Section IV - The Circulatory System.

1-29. The circulatory system is composed of the heart, blood vessels, lymphactic system and their contained fluids, blood, and lymph.

a. Arterial hypertension. Elevation of systolic and/or diastolic blood pressure, either primary (essential hypertension) or secondary. Although the etiology of essential hypertension is unknown, the family history is usually suggestive of hypertension (stroke, sudden death, heart failure). Secondary hypertension is associated with kidney disease (e.g., chronic glomerulonephritis or pyelonephritis), or occlusion of one or more of the renal arteries or their branches (renovascular hypertension). An untreated hypertensive patient is at great risk of developing fatal heart failure, brain hemorrhage, or kidney failure.

S. Primary hypertension is asymptomatic until complications arise. Complications include left ventricular failure; atherosclerotic heart disease; retinal hemorrhages, exudates, and vascular accidents; cerebral vascular insufficiency; and renal failure. Hypertensive encephalopathy due to cerebral vasospasm and edema is characteristic of hypertension.

O. Consistent diastolic pressure >100 mm. Hg in patients >60 years of age; diastolic pressure >90 mm. Hg in patients <50 years of age; or systolic pressure >140 mm. Hg regardless of age. Retinal changes will range from minimal arteriolar narrowing and irregularity to frank hemorrhages and papilledema, i.e., elevation of the optic disk or blurring of the disk margins.

A. A Dx of hypertension is not warranted in a patient under 50 years of age unless the B.P. exceeds 140/90 mm. Hg on at least three separate occasions after the patient has rested for 20 minutes or more in quiet and familiar surroundings. Secondary complications will present symptomatology of the "target organs" involved:

(1) Cardiac involvement often leads to nocturnal dyspnea or cardiac asthma (inspiratory and expiratory wheezing). Angina pectoris or myocardial infarction may develop.

(2) Renal involvement may produce nocturia and hematuria. The patient may have a uremic odor. Kidneys may be enlarged and palpable.

(3) Cerebral involvement will demonstrate neurological signs ranging from a positive Babinkşi or Hoffman reflex to paralysis.

(4) Peripheral arterial disease causes intermittent claudication (limping). If the terminal aorta is involved, pain in the buttocks and low back pain appear on walking and men become impotent.

P. Treat mild hypertension (diastolic pressure 90 to 110 mm. Hg) with an oral diuretic such as chlorothiazide (Diuril) 500 mg. b.i.d. If the diuretic does not control the hypertension, methyldopa (Aldomet) 250 mg. b.i.d. to 500 mg. q.i.d., or clonidine (Catepres) or reserpine 0.25 to 0.5 mg./day should be added. Methyldopa is preferred because its side effects are better tolerated. For moderate hypertension (diastolic pressure between 111 and 125 mm. Hg) start therapy with an oral diuretic and a sympathetic depressant (e.g., methyldopa, clonidine, reserpine, or propranolol). For severe hypertension (diastolic pressure >125 mm. Hg)

therapy should be started with an oral diuretic and guanethidine (10 mg. to 150 mg./day in a single dose) simultaneously. Methyldopa should be added if needed. Patients with acute severe hypertension (diastolic pressure >150 mm. Hg) or with pressures somewhat lower but with commanding symptoms of headache, visual disturbances, somnolence or other signs of cerebral, cardiac, or renal involvement or acute pulmonary edema should be placed on strict bed rest (semi-Fowler position) and parenteral therapy instituted immediately. Diazoxide (Hyperstat) is the drug of choice; 300 mg. IV push will reduce B.P. to normal values within 5 minutes. The drug should be used only for short periods and combined with a potent diuretic such as furosemide (Lasix) 40 to 80 mg. IV. Vital signs must be monitored continuously. Be prepared to treat hypotension (see Chapter 5, Shock). Discontinue if any sign of hearing impairment develops. When B.P. has been brought under control, combinations of oral antihypertensive agents can be added as parenteral drugs are tapered off over a period of 2-3 days.

b. Thrombophlebitis. Partial or complete occlusion of a vein by a thrombus with a secondary inflammatory reaction in the wall of a vein. It occurs most frequently in the deep veins of the legs and pelvis in postoperative and postpartum patients during the fourth to fourteenth day, and in patients with fractures or other trauma, cardiac disease, or stroke, especially if prolonged bed rest is involved. Deep venous thrombosis is usually benign but occasionally terminates in lethal pulmonary embolism or chronic venous insufficiency. Superficial phlebitis alone is usually self-limiting and without serious complications; aging, malignancy, shock, dehydration, anemia, obesity, and chronic infection are predisposing factors.

S. Approximately half of patients with thrombophlebitis are asymptomatic: Others may complain of a dull ache, tightness, or frank pain in the calf or the whole leg, especially when walking. A feeling of anxiety is not uncommon.

O. Slight swelling in the involved calf (measure); bluish discoloration or prominence of the superficial veins; warmth of affected leg when both legs are exposed to room temperature; tenderness and induration or spasm in the calf muscles, with or without pain in the calf produced by dorsiflexion of the foot (Homans' sign). With deep thrombophlebitis involving the popliteal, femoral, and iliac segments, there may be tenderness and a hard cord may be palpable over the involved vein in the femoral triangle in the groin, the medial thigh, or popliteal space; slight fever and tachycardia may be present. The skin may be cyanotic if venous obstruction is severe, or pale and cool if a reflex arterial spasm is superimposed.

A. Thrombophlebitis. Differential diagnosis: Calf muscle strain or contusion. NOTE: Pain due to muscular causes is absent or minimal on dorsiflexion of the ankle with the knee flexed and maximal on dorsiflexion of the ankle with the knee extended or during SLRs (Homans' sign); cellulitis; lymphatic obstruction; acute arterial occlusion (distal pulses are absent and there is no swelling); bilateral leg edema due to heart, kidney, or liver disease.

P. Treatment: Strict bed rest; elevate legs 15-20 degrees. Ace bandage from toes to just below the knees; moist heat. Anticoagulation therapy with heparin should be initiated if there are no contraindications to its use (contraindications are peptic ulcer, significant kidney or liver disease; Hx of cerebrovascular hemorrhage, recent head trauma, or known

clotting defect). Prior to initiation of heparin therapy, a baseline clotting time must be established. (Normal Lee-White clotting time is 6-15 minutes). The dose should be adjusted to provide 2-3 times the baseline pretreatment value. Continuous IV infusion is the preferred route. Give a loading dose as an IV bolus (2,000 units) prior to starting constant infusion at a rate of approximately 1,500 units/hour for the average-sized adult. Remember that the ultimate rate must be established on the basis of clotting times obtained q.2-3h. from an arm not being infused and verified by at least 2 successive clotting times in the therapeutic range. Subsequent clotting times are repeated q.6-10h. The required dosage will usually decrease with time. If an infusion pump is not available, give deep SQ q.6h. (use small needle and inject slowly). Start dose in the range of 7,000-9,000 units for an average-sized adult. Obtain clotting time 30 minutes before each planned dose and adjust to maintain therapeutic range. The required dose should drop to 4,000-6,000 units after a day or two of therapy. Therapy should be continued until the patient is asymptomatic and the danger of embolism has passed (normally 2-3 weeks). The diagnosis of thrombophlebitis is difficult without the use of sophisticated diagnostic aids that normally are not available (phlebography isotopic scan, etc.); therefore, maximum use must be made of past and current history and the most thorough P.E. possible. The dangers of lethal pulmonary embolism must be carefully weighed against the dangers of uncontrolled hemorrhage, and each decision is made on a sound assessment of all factors involved.

Prevention: The best cure for postoperative thrombophlebitis is its prevention. Assure that circulation is maintained by active and passive exercise while patients are bedridden. Avoid tight clothing. Elevate legs or foot of bed 15-30 degrees. Flex knees. Encourage deep breathing exercise. Ambulate patient as soon as possible (walking, not standing). Dextran, 500 ml. IV during surgery and repeated on first postoperative day, appears to have a prophylactic effect, as does ASA 1 gm daily P.O. NOTE: ASA is contraindicated once anticoagulation therapy has begun.

c. Hemorrhoids. Varicosities of the veins of the hemorrhoidal plexus, often complicated by inflammation, thrombosis, and bleeding. May be external (distal to anorectal line) or internal (proximal to anorectal line).

S. Rectal bleeding, pain (may be severe), itching, protrusion, mucoid discharge from rectum.

O. Small, rounded, purplish skin-covered masses that are soft and seldom painful unless thrombosed. When thrombosed, they are hard and often extremely painful when palpitated.

A. Hemorrhoids (internal or external). Differential diagnosis: Perianal abcess, rectal neoplasms, or colitis.

P. Use stool softners or nonirritating laxatives, such as mineral oil, and soft diet to prevent hard stools and straining. Small uncomplicated hemorrhoids are usually self-limiting and respond well to conservative or minimal treatment. Manage local pain and infection with warm sitz baths and insertion of a soothing anal suppository b.i.d.-t.i.d. Avoid the use of benzocaine and other types of similar ointments as much as possible to preclude sensitizing the patient. Use hot sitz baths t.i.d.-q.i.d. to reduce thrombosed hemorrhoids. If this is unsuccessful or

patient is in extreme discomfort, excise the thrombus under 1% lidocaine local; pack lightly with iodoform gauze initially and cover with dry sterile dressing. Change dressing daily. Continue warm sitz baths. Instruct patient to avoid trauma when cleansing the anal area after bowel movements by patting with damp tissue rather than rubbing. Instruct patient not to attempt to defecate unless there is a real urge and to avoid straining at stools.

1-30. DISEASES OF THE HEART.

a. Myocardial infarction (MI). Ischemic myocardial necrosis usually resulting from a sudden reduction in blood flow to a section of the myocardium due to occlusion of a coronary artery.

S. Sudden onset of intense, crushing substernal or precordial pain, often radiating to the left shoulder, arm, or jaw. Patients break out in a cold sweat, feel weak and apprehensive, and move about seeking a position of comfort. They prefer not to lie quietly. Lightheadedness, syncope, dyspnea, orthopnea, cough, wheezing, nausea and vomiting, or abdominal bloating may also be present, singly or in combination. The pain is not relieved by nitroglycerin.

O. Patient may be cyanotic and the skin is usually cool. The pulse may be thready and the blood pressure variable. Most show some degree of hypertension unless cardiogenic shock is developing (incidence about 8-14 percent). In a severe attack, the first and second heart sounds are faint and often indistinguishable. Arrhythmia is common. Rales may be heard on auscultation and the neck veins are often distended. Fever is absent at the onset but usually rises to 100-103o F. within 24 hours. W.B.C. will be elevated with a shift to the left by the second day. The sedimentation rate is normal at onset and will rise on the second or third day.

A. Acute myocardial infarction. Differential diagnosis: Angina pectoris, acute pericarditis, acute pulmonary embolism, reflux esophagitis, acute pancreatitis, acute cholecystitis, spontaneous pneumothorax, pneumonia.

P. Be alert for cardiac arrest, particularly during the first few hours after onset (50 percent of all MI deaths occur during this period). Be prepared to initiate CPR immediately if patient does arrest (see Chapter 3, Emergency Resuscitation). Morphine SO_4 2-5 mg. slow IV, stat. repeat q. 15 min p.r.n. unless respiration falls below 12/min. Shock position, O_2 (do not use positive pressure). Lidocaine initial bolus 50-100 mg. (1 mg./kg.) IV, then IV drip at 1-4 mg. per minute. Hospitalize with strict bed rest and complete nursing care for at least 6 weeks. Sedate with 1/2 gm phenobarbital t.i.d. Low sodium, low fat, low protein diet. Monitor vital signs constantly. Be alert for signs of left-sided heart failure (see para e, Congestive heart failure), hypotension, and cardiogenic shock (see Chapter 15, Shock); evacuate when feasible.

b. Acute myocarditis. A focal or diffuse inflammation of the myocardium occurring during or after many viral, rickettsial, spirochetal, fungal, and parasitic diseases or administration of various drugs. Severe myocarditis occurs most commonly in acute rheumatic fever, diphtheria, scrub typhus, and Chagas' disease.

S. Fever, malaise, arthralgias, chest pain, dyspnea, and

palpitations. The patient may have associated pericarditis, with chest pain characteristic of pericardial involvement (see para f, Acute pericarditis). The chest pain is frequently vague and nondiagnostic.

O. Tachycardia out of proportion to the amount of fever. The B.P. is usually normal. Ausculation may reveal a tic-tac rhythm and systolic murmur. Acute circulatory collapse, emboli, and sudden death may occur.

A. Acute myocarditis. Differential diagnosis: Viral, protozoan, or bacterial infections must be distinguished from acute toxic myocarditis due to drugs or diphtheria and from myocarditis associated with acute rheumatic fever and acute glomerulonephritis by a careful analysis of each history and clinical picture as it presents.

P. Direct treatment toward underlying cause if known. In all cases when myocarditis is suspected or apparent, complete bed rest and sedation plus continued therapy of the underlying disease are needed. Oxygen is indicated when cyanosis or dyspnea occurs. Continue bed rest until all evidence of cardiac involvement disappears.

c. Bacterial endocarditis. Bacterial infection of the lining membrane of the heart. Acute bacterial endocarditis (ABE) begins abruptly

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endocarditis (SBE) is usually due to alpha-hemolyticus streptococci and frequently follows a dental procedure. The disease is fatal if untreated.

