CHAPTER 5

NUTRITIONAL DISEASES AND DEFICIENCIES

5-1. GENERAL. Nutritional diseases and deficiencies are usually related directly to ignorance of sound nutritional practice and to poverty. Many people exist on a diet based almost exclusively on one principal starchy staple food--rice, millet, or corn for example. Another factor is parasitic and infectious diseases. These contribute to decreased intestinal absorption, sometimes to increased requirements, and usually to some degree of anorexia. These create a vicious progressive spiral where a diet deficiency is compounded. In most cases where you find evidence of a marked deficiency of one particular substance or group of substances, other deficiencies also exist. The single most important thing in the treatment of nutritional diseases is starting a completely adequate diet.

5-2. PELLAGRA (mal de la rosa, psilosis pigmentosa, Alpine scurvy, or chichism). The principal manifestation of a severe deficiency of niacin, usually complicated by deficiencies of other B vitamins. It is found worldwide and is usually associated with diets high in corn and containing little or no meat, milk, fish, or other good sources of protein. The disease is more prevalent during the spring.

S. Onset is gradual with loss of strength, loss of weight, and sore, red tongue. Dermatitis may occur. Diarrhea or alternating periods of diarrhea and constipation may occur.

O. Look for red tongue, gastrointestinal disturbances, psychic disturbances, and dermatitis. The tongue is swollen, denuded of its papillae (glossitis), and often painful and extremely sensitive. The dermatitis, characteristically, is symmetrically distributed. In most instances it is restricted to parts exposed to the sun. In the early stages the rash resembles a sunburn. This may be followed by vesiculation and bulla formation. The skin becomes thickened and roughened, and as the acute inflammation subsides, the brownish pigmentation remains. Repeated attacks lead to marked atrophy of the skin. The psychic disturbances in the early stages are that of neurasthenia, which increases in severity with progression of the disease. In advanced and long-standing cases, true psychoses occur. In these cases, spastic gait, peripheral neuritis, and other indications of organic involvement are not uncommon.

A. Pellagra (lack of nicotinic acid and tryptophan in the diet).

P. High protein, high vitamin diet. Nicotinic acid or niacinamide 50-500 mg. daily oral or injection. Give therapeutic doses of thiamine, riboflavin, and pyridoxine daily.

5-3. BERIBERI. Caused by a deficiency in vitamin B1 (thiamine hydrochloride) and other vitamins, and is found in areas where the diet consists primarily of polished rice, white flour, and other nonvitamin bearing foods. Increased need for vitamin B1; fever, high carbohydrate intake, or alcoholism may lead to deficiency.

S. Onset is usually gradual with progressive weakness of the most used muscle groups (most commonly in extensor muscles of the thigh). In many instances, patient is unable to rise from squatting position.

O. Atrophy of the muscles most used. Sensory disturbances

(hyperesthesia or hypoesthesia) usually appear at the same time but are usually less prominent. In severe cases many muscle groups may be affected and you see flaccid paralysis, muscular atrophy, with or without evidence of cardiac enlargement, and tachycardia.

With a more serious form (wet beriberi), the clinical picture is predominantly that of acute congestive heart failure with relatively little evidence of nervous system involvement. The onset is frequently rapid and acute, and the marked edema may mask the presence of muscle atrophy. Sudden collapse occurs frequently.

A. Vitamin B1 (thiamine) deficiency (beriberi). Differential diagnosis: Tabes dorsalis, post diphtheritic paralysis, and acute heart failure resulting from other causes.

P. Thiamine hydrochloride 20-50 mg. orally IV or IM in divided doses daily x 2 weeks then 10 mg. daily orally. Alternative: Dried yeast tablets (brewer's yeast) 30 gm. t.i.d. Well balanced diet of 2,500-4,500 calories a day when tolerated.

Prognosis: Recovery is rapid and complete in infants and small children. Recovery is slow in adults and there may be permanent disability, such as muscle weakness or flaccid paralysis, due to nerve cell degeneration. In the acute form of wet beriberi, deaths are frequent.

5-4. SPRUE (psilosis, Ceylon sore mouth, malabsorption syndrome). Sprue syndromes are diseases of disturbed small intestine function characterized by impaired absorption, particularly of fats, and motor abnormalities. It is not associated with any particular diet or dietary deficiency. Characteristically affects white upper-class individuals of long residence in endemic areas. Occurs in Far East, Puerto Rico, sporadically in U.S., and rarely in Africa.

S. Main symptom is diarrhea, explosive and watery at first, later stools are fewer, more solid, and characteristically pale, frothy, foul-smelling, and greasy. Patient has sore tongue and mouth and flatulent indigestion. Abdominal cramps, weight loss (often marked), pallor, irritability, muscle cramps, and weakness may occur.

O. Paresthesia (abnormal sensation from numbness to heightened sensitivity), asthenia (lack or loss of strength), abdominal distention, and mild tenderness are present. At first there are small painful ulcers on the tongue and buccal mucosa. Later the tongue becomes acutely inflamed and denuded. The ulcers can extend into the pharynx and esophagus and may cause dysphagia. Signs and symptoms of multiple vitamin deficiencies will be found in severe cases.

A. Sprue (malabsorption syndrome). Differential diagnosis: Anatomic abnormalities (fistulas, blind loops, jejunal diverticulosis) or regional enteritis.

P. Folic acid 10-20 mg. daily orally or IM for 2-4 weeks until remission of symptoms, then 5 mg. folic acid daily, tetracycline 250 mg. q.i.d. x 10 days. High calorie, high protein, low fat diet. Multiple vitamins should be given daily.

5-5. PROTEIN AND CALORIE MALNUTRITION.

a. Kwashiorkor (malignant malnutrition). Caused by inadequate proteins with adequate calories. Usually occurs in infants after weaning but may occur in children of any age and even in adults. Occurs wherever people subsist on starchy staple foods without adequate protein supplements.

S. Irritability, apathy, skin changes (rash, desquamation, depigmentation or hyperpigmentation, ulceration), inflammation of lips and mouth, conjunctivitis, sparse or depigmented hair, anorexia, vomiting, and diarrhea.

O. Growth and maturation are retarded, muscular wasting, edema (usually starts in the feet and lower legs but may affect any part of the body including the face). Liver enlargement also occurs and may or may not be palpable. R.B.C. nearly always shows moderate anemia.

A. Kwashiorkor.

P. Restore and maintain fluid and electrolyte balance. All but the most severely ill respond to a diet based on milk; dilute milk feeding can usually be introducted after 24 h. Sufficient milk should be given to supply 2-5 gm of protein/kg./day. At this stage, more calories in the form of sugar and cereal may be added to the diet to provide 150-250 k cal/kg./day. Correct remaining vitamin and mineral deficiencies. Small frequent feeding around the clock are tolerated best in early stages of recovery. Antibiotics may be indicated, but treatment of malaria and other parasitic infections should be delayed until patient is clinically improved. Whole blood is contraindicated unless Hb is < 4 gm%.

b. Marasmus. Total starvation, a protein and calorie malnutrition.

S. Constant hunger; thin, emaciated body but protuberant abdomen.

O. Retarded growth; atrophy of muscle tissue; skin is loose and wrinkled, especially around the buttocks, and when pinched between thumb and forefinger, shows almost a complete absence of subcutaneous fat. No edema; face is drawn and monkeylike. Diarrhea and anemia are frequent but not always present.

A. Marasmus.

P. Initial feedings should be slow and increased gradually. There must be adaquate intake of calories and protein; same treatment as for kwashiorkor.

5-6. SIMPLE GOITER (endemic goiter). An enlargement of the thyroid gland without either hyper- or hypothyroidism due to lack of iodine in the diet. Can be due to excess intake of goitrogenic vegetables (rutabagas, turnips, cabbage, mustard seeds).

S. In the majority of cases there are no symptoms or symptoms resulting from compression of the structures in the neck and chest (wheezing, dysphagia, respiratory embarrassment).

O. Swelling of neck, palpable thyroid gland often extremely large.

A. Simple goiter. Differential diagnosis: Toxic, diffuse, or modular goiter.

P. Iodine therapy 5 gtt. daily S.S.K.I. (saturated solution of potassium iodine) or 5-10 gtt. of a strong iodine solution in a glass of water. Continue until gland returns to normal size, then place patient on maintainance dose 1-2 gtt. daily or use iodized table salt.

5-7. OSTEOMALACIA (rickets). A calcium-phosphorus deficiency primarily of women, particularly during pregnancy and lactation; can be secondary to disorders in fat absorption (sprue, diarrhea, pancreatitis) or due to prolonged use of aluminum hydroxide gels, causing chronic phosphate depletion.

S. Usually mild aching of the bones, particularly long bones and ribs, muscular weakness, and listlessness.

O. Bony tenderness is common and severe tetany may occur. Bones become soft and flexible; deformities are more frequently caused by bones bending (bowing) rather than fractures, particularly in the legs, thorax, and spine.

A. Rickets. Differential diagnosis: Arthritis, osteoporosis, osteogenesis imperfecta.

P. Treatment can only protect against further deformities. Diet high in calcium and phosphorus, 25-100 thousand units vitamin D daily. Treat contributing disease if present.

5-8. SCURVY. Due to inadequate intake of vitamin C, but may occur with increased metabolic needs or decreased absorption. Frequently seen in formula-fed infants, elderly bachelors, and food fadists.

S. Mild or early manifestations are edema and bleeding of the gums. Severe or late manifestations are swelling of the joints, marked bleeding tendency, loosening or loss of teeth, poor wound healing, or in severe cases old scar tissue breaking down and reopening of healed wounds.

O. Mild or early manifestations are porosity of dentine and hyperkeratotic hair follicles. In severe or late cases, patient bruises easily, severe muscle changes, and anemia.

A. Vitamin C deficiency (scurvy).

P. a. Ascorbic acid 50 mg. q.i.d. x 1 wk in infantile scurvy then 50 mg. t.i.d. x 1 mo with prophylactic doses (25-30 mg./day) supplemented by orange or tomato juice. In vomiting or diarrhea, give one-half oral dose IM or IV as sodium ascorbate.

b. For adult scurvy, 250 mg q.i.d. until asymptomatic. When parenteral therapy is required, give sodium ascorbate at the same dosage. Ascorbic acid 300-500 mg./day P.O. in divided doses should be given for several months in chronic scurvy with gingivitis, repeated hemorrhagic manifestation, or joint symptoms.

5-9. VITAMIN A DEFICIENCY. Fat-soluble vitamin necessary for normal function and structure of all epithelial cells and for synthesis of visual purple in retinal rods (night vision). Toxic if too much is ingested

(e.g., seal and polar bear liver).

S. Mild or early manifestations are dryness of skin and night blindness.

O. Mild or early manifestation of follicular hyperkeratosis. In late or severe cases, softening of cornea, dryness of conjunctiva, atrophy and keratinization of the skin.

A. Vitamin A deficiency usually in conjunction with other deficiencies.

P. Oleovitamin A, 15-25 thousand units once or twice a day orally. If absorption defect is present, give same dosage IM. Care must be used as minimum toxic dose in adults is about 75-100 thousand units daily.

S and S for hypervitaminosis A are anorexia, loss of weight, dry and fissured skin, brittle nails, hair loss, gingivitis, splenomegaly, anemia, and C.N.S. manifestations.

CHAPTER 6

PEDIATRICS

6-1. The pediatric patient may mean the neonate (up to 4 weeks), the infant (1 month to 1 year), the child (1 year to 6 years), or the preadolescent (6 years to 12 years). The treatment and drug dosage of a 9-pound infant may be vastly different from an 11-year-old preadolescent. The adolescent will be treated generally as an adult (over 12 years old). For purposes of identification, specify the age and the approximate weight of the pediatric patient. In assessing the seriousness or chronicity of a disease in the pediatric patient, steadily increasing height and weight is not the sign of a very sick patient. A fat child who remains fat is generally not very sick or at least not chronically sick. A child with good appetite is rarely very sick.

a. History is the most important single factor in making a proper assessment for many pediatric problems. It should be obtained from the mother or guardian. If the child is old enough to talk, you can obtain much valuable information from him or her. Allow the informants to present the problem as they see it, then fill in the necessary past and family history and pertinent information.

b. Examination of pediatric patients, except newborn and infant, follows the same procedures as the examination of adult patients.

(1) Newborn examination.

(a) General appearance. The prime concern in the first few minutes of life is respiration. A crying baby has a good respiration.

(b) Skin color. Definite jaundice in the first 24 hours is pathologic and means infection, erythroblastosis (Rh factor), or prematurity.

(c) Extremities. All should move erratically.

(d) Reflexes. Sucking reflex should be present at birth.

(e) Digits. The fingers and toes may be cyanotic, but the trunk should be pink. A baby depressed from too much anesthesia at birth, prematurity, or difficult labor will lack some of the above. Try mildly painful stimulation (pinch); it may bring the baby out of its depression.

(2) Infant examination. Every child should receive a complete systematic examination periodically.

(a) Child should be observed from the time he or she is first brought into the room and during the entire examination.

(b) A friendly manner, quiet voice, and a slow and easy approach will usually help in the examination; if not, proceed as gently as possible in an orderly and systematic manner.

(c) Holding for examination. Before 6 months of age an infant will usually tolerate an examination table. From 6 months to 3 or 4 years of age most examinations can be performed best while the child is held in the parent's lap or over the shoulder.

(d) Parents should remove their child's clothing. If you must remove the child's clothing, do it gradually to prevent chilling or alarming the child.

(e) It is usually best to begin by examining an area unlikely to be associated with pain or discomfort. Painful/uncomfortable areas should be examined last.

(f) Take and record height, weight, and head circumference at each examination. These measurements give information regarding patterns of growth when compared with previous examination measurements.

c. The newborn generally weighs 7-1/2 pounds (3.4 kg.) in modern countries; in deprived countries, weight will probably be less than 7-1/2 pounds. Any newborn less than 5-1/2 pounds (2,500 gm or 2.5 kg.) is by definition "premature" regardless of the length of pregnancy and will require more care, have less chance of survival, and will grow and mature slower. A normal term infant's birth weight should <u>at least</u> double in 5 months and triple in 12 months.

d. Vital signs:

	Pulse/min	Respiration/min	B.P. (Systolic)
(1) Birth	140	40	60-80
(2) Six months	110	30	90
(3) One year	100	28	90
(4) Three to four years	95	25	100
(5) Five to ten years	90	24	100

e. Laboratory norms for infant and child:

	Birth	Three months	One year	Five years
(1) Hb	16-20	10-11	12-13	12.5 - 13.5
(2) W.B.C.	10-20,000	5-9,000	6-10,000	6-10,000
(3) HCT	50-60	30-33	35-36	38-41
(4) Neutrophils	45-55%	30-40%	35-45%	40-50%
(5) Lymphs	30-45%	50-60%	50-60%	45-55%

f. Calculating drug dosages (Young's Rule):

For children over 2: $\frac{\text{Child dose} = \text{age (years)} \times \text{adult dose}}{\text{age} + 12}$

For children under 2: $\frac{\text{Child dose} = \text{age in months} \times \text{adult dose}}{150}$

g. Feeding. The child must be fed by frequent intake of fluid and calories. A schedule of feeding is not necessary. A sick child must be encouraged to eat or drink.

(1) Breast feeding. This is usually superior to bottle feeding. Make sure the mother has no breast infection, she has milk, and the infant can suck properly. The infant receives all the vitamins and nutriments that are required if the mother is healthy and is receiving proper nutrition (it never hurts to give supplemental daily multivitamins to a breast-feeding mother).

(2) Bottle feeding. The infant may be fed by breast alternating with bottle or with bottle alone. If milk formula is not available, one will have to be improvised.

(3) Nutritional requirements:

(a) Calories per day. First year, 50 calories per pound (about 1,000 calories per day at age one year).

(b) Fluid. Two to three ounces per pound per day. Feedings may be given as often as possible to the sick child if the child will take it, unless some medical contraindication exists. The healthy child may eat from three to eight times daily.

(c) Caloric content:

1. Cow milk = 20 calories per ounce.

2. Evaporated milk = 40 calories per ounce.

3. Sugar = 120 calories per ounce or 60 calories per tablespoon.

(d) Milk will provide enough sodium, potassium, calcium, etc. to nourish any child temporarily, but if it is not fortified, it must be supplemented with iron and vitamin C and D. Be sure the milk is pasteurized. If there is a doubt, boil (15 seconds at a rolling boil is required).

(4) Improvising a formula. The formula should be about as thick or viscous as cow's milk. It should be reasonably palatable. Taste it yourself; if it tastes bad to you, the child may not take it. It should be comfortably warm. The bottles should be sterilized. If bottles are not available, spoon feed or drip the milk in with syringe or tubing. A good oral solution can be made using 5 percent dextrose, 1 tablespoon of sugar, and 1/2 teaspoon of salt per liter and is especially useful in a dehydrated patient who is not vomiting. It provides fluid, calories, and salt, but if it is to be used for extended periods, it must be fortified with vitamins.

6-2. THE DEHYDRATED CHILD. Newborns and infants can become dehydrated fairly rapidly due to illness or lack of fluid intake.

S. Fever; dry skin, mucous membranes, and tongue; sunken eyeballs; poor skin turgor, and depressed fontanelles.

O. Decreased or no urine output; urine dark and concentrated with a high specific gravity and a high hematocrit.

A. Dehydrated child.

P. Fluid replacement is of prime importance. If the dehydration is not severe and the patient can take fluids by mouth, then fluids should be forced. If the dehydration is severe or the patient cannot take fluids by mouth, then fluids must be replaced IV. Do not try to replace all the fluid deficit in a short period as it may throw the child into shock. Estimate the fluid deficit. Figure the daily requirement.

Maintenance fluid requirement:

0 - 10 kg. 100 cc./kg.

11 - 20 kg. $\frac{100 \text{ cc./kg.}}{10}$ + 50 cc./kg.

21 kg. and over $\frac{100 \text{ cc./kg.}}{20}$ + 50 cc./kg. + 25 cc.

Then give the daily requirement plus 1/2 of the deficit over the first 24 hours. (A good replacement fluid is 1/4 strength normal saline in 50% D5W.)

Patient should be catheterized and urine output monitored closely. You are looking for a return to good skin turgor, moist mucous membranes and tongue, and lightening of the urine. Lowering of urine specific gravity is your most important sign. Treat the cause, e.g., fever, throat infection, etc.

6-3. FEVER OF UNDETERMINED ORIGIN (FUO).

a. Fever is generally a sign of infection, but infants can spike fever for almost any reason (e.g., cutting teeth, constipation, reaction to diet, allergy, diaper rash, etc.). Fevers due to infections are usually low-grade in adults but may be much higher in infants and young children. Children often convulse with temperatures over 104o F. (occasionally at lower temperatures).

b. Treatment. Initially, lowering the temperature (if it is 104o F. or above) is of primary importance. Give Tylenol (Tempra, acetaminophen) 10 mg./kg. q.4h. if child is less than 1 year old; give aspirin 65 mg./yr of age q.6h. first if child is 1 year or older. Then give a sponge bath or alcohol bath to cool the body. The patient must be monitored closely and baths repeated as needed to keep the temperature down. If unexplained fever has been present over 24 hours, a white count and differential should be done. Ideally, the patient should be treated for the specific disease; however, if a diagnosis can't be made, broad spectrum antibiotics will often cure the infection. Tetracycline should not be used in the premature and can stain teeth in children even if used for short periods. Additional treatment consists of nursing care and maintaining fluid and caloric intake.

6-4. DIAPER RASH. A form of primary irritant contact dermatitis due to prolonged contact of the skin to a combination of urine and feces.

S. and O. Erythema; thickening on the skin in the perineal area; beefy red, sharply marginated lesions with satellites; and a history of skin contact with urine and feces.

A. Diaper rash. Differential diagnosis: Other forms of primary irritant contact dermatitis.

P. Frequent diaper changes. Avoid rubber or plastic pants. Talcum powder can be used as an absorbent. Corn starch should not be used as it is a media in which C. albicans flourishes (80 percent of cases lasting more than 4 days are caused by C. albicans). Apply Mycostatin (nystatin, Mycolog) cream or Silvadine ointment with each diaper change. In extremely inflammatory diaper rash, 1% hydrocortisone cream can be alternated with Mycostatin at every other diaper change.

6-5. CHICKEN POX (VARICELLA). Primarily a disease of childhood, but in large areas of the tropics it is principally an adult disease. Varicella and herpes zoster are caused by the same virus, with varicella being the primary infection and herpes zoster being a recurrent infection. Varicella is highly contagious (80-90 percent of exposed susceptibles are infected).

S. History of contact 10-20 days (average 12-13 days) prior to onset. Usually no prodrome, but a mild fever with itchy and runny nose is sometimes seen 1-3 days before rash appears. Onset is usually abrupt with the appearance of the rash. Systemic symptoms, if any, are mild.

O. Rash appears in crops, with faint erythematous macules rapidly developing into papules and vesicles. The vesicles are thin-walled and superficially located on the skin with distinct areolas (dewdrop on red base) that rupture easily and rapidly encrust. Successive crops (usually 3) appear in the next 2-5 days, giving rise to lesions in all stages being seen at one time. Rash is heaviest on the trunk and lighter on the extremities. If a secondary bacterial infection does not develop, the crusts fall off in 1-3 weeks, leaving no scars. Varicella can vary from a mild disease with few vesicles to a severe disease with as many as 5 crops of lesions covering most of the skin. Systemic symptoms, which are usually mild or absent, may be severe and generally parallel the extent of skin involvement. Usually laboratory tests are of little aid, although sepsis may be accompanied by an abrupt rise of neutrophilia in the W.B.C.

A. Chicken Pox. Differential diagnosis: Severe forms--smallpox, impetigo, multiple insect bites, papular urticaria, rickettsialpox, and dermatitis herpetiformis.

P. Symptomatic. Fluids, control of itching with antihistamines, attention to cleanliness (handwashing, bathing), antipyretics as needed. Treat secondary infections.

6-6. SCARLET FEVER. A formerly common ailment that is rarely seen today, probably because antibiotic therapy prevents the opportunity for the streptococcus to progress in individual patients or to create massive epidemics. Scarlet fever is associated with Group A streptococcal strains that produce an erythrogenic toxin, leading to a diffuse pink-red cutaneous blush that blanches on pressure. The rash, an additional feature of an illness that otherwise resembles streptococcal pharyngitis, is best seen on the abdomen, on the lateral chest, and in the cutaneous folds.

S. and O. Along with the characteristic manifestations of the rash are circumoral pallor surrounded by a flushed face, a "strawberry tongue" (inflamed beefy red papillae protruding through a white coating), and Pastia's lines (dark red lines in the creases of skin folds). The upper layer of the previously reddened skin often desquamates after the

fever subsides.

A. Scarlet fever due to Group A streptococcus.

P. The course and management of scarlet fever are essentially the same as for other clinically evident Group A infections.

6-7. MUMPS (PAROTITIS). A common childhood disease that is asymptomatic in 30-40 percent of cases. Most children are infected and develop lifetime immunity but a few remain susceptible throughout adolescence and adult life.

S. and O. History of contact 14-21 days prior. Bilateral or unilateral painful swelling of the parotid gland is usually the only manifestation. Systemic symptoms may consist of high fever and headache or mild respiratory symptoms or occasionally C.N.S. symptoms that appear prior to or in the absence of parotid gland involvement, or symptoms may be absent. (Mumps virus is the most common cause of meningitis in childhood.) Mild to moderate abdominal pain may be present.

The gonads may be involved (orchitis or oophoritis) in postpubertal individuals with sudden onset of fever, chills, systemic symptoms, and lower abdominal pain in females or extreme testicular pain and testicular swelling in males. Contrary to common belief, mumps, orchitis, and oophoritis do not result in sterility. Symptoms subside in 3-14 days. Mumps usually last approximately 1 week.

