depression or T wave inversion in, 322
Subcutaneous emphysema, and low-voltage QRS complex in ECG, 308
Subendocardial injury
anterior, ST segment depression or T wave inversion in, 322
inferior, ST segment depression or T wave inversion in, 322
Subepithelial injury, inferior, ST segment depression or T wave inversion in, 322
Subvalvular dysfunction, diagnostic evaluation of, 398
Sudan stain, for fecal fat, 88
Sulfasalazine
heterophile agglutination (Monospot/Paul-Bunnell) test affected by, 107
methemoglobin levels affected by, 126
Sulfonamides
glucose-6-phosphate dehydrogenase deficiency and, 97
leukocyte count affected by, 400
Sulfonylureas
glucose levels affected by, 95
plasma levels of, in hypoglycemia evaluation, 349
surreptitious use of, 349
Sun exposure
25-hydroxy levels vitamin D₃ levels affected by, 182
lack of, 25-hydroxy vitamin D₃ levels affected by, 182
Sunburn, CD4/CD8 ratio in, 68
Superficial peroneal nerve, 341–342
Suppressor T cells (CD8 cells), 68
Supraventricular nerves, 341–342
Supraventricular tachycardia, paroxysmal, 298–299
Sural nerve, 341
Surgery
bacterial meningitis after, test selection in, 200
bacterial/septic arthritis after, test selection in, 238
cardiac, cardiac troponin-I levels in, 174
cellulitis after, test selection in, 240
cortisol levels with, 76
creatine kinase levels with, 78
osteomyelitis after, test selection in, 237
SVC (slow vital capacity), 385
Sweating, excessive
chloride levels affected by, 70
sodium levels affected by, 161
Swimmer’s ear, test selection in, 207
Sympathetic tone, increased, short QT interval in, 327
Syncope, with long QT syndrome, 327
Syndrome of inappropriate antidiuretic hormone
antidiuretic hormone levels in, 53
chloride levels in, 70
serum osmolality in, 132
sodium levels in, 161
uric acid levels in, 177
Synovial fluid sampling
in bacterial/septic arthritis, 238, 390
classification of findings in, 389t–390t
examination for crystals, 35–36, 37i, 389t
normal values in, 389t
specimen handling for, 26t
Synovioma, synovial fluid sampling in, 390t
Synovitis, synovial fluid sampling in, 389t–390t
Syphilis
central nervous system. See Neurosyphilis
fluorescent treponemal antibody-absorbed test in, 92, 391t
heterophile agglutination
(Monospot/Paul-Bunnell) test in, 107
IgG index in, 112
laboratory diagnosis of, in untreated patients, 391t
Lyme disease antibody test in, 122
microhemagglutination-Treponema pallidum (MHA-TP) test in, 129, 391t
oligoclonal bands in, 131
rapid plasma reagin test in, 150, 391t
urethritis and, 233
Venereal Disease Research Laboratory Test in, 391t
cerebrospinal fluid, 179, 371t
serum, 178
Syphilitic meningitis
cerebrospinal fluid profile in, 202, 371t
test selection in, 202
Systemic lupus erythematosus (SLE)
CD4/CD8 ratio in, 68
complement C3 levels in, 75
complement C4 levels in, 75
cryoglobulin levels in, 82
double-stranded DNA antibody levels in, 84, 367t
IgG levels in, 113
neonatal, SS-A/Ro antibody in, 163
nuclear antibody levels in, 131, 367t
rapid plasma reagin test in, 150
rheumatoid factor levels in, 155
ribonucleoprotein antibody levels in, 367t
Smith antibody in, 159, 367t
SS-A/Ro antibody in, 163, 368t
SS-B/La antibody in, 163
synovial fluid sampling in, 389t
Toxoplasma antibody test in, 171
Venereal Disease Research Laboratory Test in, 178

T
T cell lymphocytic leukemia, T cell receptor gene rearrangement in, 164
T cell lymphoma, T cell receptor gene rearrangement in, 164
T cell neoplasms, T cell receptor gene rearrangement in, 164
T cell receptor gene rearrangement, 164
T wave
classes and morphologies of, 323t
in hyperkalemia, 321t
in hypocalcemia, 325t
inversion
in anterior subendocardial injury or non-Q wave MI, 322t
antiarrhythmics and, 322t
catecholamines and, 322t
drugs causing, 322t–323t, 325t
in hypokalemia, 322t–323t, 325t
in inferior subendocardial injury, 322t
in left bundle branch block, 322t
in left ventricular hypertrophy, 322t
major causes of, 322t
in myocardial infarction, 322t
in posterior subepithelial injury, 322t
in right bundle branch block, 322t
in right ventricular hypertrophy, 322t
in subarachnoid hemorrhage, 322t
in left bundle branch block, 302
in left ventricular hypertrophy, 307
in myocardial infarction, 319
anterior, 312–313
inferior, 314
in myocardial ischemia, 310
normal, 299–300
in posterior myocardial injury or infarction, 315
in right bundle branch block, 301
T3. See Triiodothyronine
T4. See Thyroxine
Tachycardia
atrial, 288t, 299
with atrioventricular block, 285t
atrioventricular block with, 286–287
QRS duration in, 285t
atrioventricular reentry, 299
AV nodal reentry, 299
junctional, 289t
multifocal atrial, 287–288
irregularly irregular QRS rhythm in, 286
QRS complex in, 285
paroxysmal supraventricular, 298–299
R-R cycle lengthening in, 286
sinus, 287
QRS complex in, 285, 290
ventricular, 289
diagnosis of, 290–296
Brugada algorithm for, 293–295
Griffith method for, 295–296
quick method for, 290–292
QRS complex in, 285, 290–296
regularity of RR intervals in, 290
torsade de pointes, 296
wide QRS complex with regular rhythm (WCT-RR), 290–296
regularity of RR intervals in, 290