S. Fever is usually present but afebrile periods may occur. Night sweats, chills, malaise, fatigue, anorexia, weight loss; myalgia; arthralgia, or redness and swelling of joints; sudden visual disturbances; paralysis; pain in the abdomen, chest, or flanks; nose bleeds; easy bruisability; and symptoms of heart failure may also occur.

O. Findings in SBE include tachycardia; splenomegaly; petechiae of the skin, mucous membranes, and ocular fundi, or beneath the nails as splinter hemorrhages; clubbing of the fingers and toes; pallor or a yellowish-brown tint of the skin; neurologic residual effects of cerebral emboli; and tender finger and toe pads. In ABE symptoms and signs are similar to those of SBE, but the course is more rapid. Suspect ABE if an otherwise healthy individual with a focal infection suddenly develops chills, high fever, and prostration. An unexplained fever in patient with a heart murmur is indicative of endocarditis. Anemia, markedly elevated sedimentation rate, variable leukocytosis, microscopic hematuria, proteinuria, and casts are commonly present in SBE and ABE.

A. Infective endocarditis due to _____.
Differential diagnosis: Lymphomas, thrombocytopenic purpura, leukemia, acute rheumatic fever, lupus erythematosus, septicemia (may be the forerunner), URIs.

P. Endocarditis due to streptococcus: Penicillin G 20-40 M.U. daily, or ampicillin 6-12 gm daily in divided doses as bolus injections q.2-4h. into an IV infusion. Probenecid 0.5 gm P.O. t.i.d. x 4-5 weeks. Streptomycin, 1 gm day; kanamycin 15 mg./kg./day; or gentamicin 5 mg./kg./per day b.i.d.-t.i.d. in divided doses. Endocarditis due to staphlococcus (penicillin resistant), nafcillin, 8-12 gm daily as a bolus

q.2h. in an IV infusion. If patient is hypersensitive to penicillin, desensitize or use vancomycin 2-3 gm IV daily in divided doses q.4h. continue Tx x 5-6 weeks. Complete nursing care. Monitor for signs of neurotoxicity and thrombophlebitis. Change injection site q.48h. and keep scrupulously clean. Evacuate if at all feasible.

d. Angina pectoris. A clinical syndrome due to myocardial ischemia producing a sensation of precordial discomfort, pressure, or a strangling sensation, characteristically precipitated by exertion and relieved by rest or nitroglycerin.

S. Squeezing or pressurelike pain, retrosternal or slightly to the left, that appears quickly during exertion and increases rapidly in intensity until the patient is compelled to stop and rest. The distribution of the distress may vary widely in different patients, but is always the same for each individual patient. The attacks usually last less than 3 minutes unless following a heavy meal or precipitated by anger, in which case they may last 15-20 minutes. The distress of angina is never a sharply localized darting pain that can be pointed to with one finger. If the patient points with one finger to the area of the apical impulse as the only site of pain, angina may almost certainly be ruled out.

O. The diagnosis of angina pectoris depends almost entirely upon the history, and it is of utmost importance that the patient be allowed to describe his symptoms to the examiner. The diagnosis is strongly supported (1) if 0.4 mg. nitroglycerin invariably shortens an attack and (2) if that amount taken immediately before hand invariably permits greater exertion before onset of an attack or prevents it entirely. Examination during an attack frequently reveals elevated B.P.; occasionally, gallop rhythm is present during pain only.

A. Angina pectoris. Differential diagnosis: Musculosketal disorders, cholecystitis, reflux esophagitis, peptic ulcer, myocardial infarction.

P. Nitroglycerin 0.3 mg. sublingually is the drug of choice. Increase dose to 0.4-0.6 mg. if smaller dose is ineffective. One amyl nitrite ampule crushed and inhaled will act in about 10 seconds. The patient should stand still or lie down as soon as the pain begins and remain quiet until the attack is over. Patients should be warned not to try to work the attack off.

e. Congestive heart failure. A clinical syndrome in which the heart fails to maintain an adequate output, resulting in diminished blood flow to the tissues and in congestion in the pulmonary and/or systemic circulation. The left or right ventricle alone may fail initially (usually the former), but ultimately combined failure is the rule. The basic causes of ventricular failure are: (1) Myocardial weakness or inflammation (e.g., myocarditis, ischemia), (2) Excess workload (e.g., hypertension, aortic insufficiency anemia, pregnancy, etc.).

S. Early manifestations of left ventricular failure include undue tachycardia, fatigue with exertion, dsypnea with mild exercise, and intolerance to cold; paroxysmal nocturnal dyspnea and cough. In advanced failure severe cough is prominent. The sputum may be tinged rusty or brown. Frank hemoptysis is rare but can occur. Acute pulmonary edema is a serious life threatening manifestation of left ventricular failure. The patient presents with extreme dyspnea, cyanosis, tachypnea, hyperpnea,

restlessness, and anxiety with a sense of suffocation. Right ventricular failure presents with increasing fatigue, awareness of fullness in the neck and abdomen, anorexia, bloating, or exertional RUQ pain. Oliguria is present in the day time; polyuria at night.

O. Signs of left ventricular failure include reduced carotid pulsation, diffuse apical impulse, palpable and audible third and fourth heart sounds, inspiratory rales, and pleural effusion. With acute pulmonary edema the pulse may be thready and the B.P. difficult to obtain. Respirations are grunting and labored with inspiration, and expiration is prolonged. Expiratory rales can be heard over both lungs. There may be marked bronchospasm or wheezing. Hypoxia is severe and cyanosis deep. Patients with right ventricular failure show signs of venous hypertension, an enlarged and tender liver, murmurs, and pitting edema of the lower extremities. CBC and sed. rate are normal in uncomplicated left heart failure. Urinalysis often shows significant proteinuria and granular casts.

A. Congestive heart failure due to _____. Differential diagnosis: Pericardial effusion, constrictive pericarditis, pulmonary disease, carcinoma of the lung, anemias, and rebound edema following the use of diuretics.

P. Bed rest (Fowler or semi-Fowler position), sedation with morphine or phenobarbital; frequent (4-6) small, bland, low calorie, low residue, sodium restricted meals with vitamin supplements. Diuretics such as hydrochlorothiazide 50 mg./day or chlorothiazide 500 mg. daily or b.i.d. are essential to management of chronic heart failure. Increase daily ingestion of foods with a high potassium content (bananas, orange juice) for potassium replacement. Administer O_2 p.r.n. for respiratory distress and hypoxia. Acute pulmonary edema is grave medical emergency demanding prompt and effective Tx. Unless in shock, the patient should sit upright with legs dangling. Give high concentrations of O_2 by mask or nasal cannula. Morphine SO_4 5-10 mg. IV or IM. Sublingual nitroglycerin 0.4-0.6 mg. q.10 min for several doses may be immediately effective. If severe, apply B.P. cuffs (or soft rubber tourniquets) to three limbs and inflate or tighten sufficiently to obstruct venous return (midway between systolic and diastolic pressure) but not arterial flow. Rotate q.15 min. NOTE: Do not apply to a limb in which an IV is running. If IV is running, deflate q.15-20 min but do not rotate. Give a rapid acting diuretic, e.g., Lasix (furosemide) 40-80 mg. IV or Edecrin 25-50 mg. IV. Aminophylline, 0.25-0.5 gm slow IV or aminophylline suppositories, 0.25-0.5 gm may be of help. Rapid digitalization is of value; however, it must be remembered that all digitalis preparations are toxic and the difference between the theraputic and toxic level is small. Do not use digitalis if there is any indication of renal failure. If renal function is normal, the following schedule may be used: Digoxin 0.25 mg. IV or P.O. stat., then 0.25 mg. q.6h. x 2 days and 0.25 mg. daily thereafter. NOTE: Digitalis maintenance may be required for the remainder of the patient's life. When stable, the patient should be carefully monitored for: (1) Status of original symptoms, (2) new symptoms or signs, (3) weight changes, (4) vital signs, (5) evidence of phlebothrombosis. Evacuate as soon as feasible.

f. Acute pericarditis. Inflammation of the pericardium. It may result from trauma, infection, or neoplasm or secondary to systemic diseases such as rheumatic fever, rheumatoid arthritis, or uremia.

S. Pleuritic or persisting substernal or precordial pain

radiating to the neck, shoulder, or back. Pain may be aggravated by thoracic motion, cough, and respiration. It is relieved by sitting up and leaning forward and may be accentuated by swallowing. Tachypnea, nonproductive cough, fever, chills, weakness, and anxiety are common.

O. Auscultation reveals to and fro friction sounds (friction rub) over 4th (L) intercostal space near sternum. Inspection and palpation sometimes reveal a diffuse apex beat. With purulent effusion may present with high, irregular fever, sweats, chills, and progressive pallor. Bulging of the precordium, increased dullness to percussion, and edema of the precordium may also be present. Leukocytosis and elevated sed. rate will be present at the onset.

A. Acute pericarditis due to_____. Differential diagnosis: Acute MI, pleurisy.

P. (1) Treat underlying condition.

(2) ASA 600 mg. P.O., codeine 15-60 mg. P.O., meperidine 50-100 mg. P.O. or IM, or morphine 10-15 mg. SQ q.4h. for pain. Sedate with phenobarbital 15-30 mg. P.O. t.i.d.-q.i.d.; 100-200 mg. phenobarbital may be given h.s. for insomnia. Prednisone 20 to 60 mg. daily in divided doses t.i.d.-q.i.d. may be required to control pain, fever, and effusion. The dose should be reduced gradually and discontinued over a period of 7-14 days. If the pericarditis is due a pyogenic infection, surgical drainage of the pericardial sac may be indicated.

1-31. DISEASES OF THE BLOOD.

a. *Anemia (general).* A condition in which there is a reduction in the number of circulating R.B.C.s and/or Hb in the blood. Fundamentally, all anemias are caused by one of the following conditions:

(1) Increased loss of R.B.C. due to:

(a) Hemorrhage.
(b) Increased rate of R.B.C. destruction (hemolytic anemias).

(2) Decreased production of R.B.C. due to:

(a) Deficiencies.
(b) Bone marrow suppression.

b. Iron-deficiency anemia. Chronic anemia characterized by small, pale R.B.C. and depletion of iron stores. In adults it is almost always due to occult blood loss (G.I. bleeding, excessive menstrual, excessive salicylate intake, etc.).

S. Easy fatigability, dyspnea, palpitation, angina, and tachycardia. Inability to swallow or difficulty in swallowing may exist in advanced cases. There often exists a craving for strange foodstuffs (dirt, chalk, paint, etc.).

O. Skin and mucous membranes are usually pale. In advanced cases the skin may have a waxy appearance; the hair and nails are brittle, longitudinal ridging with progressive concavity (spooning) may appear on the fingernails. The tongue may be smooth, and the lips inflamed and

cracked. Hb may be as low as 3 Mg% but R.B.C. is rarely below 2.5 m. W.B.C. is normal.

A. Iron deficiency anemia due to _____. Differential diagnosis: Other hypochromic anemias (anemias of infection, thalassemia, etc.) pernicious anemia, aplastic anemia.

P. (1) Treat underlying cause.

(2) Oral $FeSO_4$ 0.2 gm t.i.d. p.c. Continue for 3 months after Hb returns to normal. If there is bleeding in excess of 500 ml./wk over a sustained period, iron therapy will not work until the cause of bleeding is corrected. NOTE: Iron causes a color change in the stool (dark green or black). Advise patient not to be alarmed if this occurs.

c. Pernicious anemia. Anemia due to impaired absorption of vitamin B_{12}.

S. Same as iron deficiency. In addition the patient may complain of a "burning of the tongue"; constant, symmetric numbness of the feet; various G.I. disturbances (anorexia, constipation, diarrhea, vague abdominal pain); transient paresthesias of the upper extremities; and severe weight loss. There may be mental disturbances ranging from mild depression to delirium and paranoia.

O. Pallor with a trace of jaundice; loss of vibratory sensation in the lower extremities, loss of positional sense, loss of coordination; hyperactive deep tendon reflexes and positive Babinski. Occasional splenomegaly and hepatomegaly may be present. Differential smear will demonstrate large oval R.B.C. with a few small misshapen R.B.C. W.B.C. is usually less than 5,000. The granulocytes tend to be hypersegmented.

A. Pernicious anemia. Differential diagnosis: Anemia due to folic acid deficiency. NOTE: The oval shape of the R.B.C. and hypersegmentation of the W.B.C. are not characteristic of folic acid deficiency anemia.

P. Give 100 mg. vitamin B_{12} IM stat., then 100 mg. 3 times per week until blood picture returns to normal. If anemia is severe, give transfusion (after type and X-match) of packed red cells slowly.

d. Hemolytic transfusion reactions. Hemolysis of the recipient's or donor's R.B.C. (usually the latter) during or following the administration of solutions, plasma, blood, or blood components. Hemolytic reactions vary in severity depending on the degree of incompatibility, the amount of blood given, and the rate of administration. The most severe reaction occurs when donor R.B.C. are hemolized instantaneously by antibody in the recipient's plasma. These reactions constitute a grave medical emergency.

