THIRTEENTH EDITION

# PHYSICIAN'S HANDBOOK


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# Foreword

Since its first appearance in 1941, the Physician's Handbook has been revised every two years to include selected items of medical information as they became established as useful among physicians and medical educators. Under the authorship of Drs. Krupp, Sweet, Jawetz, and Armstrong, the Handbook has continued to enjoy a wide acceptance among students and physicians both in this country and abroad. The original authors are grateful for this acceptance and recognize the obligation it imposes upon the present authors to sort and choose among the embarrassing profusion of new data and modifications of old with which every student and physician is confronted almost daily - in the classroom, the literature, and in clinical conferences. We know from experience that the periodic revision of this manuscript is not an effortless procedure.

With the appearance of the Thirteenth Edition we wish to express our deep regret at the passing of Dr. Charles D. Armstrong. Dr. Armstrong's sections of the Handbook have been revised for the new edition under the able authorship of Dr. Edward G. Biglieri.

John Warkentin
Jack D. Lange

June, 1964

# Preface

The Physician's Handbook is essentially a compilation in convenient pocket-size format of those diagnostic and therapeutic facts and procedures which the authors feel to be of greatest daily interest and value to the student and practitioner of medicine. For the Thirteenth Edition the Handbook has been critically reviewed and brought up to date; some chapters have been extensively reorganized and revised; and a new chapter on Medical Genetics has been added. As in past editions, all of the drug information has been reviewed by Frederick H. Meyers, MD, Professor of Pharmacology, University of California School of Medicine (San Francisco).

As in former editions, the authors have drawn freely upon the skills of their colleagues in preparing this revision. The list of those whose advice and criticism has substantially contributed to the success of this Handbook has become too extensive to permit individual acknowledgement in the space available. Let each of them accept our sincere appreciation for helping us in the effort to maintain the usefulness of this volume.

Marcus A. Krupp
Norman J. Sweet
Ernest Jawetz
Edward G. Biglieri

June, 1964

## THE CLINICAL EXAMINATION

The medical history is of first importance in the clinical examination. Symptoms not only stimulate the patient to see the physician but also provide information and clues which are more likely than any other aspect of the examination to suggest or establish a diagnosis. Next in importance is observation of the patient in the course of a careful physical examination. The laboratory examination, although important, is merely an extension of the clinical examination. It goes without saying that all components of the clinical examination must be carefully performed and correctly interpreted and correlated.

## ROUTINE LABORATORY EXAMINATION*

**Urine.**

A freshly voided sample should be used for examination. Observe for color and appearance (see p. 100); test for reaction (pH) (see p. 102), specific gravity (see p. 103), and glucose (see p. 105). Examine unstained sediment after centrifuging urine sample for five minutes (see p. 116). If bacteria are present, note motility and check staining characteristics with Gram's stain.

**Blood.**

RBC (see p. 137) or hematocrit (see p. 141), WBC (see p. 147), Hgb. (see p. 136), differential smear (see p. 148), and sedimentation rate (see p. 169). Hematocrit should be taken on patients with anemia.

**Serologic Tests for Syphilis.**

Flocculation and complement fixation tests (see p. 298).

**Feces.**

Observe gross appearance (see p. 267); test for occult blood (see p. 269). Examine microscopically if indicated (see p. 270).

**Chest X-rays.**

These should be included as part of a complete physical examination. Both anteroposterior and lateral views should be taken. In routine screenings a "minifilm" may suffice.

**Pregnancy Routine.**

Laboratory examination should include complete blood count, urinalysis, serologic test for syphilis, and Rh typing (see p. 165). Diagnostic tests may be performed (see p. 235).

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*A chapter on Simplified Laboratory Procedures may be found on pp. 93 to 99.

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# 1...

# Emergency Medical Examination

The examination of the injured or unconscious patient or the patient suffering from shock, hemorrhage, or acute respiratory distress must proceed simultaneously with life-saving treatment. Detailed histories are often not available; diagnosis depends principally upon physical examination and to a lesser extent on laboratory procedures or radiologic examination.