torsade de pointes, 296
wide QRS complex with regular rhythm (WCT-RR), 290–296
regularity of RR intervals in, 290

*Taenia solium*, test selection for
brain abscess in, 197
in parasitic meningoencephalitis, 203

Tangier disease
cholesterol levels in, 71
triglyceride levels in, 172

Target cells, 29

*TBG*. See *Thyroid-binding globulin*

99mTechnetium brain scan, for brain abscess, 197
99mTechnetium hexamethylpropyleneamine oxime white blood cell scan, 281
99mTechnetium methoxyisobutyl isonitrile (sestamibi) scan, myocardial, 257
99mTechnetium-methylene diphosphonate bone scan, in osteomyelitis, 237
99mTechnetium thyroid scan, in thyroid nodule evaluation, 361

TEE. See *Transesophageal echocardiography*

Temporal arteritis, erythrocyte sedimentation rate in, 86
Temporal bone disease, computed tomography in evaluation of, 245
Tenosynovitis, with bacterial/septic arthritis, 238
Teratocarcinoma, α-fetoprotein levels in, 91
Teratomas
chorionic gonadotropin levels with, 72
α-fetoprotein levels in, 91

Terfenadine, electrocardiography affected by, 326
Test selection, 195–241
Testicular agenesis, follicle-stimulating hormone levels in, 93
Testicular biopsy, in infertility evaluation, 352
Testicular failure, semen analysis in, 159
Testicular feminization, testosterone levels in, 165
Testicular torsion, epididymitis/orchitis differentiated from, 233
Testicular tumors/cancer
chorionic gonadotropin levels with, 72
α-fetoprotein levels in, 91
Testosterone
diurnal variations in, 165
serum levels of, 165
in hirsutism, 165, 346
Tetanus, creatine kinase levels in, 78
Tetracycline, outdated, and carbon dioxide levels, 66
Tetralogy of Fallot, brain abscess with, 197
Thalassemia syndromes, 392
basophilic stippling in, 364
bone marrow iron stores in, 364
ferritin levels in, 90, 364
fetal hemoglobin levels in, 103, 392
free erythrocyte protoporphyrin levels in, 364
hematocrit in, 101
hemoglobin A2 levels in, 101
hemoglobin electrophoresis in, 102, 392
hemoglobin levels in, 103
hemosiderin levels in, 104
iron-binding capacity in, 364
mean corpuscular hemoglobin in, 123–124, 363
mean corpuscular volume in, 124, 363–364
molecular diagnostic techniques for, 375–376
red cell morphology in, 363
reticulocytosis in, 363
serum iron levels in, 115, 363–364
transferrin saturation with iron in, 116, 364
Thallium scanning, myocardial, 257
Thawed plasma, 401
Thayer-Martin media, for bacterial culture, in pharyngitis, 209