S. Sudden onset of chills and fever and pain in the vein at the local injection site or in the back, chest, or abdomen. Anxiety, apprehension, and headache are common. Under general anesthesia, spontaneous bleeding may be the only sign of a transfusion reaction.

O. Evidence of shock (see Chapter 15, Shock). Oliguria, anuria, progressing to uremia. If a hemolytic reaction is suspected, immediately take a blood sample from the patient and centrifuge it. Hemolysis will be clearly visible as a pink to dark red color in the serum.

A. Hemolytic transfusion reaction. Differential diagnosis: Minor allergic reactions. (Serum will remain clear.)

P. (1) STOP TRANSFUSION STAT.

(2) Treat for shock.

(3) To prevent renal failure, give 10% mannitol solution IV infusion at a rate of 10-15 ml./min until 1,000 ml. have been given. If diuresis occurs, continue the mannitol infusion until serum and urine are clear.

1-32. DISEASES OF THE LYMPHATIC SYSTEM.

a. Lymphadenitis. Inflammation of one or more lymph nodes. Usually secondary to a primary infection elsewhere involving the skin or subcutaneous tissue.

S. Enlarged, tender, often acutely painful lymph nodes. Systemic symptoms may be minimal or severe.

O. Primary focus of infection in the region of the affected node(s). Cellulitis, suppuration with abcess formation may occur. Low grade or chronic infections may produce firm, nontender nodes that persist indefinitely (e.g., TB and fungal infections). They may form cold abcesses or erode through the surface to create draining sinuses.

A. Lymphadenitis secondary to _____. Differential diagnosis: Lymphedema secondary to blockage of the lymph channels.

P. Treat primary infection. Apply moist heat to localize infection. Analgesics for pain. I&D abcesses.

b. Lymphangitis. Acute or chronic inflammation of the superficial or deep lymphatic channels, usually caused by streptococci or staphylococci.

S. Fever (102 to 105o F.), chills, malaise, generalized aching, and headache.

O. Patchy areas of inflammation along the path of a lymphatic channel resembling cellulitis. Lymphangitis occurring as the result of hand or foot infection presents as irregular pink, tender, linear streaks extending toward the regional lymph nodes. Lymphadenitis usually follows. Leukocytosis (W.B.C. 15,000-30,000) with shift to the left.

A. Acute lympangitis due to _____. Differential diagnosis: Acute thrompophlebitis, cellulitis.

P. Treat the original infection, but avoid all undue surgical manipulation of the wound. Use same antibiotic therapy as for acute cellulitis (Chap 1, Sec I). Antibiotics should be continued until the temperature has been normal for 72 hours and inflammation has subsided.

1-33. GENERAL. The digestive system covers the entire alimentary tract (mouth, esophagus, stomach, intestines, colon, and rectum) and all organs that aid in digestion (liver, gallbladder, and pancreas). Diseases of the mouth are covered in the dental section. Diseases of the esophagus are either minor or of such a nature that we can only treat them symptomatically.

1-34. ACUTE ABDOMEN. Usually manifested by pain, anorexia, nausea, vomiting, and fever. Physical exam shows tenderness, muscle spasm, and changes in peristalsis. Correct diagnosis depends on the precision and care in taking history and doing physical exams.

a. History.

(1) Mode of onset of abdominal pain.

(a) Patient is well one moment and seized with agonizing (explosive) pain the next; most probable diagnosis is free rupture of a hollow viscus or vascular accident. Renal and biliary colic may be very sudden in onset but are not likely to cause severe and prostrating pain.

(b) If pain is rapid in onset--moderately severe at first and becoming rapidly worse--consider acute pancreatitis, mesenteric thrombosis, or strangulation of the small bowel.

(c) Gradual onset of slowly progressive pain is characteristic of peritoneal infection or inflammation. Appendicitis and diverticulitis often start this way.

(2) Character of the pain.

(a) Excruciating pain not relieved by narcotics indicates a vascular lesion such as massive infarction of the intestine or rupture of an abdominal aneurysm.

(b) Very severe pain readily controlled by medication more typical of acute pancreatitis or the peritonitis associated with a ruptured viscus. Obstructive appendicitis and incarcerated small bowel without extensive infarction occasionally produce the same type of pain. Biliary or renal colic is usually promptly alleviated by medication.

(c) Dull, vague, and poorly localized pain usually gradual in onset strongly suggests an inflammatory process or low grade infection, e.g., appendicitis.

(d) No abdominal pain but complains of feeling of fullness that might be relieved by a bowel movement, enema provides no relief ("gas stoppage sign"). This may be present when any inflammatory lesion is walled off from free peritoneal cavity.

(e) Intermittent pain with cramps and rushes commonly seen in gastroenteritis. The peristaltic rushes have little or no relation to abdominal cramps in gastroenteritis. If the pain comes in regular cycles, rising in crescendo fashion, synchronous with the pain and then subsiding to a pain-free interval, small bowel obstruction is very likely.

(f) Radiation or a shift in localization of pain. Pain in the shoulder follows diaphragmatic irritation due to air, peritoneal fluid, or blood. Biliary pain is often referred to the right scapula and rarely to the left epigastrium and left shoulder, simulating angina pectoris. Classically, appendicitis begins in the epigastrium and settles in the right lower quadrant. A shift or spread of abdominal pain often indicates spreading peritonitis.

(g) Anorexia, nausea, and vomiting. The time of onset of these symptoms is important; if they precede the onset of pain, gastroenteritis or some systemic illness is much more likely the diagnosis than acute abdominal disorder requiring an emergency operation. The most likely possibilities are gastroenteritis, acute gastritis, acute pancreatitis, common duct stone, and high intestinal obstruction. In most other acute surgical emergencies, nausea and vomiting are not dominant symptoms though they may be present.

(h) Diarrhea, constipation, and obstipation. Some alteration of bowel function is common in most cases of acute abdominal emergencies. Diarrhea is the classic manifestation of gastroenteritis, but it may also be a dominant symptom of pelvic appendicitis. Bloody and repetitive diarrhea indicates ulceration of the colon, but you should consider bacillary or amebic dysentery first.

(i) Chills and fever. Repeated bouts of chills and fever are characteristic signs of pylephlebitis and bacteremia. Chills and fever are common in infections of the biliary or renal tract. Acute cholangitis and pyelitis present with intermittent chills and fever. In appendicitis, fever is not usually very high and there are usually no chills unless you have a perforation. In a woman with no apparent general systemic illness, a very high fever with peritoneal signs is characteristic of acute pelvic inflammatory disease (PID).

b. Routine for physical exam of the acute abdomen.

(1) General inspection (patient standing).

(2) Cough tenderness. Examine hernial rings and male genitals.

(3) Feel for spasm.

(4) One-finger palpation.

(5) Costovertebral check for tenderness.

(6) Deep palpation.

(7) Rebound tenderness.

(8) Auscultation.

(9) Rectal and pelvic examination.

1-35. DISEASES OF THE STOMACH.

a. Acute simple gastritis. This is probably the most common disturbance of the stomach and is frequently accompanied by generalized enteritis. Causes are chemical irritants (e.g., alcohol, salicylates),

bacterial infection or toxins (e.g., staphylococcal food poisoning, scarlet fever, pneumonia), viral infections (e.g., viral gastroenteritis, measles, hepatitis, influenza), and allergy (e.g., shellfish).

S. Anorexia is always present and may be the only symptom. Usually, patient complains of epigastric fullness and pressure and nausea and vomiting. Diarrhea, colic, malaise, fever, chills, headache, and muscle cramps are common with toxins or infections.

O. The patient may be prostrated and dehydrated. Examination shows mild epigastric tenderness. Hemorrhage is frequent with chemical irritants (e.g., salicylates). This may be found using a guaiac test. CBC may show a leukocytosis or in viral infections, a leukopenia.

A. Acute simple gastritis caused by _____.
Differential diagnosis: Includes peptic ulcers and appendicitis.

P. Treat the specific infection or problem. Correct fluid and electrolyte disturbance. Place patient N.P.O. until acute symptoms of pain and nausea have subided, then start giving clear liquids and progress to a soft diet as tolerated. Sedatives, Compazine, or opiates may be used as indicated. Symptoms last from 1-7 days.

b. Food poisoning and acute gastroenteritis. Food poisoning is a general term applied to the syndrome of acute anorexia, nausea, vomiting, and/or diarrhea that is attributed to food intake, especially if it affects a group of people who ate the same foods. There are numerous causative agents and organisms that have similar signs and symptoms to a greater or lesser degree. The only positive way of differentiating between these agents or organisms is by culturing the suspected food and stools of the affected individuals. Most forms of food poisoning are self-limiting and require symptomatic treatment, such as replacement of fluids and electrolytes, control of diarrhea with Lomotil, and control of nausea and vomiting with Compazine. Very rarely patients may develop hypovolemic shock and respiratory embarrassment, and this will have to be managed. Antimicrobial drugs should not be given unless the specific organism can be identified as they may aggravate the anorexia and diarrhea and prolong the course of the illness. The exception to the rule is if you suspect BOTULISM; then polyvalent antitoxin must be administered. The following chart will help in identifying the various types of food poisoning and their specific treatments.

Organism	Incubation Period (Hours)	Epidemiology	Clinical Features
Staphylococcus	1-18	Staphylococci grow in meats, dairy, and bakery products and produce entero-toxin.	Abrupt onset, intense vomiting for up to 24 hours, regular recovery in 24-48 hours. Occurs in persons eating the same food. No treatment usually necessary except to restore fluids and electrolytes.

Clostridium perfringens	8-16	Clostridia grow in rewarmed meat dishes and produce enterotoxin.	Abrupt onset of profuse diarrhea; vomiting occasionally. Recovery usual without treatment in 1-4 days. Many clostridia in cultures of food and feces of patients.
Clostridium botulinum	24-96	Clostridia grow in anaerobic foods and produce toxin.	Diplopia, dysphagia, dysphonia, respiratory embarrassment. Treatment requires clear airway, ventilation, and intravenous polyvalent antitoxin. Toxin present in food and serum. Mortality rate high.
Escherichia coli (some strains)	24-72	Organisms grow in gut and produce toxin. May also invade superficial epithelium.	Usually abrupt onset of diarrhea; vomiting rare. A serious infection in neonates. In adults, "traveler's diarrhea" is usually self-limited in 1-3 days. Use diphenoxylate (Lomotil) but no antimicrobials.
Vibrio parahaemolyticus	6-96	Organisms grow in seafood and in gut and produce toxin.	Abrupt onset of diarrhea in groups consuming the same food, especially crabs and other seafood. Recovery is usually complete in 1-3 days. Food and stool cultures are positive.
Vibrio cholerae (mild cases)	24-72	Organisms grow in gut and produce toxin.	Abrupt onset of liquid diarrhea in endemic area. Needs prompt replacement of fluids and electrolytes IV or orally. Tetracyclines shorten excretion of vibrios. Stool cultures positive.

Shigella spp. (mild cases)	24-72	Organisms grow in superficial gut epithelium and gut lumen and produce toxin.	Abrupt onset of diarrhea, often with blood and pus in stools; cramps; tenesmus; and lethargy. Stool cultures are positive. Give ampicillin, chloramphenicol, or sulfamethoxazole with trimethoprim (co-trimoxazole) in severe cases. Often mild and self-limited. Restore fluids.
Salmonella spp.	8-48	Organisms grow in gut. Do not produce toxin.	Gradual or abrupt onset of diarrhea and low-grade fever. No antimicrobials unless systemic dissemination is suspected. Stool cultures are positive. Prolonged carriage is frequent.
Clostridium difficile	?	Drug intake, e.g., clindamycin.	Especially after abdominal surgery, abrupt bloody diarrhea and fever. Toxin in stool. Oral vancomycin useful in therapy.
Campylobacter fetus	?	Organism grows in jejunum and ileum.	Fever, diarrhea; P.M.N.'s and fresh blood in stool, especially in children. Usually self-limited. Special media needed for culture. Erythromycin in severe cases with invasion.
Yersinia enterocolitica	?	Fecal-oral transmission. Food-borne.? In pets.	Severe abdominal pain, diarrhea, fever; P.M.N.'s and blood in stool; polyarthritis, erythema nodosum, especially in children. If severe, tetracycline or

gentamicin.

c. Bacillary dysentery (shigellosis). Shigellosis is a common, often mild and self-limiting disease that occasionally is serious. It is usually found in conjunction with poor sanitary conditions.

S. Abrupt onset of diarrhea (often with blood and mucus), lower abdominal cramps, and tenesmus. This is usually accompanied by fever, chills, anorexia, malaise, headache, lethargy, clouded mental condition, and in the most severe cases meningismus (S and S of meningeal irritation without actual infection), coma, and convulsions. As the illness progresses, the patient becomes weaker and more dehydrated.

O. Temperature up to 104o F., tender abdomen, and blood, mucus, and pus in the stool. Stool culture is positive for shigellae.

A. Bacillary dysentery (shigellosis). Differential diagnosis: Amebic dysentery, salmonella, gastroenteritis, E. coli, viral diarrhea, and ulcerative colitis.