All patients with a history or findings of trauma must be examined for internal and external injuries.

## INITIAL EMERGENCY EXAMINATION AND MANAGEMENT

**Initial Management.**

A. Emergency Measures:
   1. Stop hemorrhage with pressure or tourniquet.
   2. Relieve asphyxia -
      a. Clear the upper air passages -
         (1) Pull tongue forward; insert an airway.
         (2) Suction mucus or blood.
         (3) If indicated, insert an open type of endotracheal tube or perform tracheostomy.
      b. Close sucking wounds of thorax.
      c. Institute artificial respiration if necessary.
      d. Administer oxygen if necessary.
   3. Avoid spinal cord damage in any patient suspected of back injury. Maintain normal alignment of the vertebrae both in examination and transportation.

B. Follow-up Measures:
   1. Examine for concealed hemorrhage into the thorax, abdomen, gastrointestinal tract, or soft tissues.
   2. If indicated, arrange for administration of plasma, plasma substitutes, or blood.
   3. Splint fractures before moving patient.
   4. Relieve pain - Narcotics and sedatives may mask pain of diagnostic importance. Use narcotics cautiously in the presence of shock because of the danger of sudden absorption of multiple doses on recovery.
   5. Insert an adequate intravenous cannula for later medication and fluids. Suture a plastic tube into a vein and connect it to a 2 ml. syringe filled with heparin. Fill the tube with heparin and tape syringe to the extremity.

**Initial Rapid Survey Examination.**

A. History: (From relatives, friends, bystanders, police.)
   1. Present illness - Prodromes, onset, recent illness, progression of symptoms. In case of injury, elicit exact details.
   2. Past history - Previous attacks, chronic illness, habits, medications, occupation. Examine patient's personal effects (diabetic or epileptic identification card, prescription labels, etc.).

B. Physical Examination:
   1. Note position of body and extremities, evidence of external or internal bleeding, skin color, rate and quality of pulse and respiration, temperature, BP, state of consciousness, and unusual odors. Note neck vein distention; check pulsation in major arteries, paradoxic pulse.
   2. Head and neck - Injuries of skull and face, neck rigidity, breath aroma, appearance of pupils and reaction to light, ophthalmoscopic examination in coma, fluid or blood from ears or nose, mucous membranes of mouth, position of trachea, crepitation in the neck.
   3. Chest - External evidence of injury, sucking wounds, gentle compression for rib fractures, flail chest, percussion and auscultation for evidence of fluid, quality of breath sounds, loud murmurs, cardiac arrhythmias, increase in area of cardiac dullness.
   4. Abdomen - External evidence of injury, presence of rigidity or tenderness, bowel sounds. Percuss bladder.
   5. Back - Examine for injuries. Maintain normal alignment of vertebrae.
   6. Extremities - Note position and color of extremities; ask patient to move extremities, or move passively and palpate gently for evidence of fracture. Compress the wings of the ileum, and palpate the symphysis pubis. Examine the perineum.
   7. Neurologic - Deep and superficial reflexes, flaccidity or rigidity; response to pinprick or noxious stimuli.

C. Record initial and serial findings. Diagnosis often depends on changing signs.

D. Record fluid intake and urinary output.

## COMMON CAUSES OF UNCONSCIOUSNESS

Rule out shock, hemorrhage, or asphyxia at once.

**Brain Disease.**

A. Cerebral Thrombosis: Older age group; develops slowly, commonly occurs at night; localizing neurologic signs; occasionally fever with leukocytosis; spinal fluid findings

are rare; check serology for C.N.S. syphilis; moderate glycosuria may occur in either thrombosis or hemorrhage.

B. Intracerebral Hemorrhage: Hemiplegia; develops more rapidly than thrombosis and is more apt to cause unconsciousness. Hypertension, fever, and leukocytosis are often present; spinal fluid under increased pressure, and frequently contains blood.