A. Mumps. Differential diagnosis: Cervical lymphadenitis of pharynx, tonsillar or skin infection, other parotides, acute lymphoma, or lymphosarcoma.

P. Symptomatic. Control fever, pain, and discomfort. Treat orchitis or oophoritis conservatively with rest, testicular support, and analgesics. Corticosteroids may result in more rapid subsidence of testicular swelling.

6-8. VIRAL CROUP. Most commonly affects children between 3 months and 3 years of age. Characteristically occurs during late fall or early winter and is usually caused by the parainfluenza virus. It can also be caused by respiratory syncytial virus, influenza virus, rubeola virus, or adenoviruses. The major cause of symptoms is inflammation and edema in the subglottic area that can cause significant narrowing of the airway at the level of the cricoid cartilage.

S. Gradual onset, with history of several days upper respiratory tract infection prior to the onset of barking cough and harsh, high-pitched sound during inspiration (inspiratory stridor). If the lower respiratory tract is significantly involved, there may be wheezing. The child may become anxious and restless as hypoxemia and hypercapnia develop.

O. Mild temperature. Possible decreased breath sounds on ausculation. Cyanosis is a late sign and may herald complete airway obstruction. W.B.C. seldom increases to more than 15,000 with no significant leftward shift.

A. Viral Croup. Differential diagnosis: Bacterial croup (epiglottitis).

P. Cool mist therapy. (If vaporizer is not available, improvise by using steam in an enclosed room. Do not let steam go directly on patient as it may cause burns.) Monitor urine specific gravity to insure adequate hydration. Observe patient closely for signs of increasing hypoxia and impending respiratory failure. Keep patient calm and at bed rest. Do not use sedation unless an artificial airway is in place. The most effective method of keeping a child calm is having the mother or some other familiar person present. About 25-30% oxygen can be administered to relieve hypoxia. Patients starting O_2 therapy often have a marked decrease in respiratory effort and should be monitored closely for the first few minutes of oxygen administration.

Bronchial dilators (such as Bronchaid or Primatene Mist) often provide temporary relief of respiratory distress. If commercial preparations are not available, you can make a preparation of 0.5 cc. of epinephrine to 3.5 cc. of sterile water in a spray bottle.

If respiratory distress continues and there is progressively increasing cyanosis and decreasing air entry, an artificial airway must be provided. Generally, endotracheal intubation with a small endotracheal tube is used to reduce trauma to the glottis and subglottic area. (A particularly traumatic tracheal intubation can convert a reversible subglottic narrowing into a fixed nonreversible subglottic narrowing.) The best endotracheal tube care is mandatory and consists of careful tube stabilization and suctioning, postural drainage, chest percussion and humidification of inspired air. If all else fails, a tracheostomy is necessary.

6-9. EPIGLOTTITIS (BACTERIAL CROUP). The most serious form of croup syndrome. It generally affects children 3-7 years old, with no particular seasonal distribution. The most common pathogen is Hemophilus influenzae type B, but beta-hemolytic streptococci and pneumococci have been implicated in rare cases.

S. Abrupt onset over a period of only a few hours. Young children often present with high fever and respiratory distress. Older children may appear toxic and complain of difficulty in swallowing and severe sore throat. Child may have a muffled voice but usually it is not hoarse.

O. Pooling of secretions in the posterior pharynx and drooling are signs caused by extreme dysphagia (inability or difficulty in swallowing). The child, within a few hours, may be in marked respiratory distress with severe inspiratory stridor (harsh, high-pitched sound during inspiration) and retractions. The pharynx is likely to be inflamed. Diagnosis is made by markedly enlarged, friable (easily cracked or broken), "cherry-red" epiglottis. Direct visualization using a tongue blade or laryngoscope is extremely dangerous, as stimulation of the epiglottis has produced laryngeal obstruction and death. No throat cultures should be obtained until epiglottitis has been ruled out or an artificial airway is in place as this may also cause laryngospasm that causes laryngeal obstruction.

Lab findings: W.B.C. of more than 15,000 and a leftward shift is usually present.

P. Once the diagnosis is made, an artificial airway should be introduced. Because of the marked swelling and friability of the tissue,

intubation is extremely difficult. A smaller than usual endotracheal tube should be used and a tracheostomy set should be available. An IV should be initiated prior to the intubation and antibiotic therapy can be started by that route. Ampicillin 300 mg./kg./day in 6 divided doses or ampicillin and chloramphenicol are the drugs of choice.

The endotracheal tube should remain in place until the patient is able to breath around the tube easily and when there is a marked decrease in the epiglottic swellings, usually after 24-72 hours. Mortality rate may be as high as 90 without intubation and antibiotic therapy.

6-10. MENINGITIS. See Chapter 2, Section III, Bacterial.

6-11. MEASLES AND GERMAN MEASLES. See Chapter 2, Section IV, Viral.

6-12. DIPHTHERIA. An acute infection of the upper respiratory tract or skin caused by Corynebacterium diphtheriae. A toxin-producing, gram-positive rod with irregular swellings at one end giving it a club-shaped appearance. Irregularly distributed within the rods are granules that stain dark giving them a beaded appearance. The incubation period is 1-6 days.

S. and O. Pharyngeal diphtheria: Mild sore throat, moderate fever, and malaise followed fairly rapidly by severe prostration and circulatory collapse. Pulse is more rapid than temperature would seem to justify. A tenacious and gray membrane, surrounded by a narrow zone of erythema and a broader zone of edema forms in the throat and may spread into the nasopharynx or trachea, producing respiratory obstruction. High fever, prostration, difficulty in swallowing, and noisy breathing develops even without laryngeal obstruction. Cervical lymph nodes become swollen, and swelling is associated with brawny edema of the neck ("bullneck"); palatal paralysis may occur. Bleeding from the nose and mouth are common and petechiae may appear on the skin and mucous membranes.

Nasal diphtheria: Occurs in 2 percent of cases. Serosanguineous (containing serum and blood) nasal discharge and excoriation of the upper lip are characteristic and may be the only symptoms.

Laryngeal diptheria: Occurs in 25 percent of cases, and occasionally may be the only manifestation. Stridor (harsh, high-pitched sound during respiration) is apparent. The progressive laryngeal obstruction can lead to cyanosis and suffocation.

Other forms: Cutaneous, vaginal, or wound diphtheria composes less than 2 percent of all cases and are characterized by ulcerative lesions with membrane formation. They may be very hard to identify in burns or wounds.

Lab findings: W.B.C. is usually normal or slightly elevated. Urinalysis may show proteinuria of a transient nature.

A. Diphtheria. Differential diagnosis: Acute streptococcal pharyngitis, mononucleosis, occasionally other viral pharyngitis, purulent sinusitis, epiglottitis, and viral croup.

P. As the toxin causes the main damage, antitoxin should be administered ASAP. Delay beyond 48 hours must be avoided because antitoxin administered beyond that point may have little effect in altering the

incidence or severity of complication. These include myocarditis, toxic polyneuritis, and bronchopneumonia. Sensitivity to horse serum should always be skin tested for before administering the antitoxin. If positive and the diphtheria is severe, give 50 mg. Benadryl IM initially, start an IV of Ringer's lactate or D_{5W} to be used for treatment of anaphylactic shock if necessary, then and only then start an IV to administer the required antitoxin. The patient must be closely monitored for signs of reaction to the antitoxin.

Mild pharyngeal diphtheria or when the membrane is small or confined to the anterior nares or tonsils, 40,000 units. Moderate pharyngeal diphtheria, 80,000 units. Severe pharyngeal or laryngeal diphtheria, 120,000 units regardless of child's weight infused in 200 ml. of isotonic saline over a 30-minute period.

Penicillin V is the drug of choice to eliminate the organism and stop toxin production 250 mg. q.i.d. x 10 days or 600,000 units of procaine Penicillin G IM b.i.d. x 10 days. Alternate is erythromycin 25-50 mg./kg./d. in 4 divided doses orally x 10 days.

Bed rest for 10-14 days is usually required. Strict isolation until antibiotic therapy has made respiratory secretions noninfectious is also required (usually 1-7 days). IV therapy may be necessary. Warm salt water gargles or irrigation are helpful and codeine phosphate 3 mg./kg./d. in 6 divided doses may also help with the discomfort.

Prevention: Routine DPT (diphtheria, pertussis, and tetanus) immunization should be given to all infants and children.

All children exposed to diphtheria should be examined and treated if any signs of early diphtheria show.

All asymptomatic individuals, even if previously immunized, should receive diphtheria toxoid and either erythromycin 20-30 mg./kg./d. in 4 divided doses orally x 10 days or 25,000 U./kg. of benzathine penicillin G.

CHAPTER 7

GYNECOLOGY

7-1. Gynecology encompasses those diseases that are peculiar to women. History and physical examination have certain features that separate them from general ones.

a. History.

(1) Age, gravidity (number of times pregnant), parity (number of live deliveries). Medical records list these, for example, as G3P2Ab1 (three pregnancies; two deliveries; one abortion, either spontaneous or induced).

(2) Chief complaints, in the patient's words, in order of severity.

(3) Present illness. A chronological order of symptoms with details.

(4) Past medical/surgical history in chronological order from childhood through the present, with the complications and treatments for each. All operations and injuries with dates and outcomes.

(5) Obstetrical history. Number of pregnancies, duration of pregnancies and labor details, weight and sex of infants, stillbirths and abortions.

(6) Family history. Age and health of parents and siblings. Family history of any tuberculosis, diabetes, hypertension, bleeding disorders, heart disease, cancer.

(7) Marital and/or cohabitation history. Duration and compatibility of past and present relationships, ages and causes of deaths, if any, and ages and health of children, if any.

(8) Social history. Occupation, hazards, alcohol and tobacco consumption habits, drug usage, sleep and exercise habits, and general activities.

(9) Review of systems. Same as a general history, except for genitourinary. Menarche (age at onset of menstruation), last menstrual period, regularity, duration, amount and character of flow, spotting, discharges, and pain.

b. Examination. Same as the general examination except for:

(1) Breasts. Size, shape equality of both sides, masses, tenderness, scars, or nipple discharge. Breast examination is performed by gentle palpation in a circular fashion from the nipple to the outside, also covering the nodes under the arms. Attempt to express a discharge from the breast nipple as well. Perform this maneuver with the patient's arms down at the sides and over her head, in the supine position.

(2) Pelvic examination.

(a) Drape a sheet over the patient in the supine position

with her legs flexed and spread open. Have a female assistant at your side or at the patient's side for support. Obtain a good direct light source, a water-base lubricant such as KY jelly, and surgically clean gloves.

(b) Genitalia. Look for inflamed, hypertrophied, atrophied, ulcerated, or any other abnormal areas; vaginal discharge; clitoral abnormalities; skin changes over the perineum, thighs, pubis, or perianal region. Check the urethral meatus for redness, exudates from the labial gland ducts, etc.

(c) If pathological study assistance is available, obtain vaginal mucus from the posterior of the vagina for cell studies.

(d) Insert a comfortably warm speculum into the vagina. Ask the patient to relax and bear down. Carefully spread the labia with a gloved hand, insert the speculum blades slowly downward and inward, watching the insert closely. As the cervix is approached, slowly open the blades and allow the blades to straddle the cervix between them. Lock the screw lock.

(e) Inspect the cervix. Obtain cervical mucus from the cervical entrance and from any irregular lesions or sites. Insure the cervical size is not excessively large or small in proportion to the vagina. The cervix should be smooth with no large lacerations, no wide opening, of a pink color, and without blood or discharge.

(f) Unlock blades and slowly withdraw them. Watch for pink folds of the vaginal walls without blood or discharge or lesions. Leaving the blades at the introitus, or vaginal opening, ask the patient to again bear down. A drooping of the cervix indicates decensus, or loss of support, of the uterus itself. Drooping of the vaginal roof may indicate cystocele; protrusion of the vaginal floor upward may indicate rectocele. These will be explained. Take smears of any questionable exudates.

(g) Bimanual palpation.

1. Place one palm down on the abdomen as you stand between the patient's legs. Slightly flex the fingers. Press down firmly. Have the patient take shallow, rapid breaths to aid in relaxation.

2. With the other hand, gloved and coated with a small amount of lubricating jelly, slowly part the labia with the index and middle fingers. Hug the floor of the vagina with the fingers and touch the cervix with the fingertips. "Trap" the uterus between the hands and, without letting it loose, run the outside hand fingertips over the entire front and side surfaces of the uterus. It should be in the midline, be firm and smooth just above the pubis, and be somewhat movable with relatively little pain. Feel behind the cervix for any masses, fullness, or tenderness.

3. With the uterus still trapped between the hands, sweep the outside hand over to the side of the uterus to meet the fingertips of the vaginal hand. "Trap" the fallopian tube and ovary. You should not be able to feel the tube. The ovary is an almond-size, slightly tender organ attached to the side of the uterus. Feel for size, consistency, position, and contour (firm, just lateral to the uterus, smooth). Document all masses noted.

4. With the index finger of the internal hand still in the vagina, gently insert the middle finger into the rectum very slowly but firmly. Palpate as you did for the vaginal exam. This exam will aid in diagnosis of a vaginal stricture, is used in virgins, for tender masses and to explore the back of the uterus and rectal strength.

(3) Laboratory studies.

(a) Collections of Bartholin's, Skene's discharges, vaginal walls, posterior vaginal fornix, or rear pouch, or cervical opening, or os, are taken with a clean cotton applicator and treated as for a simple Gram stain unless you feel a need for culturing and these facilities are available.

(b) Wet preps. These are for vaginal discharges. Moisten a slide with a drop of sterile saline. Transfer a drop of discharge on a wooden applicator to the drop of saline on the slide. Read under a microscope immediately.

1. Trichomonas vaginalis. Look for the typical trichomonads with a whipping tail. See the laboratory plates for an example.

2. Hemophilus vaginalis. Vaginal cells may be dusted with small dark particles. These are called "clue cells." See the laboratory plates.

(c) KOH preps. Add a drop or two of 10% potassium hydroxide to a slide. Transfer a drop of discharge with a wooden applicator. The solution will dissolve R.B.C.'s, inflammatory, and epithelial cells. Candida albicans mycelia will display as hyphae and spores. Any whitish plaques in the vagina are to be scraped for this test.

(d) Pap smears. These smears of cervical cells are invaluable as a cancer screen when pathology facilities are available. With the vaginal speculum in place without lubricant other than sterile saline, transfer a specimen scraped from the center of the opening of the cervix to a slide. Smear the drops lightly across the slide. Repeat the procedure with a drop of fluid from the back of the vagina. Fix both slides immediately with 97% ethanol, Aqua-Net hair spray, or Pro-Fixx cytology fixative by spraying lightly across the slides. Be sure to have the patient's name on each slide. Pap smear readings are very difficult during active bleed.

(4) Procedures. Dilatation and curettage (D&C). This procedure involves opening of the cervix and scraping away of the endometrium or inner lining of the uterus. This procedure requires supervised practice prior to attempting the procedure yourself. Never forcefully perform this procedure. Uterine perforation can easily result. D&C is indicated for abnormal or postmenarchal questional bleeding and for spontaneous (incomplete) abortion. Contraindications include normal intrauterine pregnancy, acute cervicitis, endometritis, or pelvic inflammatory disease. The procedure may be performed under general anaesthesia, spinal (level of L3-L4 spine, inject 10-15 cc. of 0.25% Marcaine carefully), paracervical block (0.25% Marcaine injected just inside the vaginal mucosa next to the cervix on each side, 5 cc.), or 50-75 mg. Demerol IV slow while monitoring carefully.

(a) Explain the procedure to the patient.

(b) Palpate the uterine size and position. Attempt now and when "sounding" the uterus to rule out any lesions or growths that may bleed.

(c) Insert and lock down a speculum. Glove and wipe in a circular fashion outward the entire cervical stump with antiseptic sponges on transfer forceps three times. Discard the swabs and forceps. Bend the uterine sound to the estimated angle of the uterine position. Grasp the cervix with a tenaculum forcep at the six o'clock position and gently insert the sound until resistance is met. Here you will again try to note any lesions or growths as you insert the sound. Read the depth of the uterine cavity by noting the level of the mucous or blood on the sound as you would the oil level on a dipstick. Make a mental note of the depth of the uterine cavity. Starting with the smallest Hegar dilator, insert the dilator into the cervix to the dilator lip. Proceed to the next larger size until the cervix is at least as open as the loop of the largest curette, probably a #8 Hegar. Start with a small sharp curette by scraping in and out the entire diameter of the cervical canal. Fix the tissue obtained in 10% formalin. Repeat the four-quadrant scraping of the uterus by going to the depth of the uterus and scraping outward all along the uterine walls, in deep even gentle strokes to obtain long strips of endometrium. Curette the top of the uterus in an up-and-down fashion. Fix these specimens as before in formalin. If questionable specimens are obtained, fix and identify them separately. Insert a dry sterile sponge on a uterine forcep and swap the cavity with a twisting motion as you withdraw. Reinsert uterine polyp forceps and grasp for masses. Withdraw the forceps and observe for bleeding. Replace the uterus by removing the tenaculum and speculum and pushing the uterus gently but firmly upward bimanually. Place patient on bed rest for three days and limit activity for at least seven days. Excessive bleeding may require packing the uterine cavity with long continuous sterile roller gauze and shock care until out of danger and hemostasis is achieved.

7-2. THE BREAST. A modified sweat gland of duct tissue secreting nutritive fluid during the first several weeks after delivery (postpartum).

a. Postpartum mastitis (pyogenic cellulitis) generally occurs after several weeks of nursing. The infection occurs through the nipple and into the ducts. About 75 percent of all patients have unilateral involvement.

S. Chills, fever, malaise, regional pain, tenderness, and induration (hardening).

O. Gram stain of any discharge usually shows Staphylococcus aureus. A notable fluctuant mass can be palpated in the later stages. Axillary lymphadenopathy may be noted. An abscess may form in most cases.

A. Diagnosis is generally unmistakable.

P. Prevent by good hygiene. Suppress lactation (milk production) by wearing a tight binder for 72 hours, apply ice packs one hour on and one hour off. Give analgesics as needed. Broad-spectrum antibiotics such as Keflex, 250 mg. P.O. q.i.d. x 10-14 d. Incise and drain abscesses and pack with iodoform gauze.

b. Mammary dysplasia (cystic breast disease) is the most common

single breast disorder encountered.

S. Painful masses in breast, perhaps a discharge.

O. Multiple tender masses in a patient that is often 30-50 years old, often worse during menstrual periods. Sizes may go up or down. No skin retraction should be present.

A. Differential diagnosis includes breast carcinoma and adenofibroma, which require biopsy to diagnose.

P. Biopsy is needed if at all possible. If symptoms and history are classical for this disorder, infiltrate the breast locally with lidocaine 1% or procaine 1%, insert a 20-gage needle into the cyst and withdraw the watery fluid that should be straw-colored to black. Reexamine every 2-4 weeks for 3 months, then every 6-12 months. If no fluid is obtained or a persistent lump is noted, a biopsy is indicated.

7-3. VULVITIS. The vulva is subject to the same diseases as the skin elsewhere on the body. Vaginitis (covered later) is secondarily induced.

a. Eczema is a common pruritic moist dermatitis often from contact with an irritant in soap, bath oils, deodorants, clothing, dyes, etc.

S. Pruritus, occasionally a discharge, and the lesion are presented.

O. An excoriated (ulcerated) crusted lesion is noted.

A. Differential diagnosis: Includes seborrhea, psoriasis, and intertrigo.

P. Eliminate any irritant. Burow's solution b.i.d. for three days. Local application of a steroid cream (hydrocortisone, Valisone, etc.) b.i.d. until the lesion resolves. Antihistamines for itching as needed (Benadryl 25-50 mg. h.s. to q.i.d.).

b. Psoriasis is of unknown etiology.

S. Pruritus and a lesion are presented. History may be long term.

O. Erythematous, slightly elevated, flattened lesions without the typical silvery appearance of scaling seen elsewhere on the body.

A. Differential diagnosis: Includes seborrhea, eczema, and intertrigo.

P. Improved hygiene is important. Apply hydrocortisone cream 1% b.i.d. If no improvement occurs, try Valisone in the same dosage.

c. Seborrhea is based on a genetic predisposition involving hormones, nutrition, infection, and emotional stress.

S. Pruritus may present, along with a lesion that may be infected.

O. A dry, scaling lesion with underlying erythema will present.

A. Rule out fungal involvement with a KOH prep. Differential diagnosis: Includes eczema, psoriasis, and intertrigo.

P. No greasy ointments. Potassium permanganate dressings b.i.d. (soak dressing in 100 mg. of permanganate in 1 liter of water). Ammoniated mercury ointment after soaks: 5% ammoniated mercury, 3% liquid petrolatum and petrolatum q.s. ad 100%.

d. Intertrigo is caused by the macerating effect of heat, moisture, and friction. It is worse in hot humid climates and in obese patients.

S. Itching, stinging, burning sensation in a noticeable irritation.

O. Possible fissures, erythema, denuded appearance. Urinalysis may show an indication of diabetes, KOH prep may show candida, direct smear may even show many cocci.

A. Differential diagnosis: Includes eczema, psoriasis, and seborrhea which may preclude intertrigo itself.

P. Dust well with talc b.i.d. Potassium permanganate dressings prepared as above or Domeboro dressings in a 1:20 mixed ratio.

7-4. BARTHOLIN'S CYST AND ABSCESS.

S. Periodic painful swelling on either side of the introitus (vaginal opening) and dyspareunia (painful intercourse).

O. Swelling at mid to lower third of labia, usually 1-4 cm. in size, and tender and fluctuant (wavelike sensation on palpation indicating a fluid-filled sac). Rule out gonorrheal involvement by direct smear of exudate.

A. Differential diagnosis: Includes inclusion cysts, sebaceous cysts, and congenital abnormalities (these are not usually tender).

P. Local heat to the lesion. Ampicillin or erythromycin 250-500 mg. q.i.d. for 10 days. After the infection subsides, open the lesion and excise or exteriorize. If an abscess develops, incise and drain, pack with iodoform gauze.

7-5. CONDYLOMA ACUMINATA. A viral infection that does not affect a fetus.

S. Small masses on the vulva, vagina, or perineum will present with itching.

O. Pink clusters of soft narrow-based lesions that are pointed and elongated, with or without a profuse irritating discharge.

A. Rule out condyloma latum (the primary lesion of syphilis).

P. Culture any discharge for gonorrhea. Perform a darkfield exam to rule out spirochetes. Treat any secondary infection that may exist. A 25% podophyllin in benzoin tincture may be applied to the lesions only and is to be washed off in 2 hours. Do not touch the normal tissue with the podophyllin. Isolate the lesions by surrounding the lesions with mineral oil.

7-6. MOLLUSCUM CONTAGIOSUM. A virus that incubates in 1-4 weeks.

S. Asymptomatic small skin tumors will present.

O. Pink to gray, discrete, umbilicated epithelial skin tumors generally less than 1 cm. in diameter on primarily the vulva introitus.

A. Diagnosis is generally unmistakable.

P. Biopsy is indicated if the diagnosis is in question. Lightly curette away the lesions. Apply Neosporin-G cream to the curette sites and dress.

7-7. HERPES GENITALIS. A herpes type II viral infection.

S. Painful clear little "bumps" on the labia and introitus, perhaps with tender "knots" in the groin.

O. Occasional inguinal lymphadenopathy and a group of vesicles with surrounding erythema and edema. Often a history of lesions coming and going.

A. Herpes zoster is similar, but generally doesn't recur. Erythema multiforme is a larger vesicle often found on plantar surfaces that sometimes looks like tiny "targets" of concentric circles and becomes purplish as the lesions enlarge; fever is concurrent.