P. IV fluid and electrolyte replacement, place patient N.P.O.; antispasmodics (e.g., tincture of belladonna) are helpful when cramps are severe. Avoid Lomotil or paregoric; they may improve the general symptoms but prolong fever, diarrhea, and excretion of shigella in feces. Effective stool isolation and disposal should be initiated. Drug of choice is ampicillin 250 mg. q.6h. x 5-7 days; second choice is tetracycline 250 mg. q.6h. x 5-7 days. After bowel has been at rest for a short time, start patient on clear fluids for 2-3 days, then soft diet and gradually build.

d. Amebic dysentery (see Chapter 2, Section I, Parasitic Diseases).

e. Typhoid fever (see Chapter 2, Section III, Bacterial Diseases).

f. Cholera (see Chapter 2, Section III, Bacterial Diseases).

g. Infectious hepatitis (see Chapter 2, Section IV, Viral Diseases).

h. Peptic ulcer disease. An acute or chronic benign ulceration in a portion of the digestive tract exposed to gastric secretions.

(1) Duodenal ulcer. Most common type of ulcer, four to five times more prevalent than gastric ulcer.

S. Symptoms may be vague or absent. In a typical case pain is described as gnawing, burning, cramplike, aching, or as heartburn; it is usually mild to moderate, located near the midline and near the xiphoid process. Pain may radiate below the ribs into the back or occasionally to the right shoulder. Patient may have nausea and may vomit small quantities of highly acid gastric juices with little or no food. Usually occurs 45-60 minutes after meals; absent in the morning before breakfast and gets progressively worse as the day passes. May be most severe between midnight and 0200. Pain is relieved by food, milk, antacids, and vomiting within 5-30 minutes. Ulcers can spontaneously get better or worse. Causative factors may be unknown but may include physical or emotional distress, trauma, or infections.

O. Examination shows superficial and deep epigastric tenderness, voluntary muscle guarding, and unilaterial spasm over duodenal bulb. Lab

work will show occult blood in the stool and anemia in chronic ulcers. Definite diagnosis depends on X ray and endoscopic examination.

NOTE: Complications include severe hemorrhage due to ulceration into a vein or artery or even bleeding from granulation tissue; perforation into the peritoneal cavity causing peritonitis; puretration into surrounding organs, usually into the pancreas, but the liver, biliary tract or gastrohepatic omentum may be involved. In 20 to 25 percent of untreated patients, minor degrees of pyloric valve obstruction occur, but major or complete obstructions are rare.

A. Peptic ulcer disease duodenal ulcer. Differential diagnosis: functional gastrointestinal disease, gastritis, gastric carcinoma, and irritable colon syndrome.

P. 2-3 weeks rest from work if possible. Relieve or avoid anxiety whenever possible. Forbid alcohol. Discontinue or avoid drugs that aggravate ulcers (e.g., phenylbutazone, indomethacin, and large amounts of salicylates). Place patient on a dietary management program.

(a) In the acute phase, start full liquid diet with hourly antacids liberalized rapidly to a regular diet.

(b) Avoid milk as therapy.

(c) Avoid interval feeding (eating small meals every few hours).

(d) Nutritious diet.

(e) Regular meals.

(f) Restrict coffee, tea, and cola beverages.

(g) Avoid foods that are known to produce unpleasant symptoms in a given individual.

Antacids, in order to be effective, must be taken frequently. In the acute phase, antacids should be given hourly. The schedule may then be changed to a full dose 1 and 3 hours after meals and at bedtime.

(2) Gastric ulcer. In many respects it is similar to duodenal ulcer.

S. There may be no symptoms or vague and atypical symptoms. Pain is epigastric and described as gnawing, burning, aching, or hunger pangs referred at times to left subcostal area. Usually occurs 45-60 minutes after meals and is relieved by food, antacids, or vomiting. Weight loss, constipation, and fatigue are common.

O. Epigastric tenderness or voluntary muscle guarding is usually the only finding. If there has been bleeding, a guaiac test will show occult blood.

NOTE: Complications are the same as with duodenal ulcers.

A. Peptic ulcer disease, gastric ulcer. Differential diagnosis: Duodenal ulcer, irritable colon, functional gastrointestinal distress, and

gastritis.

P. Treatment is the same as for duodenal ulcer. Failure to respond in 3-4 weeks is indication for surgery.

Gastric ulcers tend to be recurrent. Recurrent uncomplicated ulcers usually heal faster than the previous ulcer.

i. Acute organic intestinal obstruction. Usually involves the small intestines, particularly the ileum. Major causes are external hernia and postoperative adhesions. Less common causes are gallstones, neoplasms, foreign bodies, intussusception, granulomatous processes, internal hernia, and vovulus.

S. Colicky abdominal pain in periumbilical area becoming more constant and diffuse as distention develops. Vomiting associated with waves of pain. If obstruction is of the distal bowel, vomiting becomes fecal in nature. Loud stomach growling, unmanageable constipation, weakness, sweating, and anxiety are often present.

O. Patient is restless, often in shocklike state with tachycardia and dehydration, tender distended abdomen (can be localized but usually generalized) without peritoneal irritation. Audible and visible peristalsis, high pitched tinkles, and pain related to peristaltic rushes may be present. Temperature is normal or slightly elevated. A tender hernia may be present. W.B.C. is normal or slightly elevated.

A. Acute organic intestinal obstruction. Differential diagnosis: Renal colic, gallbladder colic, or mesenteric vascular disease.

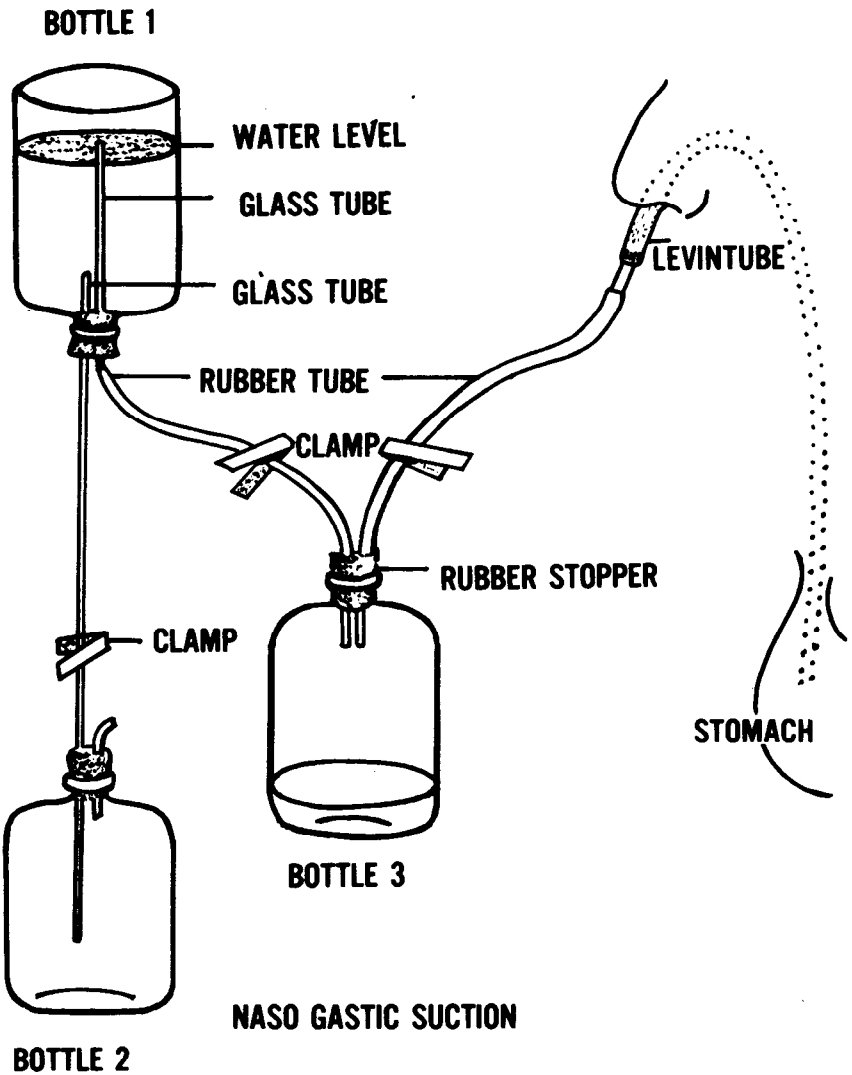
P. Place patient N.P.O. Decompress intestinal tract by nasogastric suction (see illustration on next page). Replace fluids and electrolytes by IV. Treat the cause of the obstruction. Start broad-spectrum antibiotic therapy if needed.

j. Appendicitis. One of the most frequent causes of acute abdomen. Signs and symptoms usually follow a fairly stereotyped pattern, but it can display many different manifestations that should be considered in the differential diagnosis of every case of abdominal sepsis and pain.

S. Appendicitis usually begins with generalized periumbilical or epigastric pain and 1 or 2 episodes of vomiting. Within 2-12 hours, the pain shifts to right lower quadrant where it persists as a steady soreness aggravated by walking or coughing. Patient can usually place a finger on a specific point. Anorexia, malaise, slight fever, and constipation are usual, but diarrhea occurs occasionally.

O. Rebound tenderness and spasm of the overlying abdominal muscles. Rectal tenderness is common; peristalsis is diminished or absent. Slight to moderate fever. Pain localized in right lower quadrant. W.B.C. 10-20,000 with an increase in neutrophils.

NOTE: Complications include perforation leading to generalized peritonitis, appendiceal abscess, pylephlebitis, and intestinal obstruction.


BOTTLE 1
WATER LEVEL
GLASS TUBE
GLASS TUBE
LEVINTUBE
RUBBER TUBE
CLAMP
RUBBER STOPPER
CLAMP
STOMACH
BOTTLE 3
NASO GASTIC SUCTION
BOTTLE 2

A. Appendicitis. Differential diagnosis: Acute gastroenteritis, mesenteric adenitis, Meckel's diverticulitis, regional enteritis, amebiasis, perforated duodenal ulcer, ureteral colic, ruptured ectopic pregnancy, and twisted ovarian cyst may at times mimic appendicitis.

P. Place patient under observation for diagnosis within the first 8-12 hours. Bed rest, N.P.O., start maintaining IV, avoid narcotic medication as it might mask symptoms necessary for proper diagnosis. Abdominal and rectal exam, white blood count, and differential count are repeated periodically.

(1) Once diagnosis is made, an appendectomy should be performed as soon as fluid imbalances and other systemic disturbances are controlled.

(2) Antibiotics should be administered in the presence of marked systemic reaction with severe toxicity and high fever.

(3) Emergency nonsurgical treatment when surgical facilities are not available; treat as for acute peritonitis. Acute appendicitis may subside and complications will be minimized.

k. Acute peritonitis. Localized or generalized peritonitis is the most important complication of numerous acute abdominal disorders. May be caused by infection or chemical irritation.

S. Malaise, prostration, nausea, vomiting, fever, depending on extent of involvement localized or generalized pain and tenderness, abdominal pain on coughing.

O. Elevated W.B.C., rebound tenderness referred to area of peritonitis, and tenderness to light percussion over the area. Pelvic peritonitis is associated with rectal and vaginal tenderness. Spastic muscles over area of inflammation. When peritonitis is generalized, there will be marked rigidity of the entire abdominal wall. This rigidity is frequently diminished or absent in the late stages of peritonitis, in severe toxemia, and when the abdominal wall is weak, flabby, or obese. Diminished to absent peristalsis and progressive abdominal distention is found. Vomiting occurs due to pooling of gastrointestinal secretions and gas. W.B.C. will increase to 10-20,000.

A. Acute peritonitis. Differential diagnosis: Peritonitis may present a highly variable clinical picture and must be differentiated from acute intestinal obstruction, acute cholecystitis, renal colic, gastrointestinal hemorrhage, lower labor pneumonia, porphyria, periodic fever, hysteria, and central nervous system disorders.

P. Treatment is generally applicable as supportive treatment in most acute abdominal disorders. The objectives are: Control infection; minimize the effects of paralytic ileus; correct fluid, electrolyte, and nutritional disorders.

(1) Specific measures: Identify and treat the cause; this usually entails surgery to remove sources of infection such as appendicitis, gangrenous bowl, abscesses, or perforated ulcers.

(2) General: Bed rest in medium Fowler position (semi-sitting). Nasogastric (NG) suction to prevent abdominal distention and continued

until peristalsis returns and patient begins passing flatus. Place patient N.P.O. until after NG suction is discontinued, then slowly resume oral intake. IV for fluid electrolyte therapy and parenteral feeding are required. Narcotics and sedatives used liberally to insure rest and comfort. Broad-spectrum antibiotic therapy to prevent and control infections should be initiated. Blood transfusions as needed. Watch patient for signs of toxic shock and treat as required.

1. Acute Pancreatitis. A severe abdominal disease produced by acute inflammation in the pancreas and associated "escape" of pancreatic enzymes into the surrounding tissues. The exact cause is not known, but more than 80 clinical causes have been related to acute pancreatitis, everything from alcoholism to drugs.

S. Epigastric pain generally abrupt in onset is steady and severe, made worse by lying down and better by sitting up leaning forward. Pain usually radiates to the back but may radiate right or left. Nausea, vomiting, and constipation are present, and severe prostration, sweating, and anxiety are usually found. There may be a history of alcohol intake or a heavy meal immediately before the attack.