Theophylline
therapeutic monitoring of, 188, 194
uric acid levels affected by, 177
Therapeutic drug monitoring, 187–190, 191t–194t
Therapeutic index/range
  narrow, need for drug monitoring with, 187
  reliability of, 189
Theta toxin, *Clostridium perfringens* producing, 239
Thiazide diuretics
  calcium levels affected by serum, 63
  urine, 65
  carbon dioxide levels affected by, 66
  glucose levels affected by, 95
  lithium levels affected by, 192t
  phosphorus levels affected by, 138
  serum osmolality affected by, 132
  sodium levels affected by, 161
  uric acid levels affected by, 177
Third-degree atrioventricular block, 297–298
  prolonged QT interval in, 327
Thoracentesis, in empyema, 216
Thoracic aortic dissection. See also Aortic dissection, evaluation of
Three-vessel disease, ST segment depression in, 316
Threshold approach to decision making, 16–17, 17i–18i
Throat swab
  in bacterial/septic arthritis, 238
  in encephalitis, 198
  in infectious myocarditis, 218
  in pericarditis, 217
  in pharyngitis, 209
Thrombophagocytosis, bleeding time in, 59
Thrombin, platelet aggregation by, 139
Thrombin time, 165, 377t
  inhibitor screen in evaluation of, 114
Thrombocythemia. See also Thrombocytosis
Thrombosis
  antithrombin III levels in, 56
  dural sinus, magnetic resonance venography in, 246
  factor V (Leiden) mutation in, 87, 373t
  fibrin D-dimer levels in, 91
  lupus anticoagulant and, 157
  protein C deficiency and, 145, 373t
  protein S deficiency and, 147
  ultrasound in evaluation of, 278
Thrombotic disorders, fibrin D-dimer levels in, 91
Thrombotic thrombocytopenic purpura
  lactate dehydrogenase levels in, 117
  plasma transfusion in, 401t
Thromboxane synthetase deficiency, platelet aggregation in, 139
Thyroglobulin
  antibody
    serum levels of, 166, 393t
    thyroglobulin levels affected by, 166
Thyroid
  imaging test selection and interpretation in evaluation of, 249–250
  metastases to, total body scanning in postoperative evaluation of, 250
Thyroid-binding globulin
  congenital absence of, total thyroxine levels in, 169
  decreased
    total thyroxine levels in, 169
    total triiodothyronine in, 173
increased
  total thyroxine levels in, 169
  total triiodothyronine in, 173
Thyroid carcinoma. See also Thyroid nodule
calcitonin levels in, 62
MIBG (metaiodobenzyl-guanidine) in
evaluation of, 272
radionuclide thyroid therapy for, 251
thyroglobulin antibody in, 166
thyroglobulin levels in, 166
treatment of, thyroglobulin levels in
monitoring of, 166
Thyroid function tests, 393–394
Thyroid medication, thyroid-stimulating
hormone levels in monitoring of, 168
Thyroid nodules
diagnostic algorithm for, 361i
evaluation of
  magnetic resonance imaging in, 248
  thyroid uptake and scan in, 250, 361i
  ultrasound in, 249
thyroid function tests in, 393t
Thyroid radionuclide therapy, 251
calculation of dosage, thyroid uptake
and scan in, 250
Thyroid-releasing hormone test, 393t
in hypothyroidism evaluation, 351i, 393t
Thyroid-stimulating hormone, serum
levels of, 168
in amenorrhea, 339i
in hyperthyroidism, 168, 393t
in hypothyroidism, 168, 351i, 393t
in patients on replacement therapy, 393t
Thyroid-stimulating hormone receptor
antibody, serum levels of, 169, 393t
Thyroid suppressive therapy
thyroid uptake and scan in, 250
ultrasound in, 249
Thyroid uptake and scan, 250, 393t–394t
for thyroid nodule evaluation, 250, 361i
Thyroidectomy, thyroglobulin levels after,
166
Thyroiditis
complement C3 levels in, 75
radiation, 251
thyroglobulin antibody in, 166, 393t
thyroglobulin levels in, 166
thyroid uptake and scan in, 250
thyroperoxidase antibody in, 167, 393t
Thyroperoxidase antibody, serum levels
of, 167, 393t
Thyrotoxicosis. See Hyperthyroidism
Thyrotropin. See Thyroid-stimulating hor-
monem
Thyroxine
  free, 169–170
  therapy with, for thyroid nodule, 361i
  total serum levels of, 169
Thyroxine index, free, 169–170
Tick(s), exposure to, tularemia associated
  with, test selection in, 175
Tick-borne encephalitis virus, test selec-
  tion for, in encephalitis, 198
Tick-borne relapsing fever, Lyme disease
  antibody levels in, 122
Tinea versicolor, KOH preparation in
  identification of, 33–35
Tinsdale agar, for bacterial culture, in
  pharyngitis, 209
TIPS. See Transjugular intrahepatic por-
otosystemic shunt procedure
Tissue culture, in antibiotic-associated
  pseudomembranous colitis, 224
Tissue damage, potassium levels in, 143
Tissue necrosis, lactate dehydrogenase
  levels in, 117
Tissue plasminogen activator, thrombin
time affected by, 165
Tissue trauma. See also Trauma
  magnesium levels in, 123
TLC (total lung capacity), 385t
Tobramycin
  amikacin levels affected by, 191t
  therapeutic monitoring of, 194t
Tolazamide, urine osmolality affected by,
  133
Tolbutamide, and glucose levels, 95
Tonsillitis, streptococcal, antistreptolysin
  O titer in, 55
Torsade de pointes, 296, 326
  clinical correlations of, 296
Total cholesterol, 71
Total iron-binding capacity, 116
  in anemias, 116, 363t–364t
Total lung capacity (TLC), 385t
Total parenteral nutrition, with inadequate
  replacement, magnesium levels
  in, 123
Total serum protein, 147, 378t
  in hyponatremia, 350t
Total thyroxine, 169
Total triiodothyronine, 173
Tourniquet, prolonged use of lactate levels affected by, 118 potassium levels affected by, 143
Toxicity, drug, therapeutic monitoring and, 188
Toxins electrocardiography affected by, 326 and renal tubular acidosis, 388
Toxo. See Toxoplasma antibody
Toxoplasma antibody (Toxo), serum or cerebrospinal fluid levels of, 171
Toxoplasmosis (Toxoplasma gondii infection) heterophile agglutination (Monospot/Paul-Bunnell) test in, 107
test selection for