P. (1) Rule out concurrent gonorrhea and syphilis.

(2) Virus culture for herpes species.

(3) Caesarean section patients with active lesions.

(4) Pap smear (herpes has been linked with cervical carcinoma).

(5) A 2% lidocaine ointment for pain q.i.d. for less than 2 weeks.

(6) No occlusive dressings or medications except lidocaine ointment.

7-8. VAGINITIS. An inflammation and/or infection of the vagina.

a. Atrophic vaginitis.

S. Tender, itching vagina generally in an older, postmenopausal or even a castrated patient.

O. Occasionally a clear vaginal discharge with an atrophied, erythematous, sometimes dryer appearance to the vagina.

A. Rule out other forms of vaginitis with saline and KOH preps of discharge.

P. Apply Premarin cream, 2-4 gm p.v. q.d. Use this medication cautiously with full knowledge of side effects, contraindications, etc. Use the smallest amount necessary to control the symptoms.

b. Trichomonal vaginitis. Caused by trichomonas vaginalis.

S. Vaginal symptoms of burning, itching, and tenderness with discharge.

O. Petechial spotting with erythema of the vaginal wall (with a strawberrylike appearance), usually with a thicker yellow to green frothy discharge. A stat. saline prep reading shows trichomonads.

A. Wet prep rules out other organisms.

P. Rule out other organisms including gonorrhea. Flagyl 250 mg. t.i.d. for 7-10 days. Treat the patient's sex partner at the same time.

c. Candidal vaginitis. Caused by Candida albicans, also known as Monilia.

S. Vaginal symptoms as above.

O. Erythema with a generally thick, white cheesy curdlike discharge. Thrush (whitish) patches may exist on the vaginal walls. KOH preps should show mycelia and hyphae.

A. Rule out other organisms including gonococci.

P. Nystatin vaginal suppositories 100,000 unit, 1 supp p.v. daily x 14 days.

d. Nonspecific vaginitis. Generally caused by Hemophilus vaginalis.

S. Vaginal symptoms as above.

O. Acrid, viscous, or thin watery milky discharge. Wet preps will show some epithelial cells coated with bacteria, giving a dusty appearance.

A. Rule out other organisms, including gonococci.

P. Sultrin cream, 1 applicatorful p.v. b.i.d. for 6 days, then 1 q.d. h.s. for 8 days.

7-9. CYSTOCELE. A herniation of the posterior bladder wall into the vagina.

S. Sensation of retained urine after urinating and of vaginal "looseness."

O. Presents as a reducible nontender mass that is soft and located in the anterior vaginal wall. As the patient strains, the bladder can sag downward.

A. Differential diagnosis: Includes bladder tumors and stones, both of which are firm and easily outlined. Rarely is a small bowel hernia differentiated.

P. This disorder may be alleviated by the patient manually reducing the bladder by pressing it upward from the vagina. Intermittent use of a Menge pessary placed just inside the introitus may help. Surgery,

an anterior vaginal colporrhaphy, is often the only near-permanent cure.

7-10. RECTOCELE. A herniation of the rectal pouch into the vagina.

S. Constant urge for a bowel movement and a vaginal/rectal sense of fullness.

O. A finger can be inserted rectally and cause posterior pouching of the rectum. Straining down worsens the pouching. A soft posterior vaginal fullness. Defecation may be painful.

A. Differential diagnosis: Includes enterocele (a similar disorder occurring further back in the vagina from intestinal herniation), prolapsed cervix (seen on speculum-assisted vaginal examination), and rarely a tumor, which would be firmer and more easily delineated.

P. Stool softeners or laxatives (only for short periods). Avoid straining, coughing, or lifting. Get good exercise and bowel habits, as well as good dietary habits to facilitate elimination. Surgery (colpoperineorrhaphy) is generally curative.

7-11. CERVICITIS. An inflammation/infection of the cervix. This is the most common gynecological disorder generally encountered.

S. Discharge, low back pain, dyspareunia, dysmenorrhea (painful menstruation), urinary frequency and urgency, and/or dysuria.

O. Thin, mucuslike leukorrhea (discharge); an erythematous, petechial cervix and posterior fornix (back pouch of vagina) with a discharge from the cervical os (opening). Smears show W.B.C.'s. Cervical erosion and eversion may be noticeable.

A. Rule out infectious organisms by wet preps, KOH preps, and smears.

P. Pap smear first. If no organisms present, give AVC cream, 1-2 applicatorsful p.v. h.s. or b.i.d. for 28 days, through the entire menstrual cycle. Treat specific organisms as in the forms of vaginitis. Cryosurgery (with a CO_2 wand) may be necessary in intractable cases.

7-12. CERVICAL POLYPS. Small pedicled growths on the cervix.

S. Discharge, abnormal vaginal bleeding.

O. Flesh- to red-colored rounded or flame-shaped tissue on a pedicle or strand of tissue on the cervix or, if redder, coming from the endocervix.

A. Differential diagnosis (based on pathologic studies): Includes endometrial neoplasm or growth, small submucous myoma, endometrial polyp, and products of conception from an incomplete spontaneous abortion.

P. Work up and treat any associated cervicitis. Remove at the base of the lesion. Cervical dilatation may be necessary for polyps located high up in the endocervix. Send lesion to lab for pathologic studies. Full D&C if other polyps are suspected. Warm vinegar douche q.d. for 3-7 days to reduce inflammation.

7-13. ENDOMETRITIS. An inflammation/infection of the uterine lining, generally postpartum, post-D&C, or post incomplete abortion.

S. Fever, pain in the lower abdomen in the centerline, and low back pain.

O. Occasionally a discharge from the cervical os; history of recent delivery, abortion, or D&C. W.B.C. count may be mildly elevated.

A. Rule out masses by palpation; rule out carcinoma by D&C and study of samples obtained, or by a simpler endometrial biopsy done in like fashion.

P. Endometrial biopsy and smear as indicated. Specific antibiotics for organisms (Vibramycin 200 mg. for 1 day, then 100 mg. q.d. for 9 days is useful). D&C if abortion has been suspected. This must be done in a less vigorous fashion than normally. If moderately severe systemic symptoms are present, consider a slight delay, using antibiotics first. Monitor for any systemic infection until after all symptoms subside.

7-14. UTERINE MYOMA (fibroid). The most common gynecological neoplasm. It is a round, firm, benign uterine tumor composed of smooth muscle and dense connective tissue.

S. Lower adbominal pain, bleeding, dysmenorrhea, discharge, dyspareunia, urinary frequency, sensation of pressure, and constipation.

O. Palpable enlargement of the uterus, feeling firm and rounded.

A. D&C may help confirm, as no abnormal specimens may be found. Differential diagnosis: Includes other neoplasms and benign hypertrophy; sarcoma and adherent adnexa. Surgical sections are the principal diagnostic tool.

P. Defer surgery until postpartum, if patient is pregnant, unless the uterus feels to be over two months larger than the EDC (estimated date of confinement) computes to. Watch for signs of distress. A torsioned pedicle of a myoma or intestinal obstruction may necessitate emergency surgery and blood transfusion. Excision with perhaps hysterectomy (uterus removal) is indicated if the disorder is extensive.

7-15. SALPINGITIS (pelvic inflammatory disease). An infection of the fallopian tubes, usually bilaterally, with a rapid spread to the rest of the pelvis.

S. Severe, nonradiating cramping lower abdominal pain, chills, fever, abnormal menses, leukorrhea, dyspareunia, and dysmenorrhea.

O. Thickening of the adnexal structures and palpation of the tubes (not normally paplable) on pelvic exam. Adynamic ileus (stoppage of fecal passage) may present. History of nausea and pain since last period. Discharge. Stable hematocrit and W.B.C. count to 15,000-20,000. The erythrocyte sedimentation rate will be increased.

A. Differential diagnosis: Includes appendicitis (lower fever and W.B.C. count, localized RLQ pain, nausea, and vomiting) and ectopic pregnancy (a sudden RLQ or LLQ pain, with bleeding, soft tender mass and

recent irregular menses).

P. Culture discharge (rule out tuberculosis and gonorrhea). Treat organisms appropriately (ampicillin, 500 mg. P.O. q.i.d. for 7-10 days). Control pain with analgesics and suppress menstruation with Enovid (10-15 mg. P.O. q.d. for 28 days). Treat fever and malaise symptomatically. Observe, as this disorder is potentially very dangerous. RULE OUT MASSES. Since this can be an emergency, ruling out masses helps to reduce the chance that it becomes a surgical emergency.

7-16. TUBO-OVARIAN ABSCESS. A formed abscess of the tubes that may spread to the ovaries.

S. Spikes of fever, malaise, bilateral lower quadrant pain with an acute onset, sudden and pronounced. Metrorrhagia and hypermenorrhea (later section).

O. Palpable mass, tender. Possible history of disappearing mass with softening of the abdomen, suggesting rupture of the abscess. Increased W.B.C. count and sed. rate (erythrocyte sedimentation rate). Biocept-G negative.

A. The Biocept-G rules out pregnancy and thus ectopic pregnancy. If pain is unilateral, rule out appendicitis by history and lower W.B.C. count and sed. rate. Endometriosis (endometrium growing outside the uterine cavity in its normal position) is ruled out by the cyclic nature of the pain.

P. Vibramycin 200 mg. P.O. b.i.d. for 10 days. Constant monitoring for abdominal softening. Local heat and analgesics. Surgery is indicated if rupture is suspected. If access via the cul-de-sac is possible, aspiration of abscess contents for temporary alleviation of the mass by large-bore needle may be of value.

7-17. OOPHORITIS. An infection of the ovaries, generally secondary to another infection but clinically significant from a potential infertility standpoint, since healing of ovarian tissue is not well accomplished.

S. Pain, fever, and menstrual abnormalities. Evidence of other infection as the complaints are noted.

O. Enlargement of the ovary and excessive tenderness to palpation. Anemia and increased W.B.C. count and sed rate are noted.

A. Other adnexal infections may coexist.

P. Analgesics such as codeine sulfate 30-60 mg. every 4-6 hours. Observe for systemic signs. Vibramycin 200 mg. stat., then 100 mg. q.d. for 13 days. Local heat, rest, fluids. Drain abscesses (if pointing down to the cul-de-sac, by large-bore needle aspiration). If chronic in nature, and if the patient is older, removal of the ovaries and tubes (salpingo-oophorectomy) bilaterally may be needed.

7-18. OVARIAN CYSTS AND TUMORS. Many varieties of cysts and tumors may be noted on pelvic examination and palpation of the ovaries. Rule out the known disorders in this chapter, wait one full menstrual cycle, and recheck the size and, of course, the nature of palpable adnexal masses; obtain specialist assistance if the mass has not regressed during the trial

period. If it has, make a note of all findings and recheck the patient periodically to watch for recurrence.

7-19. PREMENSTRUAL TENSION. A cyclical disorder.

S. Anxiety, agitation, insomnia, inability to concentrate fully, feelings of inadequacy, depression, and weight gain.

O. Document the symptoms. Lab and pelvic exams are inconclusive.

A. Rule out hyperthyroidism (if lab facilities permit, increased T_3 and decreased T_4 with perhaps a palpable thyroid), hyperaldosteronism (decreased serum potassium, increased serum sodium, alkalosis, and increased plasma aldosterone), and hyperinsulinism (decreased blood sugar). Also note any clinical symptoms. Psychoneurosis and psychosis are also to be considered, but they are not cyclical.

P. Reassurance is very important. Diuretics, such as Aldactone 2 mg./kg./d. in divided doses under supervision. Antidepressants as needed. Psychiatric help as needed, or assistance with differentiated disorders.

7-20. DYSMENORRHEA. Pain with menstrual periods. Secondary dysmenorrhea is a term applied to dysmenorrhea from organic causes (chronic pelvic inflammatory disease, endometriosis, etc.). This generally occurs over five years after menarche or at the beginning of having menstrual periods.

S. Pain with menstruation, abdominal bloating, breast tenderness, and a sensation of pelvic heaviness around the time for the patient's period.

O. History of intermittent premenstrual cramping through the period in the lower abdominal midline.

A. Diagnosis is based on history and absence of other pelvic exam findings.

P Analgesics as needed. Local heat and reassurance. Motrin 400 mg. P.O. q.i.d. from the onset of cramps to the end of the period.

7-21. AMENORRHEA. Failure to menstruate at the appropriate time. Primary amenorrhea is when the patient has never menstruated, while secondary amenorrhea is when over 90 days pass with no menstrual flow.

S. and O. All hinge on the absence of menstrual flow.

A. Assessment is usually unmistakable.

P. Work up the patient as follows:

(1) Perform the most accurate pregnancy test possible (the Biocept-G, if available to be done).

(2) If pregnancy test is negative, give Provera 10 mg. P.O. q.d. for 5 days.

(3) If patient bleeds, anovulation (no ovulation) occurred.

Nothing further.

(4) If the patient doesn't bleed, and if possible, draw a serum FSH and LH (follicle-stimulating hormone and luteinizing hormone). Then give Premarin 1.25 mg. P.O. q.d. for 21 days, then Provera as above.

(5) If no bleeding, trace the tract through to the uterus to target organ or outflow tract failure.

(6) If patient bleeds, get serum FSH and LH results. If they are low, then C.N.S. or pituitary failure is suspected. Refer the patient out for the C.N.S. or pituitary tumor workups.

(7) If the FSH and LH are high, then ovarian failure is suspected, dictating referral for karyotyping (chromosome studies) for genetic deficiencies.

(8) Remember, amenorrhea is complex and elusive. If at any time the disorder or its workup exceeds the practitioner's expertise or facilities, the case should be referred to a specialist with the means to work up and manage the case.

7-22. ABNORMAL UTERINE BLEEDING. A symptom of atypical menstrual flow in amount or timing. Hypermenorrhea (excessive flow) or menorrhagia; polymenorrhea (flow less than every 24 days); and metrorrhagia (flow at times other than regular time for the period) are examples.

Subjective and objective findings are as above.

A. Based on history, examination, and appropriate lab testing.

P. (1) Take a careful history and perform a careful exam. Take vaginal smears for cytology and bacteriology (fix first then add 1% HCl, which hemolyzes the red blood cells, if the bleeding is active. HCl is hydrochloric acid).

(2) Run a urinalysis, hematocrit, STS, W.B.C. count with differential, sed. rate, bleeding time, clotting time, clot retraction time, and platelets.

(3) Cervical biopsy and D&C may be critical.

(4) Hypermenorrhea. D&C, support hypovolemia, Provera 5-10 mg. q.d. for 4 days starting with the 21st day of the cycle. First day of bleeding is the first day of the cycle.

(5) Metrorrhagia. Give Enovid 10 mg. P.O. q.d. on days 5-20.

(6) Unknown or unresponsive entities should be referred for further study.

7-23. MENOPAUSE/CLIMACTERIC. Climacteric is the onset of menopausal symptoms, while menopause itself is the cessation of menses for over one year. These can of course occur due to removal or major dysfunction of the ovaries.

S. The climacteric begins at ages 40-55 with hot flushes,

diaphoresis, and depression or agitation.

O. Vaginal atrophy with dyspareunia and pruritus may exist.

A. If bleeding suddenly recurs, rule out neoplasms by pelvic exam palpation.

P. (1) Reassurance and understanding are essential.

(2) Mild sedatives as needed.

(3) If symptoms are severe or patient is fairly young, Premarin should be given low dose (0.3-2.5 mg. ranges) and adjust upward to control the symptoms that are presented to you.

7-24. CONTRACEPTION.

a. Rhythm uses basal body temperature to figure the period of ovulation. It is the only method allowed in Catholic areas.

(1) Take the temperature immediately upon awakening and before arising. Be sure to chart this reading daily.

(2) One to 1 1/2 days before ovulation, the temperature drops; 1-2 days after ovulation, the temperature rises about 0.7 degrees F. Wait 3 days after the temperature rises before allowing intercourse. The BBT thermometer is best and most accurate of all when utilizing this method.

b. Oral contraceptives. Selection is important. These medications generally work by artificial suppression of FSH secretion by the posterior pituitary. Young girls (16-20) must avoid oversuppression of the pituitary hormones, while older women must avoid thromboembolism.

(1) Ask about nausea and vomiting in previous pregnancies, fluid retention, weight gain, acne, history of varicose veins, etc.

(2) If menstrual flow is heavy and long, use more progestin (Norinyl 2 mg., or Norlestrin 2.5 mg.) to avoid breakthrough bleeding.

(3) If flow is shorter than normal, consider more estrogen and less progestin (Ovulen, Ortho-Novum, Enovid).

(4) If menstrual or other side effects are noted, increase estrogen and decrease progestin to increase the menstrual flow, or vice versa to decrease the flow. Watch the dosages of each hormone in the pills to adjust the flow in this manner.

(5) Give 1 tab P.O. q.d. If 1 day is missed, take two tabs the next day. If two or more days are missed, discontinue the tablets until the start of the next month and use another form of contraception until then.

(6) If women are very regular in timing, amount of flow, and duration of flow, try norethindrone acetate 0.2 mg. q.d. It has fewer side effects.

(7) Know the pills before prescribing, read the information, rule out any contraindications before prescribing.

c. Diaphragm and spermicidal jelly. Fitted to proper size to snugly cover the cervix and covered with jelly, this method works well when left in place after intercourse for at least 8 hours.

d. Condoms. Help prevent VD and work well with immediate postcoital withdrawal of the penis to prevent leakage of semen.

e. Foam. Spermicidal foam such as Delfen given as 1-2 applicatorsful p.v. before intercourse works well. Irritation and messiness may be noted.

f. Intrauterine devices (IUDs). The Cu-7 and Tatum-T work well when properly monitored after careful installation. Prep the cervix as for the D&C. Sound the uterus and measure the inserter to the noted depth. Turn the IUD to a position lateral so as to make it open when inserted to either side. Insert the device, pull back the inserter and withdraw the inserter. Cut the string to a couple of inches outside the cervix. Have the patient feel for the string regularly and after each period.

NOTE: This chapter is not all-inclusive and much of the data is for information only. Many of the tests cannot be performed with existing facilities. This information is useful to the practitioner becoming aware of the possibilities of disease entities and treatments in a basic way. Practice under close supervision is essential to learn properly these techniques. Be sure to refer patients to the specialists if ever in doubt or if inadequate facilities exist.

CHAPTER 8

OBSTETRICS

8-1. Obstetrics is that branch of surgery that deals with the management of women during pregnancy, labor, and the puerperium (42 days following childbirth and expulsion of the placenta; the generative organs usually return to normal during this time).

8-2. DIAGNOSIS OF PREGNANCY. In about one-third of cases it is difficult to make a definitive diagnosis before the second missed period because the variability of physical changes induced by pregnancy, possibility of tumors, obesity, and poor patient relaxation often interfere with the examination. If in doubt, schedule a reexamination in 3-4 weeks. If available the Early Pregnancy Test (E.P.T.) or in Europe, the Predictor Test, an anti-HCG test for pregnancy, can be used at least 9 days after her last period was due. This test claims a 97 percent accuracy rate.

a. The following symptoms and signs are usually due to pregnancy, but even two or more are not diagnostic. A record or history of time and frequency of coitus may be of considerable help.

(1) Symptoms. Amenorrhea (missed period), nausea and vomiting, urinary frequency and urgency (first trimester), breast tenderness and tingling (after 1-2 weeks), "quickening" (first movement of the fetus felt in the uterus; may appear about 16th week), weight gain.

(2) Signs. Skin pigmentation (after 16th week), epulis (hypertrophic gingival papillae often seen after first trimester), breast changes (enlargement, vascular engorgement, colostrum), abdominal enlargement, cyanosis of vagina and cervical portio (about the 6th week), softening of cervix (4th or 5th week), softening of cervicouterine junction (5th or 6th week), irregular softening and slight enlargement of the fundus (about 5th week), generalized enlargement and diffuse softening of corpus (after 8th week).

b. Positive manifestations. Not usually present until the 4th month, but is undeniable proof of pregnancy: Ausculation of fetal heart, palpation of fetal outline, recognition of fetal movement.

c. Differential diagnosis. All the presumptive signs and symptoms of pregnancy can be caused by other conditions and all tests for pregnancy can be positive in the absence of conception. Some examples for missed period are psychic factors (fear of pregnancy, venereal disease, emotional shock); endocrine factors (thyroid, adrenal, or ovarian dysfunctions); metabolic factors (anemia, diabetes, systemic disease); nausea and vomiting factor (acute infections, G.I. disorders, emotional disorders); urinary frequency, GU infection, pelvic tumor, emotional tension. These are just a few examples, there are many more factors that may cause a false diagnosis of pregnancy.

8-3. MINOR DISCOMFORTS OF NORMAL PREGNANCY.

a. Backache.

b. Syncope (lightheadedness and fainting).

c. Dyspnea (difficulty in breathing).

d. Urinary symptoms (frequency, urgency, and stress incontinence).

e. Heartburn.

f. Constipation (avoid enemas as they may induce labor).

g. Hemorrhoids.

h. Breast soreness.

i. Ankle swelling (restrict salt).

j. Varicose veins (provide elastic support).

k. Leg cramps (discontinue medications containing large amounts of phosphorus. Reduce dietary phosphorus intake by limiting meat to 1 meal a day and milk to 1 pint a day).

l. Abdominal pain due to pressure, round ligament tension, flatulence, distention, bowel cramping, and uterine contractions. Intra-abdominal disorders and uterine or adnexal disease can also cause abdominal pain and must be considered and treated as required.

m. Morning sickness occurs in one-half of pregnant women usually starting during 5th or 6th week and persisting until the 14th-16th week. Most severe in the morning upon rising. Treatment: Reassurance and dietary restriction; restrict fats, odorous foods, and spiced dishes. In general, dry foods at frequent intervals are indicated.

8-4. HYPEREMESIS GRAVIDARUM. Persistent severe vomiting; can be fatal if not controlled. Only about 0.2 percent of pregnant women develop hyperemesis gravidarum and cause is not known.

S. Persistent severe vomiting.

O. Acidosis, weight loss, avitaminosis, and jaundice.

A. Hyperemesis gravidarum. Differential diagnosis: Any of the diseases with which vomiting is associated, e.g., infections, poisoning, neoplastic disease, hyperthyroidism, gastric disorders, gallbladder disease, intestinal obstruction, hiatal hernia, and diabetic acidosis.

P. Hospitalize patient in a private room at complete bed rest without bathroom privileges. Allow no visitors (not even husband) until vomiting stops and patient is eating. Place patient N.P.O. x 48 hours. Maintain normal nutrition and electrolyte balance by IV therapy with vitamin and protein supplements as required. Give chlorpromazine IM or suppositories. If no response after 48 hours, institute nasogastric tube feeding of a well-balanced liquid baby formula by slow drip. As soon as possible, place patient on a dry diet of 6 small feedings daily with clear liquids 1 hour after eating. If the situation continues to deteriorate in spite of therapy, therapeutic abortion may be required. Urgent indications are delirium, blindness, tachycardia at rest, jaundice, anuria, and hemorrhage.

8-5. ECTOPIC PREGNANCY. Pregnancy outside the cavity of the uterus. Occurs in 0.5 percent of pregnancies. About 98 percent of ectopic pregnancies occur in the fallopian tubes.

S. Amenorrhea or disordered menstrual pattern followed by uterine bleeding, pelvic pain, and pelvic mass formation. May be acute or chronic. Acute (about 40 percent of cases): Sudden onset of sharp or cutting, intermittent severe lower quadrant pain that does not radiate, with backache during the attack. Scant but persistent uterine bleeding is present in approximately 80 percent of cases. At least two-thirds of patients give history of abnormal menstruation; most have been infertile. Chronic (about 60 percent of cases): Increasing pelvic discomfort, slight but persistent vaginal spotting.