O. Tender abdomen mainly in upper abdomen, usually without guarding, rigidity, or rebound. Abdomen may be distended and bowel sounds may be absent. Temperature of 101.1-102.2°F., tachycardia, pallor, hypotension, and a cool clammy skin are often present.

Mild jaundice is common. Upper abdominal mass may be present. Acute renal failure may occur early in the course of the disease. W.B.C. 10-30,000. Urinalysis shows proteinuria, casts in 25 percent of the cases, and glucosuria in 10-20 percent of the cases.

A. Acute pancreatitis. Differential diagnosis: Pancreatitis is hard to tell from common duct stone or perforated peptic ulcer. It must also be differentiated from acute mesenteric thrombosis, renal colic, acute cholecystitis, and acute intestinal obstruction.

P. Emergency measures for impending shock: Place patient N.P.O. If bowel sounds are absent, initiate nasogastric suction. Patient should be placed at bed rest and given 100-150 mg. demerol SQ as necessary for relief of pain. Atropine may be given as an antispasmodic 0.4-0.6 mg. SQ. Start IV to replace fluids and monitor urinary output. Use shock drugs if necessary; calcium gluconate must be given IV if there is evidence of hypocalcemia with tetany. Initiate prophylactic antibiotic therapy only if fever exceeds 102°F. Patient should be constantly attended and vital signs checked every 15-30 miunutes. CBC and urinalysis should be done frequently and monitored.

(1) Follow-up care: Patient should be kept N.P.O. for 48-72 hours. Examine frequently and closely for evidence of continued inflammation of the pancreas or related structures. Conduct periodic CBC and urinalysis. Hyperfeed the patient parenterally for first 48-72 hours, then gradually introduce oral feeding. When clinical evidence of pancreatitis has cleared, place the patient on a low fat diet.

(2) Prognosis: Recurrence is common. Surgery is indicated only when diagnosis is in doubt, if conservative treatment is not working, or in the presence of an associated disorder such as stones in biliary tract.

m. Acute Cholecystitis. Cholecystitis is associated with gallstones in over 90 percent of cases. It is caused by a partial or complete cystic duct obstruction. If the obstruction is not relieved, pressure builds up within the gallbladder. Primarily as a result of ischemic changes secondary to distention, gangrene may develop with resulting perforation. This may cause generalized peritonitis but usually remains localized and forms a chronic well-circumscribed abscess cavity.

S. Usually follows a large or fatty meal. Relatively sudden onset of severe, minimally fluctuating pain localized in the epigastrium or right upper quadrant frequently radiating to infrascapular area. In the uncomplicated case, the pain may gradually subside over a 12-18 hour period. Vomiting occurs in 75 percent of cases and 50 percent of these get variable relief.

O. Right upper quadrant abdominal tenderness, guarding and rebound pain. About 15 percent of cases have a palpable gallbladder and 25 percent of cases have jaundice. Fever is usually present. W.B.C. is usually 12-15,000.

A. Acute cholecystitis. Differential diagnosis: Perforated peptic ulcer, acute pancreatitis, appendicitis, hepatitis, and pneumonia with pleurisy on the right side.

P. Place patient N.P.O. Initiate IV for maintenance and feeding. Start prophylactic antibiotic therapy. Give analgesics as needed (morphine or meperidine). Smooth muscle relaxants, such as IM atropine or probanthine, should be used. Patient should be watched closely. W.B.C. should be done several times a day. Treatment is continued until symptoms subside. Cholecystectomy is usually required but not as emergency surgery unless there is evidence of gangrene or perforation.

Section VI - Genitourinary System

1-36. The genitourinary system is made up of the male and female sexual organs, the urethra, the bladder, the ureters, and the kidneys.

1-37. GENITOURINARY TRAUMA.

a. Kidney trauma. Most commonly caused by blunt external force such as blows, kicks, falls, etc., in the flank area. Other causes are wounds such as gunshot, stabs, etc.; it is very rarely caused by spontaneous rupture of a diseased kidney.

S. Pain at site of injury with a boring or tearing sensation felt in loin or upper abdomen.

O. Swelling and progressive rigidity of affected side. If there is a tear in the renal capsule, there is usually a rapidly expanding mass in the flank. From mild to gross hematuria is present in 90 percent of the cases. Shock occurs in varying degrees. W.B.C. elevates rapidly to 20,000 and higher.

A. Kidney trauma.

P. Conservervative treatment will usually provide satisfactory results in most cases where there is no penetrating wound. Bed rest for at least 2 weeks, until urine is clear. Shock and pain measures as required. Monitor urinary output closely. Patient must force fluids to insure urinary output of 25-40 ml./hr. In serious cases, an indwelling catheter should be installed and through IV therapy provide a urinary output of 25-40 ml./hr. Antibiotic therapy should be initiated in all cases as a prophylaxis. If an infection is allowed to develop, it will cause scar tissue and further complications. If at all possible, med evac all penetrating wounds and serious cases.

b. Bladder trauma. Causes include crushing injury from blows, seatbelts, etc., particularly if the injury occurs when the bladder is full; gunshot or stab wounds; or bony fragments from fractured pelvis.

S. Severe pain in lower abdomen. Slow and painful urination due to muscle spasm after injury.

O. Hematuria, often only a few drops of blood. Progressive symptoms of peritonitis depending on the extent of bladder rupture.

A. Bladder trauma.

P. Flat in bed. Treat for shock; install indwelling catheter. Prophylactic antibiotic treatment. Treat related problems (fracture, wound, etc.).

c. External genitalia trauma. Usual causes are heavy blows, cuts, direct injury, pelvic fracture, or straddle injury.

S. Intense to excruciating pain, swelling, and rapid development of a large hematoma.

O. Vary with the severity of the condition but will consist of hematuria, spasmodic contractions of the vesicle sphincter with pain, and

persistent desire to empty the bladder with involuntary ineffectual straining efforts and shock.

A. External genital trauma.

P. Indwelling catheter, cold packs, scrotal support, pain medication, and treat related problems (shock, wound, etc).

1-38. GENITOURINARY TRACT INFLAMMATION.

a. Renal calculi. Caused by a concentration of mineral salts and crystals that are formed in the calyx of the kidney. These kidney stones vary from small sandlike particles to large oval or branching (staghorn) stones that may fill the entire renal pelvis. Many factors are contributory such as infection, obstructions, dehydration, and hereditary tendency.

S. Severe intermittent colicky pain, radiating to pelvis, testicle, and/or inner aspect of the thigh. While the stone is in the kidney, the pain is dull and intensified by motion. When the stone enters the ureter, a sudden stab of excruciating pain is felt. If stone is in the bladder, the patient may be able to void only in the horizontal position.

O. Usually accompanied by chills, fever, violent movements, sweating, and shock as the stone moves through the ureter. Frequency, urgency, oliguria (diminished amount of urine formation), dysuria (painful or difficult urination), hematuria, and possibly pyuria (pus in the urine) are contributory findings. If anuria (complete urinary suppression) develops, it is indicative of renal failure.

A. Renal calculi.

P. Relieve pain (morphine 1/4 gr. q.2-3hr). Relax ureteral spasms with Pro-Banthine, 1/100-1/150 gr. atropine, or 1/100 gr. nitroglycerin. Force fluids and keep close record of intake and output. Strain all urine for stones; these should pass within 24-36 hours. At the first sign of anuria this becomes an acute emergency and patient should be evacuated to a definitive treatment facility.

b. Acute pyelonephritis. An acute infection of the kidney usually due to an ascending infection (from bladder through ureters to kidney) but may start from a systemic bacterial infection.

S. Sudden onset with chills, fever, some muscular rigidity, frequency, urgency, and dysuria.

O. Pain on percussion of the back with radiation to costovertebral angles and along the course of the ureters. Urinalysis shows albumen, pus cells, casts, R.B.C.'s, W.B.C.'s, and bacteria. W.B.C. in excess of 20,000.

A. Acute pyelonephritis. Differential diagnosis: Cystitis.

P. Bed rest, force fluids, and soft diet. Eliminate irritants such as alcohol or cocoa. Antibiotic therapy using Gantrisin, tetracycline, or penicillin/streptomycin. Symptomatic treatment.

c. Cystitis. Bladder infection usually due to bacteria.

S. Sudden or more gradual onset of burning pain on urination, often with turbid, foul-smelling, or dark urine; frequency; difficult or painful urination; and occasionally blood in the urine. Chills and fever are rare and if temperature is over 100° F., consider possibility of other causes than cystitis.

O. Usually no positive physical findings unless the upper tract is involved. Urinalysis shows pus, bacteria, and occasional hematuria. W.B.C.'s are rare unless upper tract is involved.

A. Cystitis. Differential diagnosis: Urethritis, pyelonephritis.

P. Gantrisin (sulfisoxazole) 1 gm q.i.d. x 10 days, alternate tetracycline 1-2 250 mg. tablets q.i.d. or ampicillin 1-2 250 mg. tablets q.i.d. Give Pyridium or methenamine urinary analgesic. NOTE: This may stain urine red to deep orange. Follow up in 2 weeks.

d. Urethritis. Caused by a wide range of agents that include gonococcus, Trichomonas, E. coli, and staphylococcus.

S. Burning on urination with pyuria. Discharge from urethra with a consistency from mucoid to purulent.

O. Discharge elicited by milking the penis. Gram's stain of discharge will usually show causative agent.

A. Urethritis. Differential diagnosis: Cystitis, prostatitis.

P. Ensure correct diagnosis with Gram's stain or culture. Treat causative organism with appropriate antibiotic.

e. Epididymitis. Frequent history of infection elsewhere in the general area such as urethritis, etc. Strenuous activity may precipitate spread of the bacteria.

S. Fever, malaise, nausea, tenderness, and pain that may radiate to the groin.

O. Inflammation of scrotal skin that may flake or crack. Scrotum dusky red and warm to the touch. Slight mass in the epididymis.

A. Epididymitis. Differential diagnosis: Orchitis.

P. Bed rest with scrotal elevation. Analgesics for pain, antibiotic therapy. DO NOT massage the prostate. If swelling persists, surgery will be required.

f. Orchitis. Usually results from a complication of mumps or other acute infections.

S. Fever; pain in the groin region.

O. Swelling of the affected testicle (may be bilateral).

A. Orchitis. Differential diagnosis: Epididymitis.

P. Bed rest; suspend the scrotum in suspensory or toweling

"bridge" and apply ice bags. Give codeine or morphine as necessary for pain. Inflammatory reaction can be reduced with hydrocortisone sodium succinate, 100 mg. IV followed by 20 mg. orally q.6h. x 2-3 days. Orchitis often makes the patient very uncomfortable but very rarely results in sterility.

g. Prostatitis. Caused by bacterial infection from systemic or urethral infections. Prostatitis may be acute or chronic; overmanipulation (a lot of sex) of chronic prostatitis gives rise to acute stage symptoms.

S. Acute symptoms: Perineal pain, fever, dysuria, frequency, and urethral discharge. Chronic symptoms: Lumbosacral backache, perineal pain, mild dysuria and frequency, and scanty urethral discharge.

O. Acute stage: Palpation of the prostate shows it is enlarged, boggy, and very tender. Even gentle palpation of the prostate gland results in a copious purulent urethral discharge. Chronic stage palpation of the prostate reveals an irregularly enlarged, firm, and slightly tender prostate. CBC will often show leukocytosis. Expressed prostatic fluid shows pus cells and bacteria on microscopy.

A. Prostatitis. Differential diagnosis: Urethritis. Lower urinary tract infections.

P. Bed rest, force fluids, sitz baths t.i.d. for 15 min, analgesics, and stool softeners. For acute prostatitis initial treatment may consist of sulfamethoxazole 400 mg., plus trimethoprim 80 mg. (co-trimoxazole), 6-8 tablets daily, or tetracycline 500 mg. q.i.d. x 2 weeks or ampicillin 500 mg. q.4h. x 2 weeks; two-week treatment usually results in subsidence of the acute inflammation, but chronic prostatitis may continue because most drugs fail to reach the prostatic acini. Chronic prostatitis should be treated with prolonged antibiotic therapy accompanied by vigorous prostatic massage once weekly to promote drainage.

h. Benign prostatic hyperplasia. Caused by hyperplasia (abnormal multiplication or increase in the number of normal cells in a tissue) of the prostatic lateral and subcervical lobes resulting in enlargement of the prostate and urethral obstruction.

S. Hesitancy and straining to urinate; reduced force and caliber of the urinary stream, and nocturia. Symptoms may be overlooked until the problem is well developed when the progression of the obstruction is slow.

O. Prostate is usually enlarged on palpation. The bladder may be seen and palpated as urine retention increases. Infections commonly occur as retention increases. Hematuria may occur.

A. Benign prostatic hyperplasia. Differential diagnosis: Urethral strictures, renal calculi, bladder tumor, or carcinoma of the prostate.

P. Relieve acute urinary retention by catheterization. Maintain catheter drainage if degree of obstruction is severe. Surgery is usually necessary. Treat infections that develop.

i. Carcinoma of the prostate. Rare before age 60. It metastasizes early to the bones of the pelvis and locally may produce urethral obstruction with subsequent renal damage.