in brain abscess, 197 in encephalitis, 198 in infectious myocarditis, 218 in parasitic meningoencephalitis, 203
Toxoplasma antibody test in, 171
Tracheitis, bacterial, endoscopy in, 210
Tracheobronchitis, test selection in, 210
Tracheostomy set, in epiglottitis, 211
Transbronchial biopsy, in Pneumocystis carinii pneumonia, 214
Transcatheter embolotherapy, of hepatic malignancy, hepatic angiography in evaluation of, 271
Transesophageal echocardiography in infective endocarditis, 219 in prosthetic valve infective endocarditis, 220
Transferrin electrophoresis in detection of, 146 saturation with iron, 115–116, 364 total iron-binding capacity calculated from, 116
Transfusion ABO grouping for, 44, 401t–402t action of, 401t–402t anti-D antibody formation and, 154 antibody screen for, 53 blood components for, 401t–402t calcium levels affected by, 63 ionized, 64 contraindications to, 401t–402t haptoglobin levels affected by, 99 hazards of, 401t–402t hematocrit in evaluation of need for, 101 hepatitis after, hepatitis C antibody screening in prevention of, 106 indications for, 401t–402t indirect antiglobulin test for, 54 magnesium levels affected by, 123 multiple, serum iron levels in, 115 phosphorus levels affected by, 138 platelet count affected by, 140 precautions in, 401t–402t prothrombin time affected by, 148 purpura after platelet-associated IgG in, 141 platelet count in, 140 rate of infusion, 401t–402t Rh grouping for, 154 total iron-binding capacity affected by, 116 type and cross-match for, 44, 53–54, 154, 175 type and screen for, 44, 53, 176 Transfusion reaction, 401t–402t direct antiglobulin test in, 54 hemosiderin levels in, 104 Transient ischemic attack, atypical, carotid Doppler in, 278 Transient synovitis, differentiation from bacterial/septic arthritis, 238 Transjugular intrahepatic portosystemic shunt (TIPS) procedure hepatic angiography before, 271 ultrasound for evaluation of, 278 Transplant recipients, pneumonia in, test selection in, 214 Transplantation. See also specific type or organ cytomegalovirus antibody screening for, 83 HLA typing for, 110 kidney pyelonephritis associated with, test selection in, 232 renal scan in evaluation of, 274 laryngitis in, test selection in, 209 liver hepatic angiography in preoperative evaluation for, 271 ionized calcium levels affected by, 64 pyelonephritis associated with, test selection in, 232 Transthoracic needle aspiration in anaerobic pneumonia or lung abscess, 213 in community-acquired pneumonia, 212 in mycobacterial pneumonia, 215 Transtracheal aspiration, in community-acquired pneumonia, 212
Transudates
ascitic fluid, characteristics of, 365
pleural fluid, characteristics of, 382
Transvaginal ultrasound, in salpingitis/
pelvic inflammatory disease, 236
Trauma
abdominal
angiography in, 279
computed tomography in, 259
mesenteric angiography in, 261
alanine aminotransferase levels in, 46
albumin levels in, 47
aspartate aminotransferase levels in, 56
bone scan in, 276
brain abscess after, test selection in, 197
cardiac troponin-I levels in, 174
cortisol levels in, 76
craniofacial, computed tomography in
evaluation of, 245
creatine kinase levels in, 78
creatine kinase MB isoenzyme levels in,
79
derangements in, magnetic resonance
imaging in, 278
endophthalmitis in, test selection in, 205
hepatic, hepatic angiography in, 271
magnesium levels in, 123
potassium levels in, 143
retropulsed bone fragments after, com-
puted tomography in, 277
synovial fluid sampling in, 389–390
urinary system, intravenous pyelogram
in, 273
Traumatic tap, cerebrospinal fluid profile
in, 370
Treponema pallidum
antibody
fluorescent treponemal antibody-
absorbed test for, 92, 202, 391
Lyme disease antibody and, 122
microhemagglutination test for, 129,
202, 391
infection
fluorescent treponemal antibody-
absorbed test for, 92, 202, 391
Lyme disease antibody test in, 122
meningitis
cerebrospinal fluid profile in, 202
test selection for, 202
microhemagglutination-Treponema
pallidum (MHA-TP) test in,
129, 202, 391
rapid plasma reagin test in, 150, 391
Venereal Disease Research Labora-
tory Test in
cerebrospinal fluid, 179
serum, 178
Triamterene
magnesium levels affected by, 123
potassium levels affected by, 143
Trichinella spiralis, test selection for, in
infectious myocarditis, 218
Trichinosis, myocarditis in, test selection
in, 218
Trichomonads, in vaginal wet fluid prepa-
ration, 33, 35
Trichomonas vaginalis
urethritis caused by, test selection for,
234
vaginitis/vaginosis caused by
laboratory evaluation of vaginal
discharge in, 397
test selection for, 234
vaginal wet fluid preparation in, 33,
35
Trichophyton spp., KOH preparation in
identification of, 33–35
Tricuspid valve
regurgitation, diagnostic evaluation of,
399
stenosis, diagnostic evaluation of, 399
Tricyclic antidepressants, electrocardiog-
raphy affected by, 326
Trigeminal nerve, 342
Triglycerides, serum levels of, 172
in hyperlipidemia, 172, 379–380
Triiodothyronine, total serum levels of,
173
Trimethoprim-sulfamethoxazole
electrocardiography affected by, 326
potassium levels affected by, 143
Trophoblastic disease
chorionic gonadotropin levels with, 72
testosterone levels in, 165
Tropical disease, cold agglutinin levels in,
74
Tropical sprue, fecal fat levels in, 88
Troponin-I, cardiac, serum levels of, 174,
353
Trypanosoma cruzi, test selection for, in
infectious myocarditis, 218
Trypanosomiasis, cold agglutinin levels
in, 74
TSH. See Thyroid-stimulating hormone
TT. See Thrombin time
TTP. See Thrombotic thrombocytopenic
purpura
Tube feedings, serum osmolality affected by, 132
Tuberculosis. See also Mycobacterium tuberculosis
angiotensin-converting enzyme levels in, 52