O. Acute: Palpable pelvic mass in 70 percent of cases. Collapse and shock occur in about 10 percent of cases, often after pelvic examination. Chronic: Palpable pelvic mass. Lab findings: CBC shows anemia with slight leukocytosis. Urine urobilinogen elevated in ectopic pregnancy with internal bleeding. Pregnancy tests are of little value in diagnosis.

A. Ectopic pregnancy. Differential diagnosis: Many acute abdominal illnesses, e.g., appendicitis, salpingitis, uterine abortion.

P. Hospitalize patient if there is a reasonable likelihood of ectopic pregnancy. Treat for shock. If possible, type and cross match blood. A transfusion should be started before surgery is begun. Surgical treatment is imperative. Besides normal debridement, generally a salpingectomy will be required. Iron therapy for anemia may be necessary during convalescence.

8-6. PREECLAMPSIA-ECLAMPSIA. Usually occurs in last trimester or early in the puerperium. Preeclampsia denotes the nonconvulsive form; with the development of convulsion and coma the disorder is termed eclampsia. About 10 percent of pregnancies develop preeclampsia-eclampsia and about 5 percent of cases progress to eclampsia. Ten to 15 percent of the women with eclampsia die. Cause is unknown. Predisposing factors are vascular and renal disease, sodium retention, and multiple pregnancy.

S. Preeclampsia: Headache, vertigo, malaise, irritability (due in part to cerebral edema); scintillating scotomas (irregular luminous patches in the visual field after physical or mental labor), visual impairment, epigastric nausea, liver tenderness, and generalized edema.

Eclampsia: Severe preeclampsia symptoms plus generalized tonic-clonic convulsions, coma followed by amnesia and confusion, laborious breathing, frothing at the mouth, twitching of muscle groups (e.g., face, arms), nystagmus (constant involuntary movement of the eyeball), and oliguria or anuria.

O. Preeclampsia: Persistent hypertension or a sudden rise of blood pressure, generalized edema, and proteinuria during the last 4 months of pregnancy. Opthalmoscopic examination in severe preeclampsia and eclampsia reveals variable arteriolar spasm, edema of optic disc, and with increasing severity, cotton-wool exudates and even retinal detachment.

Eclampsia: Marked hypertension preceding a convulsion, and hypotension thereafter (during coma or vascular collapse), and 3-4 + proteinuria. Opthalmoscopic examination reveals papilledema, retinal edema, retinal detachment, vascular spasm, arteriovenous "nicking," and hemorrhages. Repeated opthalmoscopic examination is helpful in judging the success of treatment.

A. Preeclampsia-eclampsia. Differential diagnosis: Primary hypertension, renal and neurologic disease.

P. Preeclampsia: Objectives are to prevent eclampsia, permanent cardiovascular and renal damage, ocular or vascular accidents, and to deliver a normal baby. Delivery should be delayed, if possible, until disease is under control or improvement is marked.

Bed rest with sedation under alert supervision, including frequent B.P. readings and urine protein determination, and careful recording of fluid intake and output. Try to achieve a zero water balance between intake and output. Give diuretics and hypertensive drugs as needed. Place patient on a low-fat, high carbohydrate, with moderate protein, salt-poor (less than 1 gm a day) diet. Opthalmoscopic examination should be done daily.

Eclampsia: Same as preeclampsia plus give magnesium sulfate 10 ml. of 25% aqueous solution IV or IM initially, then 5 ml. IV or IM q.6h. to prevent or control convulsions, lower B.P., and encourage diuresis. (Do not repeat magnesium sulfate if urinary output is less than 100 ml./h., respiration is less than 16/min, or knee jerk reflex is absent.) In case of overdose, give calcium gluconate (or equivalent) 20 ml. of 10% solution IV slowly, repeat every hour until urinary, respiratory, and neurologic depression have cleared (do not give more than 8 injections in 24 hours).

Place patient at absolute bed rest in darkened quiet room. No visitors. Use indwelling catheter, leave B.P. cuff on her arm. Do not disturb patient with unnecessary procedure (e.g., bath, enemas, douches, etc.). Patients with eclampsia often develop premature separation of the placenta with hemorrhage and are susceptible to shock.

Because severe hypertensive disease, renal disease, and preeclampsia-eclampsia are usually aggravated by continuing pregnancy, the best method of treatment is termination of pregnancy. Control eclampsia before attempting induction of labor. Labor can usually be induced by rupturing the fetal membrane. Use oxytocin (Pitocin) to stimulate labor if necessary. If the patient is not at term, if labor is not inducible, if she is bleeding, or if there is a possible disproportion, a cesarean section may be necessary. Most patients improve dramatically in 24-48 hours, but early termination of pregnancy is usually required.

8-7. ANEMIA DURING PREGNANCY. Iron deficiency anemia and folic acid deficiency anemia can be prevented and treated by administering prophylactic multivitamin plus iron capsules to all pregnant women during pregnancy and for 1 month following delivery.

8-8. ABORTION (MISCARRIAGE). At least 12 percent of all pregnancies terminate in spontaneous abortion; of these, three-fourths occur before the 16th week of gestation.

S-O. Abortion is broken down into four classifications:

Inevitable abortion: The passage of some or all of the products of conception is momentarily impending. Bleeding and cramps do not subside.

Complete abortion: All of the conceptus is expelled. When complete abortion is impending, the symptoms of pregnancy often disappear;

sudden bleeding begins, followed by cramping. The fetus and placenta may be expelled separately. When the entire conceptus has been expelled, pain ceases but slight spotting persists.

Incomplete abortion: A significant portion of the conceptus (usually placental fragments) remains in the uterus. Only mild cramps, but bleeding is persistent and often excessive.

Missed abortion: Pregnancy has been terminated for at least 1 month, but the conceptus has not been expelled. Symptoms of pregnancy disappear and body temperature is not elevated. Brownish vaginal discharge but no free bleeding. Pain does not develop. Cervix is semifirm and slightly patulous (open, distended, spread apart); uterus becomes smaller and irregularly softened.

Lab finding: Pregnancy tests are negative or positive. Blood and urine findings are those usually found in infection or anemia if these complications have occurred.

A. Abortion. Differential diagnosis: Bleeding must be differentiated from bleeding from aborting ectopic pregnancy, anovulatory bleeding in nonpregnant women, and membranous dysmenorrhea.

P. If abortion has occurred after 1st trimester, the patient should be hospitalized. In all cases, uterine contractions should be induced with oxytocin (not ergot preparations) to limit blood loss and aid in expulsion of clots and tissues. Ergotrate should only be given if complete abortion is certain. Treat for shock. If there are any signs of infection, give antibiotics. D&C is indicated to remove possibly retained tissue.

8-8. HYDATIDIFORM MOLE AND CHORIOCARCINOMA. A degenerative disorder of the chorion (develops into placenta); occurs in 1 out of 1,500 pregnancies; is five times more prevalent in the Orient than in Western countries; and more common in women over 40. Malignant change occurs in about 4 percent (higher in Asia) of cases and is often fatal when it does occur.

S. Excessive nausea and vomiting in over one-third of cases. Uterine bleeding beginning at 6-8 weeks is observed in virtually all cases and is indicative of threatened or incomplete abortion.

Choriocarcinoma may manifest itself by continued or recurrent uterine bleeding after evacuation of a mole or by presence of an ulcerative vaginal tumor, pelvic mass, or evidence of distant metastatic tumor. Diagnosis is established by pathologic examination of curettage or biopsy.

O. Uterus larger than would be expected in normal pregnancy of the same duration in one-fifth of cases. Intact or collapsed vesicles may be passed through vagina. Preeclampsia-eclampsia, frequently of the fulminating type, may develop during the second trimester, but is unusual. Vaginal smear reveals heavy cell groupings and a predominance of superficial cells.

A. Hydatidiform mole. Differential diagnosis: Hyperemesis gravidarum, multiple pregnancy (extra enlarged uterus), threatening or incomplete abortion.

P. Hospitalize, treat symptoms, evacuate the uterus; probably

will require D&C. If the uterus is larger than a 5-month pregnancy, a hysterectomy is preferred. If malignant tissue is discovered, chemotherapy is necessary.

8-9. CHILDBIRTH.

a. Signs and symptoms of impending childbirth:

(1) Nausea and vomiting.

(2) Mother displays intense anxiety.

(3) Heavy show of blood/bloody mucous.

(4) Intense desire to defecate.

(5) Rapidly occurring contractions with increasing intensity and desire to bear down.

(6) Bulging of membranes from vulva and/or spontaneous rupture.

(7) Dilation of anus with expulsion of feces.

(8) Crowning of the fetal head (figure 1).

b. Delivery of the infant: NOTE: Maintain sterile technique whenever possible, but do not endanger the mother or infant with undue delay.

(1) Place mother in dorsal position, with legs bent and hands grasping knees. Assign an assistant to stand at head of bed to monitor vital signs and offer verbal support and encouragement to the mother.

(2) Attempt to gain mother's confidence and cooperation by explaining what you are doing and what you expect of her.

(3) If time permits, put on sterile gloves and drape perineal area with sterile towels.

(4) As birth approaches, the head distends the perineum more and more with each contraction. When two to three inches of fetal scalp show, an episiotomy may be necessary to prevent serious laceration. Cut the episiotomy 1 to 1 1/2 inches long. (See figure 2.)

(5) Apply gentle pressure with palm of hand to crowning head and perineal area to prevent rapid expulsion of the head. NEVER TRY TO STOP DELIVERY BY PUSHING FORCEFULLY AGAINST THE HEAD.

(6) Encourage mother to pant during contractions to allow for slow, gentle delivery.

(7) As head is delivered, provide support with both hands and allow the head to rotate naturally to the side.

(8) Immediately slip finger around infant's neck and feel for cord that may be wrapped around the neck and choking the infant. If present, attempt to gently slip it off over the head. If it is not possible to remove the cord, clamp and cut the cord at once. (See (14)

below.)

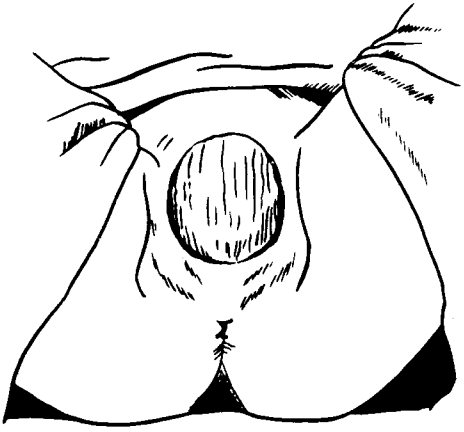


FIGURE 1

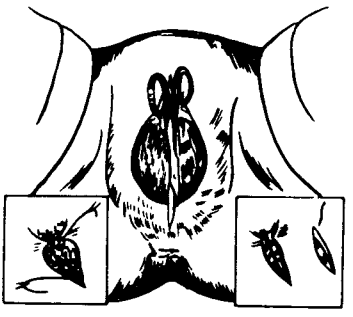


FIGURE 2

(9) If membranes are still intact over the infants face, remove by snipping them at the nape of the neck and pulling away from face and airway at once.

(10) Suction nose then mouth gently with bulb syringe to insure adequate airway. (Newborns are obligate nose breathers.) (See figure 3.)

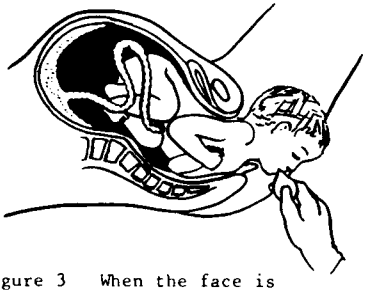


Figure 3 When the face is delivered, the shoulders reside under the pubis; the mouth and oropharynx should be aspirated.

(11) After insuring patient airway, proceed to deliver the shoulders. Place hands on either side of head and exert gentle downward pressure (toward the floor) to deliver the anterior shoulder. Then exert

gentle upward pull to permit delivery of the posterior shoulder. Support the rest of the body as it is born. (See figures 4 and 5.)

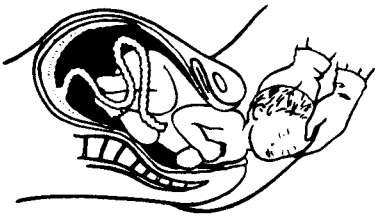


Figure 4 The head rotates to accommodate the shoulders during passage through the birth canal.

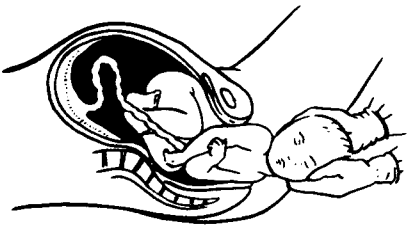


Figure 5 Following rotation, the shoulders are delivered.

(12) With firm grip on body, hold infant along length of arm, with head lower than feet and again suction the nose and mouth. Keep the infant below or equal to the level of the mother until the cord stops pulsating. DO NOT HANG INFANT BY THE FEET.

(13) If infant does not cry spontaneously, apply gentle stimulus to back and soles of feet by rubbing and gently patting.

(14) Wait for cord to stop pulsating, then tie off cord several inches apart and cut between the two ties. Apply first tie several inches from infant's body. Observe for evidence of excessive bleeding from ends of cord.

(15) Wrap infant in blanket, then place on mother's abdomen.

c. Delivery of placenta.

(1) Signs of separation of the placenta.

(a) Large gush of blood from the vagina.

(b) Umbilical cord protrudes 2 to 3 inches farther out of the vagina.

(c) Fundus rises upward in the abdomen.

(d) Uterus firming and becoming more globular.

(2) Expulsion.

(a) Ask mother to "bear down" to expel the placenta. Avoid excessive massage of the uterus.

(b) Apply GENTLE downward pressure on fundus to aid delivery, but do not apply excessive pressure or force.

(c) Check the placenta for evidence of missing portions; any section missing can mean continued uterine bleeding.

d. Care of the newborn.

(1) Maintain patient airway.

(2) Administer eye care (silver nitrate or penicillin prophylaxis).

(3) Observe cord stump for evidence of bleeding.

(4) Provide artificial respiration and cardiac support as needed.

e. Care of the mother.

(1) Observe for signs of excessive bleeding and shock.

(2) Prevent relaxation of the uterine muscles by frequent massage and close observation.

(3) Be prepared to administer IV fluid therapy as needed.

(4) Suture any lacerations and the episiotomy with chromic gut, 00 or 000. Start above apex of vaginal incision and close the vaginal mucosa with a running stitch. Suture the perineal portion as any other wound, making sure that anatomic structures are approximated. (See figure 2.) If the anal sphincter muscle or rectal wall is torn, these are repaired first. Try to get patient evacuated if lacerations are severe.

(5) Take mother's temperature 4-5 times a day. Any elevation above 100.4°F. present on successive days is evidence of infection.

(6) If membranes are ruptured more than 12 hours prior to delivery, assume infection to be present and start antibiotic therapy. If infection occurs after delivery, as evidenced by fever, foul smelling discharge, and tender uterus, start antibiotic therapy.

f. BREECH DELIVERY. (See figures 6-8.)

(1) Let baby be expelled spontaneously to the umbilicus.

(2) Cut a generous episiotomy.

(3) Deliver buttocks by gently pulling upward.

(4) Pull gently until an axilla is visible. Do not exert pressure above the iliac crests upon the abdomen (of the infant) to avoid injury to the abdominal organs.

(5) Have an assistant press downward on the fundus gently.

(6) Deliver the anterior or posterior shoulder, whichever is easier.

(7) Deliver the other arm.

(8) Deliver the head as follows:

(a) With baby lying face down on your arm, put your index finger in baby's mouth.

(b) Hook two fingers of the other hand over each of the baby's shoulders, palm on baby's back.

(c) Pull downward until occiput is under the symphisis.

(d) Bring head out by raising the baby's body up toward the mother's abdomen.

Figure 6 BREECH DELIVERY

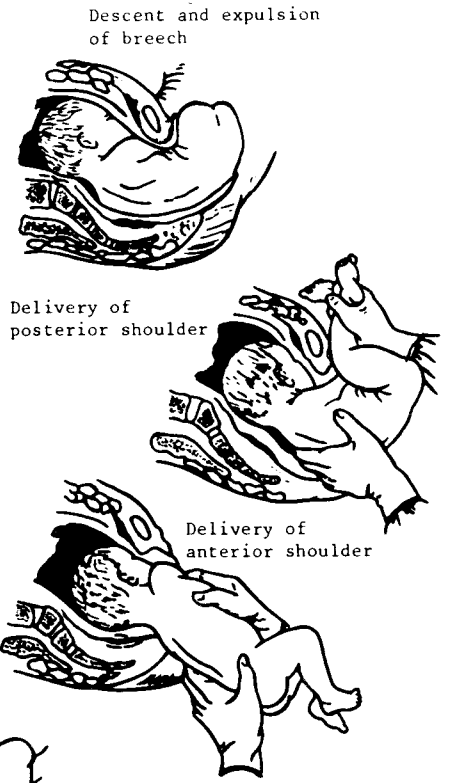


FIGURE 7 BREECH DELIVERY

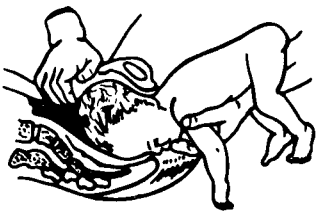


Figure 8 Wigand maneuver for delivery of head. Fingers of left hand inserted into infant's mouth or over mandible; right hand exerting pressure on head from above.

CHAPTER 9

ORTHOPEDICS

9-1. FRACTURES.

a. A fracture is a break in a bone. The break does not need to be complete to be considered a fracture; the bone may only be cracked, or in the case of stress fractures the bone tissue itself may only be torn.

b. To diagnose a fracture without X rays requires the utmost use of history and physical examination. If there is any doubt, treat as a fracture. Fractures may be suspected by one or more of the following:

(1) The patient feels or hears the bone break.

(2) Partial or complete loss of motion.

(3) Crepitus or grating.

(4) Deformity.

(5) Swelling and discoloration.

(6) Abnormal motion at fracture site (arm bending but not at the elbow).

(7) Point tenderness.

(8) Muscle spasm.

c. The main objective in fracture treatment is to prevent broken bones from moving, thus preventing further damage to tissue, nerves, and blood vessels. The basic principles of treating fractures are:

(1) Check and maintain airway (if appropriate).

(2) Determine extent of injury.

(3) Control hemorrhage.

(4) Start IV (if appropriate):

(a) Massive tissue damage.

(b) Fracture of femur.

(c) Any open fracture.

(5) Dress wounds.

(6) Immobilize (splint) fractures.

(a) Splint them where they lie. (Gross deformities may be gently corrected to alleviate circulatory inhibition if present.)

(b) Immobilize the joint above and the joint below the fracture.

(c) Pad the splint to prevent further injury or discomfort. Add extra padding over bony prominences.

(d) Traction is required on most fractures of long bones to overcome muscle contractions.

(7) Under conditions where patient cannot be evacuated, reduce fractures as soon as possible.

(a) Use anesthetics for reduction p.r.n. Fracture reduction can usually be accomplished by injecting local anesthesia into the hematoma of the fracture. An adjunct (e.g., morphine, Demerol) can be used for very painful procedures.

(b) Pad areas of pressure.

(c) Cast or splint in position of function.

(d) Bivalve all casts to allow for swelling and hold in place with ace wrap until swelling subsides (about 3 days), then replace with plaster wrap.

(e) Elevate and cool fractured extremities.

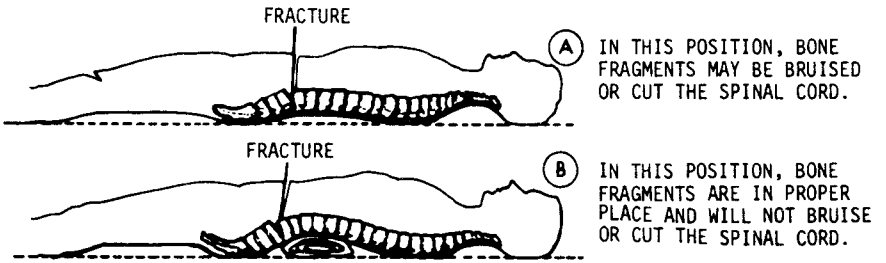
(f) Check extremities frequently for circulation loss.

d. Spinal column injuries. Any injury to the spinal column is potentially dangerous. Although a patient may have no apparent injury, moving him without proper precautions may result in spinal cord injury, causing paralysis.

(1) Fractured lower spine.

(a) Pain, tenderness, muscle spasm, deformity, paralysis, loss of bladder and/or bowel control may be present.

(b) If patient is conscious, place the patient in a swayback position (illustrated below) to avoid flexing the spine. (Flexing the spine can cause bone fragments to lacerate or compress the spinal cord.) If patient is unconscious transport in prone position with head rotated to side (be certain patient does not also have a neck injury).



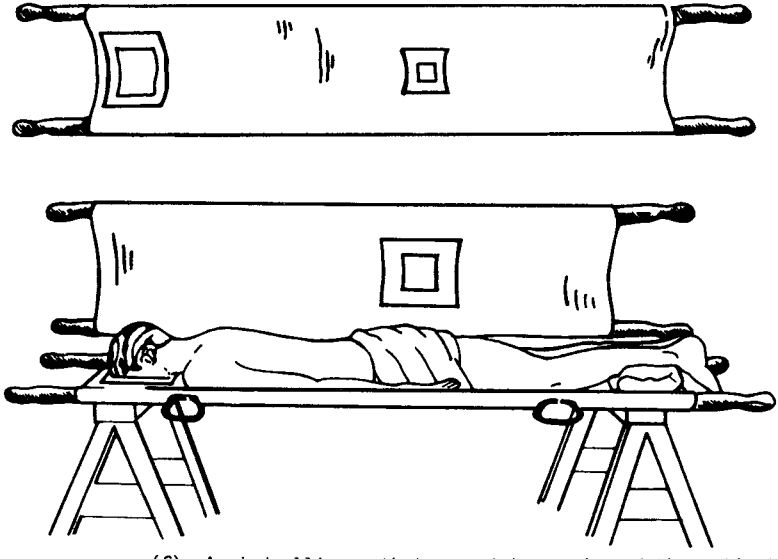
PATIENT PLACED IN A SWAYBACK POSITION.

If the patient is lying in a face-up position, place a folded blanket under the small of his back. If the patient is lying face down, place a folded blanket under his chest. This will keep the spinal column properly aligned and in a swayback position.

(c) Always move the entire vertebral column as a nonflexible unit.

(d) Use rigid litter or board longer than the patient is tall for transportation.

(e) Improvise some type of reversible bed so that the patient can be turned every 2 hours to prevent bed sores. (See illustration below.)



(f) An indwelling catheter must be used, and the patient should receive an enema daily.

(g) Patient must remain immobilized for 8-10 weeks.

(2) Fractured cervical spine.

(a) Signs and symptoms are similar to lower spinal column injury, but paralysis may include arms and upper body, even making the patient unable to breath. Any movement can cause further permanent damage.

(b) Make a thorough examination of the patient without moving his head.

(c) If patient is conscious, the first question should be, "Where do you hurt?" Suspect cervical spine injury if patient complains of severe occipital, shoulder, and arm pain, motor weakness, and numbness in arms and legs.

(d) To transport the patient. With the help of another person--

1. Hold the patient so his head and body are aligned.

2. Place the patient onto a firm surface (door or rigid stretcher). (If he is lying on his face, roll him onto the surface so he is lying on his back.) Be careful to hold the head in a neutral position.