C. Subarachnoid Hemorrhage: Sudden occipital pain followed by unconsciousness, stiff neck, and bloody spinal fluid; hypertension common; localizing neurologic signs may be absent.

D. Meningitis: Severe headache, fever, stiff neck, facial flush, rapid pulse and respiration, purpuric rash (with meningococcemia), leukocytosis, abnormal spinal fluid findings, positive blood and spinal fluid culture (see chart on p. 252).

E. Epilepsy: History of epilepsy, "epileptic identification card"; tonic and clonic convulsions followed by marked muscular relaxation, incontinence, and spontaneous recovery; signs of injury due to falling; bitten tongue.

**Systemic Diseases.**

A. Diabetic Coma: History of diabetes, "diabetic identification card"; gradual onset, blurring of vision, thirst, air hunger (Kussmaul breathing), fever, rapid pulse, dehydration, soft eyeballs, retinopathy, acetone breath, red reduction (sugar) in the urine, elevated blood sugar and low serum $CO_2$ combining power. If unable to differentiate from insulin shock, give 50% glucose I.V.

B. Hypoglycemic Shock: History of diabetes mellitus and use of insulin; "diabetic identification card"; weakness, nervousness, trembling, confusion, rapid pulse, elevated BP. Blood glucose invariably below 50 mg./100 ml. No reducing substances (sugar) in the urine. Relieved by oral sweets or I.V. glucose (50 ml. of 50%).

C. Renal Azotemia (Uremia): History of nephritis; pallor; hardened arteries in a young person; deep, rapid respirations with urinous breath; hypertension; muscle twitching and convulsions; retinal hemmorrhages and exudates, papilledema; anemia; low fixed specific gravity of urine, with protein and casts; serum N.P.N. usually over 100 mg./100 ml.

D. Hepatic Coma: History of alcoholism and malnutrition; wasted appearance, jaundiced scleras, grayish hue to the skin, sickening sweet odor of the breath, deep and rapid respiration, spider angiomas, enlarged liver (sometimes small), abdominal collateral circulation, ascites, flapping tremor, edema.

**Drug Intoxication.**

A. Alcoholic Intoxication: Rule out other causes of coma. Alcohol breath, facial flushing, ocular injection, slow pulse and respiration; patients occasionally violent, may lose deep reflexes and develop positive Babinski.

B. Barbiturates: History or evidence of barbiturate ingestion, facial flushing, shallow or deep respirations; fever common; pupils usually moderately dilated; corneal and deep reflexes may be absent, with positive Babinski. Quantitative or qualitative serum or urine barbiturate tests are difficult (see p. 115).

C. Narcotics: Pinpoint pupils, very slow respiration, slow pulse, powder-blue spots along course of veins at sites of self-injection.

D. Bromides: Diagnosis fundamentally depends on thinking of the possibility and demonstration of high blood level. Toxic delirium common; striking cyanosis in chronic users of proprietary bromides because of combination with acetanilid; striking neurologic abnormalities with no localizing pattern occasionally occur; blood bromide level (see p. 115) usually 175 mg./100 ml. or more. Look for other causes of coma if blood level is under 100 mg./100 ml.

E. Carbon Monoxide Poisoning: History of exposure, scarlet lips and flushed cheeks, fever, 30 to 60% concentration of carboxyhemoglobin in the blood.

**Other Causes.**

A. Severe febrile illness.
B. Drug poisoning (other than above).
C. Brain tumor.
D. Hypertensive encephalopathy.
E. Fainting (simple syncope).
F. Asphyxia.
G. Eclampsia.
H. Severe heart failure.
I. C.N.S. syphilis.
J. Senile cachexia.
K. Malaria.
L. Heat stroke and prostration.
M. Encephalitis.
N. Addison's disease.