HIV-associated, test selection in, 215
leukocyte count in, 400r
pleural fluid profile in, 382t
rheumatoid factor levels in, 155
Tuberculous enterocolitis, test selection in, 227
Tuberculous epididymitis, test selection in, 233
Tuberculous meningitis
cerebrospinal fluid profile in, 203, 369t
test selection in, 203
Tuberculous mycotic infections, synovial fluid sampling in, 389t
Tuberculous pericarditis, test selection in, 217
Tuberculous peritonitis
ascitic fluid profile in, 227, 365t
test selection in, 227
Tubular acidosis. See Renal tubular acidosis
Tubulointerstitial disease, renal tubular acidosis in, 388t
Tularemia
heterophile agglutination (Monospot/ Paul-Bunnell) test in, 107
Legionella antibody cross-reactivity in, 120
test selection in, in community-acquired pneumonia, 212
vaccination, Brucella antibody affected by, 60
Tularemia agglutinins, serum levels of, 175
Tumoral calcification, spinal, computed tomography in, 277
Tumors, leukocyte count in, 400r
TWAR strain. See Chlamydia pneumoniae
Two-dimensional echocardiography,
in valvular heart disease, 398t–399t
Tympanocentesis sampling, in otitis media, 206
Type and cross-match, 175
ABO grouping for, 44, 54, 175
antibody screen for, 53, 175
indirect antiglobulin test for, 54
Rh grouping for, 54, 154, 175
Type and screen, 176
ABO grouping for, 44, 53, 176
antibody screen for, 53, 176
Rh grouping for, 53, 176
Typhus, scrub, myocarditis in, test selection in, 218
U
U waves, 324
abnormal, 324
classes and morphologies of, 325t
drugs affecting, 324, 325t
in hypokalemia, 324, 325t
inverted, 324
normal, 324
Ulcerative colitis
arthritis associated with, synovial fluid sampling in, 389t
erthrocyte sedimentation rate in, 86
haptoglobin levels in, 99
neutrophil cytoplasmic antibody levels in, 130
Ulcers
decubitus, cellulitis associated with, test selection for, 240
peptic. See Peptic ulcer disease
Ulnar nerve, 341i–342i
Ultrasound
in abdomen evaluation, 258
in cholangitis/cholecystitis, 229
in diverticulitis, 228
in tuberculous peritonitis/enterocolitis, 227
in antibiotic-associated pseudomembranous colitis, 224
in chorioamnionitis/endometritis, 236
in epididymitis/orchitis, 233
in gallbladder evaluation, 266
in genitourinary tract evaluation, 273
in liver abscess, 228
in liver evaluation, 267
in neck evaluation, 249
in osteomyelitis, 237
in pancreas evaluation, 272
in parathyroid evaluation, 249
in pelvis evaluation, 275
in pulmonary embolism, 357i
in pyelonephritis, 232
in salpingitis/pelvic inflammatory disease, 236
in thyroid evaluation, 249
in vasculature evaluation, 278
Upper aerodigestive tract, evaluation of computed tomography in, 249
magnetic resonance imaging in, 248
Upper GI study, 262
Urate crystals
  in synovial fluid, 36, 36i, 389t
  urinary color affected by, 30
Urea cycle metabolic defects, ammonia levels in, 51
Urea nitrogen, blood. See Blood urea nitrogen
Urea stabilizing test, in factor XIII deficiency, 377t
Ureaplasma urealyticum, test selection for in chorioamnionitis/endometritis, 236
  in urethritis, 233
Uremia
  amikacin levels affected in, 191t
  cerebrospinal fluid profile in, 371t
  digoxin levels affected in, 192t
  gentamicin levels affected in, 192t
  glycohemoglobin levels affected by, 98
  hemostatic function tests in, 377t
  insulin levels in, 115
  lidocaine levels affected in, 192t
  lithium levels affected in, 192t
  methotrexate levels affected in, 193t
  phenytoin levels affected in, 193t
  platelet aggregation in, 139, 377t
  procainamide levels affected in, 193t
  testosterone levels in, 165
  theophylline levels affected in, 194t
  valproic acid levels affected in, 194t
  vancomycin levels affected in, 194t
Ureteral calculi, computed tomography in, 259
Ureters, x-ray of (KUB plain radiograph), 258
Urethral discharge sampling
  in epididymitis/orchitis, 233
  in salpingitis/pelvic inflammatory disease, 236
  in urethritis, 233
Urethritis
  gonococcal, test selection in, 233
  nongonococcal, test selection in, 233
Uric acid, serum levels of, 177
Urinalysis, 28–33
  dipstick testing for, 30, 31t–32t
  in diverticulitis, 228
  in epididymitis/orchitis, 233
  in perinephric abscess, 232
  postejaculate, in infertility evaluation, 352t
  in prostatitis, 231
  in pyelonephritis, 232
  specimen collection for, 28
  specimen handling for, 26t, 28–30
  in urinary tract
    infection/cystitis/pyuria-dysuria syndrome, 230
Urinary free cortisol test, 77
Urinary tract infection
  ammonia levels in, 51
  test selection in, 230
Obstruction
  blood urea nitrogen levels in, 60
  creatinine levels in, 80
  trauma, intravenous pyelogram in, 273
Urinary tract infection
  ammonia levels in, 51
  test selection in, 230
Urinary tract infection
  ammonia levels in, 51
  test selection in, 230
Obstruction
  blood urea nitrogen levels in, 60
  creatinine levels in, 80
  trauma, intravenous pyelogram in, 273
Urine
  cast of, 32, 387t, 395t–396t
  color and clarity of, 30
  composition of, in common disease states, 395t–396t
  daily volume of, 395t–396t
  microscopic examination of, 32–33, 34i
  osmolality of, 133
    normal random, with average fluid intake, 133
    in renal failure/disease, 387t
  pH of
    dipstick testing of, 30, 31t
    in renal tubular acidosis, 388t
  sediment of, in renal failure/disease, 387t
  specific gravity of, 395t–396t
    dipstick testing of, 30, 31t
  turbidity of, 30
Urine volume, residual, ultrasound in evaluation of, 273
Uroepithelial neoplasm, intravenous pyelogram in evaluation of, 273
Urokinase, thrombin time affected by, 165
Urticaria, cryoglobulins causing, 82
Uterus
  cancer of
    choriocarcinoid tropin levels in, 72
    magnetic resonance imaging in, 273
Uterus (cont.)