3. Place a small rolled towel or sheet under the neck.

4. Place sandbags or boots filled with sand or dirt on either side of the head to stabilize it, or have someone hold the head in a neutral position while transporting the patient.

(e) Definitive treatment.

1. Fit into a head halter with padding to chin and apply traction in a straight line using a 10-15 lb weight. (A head halter can be improvised, but remember the patient will be in traction for at least 3 weeks. Think of his comfort when improvising.)

2. If there is no evidence of damage to the cord, place the patient on a foam mattress or a firm air mattress.

3. Patients with spinal cord damage must be placed on a turning frame (as with lower back injuries).

4. Commonly with cervical spine injuries, some sensory loss or paralysis may appear due to swelling, transection, or compression of spinal cord. Some or all of this paralysis may disappear as the swelling goes down.

5. Meticulous skin care must be maintained to prevent pressure sores.

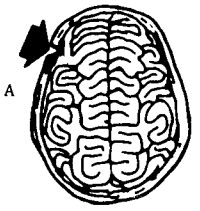
6. Place patient N.P.O., giving only IV fluids for the first few days until there is evidence of audible peristalsis.

7. Catheterize patient using indwelling Foley catheter.

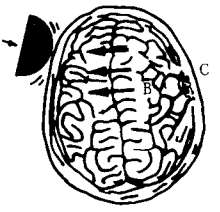
8. Usually after 3 weeks of traction, a cervical collar can be applied in cases where there is no cord damage (a collar can be made using a very well padded wire ladder splint). This should be worn for 8-12 weeks.

e. Craniocerebral injuries.

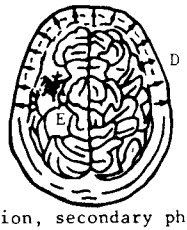
(1) Head injuries result either from penetration or impact. The damage may result from direct injury or may be secondary to compression, tension, or shearing forces caused by the injury. Note illustrations below.



A blow to the skull (direct injury) may result in fracture (A)



Or, in the absence of fracture, it may cause sufficient movement of the brain (B) to result in tearing some of the veins bridging from the cortical surface to the dura (C) with consequent development of subdural hematoma



In addition, secondary phenomena may result from the injury. Ischemia and particularly cerebral edema may ensue. Elevation of intracranial pressure secondary to ischemia cerebral edema (D), a mass lesion (E), or combination of these processes may occur and affect the outcome.

(2) Head injuries are classified as either closed or open.

(a) Closed injuries. Except for a possible bruise or contusion, there is no obvious external damage. Injury may be to the brain itself or to the pia or arachnoid meninges. Rupture of the blood vessels of the pia is particularly important in closed injuries. Blood spilled onto brain cells is a foreign substance and disturbs the functioning of these tissues. Blood collecting within the cranium exerts pressure against the brain. If there is no fracture of the skull, or if skull fracture is such that the integrity of the dura is not disturbed, the cranium is unyielding. If the skull is depressed or displaced inwardly, it may exert direct pressure on the brain even without formation of a hematoma (blood pool).

(b) Open wound. In an open wound there is obvious external damage. Open wounds of the head are subclassified according to whether or not the integrity of the dural is disturbed.

1. Nonperforated dura mater. The wound may be no more than a laceration of the scalp that, although not to be taken lightly, may not be serious. There may be one or more fractures of the skull, but the dura is not perforated. In either case, the possible internal damage is likely to be or become more serious than that of the scalp and skull. If the skull is fractured, it will hold in the same manner as a closed injury against the pressure of any hemorrhage that may occur within the cranium.

2. Perforated dura mater. With the skull and dura opened, the meninges are exposed to the open air and to pathogenic invasion. If the delicate meninges are opened, the brain itself is exposed. If the skull is fractured in such a way that it is no longer a

closed vault, part of it may be torn away, and brain tissue may be extruding through the opening.

(3) All head injuries are potentially dangerous, not only because of the immediate tissue damage and increased susceptibility to infection, but also because of the probability that some vital area or special sense is or will become involved. For these reasons, it is extremely important that all signs and symptoms referable to the nervous system be carefully noted and recorded with the time of their occurrence or observation.

(a) State of consciousness. The following descriptive adjectives should be used, as appropriate, to define the state of consciousness observed.

Conscious. Patient is alert and oriented in time and space.

Confused. Patient is alert but disoriented and excited. (For purposes of taking fluids by mouth, patient is conscious.) The disorientation and excitement, which are not in keeping with the total situation, may be temporary and have a psychological basis in addition to or instead of brain injury.

Somnolent. Patient is excessively drowsy or sleepy, but responds to stimulation.

Semicomatose. Patient responds to painful stimuli but makes no spontaneous movements. (For purposes of taking fluid by mouth, patient is considered unconscious.)

Comatose. Patient does not respond to any applied stimulus; he is unconscious in the usual sense.

(b) Pupil size. Normally, pupils of the eyes tend to become very small in the presence of strong light and to dilate as the light fades. Dilation in the presence of strong light indicates central nervous system impairment. Normally, the pupils are equal in size. When neither eye is obviously injured and the pupils are of unequal size, brain impairment should be assumed and is an ominous sign.

(c) Muscles. The musculature on one or both sides of the face may droop due to lack of stimulation from the brain through the cranial nerves serving the facial muscles. There may be loss or impairment of speech. Paralysis and lack of tone in the muscle mass of any part of the body when there is no damage to the area nor suspicion of spinal cord damage is presumptive evidence of impairment of the brain area controlling movement of those muscles.

(d) Vital signs. The vital signs--temperature, blood pressure, respiration--are especially important in head injuries since changes in these indices frequently indicate the onset of complications.

(e) Headache, nausea, dizziness, and loss of consciousness (which may be brief, intermittent, or extended) may accompany a closed head injury, depending upon the particular injury and its severity. If injury is from impact with a blunt surface, an elevated contusion (bruise) forms when blood and other fluids collect in a pocket in the subcutaneous tissue between the dermis and the skull; there may be fracture in which part of

the skull is displaced inwardly. In the more severe injuries, vomiting and paralysis of some muscle group may occur. The patient may bleed from the nose, mouth, or ears in the absence of obvious injury to these parts. Cerebrospinal fluid coming from the nose or ears indicates a grave injury. Normally a clear liquid, cerebrospinal fluid becomes cloudy when mixed with small quantities of blood. Signs of increasing intracranial pressure include: elevated blood pressure, slow pulse, restlessness, dilation of one or both pupils, decreased respiration, cyanosis, delirium or irritability, and paralysis. Unless a qualified person is available to relieve the pressure by opening the skull, increased respiratory failure, heart failure, and death may be expected.

(4) Closed head injuries may be difficult to diagnose. What may initially appear to be a minor injury with no complications may develop. (within 24 hours to 2 weeks or longer) into a life threatening problem due to gradually increasing intracranial pressure. It is important in head injuries to get a good history at the time of injury and do a complete neurological exam (see Chapter 1, Section VII, Nervous System). If there was any period of unconsciousness, the patient should be placed under observation for at least 24 hours with frequent neurological examinations. You should compare these examinations to determine if there is any deterioration in the neurologic findings.

(5) Emergency medical treatment of head wound.

(a) Assure an open airway. Clear the air passage of any vomitus, mucus, or debris as necessary; place the patient in coma position; turn the semicomatose or comatose patient from one side to the other every 20 minutes. As the patient's condition stabilizes, turning him every hour may be sufficient. Maintaining an open airway is usually not a problem for patients who have only scalp lacerations; the first consideration with these patients is to control the profuse bleeding.

(b) Control bleeding and protect wound. Place a sterile pressure dressing over the wound; do not remove or disturb any foreign material that may be in the wound; leave any protruding brain tissue as it is, and apply the dressing over this tissue.

(c) Prevent or treat shock. Apply measures for prevention or treatment of shock, with the following exceptions and modifications:

Do not put patient in head-low position.

Do not give morphine.

Give necessary fluids by mouth if possible (patient must be conscious and not nauseated). If required, give them very slowly.

(d) Observe patient. Observe the seriously injured patient for hours or until he can be transported to surgery. Take and record vital signs (which include pulse, respiration, and blood pressure) periodically. When possible, seek help from professional medical personnel if symptoms indicating intracranial injury or increased intracranial pressure appear.

f. Fracture of the femur.

(1) Usually there is a marked displacement of the fragments due to contraction of the large muscles in the thigh. This usually carries

varying degrees of shock due to trauma to the bone and soft tissue and loss of blood.

(2) First treat the patient as a whole; restore lost blood and fluid, treat for shock, relieve pain, and always make a search for associated injuries.

(3) If the fracture is an open one, it should be cleaned, debrided, and converted to a closed fracture as soon as the patient's condition permits.

(4) Traction must be used along with immobilization for all fractures of the femur. Use Thomas leg splint or improvise a traction splint of some type.

(5) Union takes at least 12-14 weeks. If there is any doubt, continue the immobilization with reduced traction for 4-8 more weeks.

(6) When union is sound, remove traction and have patient exercise the limb and joints freely in bed for several days, then allow the patient to walk using crutches until you are sure the union is sound.

g. Fracture of the lower jaw (see Chapter 19, Dental Emergencies and Treatment).

h. Fracture of the clavicle.

(1) Pain in shoulder, injured shoulder usually lower than uninjured shoulder, patient cannot raise his arm above his shoulder, patient usually supports the elbow on the affected side with opposite hand, and the fractured ends can usually be felt under the skin.

(2) Pad axillae and over the shoulder.

(3) Use two belts, strips of cloth, cravats, or roller bandages in a figure eight fashion to bring the shoulders up and back.

(4) Support the forearm with a sling and secure it to the body to reduce movement.

(5) Figure eight bandage must remain in place for 4-6 weeks.

i. Rib fracture.

(1) Pain in breathing and coughing. Pain and tenderness at fracture site are produced by hand pressure on the sternum. Sometimes the fracture can be felt. Patient usually holds his hand tightly over the break. If lung is punctured, the patient may cough up bright red frothy blood.

(2) Treat any penetrating chest wounds, hemothorax or pneumothorax (see Chapter 16, Emergency War Surgery).

(3) Control pain and apprehension, but avoid drugs that depress the respiratory and cough reflex centers. Pain is best relieved by intercostal blocks (repeated as necessary).

(a) Injection of one rib may be effective, but usually the

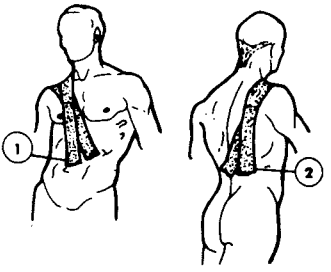
ribs above and below must also be injected to attain relief.

(b) Inject at least 5 cc. of lidocaine a hand's width proximal (toward the spine) under the margin of the rib after aspirating to insure you are not in a blood vessel.

(4) For fractures of upper ribs--

(a) Cleanse the skin and paint with tincture of benzoin.

(b) Have patient hold his breath following expiration while you apply two long 3" adhesive strips across the shoulder of the injured side. Strips should extend well down on the abdomen in the front and to the lower back in the rear (illustration below).

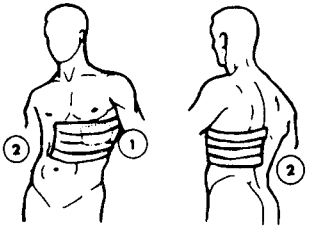


STRAPPING UPPER RIBS.

(5) For fractures of lower ribs--

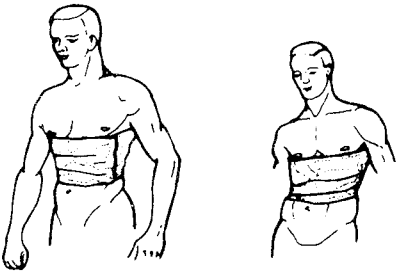
(a) Apply a piece of felt or foam rubber 1-2" thick over the fracture.

(b) Have patient hold his breath following expiration while you apply 3" adhesive strips extending beyond the midline anteriorly and posteriorly (illustration below).



STRAPPING LOWER RIB FRACTURES.

(6) An alternate method for fractures of upper and lower ribs is to apply a 6-8" elastic bandage encircling the trunk from below the costal cage to just below the level of the nipples. (See illustration below.)



STRAPPING WITH ELASTIC BANDAGE OR MUSLIN DRESSING.

(7) Union takes 4-6 weeks.

j. Fractures of fingers or toes.

(1) Manually manipulate fracture into position.

(2) Tape fractured finger or toe to adjacent finger or toe.

(3) Union takes 2-6 weeks.

9-2. SPRAINS.

a. A sprain is caused when a joint is stretched beyond its normal range of motion causing a stretching of the joint capsule and the ligaments surrounding the capsule--some fibers tear but the continuity of the structure remains intact. The amount of tearing of the ligaments determines the severity of the sprain.

b. Symptoms are very sharp pain at the time of injury accompanied by a sensation of no support in that particular joint. In addition, there is rapid swelling and loss or decrease of function in the joint.

c. Treatment.

(1) Sprains should be immobilized either by cast or taping depending on their severity.

(a) Hematomas around the sprained joint usually denote a severe sprain and should be splinted or put in a cast for at least 3 weeks.

(b) Minor sprains should be taped to support the ligaments and give them time to heal.

(2) Keep the joint at rest and elevate the part if possible.

(3) Apply cold compresses immediately after the injury and for the first 24 hours, then apply heat to relieve pain and promote circulation.

9-3. DISLOCATION.

a. A dislocation starts the same as a sprain but continues until the ligaments are torn and the bone pulls out of the joint capsule. This displacement of bone may be either partial or complete. Dislocations are

frequently accompanied by fractures, and structures such as blood vessels, nerves, and soft tissue surrounding the joint may be injured.

b. Symptoms are pain, deformity, swelling, discoloration, and usually a loss of motion. In severe cases, shock may be present.

c. Treatment.

(1) Dislocations should be reduced as soon as possible. Muscles surrounding the joint suffer a shock and you have a period of little or no pain, but as the muscles recover, they try to pull the bone back into the joint by contracting. The longer the bone is out of joint the stronger the contractions and the more damage is done to the surrounding tissue. By the same token, the stronger the contractions the more severe the pain and the harder it is to reduce the dislocation.

(a) Morphine or Demerol should be used in major dislocations to relieve pain and relax the muscles.

(b) The principle to follow in the reduction of dislocations is to pull the bone straight out and away from the joint and allow the muscles to pull the bone back into the joint by gradually releasing the pressure exerted.

(c) Once the dislocation has been reduced, the patient should feel immediate relief.

(d) Check distal capillary filling of the nail beds, blanching, pulse (pulse may or may not be present), color (look for cyanosis or pallor), and warmth of extremity to insure adequate peripheral circulation.

1. If circulation is insufficient, you will have severe pain in the flexor muscles, swelling, coldness, cyanosis or pallor, and paralysis and/or impairment of sensations.

2. Treatment should be started immediately. Treat symptomatically. Relieve anything that may cause circulatory impairment. Apply traction and ice packs (to relieve swelling). If after 2 hours circulatory impairment is not relieved, make S-shaped incision over the joint and extending distally. Incise the fascia and remove the hematoma. This may be sufficient to allow the collateral blood supply to relieve the circulatory insufficiency. (If it is necessary to repair arteries, see Chapter 16, Emergency War Surgery.)

(e) After dislocation has been reduced and blood supply is adaquate, immobilize the joint for at least 3 weeks.

9-4. STRAINS.

a. Strains are due to overstretching or overexerting a muscle or tendon, causing a tearing or rupture.

b. Symptoms are a sharp pain and cramps immediately upon injury, swelling, redness, heat, and loss of function.

c. Treatment.

(1) Place patient in a comfortable position that lessens tension and reduces pressure on the injured muscle or tendon.

(2) Apply heat.

(3) Strap injured area with adhesive tape to immobilize the area.

CHAPTER 10

BURNS AND BLAST INJURIES

10-1. BURNS.

a. How to manage situations causing burns.

(1) Patient with clothes on fire: Since flames ascend, get the patient flat on the floor, forcibly if necessary, with flames uppermost, then smother flames with coat, rug, or blanket.

(2) Scalds: Immediately rip off affected clothes so as to reduce time of application of hot fluid to skin.

(3) Patient in a burning room: Rescuer first hyperventilates, ties a wet cloth around his face and enters room, holding breath and staying low. Give oxygen to patient immediately upon rescue.

(4) Electrical: Push patient off the conductor with a nonconductor or pull him off by his belt. Do not touch his body while he is in contact with the conductor unless you are wearing insulated gloves. First check for heartbeat or pulse. If there is none, start CPR until heart resumes beating and patient is breathing on his own, or is pronounced dead or for a maximum of 3 hours.

b. Calculation of depth (degree) of burn.

(1) First degree:

(a) Examples: Sunburn, low intensity flash.

(b) Only the outer layer (epidermis) is burned.

(c) Symptoms: Tingling, painful, hyperesthetic (extremely sensitive to touch).

(d) Signs: Reddened, blanches with pressure, minimal or no edema.

(e) Course: Peeling and complete recovery within seven days.

(f) Treatment: Noxzema cream or mild analgesics.

(2) Second degree:

(a) Examples: Scalds, flash flame.

(b) Most but not all of the thickness of the skin is burned. Capillary walls are damaged with leakage of plasma into the tissues.

(c) Symptoms: Very painful; sensation to pin prick normal or slightly decreased.

(d) Signs: Blisters either intact or broken; weeping surface; mild edema.

(e) Course: Heals with no scarring or minimal scarring in 2-3 weeks. Infection may convert to third degree.

(3) Third degree:

(a) Example: Fire burns.

(b) The full thickness of the skin is destroyed. Edema is greatest.

(c) Symptoms: Painless to pin prick. Symptoms of shock may appear if edema is great enough.

(d) Signs: Skin is dry, pale white, or charred. Edema is present.

(e) Course: A scab will form and slough in about three weeks. Skin grafting will be necessary since scar, not skin, will cover the burn.

c. Treatment.

(1) First aid for all burns involves the following items:

(a) Relieve pain. (Morphine is the most active way to reach pain in severe burns. IV injection 8 to 10 mg. may bring relief).

(b) Prevent or treat shock.

(c) Prevent infection through strict asepsis.

(d) In burns due to electricity or severely swollen membranes in mouth and throat, the burn may have to be ignored while resuscitative measures are carried out, or CPR instituted to restore the heartbeat.

(2) Remove all clothing except that which is stuck.

(3) Treat chemical burns: Local treatment of chemical burns varies with the burning agents. Wash the burn with large quantities of water; acid burns should be neutralized by washing with a dilute sodium bicarbonate solution, and alkali burns with vinegar or dilute acetic solution. Otherwise the treatment is the same as for thermal burns.

(4) Examination of patient: Make careful initial evaluation combined with an accurate diagnosis.

(a) History and type of burning agent.

(b) Duration of exposure to heat.

(c) Careful examination of the depth of burn. Although burns are classified in degrees, the important factor from a therapeutic and prognostic viewpoint is whether the full thickness of the skin is affected. This may be checked in the following manner:

1. Areas of full thickness of skin loss show insensitivity to pin prick and loss of light touch.

2. If hair can be picked out with little resistance or no pain, the burn is a deep one.

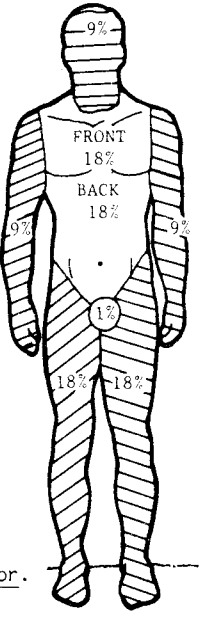
(d) Careful evaluation of the extent of the burn (percent of the body surface area burned). This may be accomplished by the "Rule of Nines" method.

d. Calculating the area of the burn.

(1) No one can treat a burn intelligently unless he is able to correctly observe and record the area of the burn expressed in percent of total body surface.

(2) RULE OF NINES:

Each upper extremity	9%
Head and neck	9%
Anterior trunk	18%
Posterior trunk	18%
Each thigh	9%
Each lower leg	9%
Perineum	1%



CAUTION: Do not overestimate percentage. Common error.

e. Classification of burns by severity.

(1) Critical burns.

(a) Second degree over 30% area.

(b) Third degree over 10% area.

(c) Third degree of hands, feet, or face.

(d) Any burn complicated by respiratory injury or other injury such as fractures or major soft tissue injury.

(2) Moderate burns.

(a) Second degree of 15-30% area.

(b) Third degree of 4-10% not involving hands, feet, or face.

(3) Minor burns.

(a) Second degree less than 15% area.

(b) Third degree less than 4% area not involving hands, feet, or face.

f. General principles of burn treatment.

(1) Cleansing: The cleaning of burned areas should be accomplished by gentle washing with pHisoHex and sterile saline when the burn is fresh. Wash away all trash, dirt, and bits of clothing. After danger of infection is past, burned area may be washed with ordinary soap and tap water.

(2) Blisters: What should be done about blisters? Leave intact blisters alone until they break themselves, then cut away with iris scissors. Any intact blister showing evidence of infection within it (purulent contents or surrounding lymphangitis) should be immediately opened and debrided in a sterile manner.

(3) All second degree or more burns should receive a tetanus booster.

(4) Ointments: There is no proven evidence that any antibiotic ointment applied to a burned surface is any more advantageous than plain vaseline or no ointment at all. However, it has been the personal experience of others that routine light application of Furacin ointment or Silvadene Cream to all second and third degree burns decreases the incidence of infection, and may prevent a deep second degree from going to a third degree burn. A very rare patient may exhibit a sensitivity to Furacin ointment or Silvadene Cream, but the benefit far outweighs the risk.

(5) Bandaging: Numerous papers variously supporting the "open method" and the "closed method" exist. You cannot go wrong by following this rule:

(a) All burns of the head, neck, and perineum should be left open.

(b) All burns of the hands, joints, and circumferential burns of the trunk and extremities should be bandaged.

(c) For burns involving a single aspect of the trunk or extremity, either method is fine.

Use own judgment, depending on the circumstances. If in doubt, bandage.

CAUTION: Extensive bandaging of a patient hospitalized in a warm room may cause hyperpyrexia.

(d) When bandaging, a nonadherent material should be placed next to the burn to prevent granulation tissue growing into the gauze only to be ripped off at the next dressing change. An ideal such material is

parachute nylon obtained from a surplus parachute. Cut it up in small pieces, package and autoclave it, and that will make an ideal nonadherent material to place next to the burn. If such is not available, use the finest mesh gauze that is available.

(e) Burns of the hand should be bandaged using a bulky dressing with the hand in the position of function (slight extension at the wrist and all fingers moderately flexed). If the fingers are burned, place bandaging between the fingers. The tips of the fingers should be exposed to allow for circulation to be checked and to preserve the patient's sense of touch.

(f) Burn bandages should be bulky so as to absorb exudate. Change the original burn bandage at 5 to 7 days if there are no complications. Change the dressing earlier if it is stained from the inside out, if there is malodor, if there is an increased pain or unexplained elevation of the patient's temperature.

(6) Burn over joints: Immobilize the joint to allow healing.

(7) Antibiotics: No systemic antibiotics are indicated for burns less than 15%. For second or third degree burns over 15%, give procaine penicillin 1.8 million units and streptomycin 1.0 gram daily in divided doses for 5 days. Thereafter, rely on sensitivity disc if infection is apparent in the wound.

(8) Pain: Small second degree burns can usually be managed with oral codeine, 1/2 grains from 1 to every 3 hours. Second degree burns over 20% usually require parenteral narcotic analgesics. The drug of choice is morphine; the second choice is Demerol.