## HEMORRHAGE AND SHOCK

**Manifestations of Hemorrhage.**

Hemorrhage may be superficial and obvious or may occur within body cavities or visceral organs, in which case diagnosis may be difficult. Arterial bleeding may lead to rapid exsanguination, and calls for immediate control and blood replacement. Blood loss in venous oozing from large surfaces may be extensive, but is slower than arterial bleeding. The general picture of severe hemorrhage is that of weakness, pallor, cold sweat, apprehension, and thirst. The pulse rate is rapid; at first full, but progressively more feeble. The

BP may fall rapidly or may remain at normal levels for hours and then fall gradually or suddenly. Confusion and disorientation eventually occur, and progress to coma in irreversible states. Serial hematocrits, pulse rates, and blood pressure determinations are essential for prompt diagnosis of inapparent hemorrhages following trauma.

A. Intracranial Bleeding Following Trauma: Progressive increase in intracranial pressure; progressive disorientation leading to coma; focal neurologic findings, papilledema, ipsilateral pupil dilatation and fixation, elevated spinal fluid pressure (with xanthochromia or red cells), and eventually Cheyne-Stokes respiration. Fracture may be demonstrated on skull films.
B. Intrathoracic Bleeding Following Trauma: Signs of accumulation of intrapleural fluid (see p. 7).
C. Gastrointestinal Bleeding: Hematemesis, tarry stool, or a positive stool test for occult blood. (See pp. 97 and 269.)
D. Intraperitoneal Bleeding: Progressive abdominal tenderness, muscle spasm, abdominal distention, shifting dullness upon percussion, appearance of blood on needle aspiration of abdomen.

**Manifestations of Shock.** (See p. 405.)

Shock is characterized by inadequate circulating blood volume leading to diminished venous return and thus a diminished cardiac output. The result is widespread tissue anoxia. Shock may follow hemorrhage, infection, pain, extensive tissue damage, or metabolic disturbances; or cardiac, renal, or hepatic failure.

Diagnosis is based on the presence of any of the above etiologic entities and the typical signs, i.e., weakness, pallor, cold sweat, tachycardia with feeble pulse, fall in BP, collapse of superficial veins, and hemoconcentration with diminished blood volume.

## COMMON INJURIES FOLLOWING TRAUMA

**Head Injury.**

Skull films are mandatory for demonstration of possible skull fracture or pineal shift.

A. Cerebral Concussion: A functional disturbance without gross brain damage. Consciousness varies from slight daze to unconsciousness. Post-traumatic and occasionally retrograde amnesia may be present. Headache is almost always present. Dilated fixed pupils, areflexia, and shock may be present. Delirium and vomiting may occur on recovery of consciousness. The prognosis is good.

B. Cerebral Contusion: Diffuse brain damage occurs, characterized by edema and capillary hemorrhages. Unconsciousness is usual and is followed by a state of drowsiness or confusion lasting days or weeks. Headache, mental symptoms, and light-headedness are a characteristic triad which may persist indefinitely. Other symptoms depend upon the damage done and include convulsions, aphasia, cranial nerve palsies, and (rarely) hemiparesis. Focal lesions are not usual. Spinal fluid usually shows elevated pressure, xanthochromia, red blood cells, and increased protein.

C. Cerebral Compression: (Subdural or epidural hematoma.) History of head injury followed in hours, days, or months by fluctuating drowsiness and confusion progressing slowly to coma. Increased intracranial pressure is evidenced by coma, slowing of pulse and respiration, irregular breathing, and rising BP. Choked disks are infrequent. Localizing neurologic signs occur, including ipsilateral dilatation of the pupil. Spinal fluid pressure is increased, and fluid may be xanthochromic or blood-tinged. X-ray evidence of skull fracture and pineal shift is common. Avoid aspiration of spinal fluid in the presence of choked disks.

D. Skull Fracture: History of head injury, usually associated with evidence of external trauma. Fracture may occur with minimal brain injury or symptoms related thereto. Obvious depressed skull fractures require surgical management. Skull films are the most reliable diagnostic aid.

E. Penetrating Wounds: Minute point of entry may conceal extensive brain damage. Surgical exploration may be indicated.

**Chest Injury.**

External evidence of injury may not be present.