S. Obstructive symptoms similar to those of benign prostatic hyperplasia are common. Low back pain occurs with metastases to the bones of the pelvis and spine.

O. Rectal exam reveals a stone-hard prostate that is often modular and fixed. Obstructions may produce renal damage and the symptoms and signs of renal insufficiency. Urine may show evidence of infection.

A. Carcinoma of the prostate. Differential diagnosis: Benign prostatic hyperplasia, urethral strictures, renal calculi, and bladder tumor.

P. Evac to a definitive care center.

j. Acute glomerulonephritis. Glomerulonephritis is a disease affecting both kidneys. It is most common in children 3-10 years old. Most common cause is a preceding infection of the pharynx or of the skin with group AB-hemolytic streptococci.

S. Malaise, headache, anorexia, low-grade fever, puffiness around the eyes and face, flank pain, and oliguria (diminished amount of urine output in relation to fluid intake). Hematuria is usually noted as "bloody" or if the urine is acid as "brown" or "coffee-colored." Respiratory difficulty with shortness of breath may occur as a result of salt and water retention and circulatory congestion. Tenderness in the costovertebral angle is common.

O. Mild generalized edema, mild hypertension, and retinal hemorrhages may be noted. There may be moderate tachycardia and moderate to marked elevation of B.P. The diagnosis is confirmed by urine examination that may be grossly bloody or coffee-colored or may only show microscopic hematuria. In addition, the urine contains protein (1-3+), red cell casts, granular and hyaline casts, white cells, and renal epithelial cells.

A. Acute glomerulonephritis. Differential diagnosis: Other diseases in which glomerular inflammation and tubule damage are present.

P. There is no specific treatment, but eradication of B-hemolytic strep is desirable. In uncomplicated cases, treatment is symptomatic and designed to prevent overhydration and hypertension. Bed rest until clinical signs abate. Blood pressure should be normal for 1-2 weeks before resuming normal activity. When protein excretion has disminished to near normal and when white and epithelial cells excretion has decreased and stabilized, activity may be resumed on a graded basis. Excretion of protein and formed elements in the urine will increase with resumption of activity, but such increases should not be great. Fluids should be restricted in keeping with the ability of the kidney to excrete urine. If edema becomes severe, a trial using an oral diuretic should be tried.

k. Phimosis.

(1) Cause and symptoms: Foreskin not pliable enough to retract over the glans penis. This causes pain on erection and may be complicated with paraphimosis.

(2) Treatment: Cut a dorsal slit in foreskin and schedule for

circumcision.

1. Paraphimosis.

(1) Cause and symptoms: Foreskin is constricted around the glans penis and cannot be reduced.

(2) Treatment: Cut a dorsal slit in the foreskin and schedule for circumcision.

Section VII - Nervous System

1-39. This section is not intended to cover all neurological problems because most neurological problems are beyond your scope for definitive treatment. It should, however, provide you with enough information to make you aware of the neurological problems you may face and enable you to make a tentative diagnosis.

1-40. COMPOSITION OF THE NERVOUS SYSTEM.

a. The nervous system is composed of (1) Central Nervous System (C.N.S) - Cerebrum, Cerebellum, Brain Stem, Spinal Cord; (2) Peripheral Nervous System (P.N.S.) - Peripheral nerves.

b. Review of the twelve cranial nerves.

(1) First: Olfactory. Sense of smell. Injury causes loss of sense of smell.

(2) Second: Optic. Sense of sight. Injury causes optic disturbances to loss of sight in one or both eyes.

(3) Third: Oculomotor. Supplies all the muscles of the orbit except the superior oblique and external rectus; also supplies the sphincter muscle of the iris and the ciliary muscle. Injury causes dilated and fixed pupils, slight prominence of the eyeball, and drooping of the upper eyelid.

(4) Fourth: Trochlear. Supplies the superior oblique muscle (smallest of the cranial nerves). Injury makes patient unable to turn eyes downward and outward. If attempted, affected eye is twisted inward causing double vision.

(5) Fifth: Trigeminal. Innervates facial sensation and motor to muscles of mastication (largest cranial nerve). This nerve also supplies the eye, nose, teeth, gums, palate, etc." Injury can cause numerous problems from dryness of the nose and eyeball to impaired action of the lower jaw.

(6) Sixth: Abducens. Supplies the external rectus muscle. More frequently involved in base of the skull fractures than any other nerve. Injury causes an internal or convergent squint often with a certain amount of contraction of the pupil.

(7) Seventh: Facial nerve. Motor nerve of all the muscles of facial expression: the platysma and buccinator; external ear muscles; posterior belly of the digastric and stylohyoid; nerve of taste for the anterior two-thirds of the tongue; the vasodilator nerve of the submaxillary and sublingual glands; and tympanic branch supplies the stapedius muscle. Most common effect of injury is Bell's facial palsy.

(8) Eighth: Auditory. Sense of hearing. Injury causes deafness.

(9) Ninth: Glossopharyngeal. Nerve of sensation to pharynx, fauces, and tonsil. Also sensation of taste to posterior third of tongue.

(10) Tenth: Vagus. Supplies the organs of voice and

respiration with motor and sensory fibers and the pharynx, esophagus, stomach, and heart with motor fibers.

(11) Eleventh: Spinal accessory. Consists of accessory portion which is motor to larynx and pharynx and spinal portion which is motor to sternocleidomastoid and trapezius muscles.

(12) Twelfth: Hypoglossal. Motor nerve of the tongue.

1-41. RECOGNITION OF NEUROLOGICAL PROBLEMS.

a. Not all problems have neurological origin. Your first task is to recognize the potential neurologic origin of the patient's complaint. There are eight different complaints or problems that point to neurologic disease. Although each of these complaints may be produced by diseases that do not involve the nervous system, differentiating between neurological and non-neurological causes is usually easy (e.g., a patient's leg may not move correctly because it is broken; he can't see properly because he needs glasses; or he has a headache and fever after taking a typhoid immunization). The eight complaints/problems are:

(1) Something doesn't move right.

(2) Something doesn't feel right (including disorders of other sensory modalities).

(3) I can't see properly.

(4) I can't think or communicate properly.

(5) I have spells.

(6) I am dizzy.

(7) My head hurts.

(8) Patient is unconscious, unrousable, or excessively drowsy.

1-42. NEUROLOGIC HISTORY. Most patients with neurologic disease will tell their physician what is wrong with them if he can properly interpret what they are trying to say and expands the history with skillful questioning. The history should give a profile of the disorder. This provides a valuable clue to the basic disease process. A few general principles are worth mentioning.

a. Seizures (convulsions) develop more rapidly than any other form of neurologic disorder. In many cases they develop in less than one second and may disappear as quickly as they come. Neuralgias are the only other group of disorders this abrupt. Vascular disorders including stroke and migraine usually take seconds to minutes to develop. Instead of clearing rapidly they melt away over hours or days. Demyelination seldom develops as rapidly as stroke but may progress over hours to days. Tumors usually develop in weeks to months and degenerative disorders in months to years. Toxic, metabolic, and infectious disorders are variable and more likely to leave their mark on other organ systems.

b. A brief neurologic review of systems should be made. It helps the medic be sure that the neurologic disorder is restricted to the problem

area he is evaluating. Possible intellectual defects can be elicited by asking about any difficulty in thinking or remembering, comparing recent job or school performance with past achievements may be helpful, asking whether he has any difficulty understanding what is said to him or expressing himself in oral or written language. Other possible complaints relative to the head are logically explored next. These include a discussion of the patient's headaches. He should be asked about any spells, attacks of dizziness, or alteration of consciousness he may have had. Visual complaints including diplopia, scotomata, and loss of visual acuity should be solicited.

1-43. NEUROLOGICAL EXAMINATION.

a. The following checklist will help you make a neurological examination: See para b, below, for details.

(1) Mental status.

(a) Affect and mood
(b) Orientation
(c) Memory
(d) Calculation and abstraction
(e) Aphasia

(2) Patient standing.

(a) Routine gait - note:

1. Arm swing
2. Width of gait
3. Limp or other abnormality

(b) Toe walking
(c) Heel walking
(d) Tandem walking
(e) Romberg's test

(3) Patient seated on exam table.

(a) Cranial nerve tests:

1. Visual acuity
2. Visual fields to confrontation
3. Ocular fundus
4. Extraocular movements
5. Pupillary reactions
6. Smiling, voluntary and emotional
7. Tongue protrusion
8. Voluntary palate movement
9. Hearing

(b) Arm strength and coordination

1. Strength

a. Shoulder abduction
b. Elbow flexion - extension
c. Thumb adduction

d. Thumb opposition
e. Wrist dorsiflexion
f. Handgrip

2. Reflexes
   a. Biceps
   b. Triceps
   c. Radial - periosteal

3. Coordination
   a. Finger to nose
   b. Rapid alternating movements
   c. Muscle tone

(4) Patient lying down.

(a) Leg strength and coordination

1. Strength

   a. Hip flexion
   b. Knee extension
   c. Dorsiflexion of the foot

2. Reflexes

   a. Abdominal
   b. Knee jerk
   c. Ankle jerk
   d. Babinski

3. Heel to shin test

(b) Sensory examination

1. Pain

   a. Face
   b. Extremities

2. Vibration - extremities
3. Light touch

   a. Cornea
   b. Face
   c. Extremities

4. Position
   a. Fingers
   b. Toes

b. Further details on neurological examination.

(1) Mental status exam. The medic who is evaluating a patient's mental status is usually looking for elements of dementia, aphasia, depression, or anxiety. These can often be observed during history taking.

(a) Affect and mood should be observed and recorded.

Affect is how the patient transmits his feelings and mood is what he is trying to transmit. In most individuals a depressed affect reflects a depressed mood and vice versa. Flattening or dulling of affect is seen in most depressed, schizophrenic, or parkinsonian patients.

(b) Orientation to time, place, and person should be recorded.

(c) Memory can usually be judged from the quality of the history, but should be commented on. Formal memory testing is unnecessary unless there is some reason to suspect difficulty.

(d) Calculation and abstraction should be tested in patients over 50 years of age. Serial 7's and a well-known parable (such as "why shouldn't people who live in glass houses throw stones?") are usually adequate.

(2) Gait and station. Four types of gait are routinely tested: ordinary gait, heel walking, toe walking, and tandem gait. Ordinary gait is observed for gross abnormalities of carriage and width of base. Arm swing may be deficient if there is weakness (especially hemiparesis) or a basal ganglion disease such as Parkinson's disease. Asymmetric heel elevation during toe walking indicates weakness in plantar flexors of foot while asymmetric toe and foot elevation in heel walking suggests weakness of the dorsiflexion of foot and toes. Tandem walking brings out gait ataxia (broad-based gait) seen in midline cerebellar disorders. Romberg's test is an evaluation of position sense. The patient is told to stand with his feet as close together as possible. If, with his eyes open, he can only stand with a wide base, the problem is most likely cerebellar. If he stands firm with eyes open, but tends to fall upon closing his eyes, the problem is position sense (posterior column or peripheral nerve) and Romberg's sign is present. While performing the Romberg test, it is convenient to examine for arm drift, a useful test of mild shoulder weakness or proprioceptive loss. Before the patient closes his eyes, have him extend both arms, palms up and elbows stiff in front of him. If while his eyes are closed he displays a tendency for either hand to pronate or either arm to "drift" downward, you may have discovered a significant defect. About 20 seconds of holding against gravity is sufficient.

(3) Cranial nerves. Now the patient can be seated and cranial nerves tested. Smell and taste need not be routinely evaluated. Vision requires more attention. Acuity should be checked first. With glasses on, the ability to read newsprint at about two feet constitutes 20/30 vision; and at 14 inches 20/50 vision. Each eye should be tested separately. Visual fields should also be tested in each eye separately. Always check all four quadrants. In patients over 50 years, check simultaneous stimulation by quadrants, preferably by superior temporal against the inferior nasal and then inferior temporal against the superior nasal. The optic nerve head is routinely examined as part of the ophthalmoscopic exam. A simple tuning fork test for hearing should be included. Extraocular movements and pupillary reactions should always be tested. Emotional and volitional face movements should be observed. Tongue protrusion, voluntary palate elevation, and voice timbre should be examined, but these are usually included as part of the routine oropharyngeal exam. Corneal reflexes, Myerson's sign, and snouting responses should be tested. Ordinary sensation in the face is best checked later with the rest of the general sensory exam.

(4) Motor strength and coordination in the upper extremities. Acceptable techniques of muscle testing consist of the examiner trying to move a joint against resistance or evaluation of a maximum effort by the patient to overcome the examiner. I generally prefer to have the patient exert a maximum effort against my resistance for internal and external rotation at the shoulder, flexion and extension of the elbow, and flexion and extension of the knee. I usually try to overcome the patient's fixation for shoulder abduction, wrist flexion and extension, hip flexion, and foot dorsiflexion. When a patient is making a maximum effort and the examiner is able to overcome the force of his muscle contraction, gradual movement of the joint will be felt. There should be no sudden "give" or relaxation, suggesting a lack of full cooperation. There are several numerical and descriptive scales for recording weakness. Like describing unconsciousness as coma, semicoma, and lethargy, they suffer from a lack of consensus among physicians as to what the numbers mean. At this stage in the examination, shoulder abduction, elbow flexion and extension, wrist dorsiflexion, and thumb opposition and adduction should be tested bilaterally. Biceps, triceps, and radial-periosteal reflexes may be tested at this time or deferred until the patient is supine. Coordination and muscle tone should be checked. Three maneuvers are essential. The first is the familiar finger to nose test. While this is being done, watch for any tremor or involuntary movement. Rapid alternating movements consisting either of opening and closing the hands or touching the tips of each finger with the tip of the thumb is tested next. Finally, passive circumduction of each wrist should be tried while the patient opens and closes the other hand as fast as he can. This will bring out any latent muscle rigidity.