NOTE: If hypotension or shock exist, which is possible if the burn is over 20%, give the analgesic intravenously in 1/3 to 1/2 of the IM doses. The reason for this is that subcutaneous or IM medications are not picked up by the patient in shock because of decreased blood flow. Therefore, the medications will not give pain relief even if repeated and will accumulate in the extravascular spaces until such time as the shock is corrected. Then they will pick medication up into the circulation all at once and constitute an overdose.

(9) Environmental temperature: The ideal environmental temperature for treatment of a large burn is 75 degrees to 80 degrees F.

(10) Burns of the genitalia: The urethra may close off from excessive edema in one-half hour, therefore place a Foley catheter as early as possible.

(11) Transportation of burned patients: If transportation to a hospital requires less than one-half hour, the only treatment required is about 1/8 grain morphine intravenously. If transportation is expected to require more than one-half hour, start an IV of Ringer's lactate or Saline. The patient does not tolerate prolonged transportation as well after 48 hours as he does before that time.

(12) Establishment of intravenous lifeline: All patients with burn over 20% must have an 18-gage needle (or preferably an intravenous catheter) placed and anchored securely in a vein as soon as the diagnosis is made because intravenous fluids and whole blood will be required. The

most practical site for a corpsman to do a cutdown is the greater saphenous vein just anterior to the medial malleolus of either ankle.

(13) Oral intake: Burned patients are usually quite thirsty and demand large quantities of water. Fluid replacement in burns less than 15% can usually be administered entirely by mouth. In more extensive burns, paralytic ileus and vomiting are quite common during the first few days. In an extensively burned patient, all oral intake is withheld and the entire replacement therapy is intravenous.

(14) Respiratory tract burns: A burn of the trachea or bronchi is a very serious complication of a burn injury. It is most apt to occur in flame burns about the face, or if the burn was sustained in a close-fire explosion, or if live steam is involved. Edema of any of the upper respiratory passages after such a burn may quickly cause death. As soon as the burned patient is seen, determine if respiratory tract damage has occurred.

(a) The symptoms of respiratory tract burns are hoarseness, coughing, rapid respiration, or cyanosis. Redness in the posterior pharynx may be present. Rales or rhonchi in the lung may be or may not be present. If a respiratory tract burn exists, a tracheostomy should be performed as soon as pain is relieved and replacement therapy has been started (fluids or whole blood). Give oxygen.

(b) Recent evidence clearly shows that a tracheotomy should not be done in burn patients unless there is a clear indication for it. The concept of "prophylactic tracheotomy" in the burned patient should be abandoned. If the tracheotomy is done, be sure it is done into the second or third tracheal rings, located just below the cricoid cartilage and above the isthmus of the thyroid gland. A "low" tracheotomy below the fourth ring is dangerous; the endotracheal cannula may rest on the carina or enter a main stem bronchus.

g. Treatment of pulmonary edema.

(1) Semi-Fowler position (sitting up).

(2) Morphine grains 1/6, IM or IV.

(3) Oxygen in high concentration (8-9 liters per minute).

(4) Aminophylline 250-500 mg. IV slowly.

(5) Reduction in the blood volume by venesection (300-500 ml.) or tourniquets on two of the four extremities applied with sufficient pressure to obstruct venous but not arterial flow. Rotate every 15 minutes.

h. Pathologic physiology.

(1) Edema inevitably follows burning. Heat causes capillary injury, resulting in vasodilation and increased capillary permeability.

(2) A burn is a three-dimensional wound consisting of length and width (area) and depth. Depth cannot be visualized and is difficult to determine accurately, but is an extremely important factor affecting the volume of edema that will occur. Examples: In a first degree burn,

vasodilation is the only change that occurs, and edema is minimal. A second degree burn, being deeper, involves a large volume of tissue plus more extensive capillary damage so that edema occurs. Although there is no edema in the charred eschar of a third degree burn, the volume of edema around and under it is greater than the volume of edema in a second degree burn of comparable area. Remember that the greatest fluid losses occur deep in the wound, hence appearance is misleading.

i. Edema time factors.

(1) The rate of edema formation (intravascular fluid loss) is greatest in the first 8 hours after burning. Edema continues to form, but at a less rapid rate, until about 36 to 48 hours after burning, at which time the total edema is maximal.

(2) Resorption of edema then occurs and proceeds slowly over 5 to 7 days, but burn edema may persist for 2 or 3 weeks.

(3) From the above statement it is apparent that the danger of shock is greatest in the first 24 hours after burning and is almost never a problem after 36 to 48 hours, if adequate fluid replacement therapy has been given. After the possibility of respiratory tract burn has been considered, all efforts in therapy are then directed towards proper fluid therapy to replace the fluid extravasated into the tissues as edema.

j. Fluid replacement in minor burns.

(1) In general, there is not a significant danger of shock in burns less than 20% and these can be handled with an oral fluid replacement therapy consisting of a solution of 1/2 teaspoon of salt and 1/2 teaspoon baking soda in one quart of water.

(2) The solution should be thoroughly chilled for optimal patient tolerance. If vomiting occurs, discontinue oral intake and use the intravenous route.

(3) In a disaster, when IV fluids may not be available, oral electrolyte replacement solution may be a lifesaving measure for all patients with burns up to 35%. The recommendation limiting the use of oral therapy to patients with less than 20% burns is conservative and assumes availability of IV fluids.

(4) If both IV and oral fluids are given, the oral intake must be included in the calculated 24-hour fluid replacement plan.

k. Calculation of fluid replacement therapy in moderate or serious burns.

(1) Fluid replacement requirements are governed by many complex variables and it is impossible to state in a formula exact replacement requirements. The burn formula below is a very practical and valuable as an initial rough estimate fluid replacement guide.

(2) Brooke formula:

(a) Ml. of fluid to be given in the first 24 hours = (% body burn) x (wt. in kilograms) x (0.5 cc.) colloid plus (% body burn) x (wt. in kilograms) x (1.5 ml.) Ringer's lactate solution plus 2,000 ml. 5%

dextrose in water.

(b) Expressed in terms of pounds instead of kilograms, the formula becomes: ml. first 24 hours = (% burn) x (wt. in pounds) x 0.23 ml. colloid + (% burn) x (wt. in pounds) x (0.67 ml.) Ringer's lactate solution plus 2,000 ml. of 5% dextrose in water.

(c) Give one-half of the total calculated 24-hour requirement in the first 8 hours after the burn, starting from the time the burn occurred. Give the remainder evenly over the remaining 16 hours.

(d) In applying the formula to burns over 50%, calculate as though only 50% had been burned.

(e) Do not count first degree burns in computing the fluid requirement.

(f) During the second 24 hours, give one-half of the volume of colloid and Ringer's lactate as calculated for the first 24 hours, plus 2,000 ml. of 5% dextrose in water.

Problem: A 150-lb man sustains a total body burn of 35% consisting of 20% first degree, 25% second degree, and 10% third degree. Plan fluid therapy. Assume you have dextran.

Ml. first 24 hours = 35 x 150 x 0.23 ml. colloid + 35 x 150 x 0.67 ml. Ringer's lactate plus 2,000 cc. 5% dextrose in water which equals 1,207 ml. dextran plus 3,517 ml. Ringer's lactate plus 2,000 ml. dextrose in water.

The total volume of fluids to be given in the first 24 hours after the burn is 6,724 ml. One half of this amount, or about 3,500 ml., should be given in the first 8 hours after the burn. Therefore, appropriate fluids for the first 8 hours after the burn would be 2,000 ml. of Ringer's lactate, then 500 ml. Dextran, then 1,000 ml. dextrose 5% in water. If IV fluids are started late and you are trying to catch up, you can give as much as 200 ml. in 1 hour (300 drops per minute) without overloading the circulation. Or if treatment has been delayed and the patient is already in shock, fluid administration by two separate veins may be necessary.

(g) By far the most accurate guide to adequacy of administered fluids is the rate of urinary output. Therefore, all patients receiving prolonged therapy should have a Foley catheter in place and urinary output measured at least hourly. A urinary output of 25 to 40 ml. per hour is adequate. A rate much over this indicates fluids are being given too rapidly.

1. Oliguria (very low rate of urine output).

(1) A special problem is posed by the severely burned patient who has oliguria or anuria even after fluid therapy has been started, since acute renal failure is a rare complication of severe burns. Should the case be considered one of renal failure or should it be assumed that fluid therapy has been insufficient? This question is crucial since the accepted method of treatment for renal failure is rigid fluid restriction, a plan that would be disastrous if in fact the oliguria is due to inadequate fluids. In this circumstance the correct course of action is intensive therapy with whatever fluid appears to be deficient on the assumption that oliguria is caused by inadequate fluids. If oliguria persists, then he has

a renal shutdown and you stop the flow of IV fluids after 1,000 ml. of colloid and 1,000 ml. of electrolyte solution have been given rapidly (150-300 drops per minute), then renal failure due to organic changes is likely to be present.

(2) Oliguria is often encountered in extensive second degree burns, while anuria is more commonly a complication of extensive third degree burns.

m. Other care in the first 24 hours.

(1) Record the urine output, pulse, and blood pressure at least hourly. It is preferable not to give anything by mouth for the first 48 hours in severely burned patients. If there is severe thirst, small amounts of water may be given, but the amount must be recorded and subtracted from the total allowance.

2. Since acute gastric dilation is a common complication, examine the abdomen frequently for distention. It may be necessary to pass a nasogastric tube.

n. The second 24-hour period.

(1) The fluid regimen consists of 1/2 of the colloid and electrolyte solutions given during the first 24 hours, plus 2,000 ml. 5% dextrose in water.

(2) Fever of 101-102°F. (orally) is not uncommon even in the absence of infection.

o. Treatment after 48 hours. By 48 hours, edema is maximal but its production has ceased. The physiology of electrolyte imbalance that may occur after 48 hours is too complicated to be considered in this manual. A general rule is to give only 5% dextrose and water in order to dilute the large amount of sodium being immobilized from the burn edema.

p. Outline of immediate treatment plan.

(1) Relieve pain.

(2) Obtain history, including weight of patient.

(3) Map area and degree of burn.

(4) Determine need for tracheotomy.

(5) Start Ringer's lactate of dextran, using a large-bore needle (an 18-gage is preferable).

(6) Do cut down if necessary.

(7) Insert Foley catheter.

(8) Initiate local care, like cleansing and dressing.

(9) Give penicillin and streptomycin if indicated.

(10) Give tetanus toxoid.

(11) Plan fluid replacement requirement.

10-2. BLAST INJURIES.

a. General information. The human body is not constructed to tolerate very marked or sudden increases in pressure. This is obvious from our past experiences in wars and from the experiences of deep-sea divers. The effects of a blast depend upon the wave length and the substance this blast or "shock" waves are transmitted by. Long slow waves are very low pitched and do very little damage since usually only one or two waves pass through the body. A sudden increase of 7 psi may rupture the tympanic membrane; however, it will take a sudden increase in excess of 30 psi to injure the hollow organs or cavities of the body.

b. Types of blast.

(1) Air. The waves travel slowest in air and do not do as much damage to the human body. Most injuries from an air blast are not true blast injuries but are caused by flying debris, etc.

(2) Water. Blast waves travel much faster in water than in air and will cause damage at greater distances. The human body has essentially the same density as sea water, which allows blast waves to pass through solid tissue without injury. Most of the damage from a water blast occurs to the hollow viscera, lungs, abdomen, and gas-filled cavities.

(3) Solid. Blast waves travel fastest through solid objects. The denser the substance, the faster they travel. These waves traveling through the deck of ships, etc., produce breaks in major blood vessels often without a break in the skin.

c. Classification of blast injuries.

(1) Primary. Injuries caused by the effect of blast waves on the body such as ruptured tympanic membranes, damage to hollow viscera, etc.

(2) Secondary. Injuries caused by flying debris such as shrapnel, bricks, chunks of plaster, etc. This classification also covers those who were trapped and injured in a building that was blown up around them.

(3) Tertiary. Injuries caused by the blast picking up the body and hurling it through the air striking some other object.

NOTE: It is frequently very hard to determine just which classification is proper and there are many times when more than one classification of injury will coexist in the same patient.

d. Common blast injuries. It is necessary to suspect blast injuries after any incident that would cause them. With no external marks or visible symptoms, the victim might be required to do something that could prove fatal to him. If there are no visible signs of injury and the patient is developing shock, it is a good indication of blast injury. Victims are often treated as "walking wounded" and only when shock, dyspnea, apprehension, tremulousness, and fear are apparent, is the correct diagnosis made.

(1) Ruptured tympanic membrane. As previously noted, this is the most common blast injury and occurs when there is as little as 7 psi sudden increase in pressure.

(a) Symptoms: A sudden, severe, lancinating pain in the ear. There may be bleeding from the affected ear and various degrees of hearing loss.

(b) Treatment: Clean the opening or meatus gently then "leave it alone." Do not pack, syringe, or instill any medication.

(2) Blast lung. When the symptoms of pain and clinical signs are first in and remain localized in the upper abdomen, the chances are very good that the blast injury is thoracic.

(a) Symptoms: In addition to the routine blast symptoms there is usually cyanosis, rapid pulse, and pain in the chest and upper abdomen with moderate abdominal rigidity. The patient may be coughing with ineffective expectoration of bloody, frothy mucous. There are usually multiple hematomas along the anterior costal lines.

(b) Treatment: Move as little as possible and it is best to stabilize patient for 48 hours before even evacuating him if possible. Administer oxygen for relief of cyanosis and dyspnea. Always suspect pulmonary edema from alveolar hemorrhage, and if it is mandatory to use IV fluids, use with extreme caution and run at slow rate. Atropine sulfate may be given in small doses to help diminish secretions. Avoid any ether or gaseous anesthetic agents. Antibiotics are useful for serious blast lung cases.

(3) Blast abdomen. Many persons describe the sudden onset of pain as a "kick in the belly," followed by a remission then by a recurrence. When clinical signs occur first in the upper abdomen then spread to the lower abdomen, then abdominal blast injury is most certain. When clinical signs remain from the onset in the lower abdomen, there is little doubt of intraperitoneal damage.

(a) Symptoms: Sudden occurrence of abdominal pain, a brief period of remission, then reoccurrence of severe, unremitting, and most of all increasing pain. Frequent bowel evacuations with difficulty in urination. Melena or frank passage of bright red blood in the stool.

(b) Treatment: The serious cases can be treated only with surgery. Place them high on the evacuation list. If hemorrhage or perforation is suspected with good reason, then request advice on dosage of antibiotics to sterilize the bowel. Keep N.P.O., insert nasogastric (NG) tube, catheterize with indwelling catheter, and consider pain relief. Withhold morphine until a careful assessment is made of the injury and treatment schedule. For example, do not give morphine if he will be on anesthesia and surgery within the following 1 to 2 hours.

(4) Other blast injuries. For fractures and other tissue trauma, treat the same as at any other time. Contusions of the scrotum and testicular pain are common; treat with adequate support. The transient paresis of the limbs that has been described in association with blast injuries is probably due to minor vascular disturbances in the spinal cord.

CHAPTER 11

HEAT AND COLD INJURIES

11-1. HEAT INJURIES.

a. Factors that govern heat injuries.

(1) Water.

(a) The human body is absolutely dependent upon water to cool itself in hot environments. In severe heat it is possible for a person to lose a quart of water each hour. Water lost must be replaced or an individual can become a heat injury. The activity will determine the amount of water necessary to maintain proper body functions, as illustrated below.

ACTIVITY	ILLUSTRATIVE DUTIES	Quarts per man per day for drinking purposes (a guide for planning only) WBGT or WD index* Less than 80°	Greater than 80°
Light	Desk	6	10
Moderate ...	Route march	7	11
Heavy	Forced marches; stevedoring; entrenching; or route marches with heavy loads or in CBR protective clothing.	9	13

*80° wet bulb globe temperature (WBGT) or WD index is approximately equivalent to a dry bulb temperature of 85° in a jungle or 105° in a desert environment.

Water requirements.

(b) The myth that humans can be taught to adjust to decreased water intake has been disproven. When water is in short supply, significant water economy can be accomplished only by limiting physical activity to the coolest part of the day or night.

(2) Salt.

(a) Ordinarily one's normal food intake will contain adequate salt; however, in heat stress situations, unacclimatized persons may require additional intake.

(b) Unless one is sweating continuously or repeatedly, salt tablets will not be required. Extra salt in the cooking, at the table, and in the water is all that is required.

(c) Older people and acclimatized persons tend to have less acute needs for salt replacement.

(d) A convenient way to provide adequate salt is to salt the drinking water 0.1%, in amounts shown below.

Salt	Diluting Water
2 ten-grain salt tablets--dissolved in	1-quart canteen
4 ten-grain salt tablets--dissolved in	2-quart canteen
1 1/3 level mess kit spoons salt--dissolved in	5-gallon can
9 level mess kit spoons salt--dissolved in	lister bag
1 level canteen cup salt--dissolved in	250-gallon water trailer

Preparation of 0.1 percent salt solution.

(3) Acclimatization. It takes a period of about 2 weeks to become acclimatized, regardless of the physical condition. An acclimatization program should consist of a person being exposed to progressively increasing heat and physical exertion in a new climate condition. Careful and fully developed acclimatization increases resistance, but it does not give complete protection from the ill effects of heat.

(4) Physical conditioning has a significant bearing on the reaction to heat stress.

(a) Debilitating diseases and injuries enhance the likelihood of heat injuries.

(b) Overweight personnel have a much higher incidence of heat injuries.

(5) Environmental factors.

(a) Although heat injuries can occur at temperatures below 0°F., e.g., overexerting and overdressing, most heat injuries occur during periods of high temperature and humidity. As the temperature rises, physical activity should be curtailed, as shown in heat categories below.

GUIDELINES FOR PHYSICAL ACTIVITY

CATEGORY	WBGT INDEX	NONACCLIMATED PERSONNEL	ACCLIMATED PERSONNEL
I	82-84.9°F.	Use discretion in planning intense physical activity. Limit intensity of work and exposure to sun. Provide constant supervision.	Normal duties.
II	85-87.9°F.	Strenuous exercises such as close order drill and physical training will be canceled. Outdoor classes in the sun will be canceled.	Use discretion in planning intense physical activity. Limit intensity of work and exposure to sun. Provide constant supervision.
III	88-89.9°F.	All physical training, strenuous activities, and parades will be canceled.	Strenuous outdoor activities will be minimized for all personnel with less than 12 weeks

			training in hot weather.
IV	90°F. and above	Strenuous activities and nonessential duty be canceled.	Strenuous activities and nonessential duty will be canceled.

Heat categories.

(b) The four basic factors that determine the degree of heat stress exerted by the environment are air temperature, relative humidity, air movement, and heat radiation. These factors can be measured by using a WBGT Index. The WBGT Index is computed as follows:

WBGT = 0.7 x wet bulb temperature

+ 0.2 x black globe temperature

+ 0.1 x dry bulb temperature.

(c) To make a WBGT apparatus, see sketch on following page.

b. Heat cramps. Caused by excessive salt loss from the body.

S. Painful cramps of the voluntary muscles usually in paroxysms lasting from 3-10 minutes with periods of relative comfort between the spasms. Patient may be grimacing and thrashing about with arms and legs drawn up. Skin is usually hot and moist.

O. Blood pressure and temperature will usually be normal. The pulse may be slightly elevated.

A. Heat cramps. Differential diagnosis: Heat exhaustion.

P. Remove to shaded area and give salt in any form to balance loss. IV normal saline 500-1,000 cc. in acute cases, 0.1% salt solution in cool water orally will afford both relief and continued protection. Massaging of cramped muscles will usually help afford immediate relief.

Do not use hot packs on the cramped muscles; that will only make it worse. Do not use saline enemas as that only draws more salt and water from the tissue.

c. Heat exhaustion. Caused by failure of peripheral circulation due to salt loss and dehydration.

S. Profuse sweating (diaphoresis) with cold, wet, and pale skin. Headache, mental confusion, vertigo, incoordination, drowsiness, extreme weakness, anorexia, nausea and vomiting, with visual disturbances. Occasionally, cramps of the extremities or abdominal muscles occur.

O. Temperature taken orally may be subnormal or slightly elevated, rectal temperature is usually elevated (100-101°F.), rapid pulse (140-200) with blood pressure usually lowered.

A. Heat exhaustion. Differential diagnosis: Heat cramps,

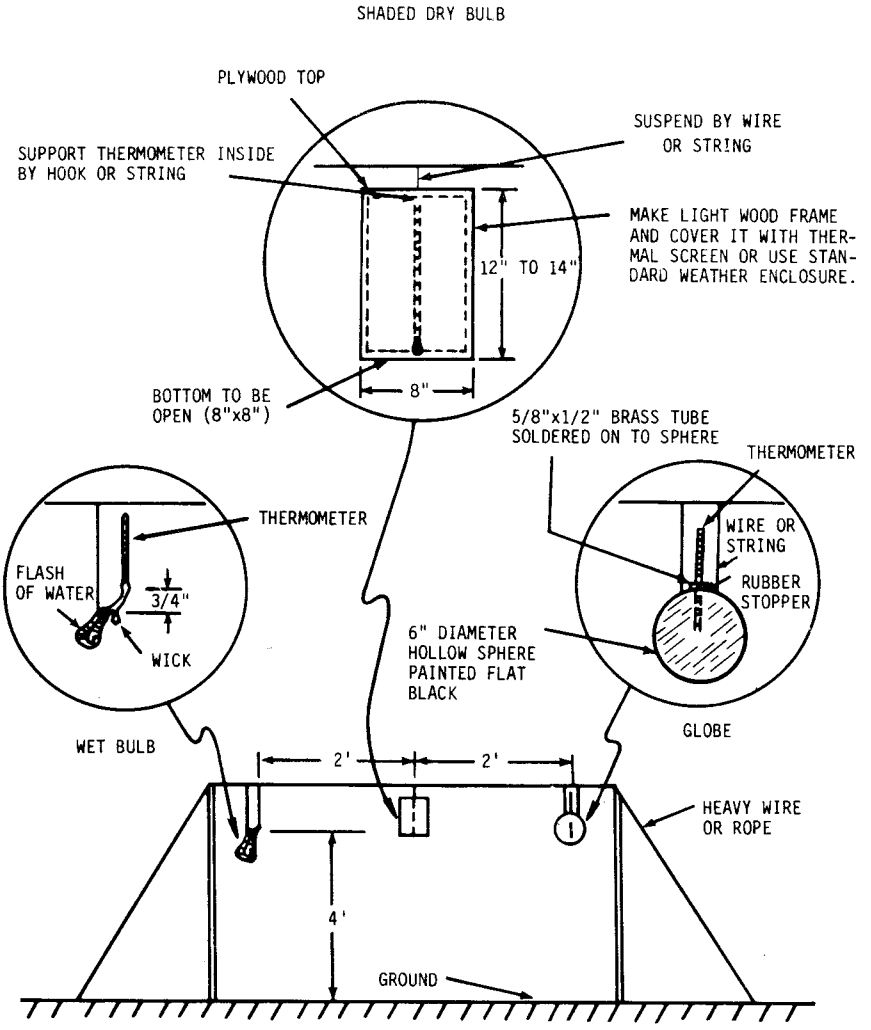


FIGURE 1. WBGT Index of-field apparatus.

heatstroke.

P. Remove patient to a cool, shaded area. Replace fluids and salt by giving patient cool water with 0.1% salt solution; or if he cannot take by mouth, give 1,000 to 1,500 cc. 5% dextrose in normal saline or a normal saline IV (an IV should be started in any case). Stimulation may be required such as tea, coke, coffee (caffeine) or even IM injection of .3 to .5 cc. of 1:1,000 epinephrine. Avoid immediate reexposure to heat.

d. Heatstroke. Caused by a breakdown of the body heat regulating mechanism.