A. Rib Fracture: Localized pleuritic pain (sharp pain with breathing), localized severe pain with pressure at fracture site, or pain with compression of sternum or lateral chest. Pleural friction rub may be present. Examine for fluid (hemothorax) and pneumothorax.

B. Flail Chest: Crushing injury to the chest with multiple rib fractures results in separation of an area of chest wall which then functions independently of the rib cage proper. With inspiration, the segment is sucked in, which limits expansion of the lung on the involved side. Flail chest is characterized by pain, dyspnea, cyanosis, and paradoxic motion of the involved segment. Immediate immobilization is required.

C. Pneumothorax: Pneumothorax is classically characterized by dyspnea and cyanosis, chest lag, absence of

fremitus, hyperresonance, and absence of breath and voice sounds on the involved side. X-ray will confirm the diagnosis. The classical picture varies greatly with the amount of air. If fluid is also present, findings of pneumothorax predominate. There are three types: spontaneous, tension, and open.

1. Spontaneous pneumothorax - Rupture of bleb with leakage of air into the pleural cavity; source of leak seals spontaneously. Characterized by sudden onset of dyspnea and/or pleuritic pain; respiratory lag, hyperresonance, and absent breath and voice sounds on the involved side. Symptoms and signs do not progress.
2. Tension pneumothorax - Failure of the lung leak to seal results in increasing amount of air in the pleural space with each breath. This causes rapidly progressive dyspnea, cyanosis, and physical findings as noted above. Marked intrathoracic pressure may prevent hyperresonance. Tension pneumothorax may result from lung trauma (penetrating wounds) or may occur with spontaneous pneumothorax. Immediate measures must be taken to aspirate air and to allow continued egress of trapped air.
3. Open pneumothorax - Characterized by presence of an open wound, severe respiratory distress with cyanosis, audible sucking sounds, and ingress or egress of frothy, blood-tinged fluid with each breath. Opening must be closed **at once** with an emergency airtight bandage and the patient placed on the injured side.

D. Hemothorax: Signs of pleural fluid as evidenced by absent fremitus, loss of resonance, absent breath and voice sounds, and tracheal shift to the opposite side, together with general symptoms of hemorrhage following chest injury; may be associated with pneumothorax. Physical findings of pneumothorax may obscure those of hemothorax. Confirm by needle aspiration.

E. Penetrating Wounds of Chest: May be closed or open.
   1. Closed wounds - A minute point of entry may be associated with extensive intrathoracic damage. Check for rib fracture, pneumothorax, hemothorax, subcutaneous (palpation) or mediastinal emphysema (crunching sound with each heartbeat), and cardiac contusion.
   2. Open wounds inevitably produce critical pneumothorax; see above.

F. Cardiac Injury: May consist of simple contusion, a penetrating wound, valve rupture, or cardiac tamponade. Contusion may be associated with arrhythmia or nonspecific electrocardiographic findings. Rupture occurs most commonly in the aortic valve and is manifested by a loud, "cooing" diastolic murmur; signs of acute left heart failure may be present.

**Cardiac tamponade** due to blood in the pericardial sac progresses to limitation of diastolic filling of the heart with resultant progressive narrowing of pulse pressure, increase in pulse rate, paradoxic pulse, engorged neck veins, and eventually critically low cardiac output. Pericardial paracentesis may be life-saving (see p. 241).

**Nonpenetrating Abdominal Injuries.**

May be accompanied by varying degrees of shock, hemorrhage, or peritonitis. The nature and direction of injury should be ascertained. Serial examinations are imperative.

A. Liver Rupture: Manifestations are due to hemorrhage, shock, and possible bile peritonitis. Liver rupture is characterized by a history of injury followed immediately or after a few hours by right upper quadrant pain, tenderness, and signs of hemorrhage. Shock and rapid exsanguination may occur.