(5) Completion of the motor and reflex exam. The patient should now be placed in the supine position. Up to this point we have been deliberately sloppy in testing strength. We have been testing it without providing fixation of the limb. As a screening procedure, this is fine. If any weakness has been suspected, shoulder rotation and elbow movements should be retested with the shoulder fixed against the examining table. Wrist and hand movements can similarly be isolated. Extension of the hip, extension of the knee, and dorsiflexion of the foot should be routinely examined. If there is a question of knee weakness, have the patient assume the prone position, fix the thigh against the table, and retest flexion and extension.

Biceps, triceps, and radial-periosteal reflexes in the arm should be tested if they have not been previously. Knee jerks, ankle jerks, and abdominal reflexes should be tested, and Babinski's sign sought. All reflexes require three elements: a sensory limb, some form of central integration, and a motor response. Reflexes will be altered if any of these three elements are disturbed. Any peripheral sensory disturbance or disturbance of the lower motor neuron or muscle can abolish reflexes. The only thing that will exaggerate reflexes is a disorder of the corticospinal system (the upper motor neuron syndrome). Finally, leg coordination should be observed with the heel-to-shin test.

(6) Sensory exam. It is convenient to perform the entire sensory exam at one time with the patient lying supine. During an ordinary screening exam, pain sensation with a sharp pin and fine touch with a wisp of cotton or Kleenex should be checked on both cheeks, both hands, and both feet. Position sense should be tested at least in the toes, and vibration sense in both feet and hands. A tuning fork should be used to test vibratory sensation on bony prominences.

1-44. EPILEPSY. Any recurrent seizure pattern. Violent, involuntary contractions of the muscles, occurring singly or in series, often accompanied by sudden loss of consciousness.

a. Grand mal attacks.

(1) Focal or jacksonian seizures. Initiated by specific focal phenomena (motor or sensory). Seizures are one-sided or localized. Head and eyes may turn to one side (that opposite the lesion). Jerking of the limbs may be one-sided. This is an acquired type of epilepsy. Convulsive movements start in small muscle groups (e.g., the hand) and slowly spread to other areas, it is termed the jacksonian "march." Loss of consciousness results when it becomes a generalized convulsion. Indicates specific portion of the cerebrum where lesion is located. May have an "aura," often referred to as a warning, but in reality it is a part of the seizure. The focal point indicates area of the brain where attack originates. Should be considered the focal trigger for the seizure.

(2) Typical grand mal seizures are characterized by a cry; loss of consciousness; falling; tonic then clonic muscle contractions of the extremities, trunk and head; urinary and fecal incontinence; frothing in the mouth; biting of the tongue. About 50 percent have an aura (auditory, visual, olfactory, visceral, or mental) disturbance. Losing consciousness after crying out, the person falls making no effort to protect himself.

(a) Tonic phase: sustained contraction of all muscles; body is rigid, jaws fixed, hands clenched, legs are extended, dilated pupils, face is red or cyanotic due to spasm of respiratory muscles.

(b) Clonic phase follows tonic phase in less than a minute with jerky movements due to alternating contraction and relaxation of muscles. The attack lasts 2 to 5 minutes usually. These attacks may be followed by deep sleep, headache, or muscle soreness.

b. Petit mal attacks. Fleeting attacks of staring into space without loss of consciousness (absence attack) for 1 to 30 seconds. Can occur with loss of muscular tone. Occurs predominantly in children and can recur as frequently as 100 attacks per day. Petit mal may eventually develop into grand mal later in childhood or adolescence.

c. Status epilepticus (continuous seizures).

(1) A serious condition in which seizures of the grand mal type follow in rapid succession with no intervening period of consciousness.

(2) Treatment of this particular condition: Give sodium phenobarbital (Luminal) 0.4 to 0.8 gm or paraldehyde 3 to 6 ml. intravenously to produce brief anesthesia and to help prevent further attacks.

d. Psychomotor seizures do not conform to the classic criteria of grand mal, petit mal, or jacksonian seizures. These are minor seizures with loss of contact with environment for 1 to 2 minutes. The patient does not fall but may stagger around performing automatically and does not understand what is being said. He may resist aid. Mental confusion continues for 1 to 2 minutes after attack has ended. May develop at any age. Usually associated with brain damage.

e. Treatment of convulsive seizures.

(1) Prevent the patient from injuring himself by placing a tongue depressor, handkerchief; or padded gag between teeth to prevent biting of the tongue. Do not restrain patient. Do not' leave him alone. If possible, before seizure, place a gag between the. teeth, but do not use a metal object. Do not pry the teeth open. Loosen clothing, especially around the neck. Turn head to the side, allowing mucus to flow from mouth and throat. After the attack, give phenobarbital 15-30 mg. t.i .d.

(2) Patient should be hospitalized. If hospitalization is not possible, you will have to control the seizures using anticonvulsant drugs such as Dilantin 100 mg. t .i.d. to q.i.d. P.O. or IM. If seizures add phenobarbital 15-30 mg. t.i.d. to q.i.d. What you want is the lowest dose possible to prevent seizures. To accomplish this start with a low dosage and if the patient has another seizure add a little to the dosage until seizures disappear canpletely. Patient ***must not*** drink alcohol.

1-45. HYSTERICAL ATTACKS VS. GRAND MAL ATTACKS.

a. May resemble grand mal epilepsy. With hysterical attacks the onset is slower and movements are purposeful, incontinence and cyanosis are absent, pupils do not dilate, patient does not injure himself when he falls, does not bite his tongue, usually has history of emotional upset and neurosis .

b. Treatment is the same as (1) of epilepsy treatment.

1-46. BELL's PALSY. A paralysis of the muscles of one side of the face sometimes precipitated by exposure, chill, or trauna. Can occur at any age but most common from 20-50.

S. and 0. One side of the face sags--eyelids, lips, eyebrows, or entire face.

A. Dell's palsy.

P. Keep face warm and avoid further exposure, especially to wind and dust. Protect eye with patch if necessary. Gentle upward massage of the involved muscles-5-10 minutes 2-3 times a-day helps maintain muscle tone. Prednisone 40 mg. daily x 4 days, then taper to 8 mg. a day in 8 days may help. In most cases partial or complete recovery occurs usually in 2-8 weeks (1-2 years in older patients).

1-47. The endocrine system is made up of glands of internal secretion (ductless glands). The secretions (hormones) enter directly into the blood or lymph circulation. Very small quantities of hormones are produced, only a trace being necessary to produce an effect, and some of them influence the body as a whole. Because of this and the fact that endocrine disorders can mimic a wide variety of primary disease states, the diagnosis of endocrine diseases is extremely difficult to make. The hormone producing glands include the pituitary, thyroid, parathyroids, ardrenals, gonads, and pancreas.

1-48. GOITER (see Chapter 5, Nutritional Diseases and Deficiencies).

1-49. DIABETES MELLITUS. A chronic metabolic disorder, characterized by abnormal insulin secretion and a variety of metabolic and vascular manifestations reflected in a tendency toward abnormally elevated blood glucose levels, large vessel disease, microvascular disease, and neuropathy.

S. Polyuria, increased thirst and hunger, paresthesia, and fatigue. Bed wetting may signal the onset of diabetes in children. Vaginitis and pruritus vulvae are frequent initial complaints of adult females. There may be marked weight loss despite normal or increased appetite. Diabetes should be suspected in obese patients, patients with a positive family Hx of diabetes, and in women who have delivered large babies (over 9 lbs) or who have had unexplained fetal losses.

O. In mild or moderate diabetes there may be no abnormal signs at onset, whereas the patient with severe insulin deficiency may present with loss of SQ fat, dehydration, muscle wasting, anorexia, nausea, vomiting, air hunger, and if untreated, coma and death. The retina may show microaneurysms, intraretinal hemorrhages, and hard exudates. Cardiovascular signs include signs of circulatory embarrassment of the lower extremities and hypertension. Neurological signs are predominantly sensory in nature with dulled perception of vibration, pain, and temperature, particularly in the lower extremities. The ankle jerk is often absent, but the knee jerk may be retained. Urinalysis is positive for glucose and ketones with specific gravity 1.02u-1.040. NOTE: Certain common therapeutic agents, e.g., ascorbic acid, salicylates, methyldopa, and levodopa, when taken in large doses, can give a false positive for glucose when using Clintest measurements or false negatives when using glucose oxidase paper strips (Clinistix, Tes-Tape, etc.). Despite the importance of the above signs and symptoms to the diabetic syndrome, none constitute the basis for a conclusive diagnosis. Whenever diabetes is suspected, it should be confirmed by a fasting blood or serum glucose and a glucose tolerance test if indicated.

A. Diabetes mellitus. Differential diagnosis: Nondiabetic (renal) glycosuria, hyperglycemia due to end organ insensivity to insulin.

P. A well balanced (sugar free) 1,000-1,200 calorie diet and weight reduction will manage many cases of mild to moderate diabetes, especially in obese patients who demonstrate symptomatology at age 40 or above. If glycosuria persists, the use of hypoglycemic agents such as insulin or tolbutamide (Oranase) is indicated. The ultimate choice of agents, route, dose, and interval must be determined by a careful analysis of serum glucose levels.

1-50. COMPLICATIONS OF DIABETES.

a. Hypoglycemia (insulin shock). An abnormally low blood sugar level and the most common complication of patients on insulin therapy.

S. Sudden onset (slower with long acting insulins) of mental confusion, bizarre behavior, sweating, palpitations, and tremulousness that may lead to coma, convulsions, and death.

O. Skin is moist, pale, and cool. There may be drooling from the mouth. Respirations are normal or shallow and the breath is usually odorless. B.P. is normal with a full bounding pulse. The urine is negative for glucose and ketones by the second voiding (there may initially be some residue from earlier hyperglycemia.) Serum glucose is <60 mg./100 ml.

A. Hypoglycemia due to insulin reaction. Differential diagnosis: Diabetic ketoacidosis, alcohol or drug induced coma, head injury, and cerebrovascular accidents. NOTE: If serum glucose is <50 mg./100 ml. the Dx is confirmed.

P. If still conscious and able to swallow, give orange juice, glucose, or any beverage containing sugar. If stuporous or unconscious, give 20-50 ml. 50% glucose IV stat. Then continue infusion at a rate of 10 gm/hr. If patient is still hypoglycemic, give a second bolus of 25 ml. 50% glucose. If unable to start IV, give 1 mg. glucagon IM or SQ then sugar by mouth when patient is awake and can swallow. If neither glucose nor glucagon is available, give 30 ml. syrup or honey in 500 ml. warm water rectally. Monitor patient response and plasma glucose level carefully.

b. Diabetic ketoacidosis. Hyperglycemic coma. Usually occurs in insulin dependent and juvenile (age <30) onset diabetics.

S. Gradual onset (1-2 days). Nausea, vomiting, abdominal pain, polyuria, intense thirst, and marked fatigue progressing to mental stupor and finally coma and death, if untreated.

O. Skin is hot, dry, and flushed with a loss of turgor. Mouth is dry. Respirations are deep, rapid, and labored. A fruity (acetone) odor is usually present on the breath. There may be signs of shock (see chapter 15). The eyeballs are soft. Urine glucose and ketones are strongly positive. Plasma glucose is >300 mg./100 ml. and ketones are strongly positive. NOTE: A rapid blood glucose determination can be made using commercially available glucose test strips (Dextrostix) and a rough quantitation of serum or plasma ketone can be made using either Ketostix or Acetest tablets. The presence of ketone may be masked if there is a strong level of lactic acid present.

A. Diabetic ketoacidosis. Differential diagnosis: Hypoglycemia, lactic acidosis due to septic, cardiogenic, or hypovolemic shock. NOTE: With lactic acidosis, the clinical picture will be approximately the same without the acetone breath or ketonuria. Blood glucose is variable.

P. (1) Diabetic ketoacidosis. Start IV .5 N saline at rate of 1 L./hr x 2 hrs, then adjust to 5-8 L. (total) over a period of 24 hours. If patient is already in shock, give N saline. Insulin (regular) 5-10 units/hr slow IV drip or IM. When blood glucose is <250 mg./100 ml., start

IV D5W at a rate of approximately 200 ml./hr with insulin q.2-4h. p.r.n. to maintain glucose level between 200 and 250 mg./100 ml.