S-O. There may be early symptoms of headache, dizziness, mental confusion, weakness, nausea, involuntary urination, and diminished or absent sweating. There may even be a false sense of exhilaration. Usually, however, the onset is dramatically sudden with collapse and loss of consciousness. Convulsions may occur. The skin is hot, red, and dry. The pulse is full and rapid, with blood pressure normal or elevated. Respirations are rapid and deep. The body temperature is markedly elevated (106-110oF.). As the patient's condition worsens, cyanosis is usually noted. The breathing becomes shallow and irregular. Pulmonary edema, involuntary urination and defecation, vomiting, hemorrhagic tendencies, disturbances of muscle tone, and jaundice and meningitislike symptoms to include tetanuslike body arching. Death may come very rapidly, but if patient survives until the second day, recovery usually occurs. Severe relapses may occur.

A. Heat stroke.

P. Lower the patient's body temperature as rapidly as possible. The longer the body temperature is high the greater the threat of permanent damage or death. Remove the patient's clothes and immerse him in cold water (tub of ice water, if possible). If not available, wet patient down with ice, water, or alcohol and fan him. Rub patient's extremities and trunk briskly to increase circulation to the skin. Temperature must be monitored closely and when temperature drops to 102oF. stop cooling, dry patient off, and wrap him in blankets or place him on heating pads. Usually the temperature will continue to fall, and sometimes will reach as low as 94oF. before it starts to rise again. If the patient's temperature falls below 97oF. be prepared to start the cooling process again when his temperature rises to 97oF. Constantly monitor the patient's body temperature and alternate heating and cooling until his temperature stabilizes. Continue monitoring the temperature every 10 minutes for the next 48 hours.

Care must be used in giving heatstroke patients medication. Sedative drugs disturb the heat regulating center and should be avoided if possible. When sedatives are necessary (as with convulsions), a short acting barbiturate such as sodium Pentothal IV is the drug of choice. If a longer acting drug is needed, phenobarbital should be administered IM. Epinephrine, sodium amytal, and morphine are contraindicated. Atropine or other drugs that may interfere with sweating are also contraindicated. An IV of normal saline or as second choice Ringer's lactate should be started and 1,000-2,000 cc. should be given initially. Subsequent IV infusion is determined by hourly urinary output and serum electrolyte determinations. It is important to recognize that the heat regulating centers may not function correctly for many weeks after an attack. This means the patient must be kept in a fairly controlled environment during this period and

monitored at regular intervals. One attack of heatstroke predisposes an individual to further attacks.

11-2. COLD INJURIES.

a. Factors governing cold injuries.

(1) Weather, temperature, humidity, precipitation, and wind modify the rate of body heat loss. Low temperatures and low humidity favor frostbite, whereas higher temperatures together with moisture are usually associated with trench foot. Wind velocity accelerates body heat loss under both wet and cold conditions. (See chart.)

Cooling Power of Wind on Exposed Flesh Expressed as an Equivalent Temperature (under calm conditions)

Estimated wind speed (in mph)	Actual Thermometer Reading (°F.)											
	50	40	30	20	10	0	-10	-20	-30	-40	-50	-60
	EQUIVALENT TEMPERATURE (F.)											
calm	50	40	30	20	10	0	-10	-20	-30	-40	-50	-60
5	48	37	27	16	6	-5	-15	-26	-36	-47	-57	-68
10	40	28	16	4	-9	-21	-33	-46	-58	-70	-83	-95
15	36	22	9	-5	-18	-36	-45	-58	-72	-85	-99	-112
20	32	18	4	-10	-25	-39	-53	-67	-82	-96	-110	-124
25	30	16	0	-15	-29	-44	-59	-74	-88	-104	-118	-133
30	28	13	-2	-18	-33	-48	-63	-79	-94	-109	-125	-140
35	27	11	-4	-20	-35	-49	-67	-82	-98	-113	-129	-145
40	26	10	-6	-21	-37	-53	-69	-85	-100	-116	-132	-148
(wind speeds greater than 40 mph have little additional effect)	LITTLE DANGER (for properly clothed person)				INCREASING DANGER			GREAT DANGER				
	Danger from freezing of exposed flesh											

Trench foot and immersion foot may occur at any point on this chart.

(2) Clothing should be worn loose and in layers. Loose layers of clothing with air space between them worn under an outer wind- and water-resistant garment provide maximum protection. The loose inner layers can and must be removed during periods of strenuous physical exertion to prevent overheating and accumulation of perspiration. Wet clothing loses much of its insulation value.

(3) The very young and very old are more susceptible to cold injuries.

(4) Previous cold injuries definitely increase the risk of subsequent cold injury, not necessarily involving the part previously injured.

(5) Fatigue may cause apathy leading to neglect of acts vital to

survival.

(6) Other injuries resulting in significant blood loss or shock reduce blood flow to extremities and predispose the extremities to cold injury.

(7) Studies show blacks are more vulnerable than whites to cold injuries.

(8) Starvation or semistarvation predisposes to cold injury.

(9) Any drug or medication that affects peripheral circulation or sweating can lead to cold injury.

(10) Alcohol dilates the peripheral blood vessels causing body heat loss, which increases the dangers of hypothermia and frostbite.

(11) Heat injuries, as strange as it sounds, may occur even in extreme cold due to overdressing and overexertion. When this happens, the body temperature regulating mechanism is damaged and the patient can rapidly develop hypothermia leading to death.

b. Clinical manifestations.

(1) Symptoms during exposure.

(a) The lack of warning symptoms emphasizes the insidious nature of cold injury.

(b) There may be tingling, stinging, or at most a dull aching of the affected part followed by numbness.

(c) The skin briefly may appear red and then becomes pale or waxy white. At this stage the part may feel "like a block of wood." If freezing has occurred, the tissue appears "dead white" and is hard or even brittle with complete lack of sensation and movement.

(2) Differentiation. Terms such as chilblain, trench foot (immersion foot), and frostbite are only used to describe how the injury occurred. After rewarming, the tissue injury, which is largely the result of vascular damage, is similar in all forms of cold injury. The major variable is the degree (severity) of injury. Early evaluation of the degree of cold injury is extremely difficult even to the most experienced doctor. Definitive classification of severity into first, second, third, and fourth degree is possible only in retrospect.

(a) First degree. After rewarming, the skin becomes mottled, red, hot, and dry. The skin blanches poorly on pressure and capillary filling is sluggish or absent. There is frequently intense itching or burning and a later deep-seated ache. Swelling begins within 3 hours and may persist for 10 days or more if patient remains on duty, but usually disappears in less than 5 days if patient is kept at bed rest. Peeling of the superficial layers of the skin may begin within 5-10 days after the injury and last for a month.

(b) Second degree. After rewarming, the skin becomes deep red, hot, and dry. Light touch and position sense are frequently absent. Blisters and even huge blebs may appear within 6-12 hours and may extend

nearly to the tips of the involved digits. These blebs are a valuable sign identifying the injury as second degree. They dry forming black eschars within 10-24 days; the eschars gradually separate revealing intact skin that is thin, soft, poorly keratinized, and easily traumatized. During rewarming there may be a tingling and burning sensation that increases in intensity to a deep aching and burning sensation. This pain may increase to the point the patient will require medication.

(c) Third degree. Necrosis of skin and cutaneous tissue. Vesicles may be present but they contain blood, are smaller, and do not extend to the tip of the involved digits. Edema of the entire involved area (entire hand or foot) usually appears in an average of 6 days. Most patients have a period of anesthesia lasting from 5-17 days followed by burning, aching, throbbing, or shooting pains lasting for months and recurring during exposure to cold sometimes for the rest of their lives. The skin overlaying the injury forms a black, hard, dry eschar that eventually separates exposing underlying granulation tissue. Healing occurs in an average of 68 days. Trauma and infection due to injury other than cold may result in extensive tissue loss, systemic infection, and even wet gangrene requiring emergency amputation of the affected part.

(d) Fourth degree. Complete necrosis of the entire thickness of the part including bone, resulting in loss of the entire injured part. Upon rewarming the skin may turn deep red, purple, or mottled and cyanotic. In some cases edema develops rapidly reaching a maximum within 6-12 hours; the area may show no significant increase in volume, but rapidly progresses to dry gangrene and mummification. In other cases edema develops slowly and is more pronounced, and the eschar formation is not evident until 2-3 weeks after rewarming. The line of demarcation becomes apparent in an average of 36 days but it takes 60-80 days to extend down to the bone. Usually there is no feeling in the injured area for 3-13 days, then ghost pain begins that may become severe.

(3) Early diagnosis and prognostic signs. As previously pointed out, classification of cold injuries as to degree is a retrospective diagnosis. In the early stages (first 48-72 hours) after rewarming, you can only differentiate between superficial (loss of skin or less) or deep (loss of skin and tissue) cold injuries.

(a) Signs of superficial cold injury.

1. Early development of large, clear blebs extending to tips of the digits.

2. Rapid return of sensations.

3. Return of normal (warm) temperature in injured area.

4. Pink or mildly erythematous skin color that blanches rapidly.

(b) Signs of deep cold injury.

1. Hard, white, cold, and insensitive.

2. Absence of edema.

3. Dark hemorrhagic blebs or lack of blebs or blisters.

4. Early mummification.

5. Systemic signs of tissue necrosis (fever, tachycardia, prostration).

6. Superimposed trauma.

7. Cyanotic or dark red skin color that does not blanch on pressure.

c. Treatment. Because of the progressive nature of cold injuries, the earlier they are detected and treatment started the better.

(1) Individual. A fairly reliable symptom of incipient frostbite of fingers, toes, and exposed skin is the sudden and complete cessation of the sensation of cold or discomfort in the part, often followed by a pleasant feeling of warmth. Prompt and immediate care will usually prevent the development of a more serious cold injury. The part must be rewarmed immediately. To rewarm an ear, nose, or cheek, remove your glove and hold (do not rub) your warm hand against the part until it is rewarmed, then protect the area with a scarf or ear flaps, etc. Fingers can be warmed by placing them under the clothes against the skin of the abdomen or the armpit. Toes can be rewarmed by holding them against a companion's chest or abdomen under his outer clothing.

(2) Initial or emergency treatment. The patient should be restricted from his usual duties or activities until the severity of the injury can be evaluated. All constricting items of clothing (boots, socks, gloves) should be removed from the injury site, and the area must be protected from further cold injury by blankets or available loose clothing. Smoking, drinking of alcohol, and use of medications (salves, ointments) on affected area are prohibited. Do not drain blisters; cover them with loose dry dressing. Give plenty of hot liquids to the patient (soup, coffee, tea, etc.). If a lower extremity is involved, treat the patient as a litter patient with the affected limb level or slightly elevated. If travel by foot is the only means of evacuation, do not thaw frostbitten feet until the patient reaches an aid station and medical help. Once the patient has reached an area of shelter (aid station, hospital) if freezing has occurred and the affected tissue is still frozen, it should be thawed as rapidly as possible in water 104-109°F. (40-42°C.). Thawing is determined by return of sensations (usually), return of color (frequently dark red or purple), and the observation that the tissue is soft. Under no circumstances should snow, ice water, grease, massage, walking, or dry heat be used. Warming above 98°F. (37°C.) is not recommended for nonfreezing cold injuries. Cold injury is no contraindication for narcotics or other pain medications, but accompanying injuries may govern the choice of medications. Tetanus toxoid booster should be given. Prophylatic antibiotics should not be used, but if an infection develops, suitable antibiotics should be started.

(3) Definitive treatment. Absolute bed rest is mandatory for any cold injury involving the feet. Debridement should be postponed until the eschar is completely formed, which in fourth degree cold injuries can take 60-80 days to extend to the bone. Patience, understanding, and constant encouragement are essential to good results.

d. Hypothermia--lowering of the body temperature below normal.

(1) Usually caused by exposure (atmospheric or immersion) to prolonged or extreme cold. Immersion in water 48°F. for 1 hour will usually lower body temperature enough to cause death, but hypothermia in cold environments can be caused by unconsiousness due to wounds, disease, alcohol, etc., in individuals who are inadequately protected.

(2) When the internal body temperature is about 95°F., there is a breakdown of the temperature control centers and the body cannot produce enough heat to maintain temperature balance. Further decline of body temperature is quite rapid. Death usually occurs by the time the internal body temperature has reached 80°F.

(3) Symptoms.

(a) As the body temperature drops, the patient may become delirious, drowsy, or comatose; the skin is pale and cold. Respirations may be markedly reduced in frequency and so shallow that casual observation may fail to note any respiratory movement. The pulse and blood pressure become difficult to take or even unobtainable. Pupils become unreactive to light but usually not dilated, and the patient becomes unresponsive to painful stimuli. The tissue becomes semirigid and passive movements are difficult. Death usually follows due to cardiac arrest or ventricular fibrillation.

(4) Treatment. The primary intent is to raise the body temperature.

(a) First aid. If patient is wet, strip and dry him. Heat him by a fire or by stripping and bundling in blankets or sleeping bag to share body heat. If patient is conscious, give plenty of hot fluids (tea, coffee, soup).

(b) Definitive treatment. Patients with moderate or severe hypothermia (core temperature of less than 32°C. (89.6°F.)) often require aggressive rewarming with individualized supportive care. Either heated blankets or warm baths may be used. Bath should be 40-42°C. (104-107.6°F.) with a rate of rewarming of 1-2°C. per hour. The patient must be closely monitored as active external warming may cause marked peripheral dilatation that predisposes to ventricular fibrillation and hypovolemic shock. CPR may be required. An IV should be initiated as soon as possible and urinary output closely monitored. Metabolic acidosis, pneumonia, renal failure, and ventricular fibrillation may occur even several days after an apparently successful resuscitation and restoration of body temperature. Because of this the patient's vital signs should be closely monitored for several days after rewarming. With proper early care 50-70 percent of moderate to severe hypothermia cases can be saved.

e. Snowblindness.

(1) Cause/definition. The eye is sensitive to ultraviolet radiation just as the skin is. In areas of unbroken ice or snow, approximately 75 percent of the incident ultraviolet radiation is reflected so that the eyes are exposed to reflected as well as direct rays from the sun. The eyes can be exposed to excessive ultraviolet radiation even on grey, overcast days or in forested areas. Such excessive exposure can result in sunburn of the tissues comprising the surface of the eye, as well

as the retina, producing snowblindness.

(2) Signs/symptoms.

(a) Symptoms may not be apparent until as much as 8 to 12 hours after exposure.

(b) Initially, the eyes feel irritated and dry, but as time passes, the eyes feel as though they are full of sand. Blinking and moving the eyes becomes extremely painful, and even exposure to light may cause discomfort. Redness of eyes and excessive tearing may occur. The eyelids are usually red, swollen, and difficult to open.

(3) Complications. A mild case of snowblindness may completely disable an individual for several days; however, in the more severe cases, the damage to the eye may be permanent.

(4) Treatment.

(a) A mild case of snowblindness will heal spontaneously in a few days; however, the pain may be quite severe if the injury is not treated.

(b) Cold compresses and a lightproof bandage should be applied in order to relieve pain.

(c) If available, an opthalmic ointment should be applied hourly, not only to provide relief from pain but also to lessen the inflammatory reaction and course of the injury.

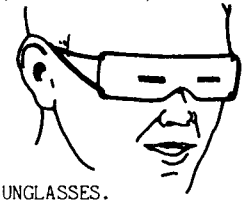
(d) The individual SHOULD NOT rub his eyes.

(e) Local anesthetic agents SHOULD NOT be used. These agents rapidly lose their effectiveness when applied to the eyes, and they may further damage the eye surface.

(5) Prevention.

(a) Snowblindness can be prevented by the consistent use of proper goggles or sunglasses when in areas of unbroken ice or snow. These glasses should be large and curved or have side covers to block reflected light coming from below and from the sides.

(b) If sunglasses or goggles are broken or lost, an emergency pair should be made from a thin piece of leather, cardboard, or other material that is cut the width of the face and provided with horizontal slits over the eyes. The improvised eye protectors can be held in place with string attached to the sides and tied at the back of the head. (See sketch below.)



IMPROVISED SUNGLASSES.

CHAPTER 12

BITES (SNAKE, INSECT, AND ANIMAL)

12-1. SNAKE BITES.

a. Classification of poisonous snakes.

(1) Crotalidae (viperine). Frequently called pit vipers (rattlesnake, moccasins, copperhead, bushmaster, fer-de-lance, habu, Russel's viper, etc.).

(2) Elapidae. This family is composed of coral snakes, kraits, cobras, mambas, asps, and others.

(3) Hydrophidae (sea snakes). All are extremely poisonous and many have more toxic venom than cobras.

(4) Colubridae. This family is represented by the backfanged boomslang.

b. Classification of snake venom. Snake venom is broken into two categories: hemotoxic and neurotoxic. Unfortunately snakes are not just hemotoxic or neurotoxic. They are primarily one or the other, but contain elements of both.

(1) Hemotoxic. Members of the Crotalidae family are primarily hemotoxic with the following substances in the venom:

(a) Thrombase. Action mainly at the site of the bite, causing local thrombosis, gangrene, and intravascular clotting.

(b) Hemorrhagin. This is the predominant substance in the venom, causing lysis of the capillary cells with resultant leakage into the tissue. This starts locally and then becomes generalized. Convulsions due to small hemorrhages in the brain sometimes occur.

(c) Anticoagulin. Causes a breaking down of proteins in the fibrin network of the clot.

(2) Neurotoxic. Members of the Elapidae, Colubridae, and Hydrophidae families are primarily neurotoxic with the following substances in the venom:

(a) Neurotoxin. Has paralytic effect on the respiratory center and the 9th, 10th, 11th, and 12th pairs of cranial nerves.

(b) Hemolysin. Found in some varieties; causes lysis of blood cells.

(c) Cardiotoxin. Causes toxic cardiac arrest.

c. Diagnosis of snakebite.

(1) Crotalidae. Symptoms are very marked and onset is rapid.

(a) Tissue swelling at site of bite, gradually spreading to surrounding area. Swelling begins within 3 minutes and may continue for an

hour with enough severity to burst the skin.

(b) Excruciating pain at site of bite.

(c) Often presence of fang marks.

(d) Bleeding from major organs that may show up as blood in the urine.

(e) Destruction of blood cells and other tissue cells.

(f) Severe headache and thirst.

(g) A marked fall of B.P. with a corresponding rise in pulse.

(h) Bleeding into surrounding tissue.

NOTE: Death may occur within 24-48 hours if bite is serious and untreated. Even with proper treatment, there is grave danger of loss of a portion of the extremities.

(2) Elapidae and Colubridae. Symptoms not as marked and onset is usually slower than Crotalidae.

(a) Impairment of circulation with irregular heartbeat, drop in B.P., weakness, and exhaustion terminating in shock.

(b) Severe headache, dizziness, blurred vision, hearing difficulty, confusion, and unconsciousness.

(c) Muscular incoordination and muscular twitching.

(d) Respiratory difficulty leading to respiratory paralysis.

(e) Irregularities of skin sensations such as tingling, parasthesia, excessive perspiration, and numbness of the lips and the soles of the feet.

(f) Chills and often rapid onset of a fever.

(g) Nausea, vomiting, and diarrhea.

(3) Hydrophidae. Neurotoxic, bite is usually painless, does not swell, and often there is no clue that treatment should be started. Poisoning should be suspected in those who have been in coastal waters frequented by sea snakes within 1-2 hours before complaining of:

(a) Muscular aches, pains, and stiffness of movement.

(b) Pain on passive movement of arm, thigh, neck, or trunk muscles.

(c) Urine becomes reddish brown within 3 hours.

(d) There is a consistent appearance of neurotoxic symptoms as outlined in Elapidae diagnosis.

NOTE: Without treatment death usually occurs within 12-24 hours.

d. Treatment of snakebite.

(1) General treatment for all snakebites.

(a) Kill the snake if possible, but do not spend more than a few minutes and avoid overexertion in the attempt. Try not to crush the head as this is the primary source of exact species identification.

(b) Have patient lie down. Immediately immobilize injured part. Keep patient warm and quiet.

(c) Tetanus booster and antibiotics are indicated.

(d) Symptomatic treatment as necessary.

(2) Treatment for Crotalidae (viperine).

(a) General treatment for all snakebites (see para d (1) above).

(b) If bitten on a large area of the body (i.e., thigh, calf, forearm, etc.), make an incision 1/8 to 1/4 inch deep along or in the direction of muscle (not across the tissue) through the puncture sites. (Do not make an X cut. Do not cut into joints, tendons, etc.) Then suction using a mechanical device; use mouth only as a last resort and then only if you have no cavities, cuts, or sores in the mouth.

NOTE: Incision and suction should not be used if antivenom can be given within 1 hour or if 1 hour or more has elapsed since the bite.

(c) Do not use a tourniquet, constricting bands, or cold packs.

(d) Do not allow the patient to eat any food or to drink alcohol.

(e) Have patient drink small amounts of water at frequent intervals.

(f) Initiate IV D5W, normal saline, or Ringer's to help prevent hemolytic shock.

(g) Administer specific antivenom, if available and species is known, or polyvalent antivenom as soon as possible.

1. Inject 0.1 cc. subcutaneously and observe patient for 15 minutes for symptoms of allergy such as itching, swelling, and redness at injection site.

2. If patient is not allergic, inject the antivenom in one dose IM at a site other than the bite area.

3. If patient is allergic to the antivenom, but there is no doubt that he has an effective bite by a very dangerous species and will surely die without the antivenom, inject divided doses of 1.0 cc. IM very slowly. Be prepared to treat anaphylactic reactions should they

occur.

(h) Use morphine or other suitable pain relievers as necessary.

(3) Treatment for Elapidae and Colubridae (boomslang).

(a) Apply a tourniquet around the affected limb, over a single bone (above the knee or elbow) proximal to the bite, tight enough to stop arterial flow. This tourniquet should be released for 30 seconds every 20 minutes to allow fresh blood into the affected area.

(b) Administer antivenom using the same rules and precautions as for viperine bites.

(c) General treatment for all snakebites (see para d (1) above).

(d) Do not use morphine or any drugs that cause respiratory depression.

(4) Treatment for Hydrophidae (sea snakes).

(a) Antivenom is the only treatment other than symptomatic care.

(b) Incision and suction are of no value.

12-2. INSECT AND SPIDER BITES.

a. Insect bites. Of all deaths per year due to bites, 40% are caused by insect bites compared to 33% for snake bites, 18% for spider bites, and 9% for animal bites.

(1) Bees, wasp, hornets, yellow jackets, and ants. Most of this group sting their victims and often leave the stingers and venom sac embedded in the skin. The stinger should be removed immediately to prevent more venom from entering the victim. Toxins from this group are similar to the venom of viperine snakes in having a hemolysin factor, but their primary effect seems to be the strong histamine they contain.

(a) Symptoms. Stinging, burning sensation with swelling. This swelling, when caused by stings around the head and neck, may be severe enough to impair the airway.

(b) Treatment.

1. Apply a paste of baking soda (sodium bicarbonate) or apply strong household ammonia to reduce discomfort. Infiltration of lidocaine into sting area often helps.

2. In severe cases give Benadryl 4 mg. per kg. IV stat. with 10 cc. of 10% calcium gluconate. Inject 2 to 4 cc. fairly fast until patient has a burning sensation in the tongue, palm, or soles of his feet. Then slow the injection of the remainder to avoid flushing.

3. If patient is allergic to the venom, treat the anaphylactic reaction.

(2) Centipedes, millipedes, and caterpillars.

(a) Centipedes are venomous with hollow fangs like snakes. If bitten, the patient will have immediate severe pain followed by redness and swelling. Sometimes necrosis with ulcer formation may occur.

(b) Millipedes secrete a toxin by glands in the body. When the fluid touches the skin, it produces burning and itching.