- enlarged, ultrasound in evaluation of, 275
- leiomyomas of, red cell volume in, 151

V

Vaccination

- cholera, Brucella antibody affected by, 60
- pertussis, encephalitis after, test selection in, 198
- Q fever, Q fever antibody levels affected by, 149
- rabies, encephalitis after, test selection in, 198
- rubella, rubella antibody titer affected by, 156
- tularemia, Brucella antibody affected by, 60

Vaginal bleeding, ultrasound in evaluation of, 275

Vaginal cancer, magnetic resonance imaging in, 275

Vaginal discharge/secretions

- amniotic fluid contaminated by, lecithin/sphingomyelin ratio affected by, 119
- laboratory evaluation of, 397t
  - in mucopurulent cervicitis, 235, 397t
  - in vaginitis/vaginosis, 234, 397t

Vaginal fluid KOH preparation, 33–35, 397t

- positive, 36i
  - in vaginitis/vaginosis, 33–35, 35i, 234, 397t

Vaginal fluid wet preparation, 33

- positive, 35i
  - in vaginitis/vaginosis, 33, 35i, 234

Vaginitis

- atrophic, test selection in, 234
- laboratory evaluation of vaginal discharge in, 234, 397t
- test selection in, 234

Vaginosis, bacterial (Gardnerella vaginallis-associated)

- laboratory evaluation of vaginal discharge in, 234, 397t
- test selection in, 234
- vaginal fluid KOH preparation in, 33, 234, 397t
- vaginal fluid wet preparation in, 33, 35i, 234

Vagotomy

- antrectomy with, gastric levels affected by, 95
- gastric emptying study in, 265
- glucose tolerance test in, 96

Valproic acid, therapeutic monitoring of, 194t

Valvular heart disease, diagnostic evaluation of, 398–399t

Vancomycin, therapeutic monitoring of, 194t

Vanillylmandelic acid, urinary levels of, 178

Varicella-zoster infection, encephalitis after, test selection for, 198

Varicella-zoster virus, test selection for in aseptic meningitis, 199

- in conjunctivitis, 204
- in epididymitis/orchitis, 233
- in impetigo, 239
- in infectious esophagitis, 222
- in keratitis, 205