(2) Lactic acidosis. . Start IV .5 N saline at rate of 1 L./2 hours, then 1 L./2-3 hours. Na bicarbonate 2 ampules (90 mEq.) stat. Repeat with 3-4 ampules if necessary. Stop when breathing returns to normal.

c. Prevention of soft tissue complications. Diabetics are susceptible to bedsores, infection, and gangrene. Because of poor circulation, feet should be kept scrupulously clean and dry. Extreme care should be used when trimming toenails, and corn and callouses should be removed by soaking, not cutting. Use oil or lanolin to keep feet soft and avoid tight shoes. Do not apply local heat to legs and feet. Instruct the patient to brush teeth at least three times a day. Take warm baths daily and seek prompt attention for any bruise or break in the skin.

1-51. ACUTE ADRENAL INSUFFICIENCY. A clinical syndrome caused by marked deprivation or insufficient supply of adrenocortical hormones following trauma, surgery, overwhelming sepsis (principally meningococcemia), or sudden withdrawal of corticosteroid drug therapy. Acute adrenal insufficiency constitutes a grave medical emergency and is rapidly fatal if not treated.

S. Headache, lassitude, nausea, vomiting, abdominal pain, C.V.A. pain, and tenderness. Confusion or coma may be present.

O. Fever 105°F. or more, B.P., cyanosis, petechiae (especially with meningococemia), dehydration, abnormal skin pigmentation, and lymphadenopathy marked eosinophilia. NOTE: A high eosinophil count in the presence of severe stress due to trauma, infection, or other mechanisms is strongly suggestive of adrenal failure.

A. Acute adrenal insufficiency due to _____. Differential diagnosis: Diabetic coma, cerebrovascular accident, acute poisoning.

P. IF ADRENAL FAILURE IS SUSPECTED, TREAT AT ONCE WITHOUT WAITING FOR CONFIRMATION BY LAB RESULTS. Treat for shock (see chapter 15). Start IV fluids stat., vasopressor drugs and O_2 p.r.n. Do not give narcotics or sedatives. 100 mg. Solu-Cortef IV stat. and ~~continue~~ IV infusion of 50-100 mg. q.6h. x 1 day, then same amount q.8h. x 1 day. Continue to give q.8h. with a gradual reduction in dose until the patient is able to take food by mouth, then give oral cortisone 12.5-25 mg. q.6h. and reduce to maintenance levels p.r.n. Monitor B.P. and observe for signs of edema and hypertension. If signs of cerebral edema (unconsciousness or convulsions) or pulmonary edema occur, withhold sodium and fluids and treat these conditions. If signs of hypokalemia occur, give potassium salts or food high in potassium content (orange juice or bananas). Evacuate when feasible.

1-52. EYE DISORDERS.

Conjunctivitis. Conjunctivitis is the most common eye disease. It may be acute or chronic. Most cases are due to bacterial, viral, or chlamydial infections. Other causes are allergy, chemical irritation, and fungal or parasitic infection. The mode of transmission is usually direct contact via fingers, towels, etc.

a. Bacterial conjunctivitis.

S. Copious purulent discharge and redness with no pain or blurring of vision.

O. Gram's stain of discharge usually shows streptococcus or staphylococcus organisms.

A. Bacterial conjunctivitis. Differential diagnosis: Iritis, glaucoma, corneal trauma, keratitis, and other causes of conjunctivitis.

P. Disease is usually self-limiting, lasting 10-14 days if untreated. Sulfonamide or antibiotic ophthalmic ointment applied locally t.i.d. usually clears the infection in 2-3 days.

b. Viral conjunctivitis.

S. Redness, copious watery discharge, and scanty exudate from the eye. Usually associated with systemic symptoms, pharyngitis, fever, malaise, and adenopathy.

O. Children are more often affected. Contaminated swimming pools are a major cause.

A. Viral conjunctivitis. Differential diagnosis: See bacterial conjunctivitis.

P. No specific treatment. Use antibiotic ophthalmic ointment to prevent secondary infections. Usually lasts at least 2 weeks.

c. Chlamydial keratoconjunctivitis (trachoma). Trachoma is a major cause of blindness. In endemic areas it is contracted in childhood. It is usually insidious with minimal symptoms. In adults it is acute.

S. Redness, itching, tearing, and slight discharge.

O. Bilateral follicular conjunctivitis, inflammation of the cornea, and pannus (cloudy, uneven, newly formed vascular tissue over the cornea). In the later stages, scarring of the eyelid margin may cause inversion of the eyelid and the eyelashes causing them to rub against the cornea thereby scratching and scarring the cornea. This decreases the vision, leading to blindness. Giemsa stain scraping from conjunctiva shows typical cytoplasmic inclusions in the epithelial cells. In active trachoma, the smear may also include polymorphonuclear leukocytes, plasma cells, and debris-filled macrophages.

A. Trachoma. Differential diagnosis: Other eye infections.

P. Oral tetracycline 250 mg. q.6h. x 3-5 weeks, good hygiene practice.

1-53. EAR DISORDERS.

a. External otitis. An infection of the external ear canal, usually bacterial, with occasional secondary fungal infection. In many cases there is no infection; it is a contact dermatitis or a variant of seborrheic dermatitis.

S. Itching and pain, dry scaling ear canal; there may be a watery or purulent discharge and intermittent deafness. Pain may become extreme when ear canal becomes completely occluded. Adenopathy and/or fever indicates increasing severity of infection.

O. Crusting, scaling, erythema, edema, and pustule formation. Cerumen may be absent. Lab: W.B.C. may be elevated or normal.

A. External otitis. Differential diagnosis: Draining otitis media.

P. Clean ear, then apply antibiotic ointment or ear drops with a cotton wick for 24 hours, followed by ear drops twice daily. If there is systemic involvement, systemic antibiotics may be necessary.

b. Otitis media. Infection of the middle ear.

(1) Acute otitis media.

S. Ear pain, deafness, fever, chills, hearing loss, and a feeling of fullness and pressure in the ear. If the eardrum ruptures, discharge is found in the ear.

O. Exam shows a loss or normal landmarks and a bulging of the eardrum as the pressure increases. Lab: W.B.C. usually increased; Gram's stain of drainage may reveal infecting organism.

A. Acute otitis media. Differential diagnosis: External otitis, chronic otitis media.

P. Bed rest, analgesics, and systemic broad-spectrum antibiotics. Ear drops are of limited value; local heat may help resolve the infection. Most important is a myringotomy (incision of the tympanic membrane) if there is continued bulging of the eardrum, continued pain, fever, increasing hearing loss, or vertigo.

(2) Serous otitis media.

S. Hearing loss, full or plugged feeling in the ear, and an unnatural reverberation of the patient's voice.

O. Eardrum retracted often with a characteristic "ground glass" amber discoloration. Air-fluid bubbles or a fluid level can sometimes be seen on the eardrum. Absence of fever, pain, and toxic symptoms. Serous otitis media is caused by eustachian tube blockage.

A. Serous otitis media. Differential diagnosis: Acute otitis media.

P. Nasal decongestants to keep eustachian tube open. Antihistamines if there is any suggestion of nasal allergy. Treat cause of blockage, e.g., tonsillitis or sinus infection. If all else fails to relieve the fluid, a myringotomy is necessary to drain the ear. Indwelling plastic tubing for drainage can be used in persistent cases.

c. Diseases of the inner ear.

(1) Meniere's disease. Characterized by recurrent episodes of severe vertigo associated with deafness and tinnitus. Meniere's disease is usually encountered in men 40-60 yrs old. Cause is not known.

S. Intermittent severe vertigo that may cause the patient to fall. Nausea, vomiting, and profuse perspiration are often associated. These attacks may last from minutes to several hours. Frequency of attacks varies. Headache, hearing loss, and tinnitus occur during and persist between attacks. Hearing loss may be progressive and in 90 percent of the cases unilateral. Involuntary eyeball movement may occur during attacks of vertigo.

O. Increased sensitivity to loud sounds and decreased speech discrimination. Marked psychic disturbance is found in many patients.

A. Meniere's disease. Differential diagnosis: Systemic infections, psychiatric disorders, and cerebrospinal injuries or disorders.

P. Reassurance, salt-free diet; antihistamines (Benadryl and Dramamine) 50-150 mg. orally 3-4 times daily may help some patients. Parenteral Dramamine, Benadryl or 0.6 mg. atropine sulfate may stop acute attacks. Meniere's disease is chronic, recurrent, and may persist for years.

(2) Acute nonsuppurative labyrinthitis.

S. Usually follows respiratory tract infections. Manifested by intense vertigo, usually with marked tinnitus, a staggering gait, and involuntary eyeball movement.

O. Hearing loss is often not present.

A. Acute nonsuppurative labyrinthitis. Differential diagnosis: Meniere's disease.

P. Bed rest, preferably in a darkened room until severe symptoms subside. Antibiotics are of little value unless there is an associated infection of the middle ear or mastoid bone. Antihistamine (Benadryl or Dramamine) may be of value. Phenobarbital 15-60 mg. 3-4 times a day is generally helpful. Thorazine HCl 50 mg. IM is useful in the acute early phase. Attacks may last for several days but recovery is usually complete.

1-54. NOSE DISORDERS.

a. Sinus infection.

S. History of an acute upper respiratory infection, swimming or diving, dental abscess or extraction, or nasal allergies. Pain, tenderness, redness, swelling over the involved sinus, fever, chills, malaise, and headache.

O. Nasal congestion and purulent nasal discharge. Lab: Smear of nasal discharge may show causative organism; white count may be elevated.

A. Sinus infection. Differential diagnosis: Acute dental infection.

P. Bed rest, sedatives, analgesics, light diet, force fluids, nasal decongestants (nose spray or drops) 2-3 times a day, local heat, and systemic antibiotics will usually clear up the infection.

b. Common cold. Caused by a wide variety of viruses, all of which exist in multiple antigenic types, and recurrent infection is common.

S. Malaise, fever, headache, nasal discomfort with watery discharge and sneezing followed by mucoid to purulent discharge, and nasal obstruction. Throat symptoms are dryness and soreness rather than actual pain and hoarseness.

O. Nasal mucosa is reddened and swollen. Pharynx and tonsils show mild to moderate infection usually without edema or exudate; cervical lymph modes may be enlarged and slightly tender. Lab: Not remarkable unless there is a secondary bacterial infection.

A. Common cold. Differential diagnosis: Flu or URI.

P. General measures: rest, forced fluid, symptomatic treatment, e.g., asiprin for headache, etc.

c. Allergic rhinitis (hay fever).

S. Nasal congestion; a profuse, watery nasal discharge; itching nose often leading to paroxysms of violent sneezing; nasal mucosa is pale and boggy; itching watery eyes; conjunctiva is often red and swollen.

O. Gram's stain of nasal secretion reveals numerous eosinophils, C.B.C. shows 5-40% eosinophilia.

A. Hay fever. Differential diagnosis: Other common upper respiratory infections.

P. Antihistamines give relief in 60-80 percent of cases but effectiveness wanes as the allergy season progresses. Sympathomimetic drugs such as ephedrine are effective by themselves or in combination with antihistamines. Sedation may be of value for tense or nervous patients.

1-55. THROAT DISEASES.

a. Acute tonsillitis is nearly always a bacterial infection, often due to streptococci.

S. Sudden onset of sore throat, fever, chills, headache, anorexia, and malaise.

O. Swollen and red tonsils with pus or exudate. Cervical nodes are frequently enlarged and tender. White count may be elevated. Gram's stain of pus or exudate may show causative organism; throat culture will.

A. Tonsillitis. Differential diagnosis: Simple pharyngitis, infectious mononucleosis, Vincent's angina, and diphtheria.

P. Bed rest, fluids, light diet, warm salt water gargles, analgesics, and antibiotics as required.

b. Simple pharyngitis. Usually bacterial or viral in nature; may be part of the syndrome of an acute specific infection (e.g., measles, scarlet fever, etc.).

S. In acute pharyngitis the throat is dry and sore; systemic symptoms are fever and malaise. Chronic pharyngitis may produce few symptoms, e.g., throat dryness with thick mucus and cough or recurrent acute episodes of more severe throat pain, dull hyperemia.

O. Acute pharyngitis, red mucosa slightly swollen with a thick sticky mucus. Chronic pharyngitis, mild swelling of the mucosa with a thick tenacious mucus often in hypopharynx.

A. Simple pharyngitis. Differential diagnosis: Other upper respiratory infections and part of the syndrome of an acute specific infection (e.g., measles, whooping cough, etc.).

P. Symptomatic treatment; rest, light diet, analgesics, warm saline gargles, and antibiotics if it is a bacterial infection.

c. Influenza transmitted by respiratory route. Although sporadic cases occur, usually occurs as pandemic or epidemic in the fall or winter. Incubation period is 1-4 days.

S. and O. Abrupt onset of fever, chills, malaise and muscular aching, substernal soreness, headache, sore throat, nonproductive cough, nasal stuffiness, mild pharynged infection, flushed face, conjunctival redness, and occasional nausea. Fever lasts 1-7 days (usually 3-5). If fever persists more than 4 days, cough becomes productive or if W.B.C. rises to about 12,000, secondary bacterial infection should be ruled out or verified and treated. Most fatalities are due to bacterial pneumonia.

Lab findings: Leukopenia is common and proteinuria may be present.

A. Influenza.

P. Symptomatic, bed rest to reduce complications, forced fluids, analgesics, and sedative cough mixture. Do Not Use antibiotics unless secondary bacterial infection develops.