B. Splenic Rupture: Manifestations are due to hemorrhage and shock. Splenic rupture is characterized by a history of injury followed immediately or after days (subcapsular hemorrhage) by left upper quadrant and shoulder pain, rebound tenderness, muscle rigidity, signs of bleeding (including shifting dullness), a mass in the left upper quadrant, and shock. Spontaneous rupture may occur with malaria or infectious mononucleosis.

C. Intestinal Rupture: Manifestations are due to localized peritonitis or to gangrene of the bowel following a mesenteric tear with impairment of blood supply. Characterized by history of injury followed by symptoms due to peritonitis, anemia, or gangrene of bowel.

D. Kidney Rupture: Manifestations are due to perirenal bleeding and urinary extravasation or intrarenal bleeding. Characterized by history of injury followed by flank pain, hematuria, local costovertebral angle tenderness, swelling, muscle spasm, a palpable mass, non-shifting flank dullness, shock, and ecchymosis. An intravenous urogram is valuable for confirmation and to determine the extent of injury.

E. Bladder Rupture: Manifestations are due to local injury with intra- or extraperitoneal extravasation of urine or blood, which may become infected. Rupture is caused by trauma to a full bladder. Characterized by history of injury to the lower abdomen, followed by persistent pain, suprapubic tenderness, muscle spasm, and hematuria. Signs of free fluid in peritoneal cavity may occur. A boggy suprapubic mass may be felt or percussed. X-rays of the pelvis should be taken to determine if fracture has occurred. A simple procedure is to empty the bladder, instill 300 ml. of sterile saline solution, and measure the return. A cystogram is the most dependable test

for bladder injury: instill 350 ml. of sterile 5% sodium iodide and take anteroposterior and oblique views.

F. Urethral Rupture: Manifestations depend upon the segment of urethra involved; extravasation of urine or blood may be around the bladder, in the anterior abdominal wall, periprostatic, or perineal. An abdominal or perineal injury is followed by pain, blood at the urethral meatus, difficulty in voiding, and signs of extravasation (see above). Urethrograms (25 ml. radiopaque material instilled into urethra by catheter) should be taken to confirm and localize the site of rupture.

**Penetrating Abdominal Injuries.**

A minute entry wound may mask extensive internal damage. Penetrating abdominal wounds always require exploratory laparotomy. The patient should be stripped and carefully examined for entry and exit wounds and for evidence of associated injuries or bleeding contributing to shock. Symptoms, signs, and laboratory evidence of severe hemorrhage must be evaluated promptly so that life-saving surgery may be done - in spite of shock, if necessary.

The status of the patient depends upon (1) the organs involved, as suggested by the type and direction of the injury and specific symptoms and signs; (2) the severity of hemorrhage, shock, and peritonitis; (3) the time elapsed since injury; and (4) treatment already administered.

Symptoms and signs of specific organ involvement are reviewed above. Manifestations of hemorrhage and shock are reviewed on pp. 4 and 5.

**Fractures.**

A. General Features: Clinical manifestations of fracture include pain, local tenderness, ecchymosis, deformity due to swelling and bone displacement, impaired function, abnormal motion, and crepitus at the site of fracture. In some instances only pain is present. Simple inspection is often diagnostic. X-ray confirmation is mandatory. Evaluate sensory changes and voluntary motion of joints distal to the fracture for evidence of nerve damage. Check distal portion of extremity for evidence of impaired blood supply.

B. Spinal Injuries: Vertebral fractures and spinal cord injuries are suggested by the nature of the injury, back pain, and abnormal position or mobility of the neck, back, or extremities. All unconscious patients or those who complain of back pain should be treated as potential spinal cord injury cases. Every effort should be made to maintain the normal alinement of the spine both in examination and transportation. Never transport such patients in a sitting or semi-reclining position; use a flat

stretcher without a pillow. By asking the patient to move his toes, legs, and hands, one can roughly determine the presence of significant cord injury and its approximate location. Loss of sensation to pain will further identify the level of the cord injury.