Varices

- esophageal, upper GI study in evaluation of, 262
- fecal occult blood in, 89

Vascular ectasia, fecal occult blood in, 89

Vascular malformations, hepatic angiography in, 271

Vascular occlusion, pulmonary angiography in, 255

Vascular purpura, cryoglobulins causing, 82

Vascular surgery, baseline prior to, carotid Doppler in, 278

Vascular tumors, blood supply to, magnetic resonance angiography of, 246

Vasculature, imaging test selection and interpretation in evaluation of, 278

Vasculitides, pulmonary angiography in, 255

Vasculitis

- evaluation of, mesenteric angiography in, 261
- neutrophil cytoplasmic antibody levels in, 130, 368t
- SS-A/Ro antibody in, 163, 368t

Vasectomy, semen analysis after, 159

Vasopressin. See Antidiuretic hormone

VDRL. See Venereal Disease Research Laboratory Test
Veillonella spp., test selection for, in anaerobic pneumonia or lung abscess, 213
Vein graft donor site, cellulitis at, test selection for, 240
Veins, patency of, ultrasound in evaluation of, 249
Venereal Disease Research Laboratory Test, 391

tcerebrospinal fluid, 179
for spirochetal meningitis/neurosyphilis, 202, 371
in salpingitis/pelvic inflammatory disease, 236
serum, 178
for spirochetal meningitis/neurosyphilis, 202
in urethritis, 233
Venography, magnetic resonance, 246
Venous sampling, in pheochromocytoma evaluation, 355
Venous thrombosis. See Thrombosis
Ventilation
alveolar, decreased, pH in, 137
artificial, excessive, pH in, 137
Ventilation-perfusion mismatch, partial pressure of oxygen in, 133
Ventilation-perfusion scan, 253
in pulmonary embolism, 356–357
Ventricular aneurysm, ST segment elevation in, 320
Ventricular complexes, definition of, 289
Ventricular conduction, aberrant, 286
Ventricular hypertrophy, 306–308. See also Left ventricular hypertrophy; Right ventricular hypertrophy
Ventricular preexcitation, 305–306
Ventricular rhythms, 289
accelerated, 289
QRS duration in, 285
escape, 289
QRS duration in, 285
major types of, 289
Ventricular shunt patency, cisternography in evaluation of, 247
Ventricular tachycardia, 289
diagnosis of, 290–296
Brugada algorithm for, 293–295
Griffith method for, 295–296
quick method for, 290–292
QRS complex in, 285
regularity of RR intervals in, 290
torsade de pointes, 296, 326
Ventriculography, radionuclide, 257
Verapamil
digoxin levels affected by, 192
phosphorus levels affected by, 138
Very low density lipoprotein (cholesterol), serum levels of, in hyperlipidemia, 71, 379–380
Vesicle culture, in aseptic meningitis, 199
Vibrio spp., test selection for, in infectious colitis/dysentery, 223
in otitis externa, 207
Vibrio vulnificus, test selection for, in cellulitis, 240
Videofluoroscopy, in aspiration pneumonia, 213
Vincristine, urine osmolality affected by, 133
Viral culture
in keratitis, 205
in laryngotracheobronchitis, 210
Viral keratitis, test selection in, 205
Viridans streptococci, test selection for, in bacteremia of unknown source, 241
in bacterial/septic arthritis, 238
in brain abscess, 197
in chorioamnionitis/endometritis, 236
in endophthalmitis, 205
in infective endocarditis, 219
in osteomyelitis, 237
in prosthetic valve infective endocarditis, 220
in sinusitis, 208
Virilization, testosterone levels in, 165
Virus(es). See also specific virus infections
leukocyte count in, 400
β2-microglobulin levels in, 128
rheumatoid factor levels affected by, 155
test selection for
in community-acquired pneumonia, 212
in encephalitis, 198
in laryngitis, 209
in pericarditis, 217
in pharyngitis, 209
in sinusitis, 208
Visceral ischemia, angiography in, 279
Vitamin A intoxication, calcium levels in, 63
Vitamin B₆ deficiency (pyridoxine deficiency)
alanine aminotransferase (ALT) levels in, 46
Vitamin B6 deficiency (pyridoxine deficiency) (cont.)
aspartate aminotransferase levels in, 56
transferrin saturation with iron in, 116
Vitamin B12 deficiency
folic acid levels in, 93
hematocrit in, 101
hemoglobin levels in, 103
laboratory and clinical findings in, 363
lactate dehydrogenase levels in, 117
leukocyte count in, 121, 400
mean corpuscular volume in, 124
methylmalonic acid levels in, 126
recovery from, reticulocyte count in, 153
vitamin B12 absorption test (Schilling’s test) in, 180–181
vitamin B12 levels in, 180
serum levels of, 180
Vitamin B12 absorption test (Schilling’s test), 180–181
Vitamin C, glucose-6-phosphate dehydrogenase deficiency and, 97
Vitamin D
calcium levels affected by, 63, 347
deficiency
calcium levels in, 63
1,25-dihydroxy vitamin D3 levels in, 183
25-hydroxy vitamin D3 levels in, 182
intoxication/overdose/toxicity
calcium levels in, 347
1,25-dihydroxy vitamin D3 levels in, 183
25-hydroxy vitamin D3 levels in, 182
urine calcium levels in, 65
phosphorus levels affected by, 138
Vitamin D3
1,25-dihydroxy, serum or plasma levels of, 183, 347
25-hydroxy, serum or plasma levels of, 182, 347
Vitamin K deficiency
protein C levels in, 145
protein S antigen levels in, 147
prothrombin time in, 148
Vitiligo, smooth muscle antibody levels in, 160
VLDL. See Very low density lipoprotein
Volume contraction, carbon dioxide levels in, 66
Volume depletion, pH in, 137
Volume of distribution, and therapeutic drug monitoring, 189
Volume overload, carbon dioxide levels in, 66
Volvulus, Hypaque enema in evaluation of, 264
Vomiting
chloride levels affected by, 70
hematocrit affected by, 101
hemoglobin levels affected by, 103
pH affected by, 137
phosphorus levels affected by, 138
potassium levels affected by, 143
serum osmolality affected by, 132
sodium levels affected by, 161
von Willebrand’s disease
activated clotting time in, 73
bleeding time in, 59, 377
cryoprecipitated anti-hemophilic factor for, 402
factor VIII assay in, 88, 377
hemostatic function tests in, 377
partial thromboplastin time in, 136
platelet aggregation in, 139
von Willebrand’s factor protein levels in, 184
von Willebrand’s factor protein, immunologic, plasma levels of, 184

W
Waldenström’s macroglobulinemia
cold agglutinin levels in, 74
cryoglobulin levels in, 82
IgM levels in, 113
immunoelectrophoresis in, 112
protein electrophoresis in, 146
rheumatoid factor levels in, 155
Walnuts, 5-hydroxy-indoleacetic acid levels affected by, 111
Warfarin
partial thromboplastin time affected by, 136
protein C levels affected by, 145
protein S antigen levels affected by, 147
prothrombin time for monitoring therapy with, 148, 188
Water balance, antidiuretic hormone release and, 53
Water intake, inadequate
serum osmolality in, 132
sodium levels affected by, 161
Water intoxication
chloride levels in, 70
sodium levels in, 161
Watson-Schwartz test, 142
Waveforms, cardiac, morphological diagnosis of, electrocardiography in, 284, 299–330

WBC count. See Leukocyte count

WCT-RR (wide QRS complex tachycardia with regular rhythm), 290–296

Wegener’s granulomatosis, neutrophil cytoplasmic antibody levels in, 130, 368t

Welders, lead poisoning in, 119

Wenckebach atrioventricular block, 287, 297

Western blot analysis
  for HIV antibody, 110
  for Lyme disease antibody, 122
  for neuroborreliosis, 202
  for spirochetal meningitis, 202

Western equine encephalitis, test selection in, 198

Whipple’s disease, glucose tolerance test in, 96

White blood cells. See Leukocyte(s)

Whole blood, 401t

Whooping cough, test selection in, 210

Wide QRS complex tachycardia with regular rhythm (WCT-RR), 290–296
  atrioventricular dissociation with, 290, 295

Wilson’s disease (hepatolenticular degeneration)
  ceruloplasmin levels in, 69
  Kayser-Fleischer rings in, 69
  urinary calcium levels in, 65
  Wiskott-Aldrich syndrome, IgG levels in, 113

Wolff-Parkinson-White patterns, 305–306, 310, 320t, 329–330
  left lateral accessory pathway, 330
  posteroseptal accessory pathway, 330
  Wolff-Parkinson-White syndrome, anterograde conduction of atrial fibrillation in, 285t

WPW. See Wolff-Parkinson-White patterns; Wolff-Parkinson-White syndrome

Wright stain
  microscopic examination of, 27–28, 29i
  of peripheral blood smear, 27–28, 29i
  preparation of smear for, 27
  technique for, 27

X

X-ray
  abdominal, 258
    in diverticulitis, 228
  bone, in osteomyelitis, 237
  chest, 252
    in HIV-associated tuberculosis, 215
    in liver abscess, 228
    in Pneumocystis carinii pneumonia, 214
    in pulmonary embolism, 356i–357i
    in valvular heart disease, 398t–399t
  KUB (kidneys, ureters, bladder), 258
  neck, in epiglottis, 211

Xanthelasma, in hyperlipidemia, 379t

Xanthine oxidase deficiency, uric acid levels in, 177

Xanthine oxidase inhibitor, uric acid levels affected by, 177

Xanthomas, in hyperlipidemia, 379t–380t

Xerocytosis, mean corpuscular hemoglobin concentration in, 124

D-Xylose absorption test, 185

Y

Yeasts
  culture, in prosthetic valve infective endocarditis, 220
  on Gram-stained smear, 27, 28i
  KOH preparation in identification of, 33–35, 36i

Yellow-top tubes, 42

Yersinia enterocolitica
  infection, Brucella antibody in, 60
  test selection for
    in HIV-associated diarrhea, 225
    in infectious colitis/dysentery, 223

Yersinia pestis, Legionella antibody cross-reaction with, 120

Z

Zollinger-Ellison syndrome
  calcitonin levels in, 62
  gastrin levels in, 95

Zygomycetes, test selection for, in sinusitis, 208