## COMMON CAUSES OF ACUTE RESPIRATORY DISTRESS

**Heart Failure.**

A. Left Heart Failure: Symptoms depend upon the degree of failure. Paroxysmal nocturnal dyspnea and pulmonary edema may both occur as the result of acute left ventricular failure of varying degree. Acute left heart failure may occur with hypertensive cardiovascular disease, coronary artery insufficiency, mitral incompetence, and aortic insufficiency and/or stenosis.
   1. Paroxysmal nocturnal dyspnea - Manifested by a sudden sense of suffocation, usually during the night, whereupon the patient sits upright acutely short of breath. Cough is usually present, and wheezing on auscultation. Cold sweat and cyanosis are common. BP is usually elevated. The attack ceases spontaneously in 10-20 minutes.
   2. Acute pulmonary edema - Manifestations are the same as those of paroxysmal dyspnea except that rales are heard throughout the lung fields; frothy pink or white sputum is coughed up; and the episode may last for hours or until relieved by appropriate medications.

B. Right Heart Failure: Pure right heart failure is not accompanied by pulmonary congestion, and the respiratory distress is not as marked. In its pure form right failure results from pulmonary hypertension, pulmonary embolism, pulmonary stenosis, or left to right shunt. It is characterized by elevated venous pressure, enlargement and tenderness of the liver, and peripheral edema.

C. Congestive Heart Failure: Combined right and left heart failure. The clinical pictures of both are combined; pleural effusion is common.

**Acute Pulmonary Edema (Noncardiac).**

The clinical picture described above for pulmonary edema may occur without heart failure. Common causes are infectious pneumonitis, shock, and aspiration pneumonitis. Pulmonary embolism and irritant gases may also produce this clinical picture. Pulmonary edema due to these causes is not necessarily nocturnal and is not precipitated by the horizontal position. Duration and treatment depend upon the precipitating disease.

**Bronchospasm.**

A. Allergic (Bronchial Asthma): Usually begins in childhood. There may be a history of other allergic manifestations, and occurrence may be seasonal. Characterized by acute respiratory distress, use of accessory muscles of respiration, pinched facies, and bouts of nonproductive coughing. Auscultation reveals inspiratory and expiratory wheezing and rhonchi throughout, with forced, prolonged expiratory effort. Pulmonary emphysema may be present.

B. Nonallergic (Asthmatic Bronchitis): Usually occurs in elderly adults who have pulmonary emphysema, barrel chest, and a chronic cough. Scattered wheezes and rhonchi may be heard in the nonacute phase. The acute phase, usually precipitated by respiratory infection, is characterized by acute respiratory distress, spasms of dry cough, cyanosis, inspiratory and expiratory rhonchi and wheezing, and tachycardia.

**Pulmonary Embolism.** (See p. 414.)

Originates from a free-floating clot in the deep veins of the lower extremities or pelvis, or in the right heart. Factors contributing to clot formation are slowing of the circulation due to any cause, inflammatory changes in the vein wall, or increased coagulability of blood. Emboli may or may not produce infarction. Acute, severe respiratory distress usually occurs only with large emboli but may occur with smaller emboli if other causes of pulmonary or cardiac dyspnea are present also.

A. Large emboli are manifested by sudden severe dyspnea, severe substernal or generalized chest pain, sweating, pallor, cyanosis, abdominal discomfort, mental symptoms, and occasionally convulsions. Hypotension, a loud pulmonary second sound with a systolic murmur, and distended neck veins may be present. Death usually occurs in minutes or hours and before infarction occurs. The Ecg. occasionally shows a pattern of right ventricular strain. This clinical picture occurs most commonly in association with a major operative procedure, thrombophlebitis or phlebothrombosis, or with congestive heart failure (especially with atrial fibrillation).

B. A smaller embolus in a patient with congestive failure or pulmonary hypertension may produce a similar picture, a lesser degree of shock, tachycardia, development of infarct with pleuritic pain and rub, hemoptysis, and pulmonary effusion. The prognosis is better than in the case of the larger emboli.