(c) Many caterpillars have hollow venom-containing hairs on their bodies. If these hairs contact the skin, they cause severe burning pain, redness, swelling, and necrosis of tissue. Scotch tape on the sting is effective in removing the broken off hairs from the skin.

(d) Treatment. Very similar to that of bee, wasp, and hornet stings. Antihistamines, ice, and pain medication are helpful. Treat anaphylactic reactions.

b. Spider and scorpion bites.

(1) Black widow spider. Only the female bites and has a neurotoxic venom. Identified by red hourglass on abdomen.

(a) Symptoms. Initial pain is not severe, but severe local pain rapidly develops. The pain gradually spreads over the entire body and settles in the abdomen and legs. Abdominal cramps and progressive abdominal rigidity may occur. Weakness, tremors, sweating, salivation, nausea, vomiting, and/or a rash may occur. Anaphylactic reactions can occur. Symptoms usually begin to regress after several hours and are usually gone in a few days.

(b) Treatment.

1. Calcium gluconate 10 cc. of 10% solution IM or injected slowly IV.

2. Robaxin 10 cc. given slowly IV over a 5-10 min period followed by 10 cc. in 250 cc. of D5W in IV drip over 4 hours.

3. Patients under 14 and over 50 should receive the specific antivenom if they are not allergic to horse serum.

4. Supportive care as necessary, tetanus booster, antibiotics, etc.

(2) Brown house spider (recluse). Identified by dark brown violin on the back of a small light brown spider.

(a) Symptoms. There is no pain or so little pain that most of the time the patient is not aware he is bitten. A few hours later a painful, red area with a mottled cyanotic center appears. A macular rash sometimes occurs. Necrosis does not occur in all bites, but usually after 2-3 days there is an area of discoloration that does not blanch with finger pressure. The area turns dark and mummified in a week or two. The margins separate and the eschar falls off leaving an open ulcer. Secondary infection and regional lymphadenopathy usually become evident at this stage. Many times the patient is unaware of any cause for the ulcer. The outstanding feature of brown recluse bites is the ulcer does not heal, but

persists for weeks or months. Physical exam reveals a hard indurated area of skin and superficial fascia with undermined edges.

In many cases there is a systemic reaction, in addition to the ulcer, that is serious and may lead to death. The systemic reactions occur chiefly in children and are marked by fever, chills, joint pain, splenomegaly, vomiting, and a generalized rash. These systemic reactions may occur at any time as long as the ulcer is present.

(b) Treatment. There is no antivenom for brown recluse bites. It is necessary to excise all the indurated skin and fascia before healing will start. If the ulcer is not excised, it may continue to grow until it is several inches in diameter.

Tetanus prophylaxis and antibiotics are necessary to control secondary infection. Cortisone will arrest the systemic reaction but will not affect the ulcer. Anaphylactic reactions may also occur and must be managed.

(3) .Scorpions. All are poisonous to a greater or lesser degree. Fortunately none of the very poisonous varieties are found in the US, but deaths have been reported due to scorpion stings in the US.

(a) Symptoms. There are two different reactions depending on the species.

1. Severe local reaction only, with pain and swelling around area of sting. Possible prickly sensation around the mouth and a thick feeling tongue.

2. Severe systemic reaction with little or no visible local reaction. Local pain and hyperesthesia may be present. Systemic reaction includes respiratory difficulties, thick feeling tongue, tetanuslike body spasm, drooling, gastric distention, double vision, blindness, involuntary rapid movement of the eyeball, involuntary urination and defecation, hypertension, and heart failure. Death is rare, occurring mainly in children or adults with hypertension.

(b) Treatment.

1. DO NOT give morphine or morphine derivatives, including Demerol, because it has a synergistic effect with scorpion venom. Effective pain relief can be obtained by specific nerve blocks using lidocaine.

2. Ice packs or cold water helps slow spread of toxin and relieve pain.

3. Tetanus prophylaxis and antibiotics are indicated.

4. Specific antivenoms are available for the more toxic varieties.

5. Symptomatic care.

12-3. ANIMAL BITES.

a. Animal bites themselves are not usually serious. The main

problem is the diseases that can be transmitted by the bites. Number one among these is rabies.

b. Protective measures for bites.

(1) Capture and isolate animal for 8-10 days.

(a) An animal that is rabid should show unmistakable signs of rabies within 8 days.

(b) If the animal dies, cut off the head, freeze it, and ship it frozen to the nearest laboratory having facilities for rabies determination.

(2) Bites from animals that can't be captured and isolated should be considered as rabid, and patient should receive antirabies vaccine.

c. Treatment.

(1) All bites must be promptly and thoroughly cleaned with soap, Betadine or hexachlorophene and water. Then apply either 40-70% alcohol, tincture of iodine, or 1:10,000 benzalkonium chloride directly into the bite. This mechanical cleansing and disinfecting has been credited with blocking many cases of rabies as well as lessening the chances of other types of infection.

(2) Antitetanus prophylaxis is indicated.

(3) Avoid suturing or cauterizing the wound; use delayed secondary closure if at all possible.

(4) If suturing is absolutely necessary, infiltrate 50% of the first dose of rabies vaccine into wound area.

(5) Immediate judgment as to the advisability of administering antirabies serum is required. Take into account the circumstances of the bite and prevalance of rabies in the area.

(6) Symptomatic treatment as required.

CHAPTER 13

OVERDOSE AND POISONING

13-1. GENERAL PRINCIPLES.

a. ABCs in severe OD.

b. Gradation of coma.

Stage 0: Asleep but arousable.
Stage I: Comatose - withdraws from painful stimuli.
Stage II: Reflexes present - does not withdraw from painful stimuli.
Stage III: Reflexes absent - no respiratory or circulatory depression.
Stage IV: Reflexes absent - respiratory depression and/or circulatory collapse.

c. Removal of toxic drug.

(1) Emesis.

(a) More effective than lavage in awake patient.

(b) Ipecac: 15-30-45 cc. of syrup (not tincture) followed by 1 liter of warm water. Activity helps stimulate vomiting. Never leave ipecac in (cardio toxic).

(c) Apomorphine: 0.1 mg./kg. IV or SQ. Vigorous vomiting difficult to control. May act as respiratory depressant: counteracted by Narcan. (Rarely indicated.)

(2) Lavage.

(a) Use normal saline - 200-300 cc. per pass, or 10 cc./kg. per pass.

(b) Y tube set up with 28-32 Fr. Ewald Tube, larger for undissolved tabs.

(c) If no gag use endotrachial tube to protect airway.

(3) Contraindications to emesis and/or lavage.

(a) Caustics.

(b) Hydrocarbon ingestions that are not potentially lethal.

(4) Activated charcoal.

(a) Dose = 30-50 grams.

(b) Will deactivate ipecac. (Wait until patient stops vomiting.)

(c) Can be given prior to and after lavage.

(5) Purgatives.

(a) Castor oil or mineral oil: contraindicated in pesticide poisoning and hydrocarbon ingestion. Helpful in glutethimide.

(b) Mag citrate: 10 oz. Contraindicated in renal failure.

(c) Sodium sulfate: 250 mg./kg. diluted 1:2 or 1:4.

(6) Forced diuresis with alkalinization of urine.

(a) Especially useful in barbiturate and ASA OD.

(b) Protocol.

500 cc. D_5W x 2
500 cc. NS + 20 KCl at 200-500 cc./hr.
1 Amp $HaHCO_3$/2-3 liters.
Lasix 40 mg. IV q. 4-6 h.
Foley for urethral drainage.

(7) Forced diuresis with acidification of urine.

(a) Especially useful in amphetamine and PCP ODs.

(b) Protocol.

1. Ascorbic acid: 1-2 gm in 500 cc. NS IV q.i.d. and/or

2. Ammonium chloride: 2.75 mEq./kg./dose in 60 cc. saline P.O q.6h. until urine pH less than 5.

(8) Peritoneal and hemodialysis.

13-2. HYPNOTICS.

a. Barbiturates signs and symptoms.

(1) C.N.S. depression and/or agitation (coma is major toxicity).

(2) Temperature usually decreased.

(3) Pulse usually normal, may be increased.

(4) B.P. normal or decreased.

(5) Respirations normal or decreased (if increased, consider aspiration).

(6) "Barb burns" - skin necrosis at high dosage.

(7) Reflexes normal or decreased.

(8) Myocardial toxicity at high dosages.

(9) Ataxia, nystagmus, and vertigo in early OD.

b. Treatment for barbiturates.

(1) General OD protocol.

(2) Hemodialysis.

c. Nonbarbiturate hypnotics.

Glutethimide (Doriden)
Methyprylon (Noludar)
Quaalude (methaqualone)

(1) Signs and symptoms.

(a) Lethargy.

(b) Mydriasis.

(c) Decreased B.P.

(d) Flaccidity (except Quaalude).*
* Quaalude, Placidyl, or phencyclidine can cause hyperreflexia in the presence of coma.

(e) Respiratory depression.

(2) Treatment as for general OD: Glutethimide especially lethal because of enterohepatic circ and varying levels of coma. Use duodenal NG and cholestyramine.

13-3. C.N.S. STIMULANTS.

Amphetamines and cocaine.

a. Signs and symptoms.

(1) Agitation.

(2) Euphoria.

(3) Tachycardia.

(4) Hypertension.

(5) Hyperpyrexia.

(6) Cramps.

(7) Hallucinations (auditory and visual).

(8) Convulsions and coma.

(9) Perforated nasal septum suggests cocaine.

(10) Toxicity begins at 20-25 mg./kg. for amphetamines.

b. Treatment.

(1) Support and calming, avoid stimulation.

(2) Dialysis for amphetamines, but not for cocaine.

(3) Thorazine 25 mg. or valium 5 mg. as calmative.

(4) Acidify urine for amphetamines.

13-4. ANTICHOLINERGICS.

a. Examples.

(1) Atropine.

(2) Scopolamine.

(3) Belladonnna alkaloids.

(4) Tricyclic antidepressants.

(5) Phenothiazines.

(6) Antihistaminics.

(7) Antispasmodics (Pro-Banthine).

(8) Antiparkinsonian agent.

(9) Toxic plants.

(a) Jimsonweed.

(b) Morning glory seed.

(c) Deadly nightshade.

(d) Certain mushrooms.

(e) Potato leaves and sprouts.

b. Signs and symptoms.

(1) Tachycardia.

(2) Dry flushed skin.

(3) Mydriasis.

(4) Dry mouth.

(5) Nausea and vomiting.

(6) Urinary retention.

(7) Increased intraocular pressure.

(8) Confusion.

(9) Disorientation.

(10) Bizarre behavior.

(11) Paranoia.

(12) Hallucinations.

(13) Hyperpyrexia.

(14) Hypotension.

(15) Convulsions.

c. Treatment.

(1) Cooling.

(2) Support.

(3) Sedation.

(4) Physostigmine in severe OD.

13-5. MAJOR AND MINOR TRANQUILIZERS.

Major - Phenothiazines.
Minor - Librium, valium, Placidyl, meprobamate, etc...

a. Signs and symptoms.

(1) Hypotension.

(2) Lethargy.

(3) Respiratory depression.

(4) Coma.

(5) Seizures.

(6) Extrapyramidal ------ Phenothiazines.

(7) Loss of temperature control.

b. Treatment.

(1) Support and general OD measures.

(2) Gastric lavage or emesis.

(3) Control shock as necessary.

(4) Use barbiturates <u>cautiously</u> for control of convulsions.

13-6. USE OF PHYSOSTIGMINE.

Anticholinesterase - i.e., cholinergic drug counteracts

anticholinergic drugs.

a. Actions.

(1) Pupillary constriction.

(2) Contracts bronchioles, gut, and bladder.

(3) Stimulates salivation and sweating.

(4) Slows heart.

(5) Increases muscle contraction.

(6) C.N.S. stimulant.

b. Definite antidotes for:

(1) Atropine and belladonna alkaloids (jimsonweed).

(2) Tricyclic antidepressants.

c. Contraindications.

(1) Mechanical G.I. obstruction - absolute.

(2) Mechanical GU obstruction - absolute.

(3) Asthma - relative.

(4) COPD - relative.

(5) ASCVD - relative.

(6) Should be used to counteract life-threatening central and peripheral anticholinergic signs, or to reverse coma in the presence of those signs.

d. Central anticholinergic signs.

(1) Short-term memory loss.

(2) Disorientation.

(3) Hallucinations (visual and auditory).

(4) Anxiety and agitation.

(5) Psychosis.

(6) Coma.

(7) Twitchy and jerky movements.

(8) Pyramidal signs (Hyperreflexia, hypertonus, clonus).

e. Peripheral anticholinergic signs.

(1) Tachycardia.

(2) Mydriasis with decreased light reflex.

(3) Dry mucous membranes.

(4) Flushed dry skin.

(5) Decreased bowel sounds.

(6) Urinary retention.

(7) Hyperpyrexia.

f. Physostigmine dosage.

(1) 2 mg. in 10 cc. IV over 2 minutes repeated x 2 at 5-10 minutes.

(2) 2 mg. undiluted IM q. 20 minutes.

(3) 0.5 mg. in children.

g. Dangers of Physostigmine.

(1) Precipitate cholinergic crisis and seizures.

(2) Central (medullary) and peripheral respiratory collapse.

(3) Excess salivary and tracheobronchial secretions.

(4) Bronchospasm and laryngospasm.

(5) Muscle twitching, fasciculations, and paralysis.

(6) Reversible - treat with atropine 1/2 physo dose IM.

h. Indications.

(1) Known OD on belladonna alkaloids or tricyclics who are in marked distress.

(2) Anticholinergic findings and hypertension, hallucinations, coma, convulsions, and dysrhythmias.

13-7. HYDROCARBONS.

a. 1 cc./kg. of hydrocarbon, or less if chlorinated or metal containing solvent. Leads to C.N.S. or respiratory depression.

b. If depression has occurred - intubate with cuffed tube gastric lavage and use general OD procedures.

c. Potentially lethal dose but no depression, use ipecac.

d. If less than 1/2 cc./kg., cathartic only - no emesis or lavage.

e. Activated charcoal is useless.

f. Don't use oil-based cathartics. May lead to lipoid pneumonias.

g. X ray initially and at 6-12 hours if evidence of aspiration (patient coughing).

h. Epinephrine contraindicated (dysrhythmias).

i. Steroids are questionably useful.

j. No antibiotics unless infection documented.

13-8. SALICYLATES: ASA AND METHYL SALICYLATE (OIL OF WINTERGREEN).

a. Symptoms at 100 mg./kg. (2 grains/lb toxic - 3-4 gr/lb fatal).

b. Is severe to fatal at 250-400 mg./kg.

c. Adult ingestion of 10 grams should be considered serious.

d. Symptoms.

(1) Early: Headache, dizziness, tinnitus, blurred vision, confusion, lethargy, diaphoresis, thirst, nausea and vomiting, diarrhea, abdominal pain, hyperpnea.

(2) Severe: Restlessness, incoherence, vertigo, tremor, diplopia, delirium, convulsions, coma, fever to 106°F. in children, respiratory alkalosis in adults and children, occasionally hypoglycemia.

e. Treatment.

(1) Basic principles, especially activated charcoal.

(2) Correct acid-base and electrolyte disturbances.

(3) Alkalinization of urine.

13-9. ACETAMINOPHEN OVERDOSAGE (PARACETAMOL).

a. In adult dosages over 5 grams, should be admitted for observations.

b. 10 grams ingestion is associated with severe hepatotoxicity and death.

c. Peak plasma levels usually occur in 40-120 minutes, may be prolonged in OD.

d. Metabolized in liver and conjugated with glucuronide, sulfate, cysteine, and mercapturic acid.

e. Effects are potentiated by drugs utilizing the same conjugating systems, i.e. ETOH and barbiturates.

f. Plasma levels 4 hours after ingestion.

(1) Greater than 300 mg./ml. leads to liver damage.

(2) Less than 120 mg./ml. does not lead to liver damage.

g. Manifestations.

(1) Pallor.

(2) Nausea, vomiting, and diarrhea.

(3) Hepatotoxicity.

(a) May not appear clinically for 2-6 days.

(b) Right upper quadrant pain.

(c) Increased liver enzymes.

(d) Jaundice.

(e) Encephalopathy.

h. Treatment.

(1) Emesis early.

(2) Charcoal early questionably useful.

(3) N-acetylcysteamine (Mucomist) - not yet approved.

(a) 140 mg./kg. P.O. loading dose.

(b) 70 mg./kg. P.O. q.4h. x 17 doses.

13-10. CAUSTICS.

a. Examples.

(1) Draino, Liquid Plummer, etc...

(2) Liquid and dry bleach.

(3) Acetest tablets.

(4) Lye.

(5) Any strong acid or base.

b. Treatment.

(1) No emesis.

(2) No lavage.

(3) Dilute with cool water.

(4) Do not neutralize with weak acid or base (generates heat and gas).

(5) All caustic ingestions should be scoped and followed by G.I.

or ENT.

c. N.B. - Absence of intra or perioral burns does not preclude ingestion.

13-11. ORGANOPHOSPHATES.

a. Examples. (insecticides replacing DDT).

(1) Organophosphate insecticides - chlorathion, Di-Captan, ethion, disyston, malathion, methyl parathion, parathion, phosphamidon, etc...

(2) Carbamate insecticides - cabofuran, dimeton, mexecarbate, etc...

b. Signs and symptoms consist of increased cholinergic stimulation.

(1) Bronchoconstriction with increased bronchial secretion and pulmonary edema.

(2) Nausea and vomiting.

(3) Abdominal cramps and diarrhea.

(4) Increased sweating.

(5) Increased salivation and lacrimation.

(6) Bradycardia and hypotension.

(7) Miosis (may be unilateral) and blurred vision.

(8) Urinary incontinence.

(9) Muscle cramps, weakness, fasciculations, and arreflexia.

(10) Headache.

(11) Restlessness.

(12) Convulsions.

(13) Coma.

c. Treatment.

(1) Mild intoxication requires removal from further exposure.

(2) Severe poisoning.

(a) Support (including removal of secretions).

(b) Decontamination.

(c) Administration of anticholinergic agent.

1. Atropine - 2-4 mg. IV slow q. 15 minutes.

2. PAM pralidoxime - only useful if administered within 24 hours (1 gram over several minutes, children 10-12 mg./kg.)

13-12. CYANIDE.

a. Sources of cyanide (amygdalin).

(1) Nitroprusside therapy.

(2) Laetrile.

(3) Various fruit pits such as peach, apricot, chokecherry, plum, lima beans, apple seeds, various grasses.

(4) Synthetic rubber.

(5) Some fumigant gasses.

(6) Photographic chemicals.

(7) Salts for electroplating, gold and silver extraction, metal cleaning, dehairing hides.

(8) Cyanamide (for fertilizing).

b. Signs and symptoms of cyanide poisoning.

(1) Smell of bitter almonds.

(2) Headache.

(3) Vertigo and faintness. C.N.S.

(4) Excitability.

(5) Opisthotonus and trismus.

(6) Convulsions and coma.

(7) Burning tongue.

(8) Salivation. Oral ingestion

(9) Nausea.

(10) Hypertension with bradycardia and blocks early.

(11) Hypotension, tachycardia, and cardiovascular collapse - late.

c. Treatment.

(1) 100% O_2 by bag and mask.

(2) Cyanide antidote kit:

(a) Amy nitrite by inhalation followed by,

(b) Sodium nitrite IV 10 ml. over 4 minutes (0.2 ml./kg. IV for peds), and

(c) 50 ml. of 25% sodium thiosulfate IV (1 ml./kg. IV for peds).

13-13. ARSENIC.

a. Sources.

(1) Herbicides.

(2) Insecticides.

(3) Rodenticides.

(4) Fungicides.

(5) Paints.

(6) Tanning agents.

(7) Some veterinary medicines.

b. Signs and symptoms (levels in urine, hair, and nails).

(1) Smell of garlic.

(2) Mees' lines in nails after 2-3 weeks.

(3) Hyperpyrexia.

(4) Tremor and convulsions.

(5) Coma.

(6) Nausea, vomiting, and diarrhea.

(7) Liver and kidney damage.

(8) Polyneuropathy.

(9) G.I. complaints may preceed neuropathy, and by 2-3 weeks in acute cases.

c. Treatment.

(1) If acute, lavage or emesis.

(2) General support.

(3) Dimercaprol (BAL). (See chapter 14.)

(4) Follow urine arsenic levels.

13-14. METHEMOGLOBINEMIA.

a. Hemoglobin with Fe in the ferric state.

b. Incapable of reversible transport.

c. Causative agents (oxydizers).

(1) Inorganic agents:
Copper chlorates, chromates, nitrates, nitrites.

(2) Drugs:
Acetanilid, phenacetin, PAS, sulfonamide, lidocaine, benzocaine, nitroglycerin, chloroquine, menthol, primaquine, etc...

(3) Miscellaneous:
Alloxan, naphtalene, quinones anilines nitrosobenzene.

d. Symptoms.

(1) Cyanosis with level 727 (slate gray).

(2) Normal pO2·

(3) Dyspnea.

(4) Tachycardia.

(5) Stupor and coma.

(6) Nonspecific symptoms.

(7) Blood looks like Hershey's Chocolate.

e. Treatment.

(1) Support.

(2) 02·

(3) Time.

(4) Methylene blue - 1-2 mg./kg. IV slowly or 3-5 mg./kg. orally.

13-15. CARBON MONOXIDE.

a. Sources.

(1) Car exhausts.

(2) Poorly ventilated furnaces and fireplaces.

(3) Smoke inhalation.

b. Symptoms.

(1) Depends on level - become toxic over 20%, 10% if found in smoking.

(2) Cherry red lips and mucous membranes.

(3) Headache.

(4) Muscle weakness.

(5) Palpitations.

(6) Dizziness.

(7) Confusion.

(8) Coma and death.

c. Treatment.

100% O_2 under hyperbaric conditions for at least 1 hour; maintain body warmth and blood pressure. Give 50 ml. 50% glucose p.r.n. for brain edema.

13-16. LEAD POISONING (PLUMBISM).

a. Sources.

(1) Lead based paint.

(2) Cooking utensils - ceramic or earthenware with lead glaze.

(3) Plumbing.

(4) Stills.

(5) Industrial exposure:

(a) Smelters.

(b) Battery workers.

(c) Painters (auto).

(d) Demolition experts.

b. Signs and symptoms.

(1) Chronic.

(a) Vague aches and pains (may mimic other neuropathies).

(b) Wrist and ankle drop.

(c) Chronic nephritis.

(d) Anemia Hg 10 with basophilic stippling.

(e) Increased urinary aminolevulinic acid (ALA).

(f) Abdominal lead and leadlines on X ray.

(2) Acute.

(a) Metallic taste.

(b) Anorexia.

(c) Constipation and vomiting.

(d) Abdominal pain.

(e) Personality changes.

(f) Lethargy.

(g) Clumsiness.

(h) Ataxia.

(i) Convulsions.

(j) Coma.

(k) Anemia with basophilic stippling.

(l) Increased urinary ALA and coproporphyrins.

(m) Abdominal lead and lead lines on X ray.

(n) Blackstools (leadsulfide).

c. Treatment.

(1) Isolation from further contaminations.

(2) General OD principles.

(3) General support.

(4) Chelation.

(a) BAL 4 mg./kg. IM over 4 h.

(b) EDTA 50-75 mg./kg./day 1 m. 5-7 day courses.

(c) D-penicillamine 20-40 mg./kg./day as outpatient.

13-17. IRON INTOXICATION.

a. Source is usually prescription or over the counter Fe containing preparations.

b. Toxic to lethal dose of 150-300 mg./kg.

c. Signs and symptoms.

(1) Stage I.

(a) 30-120 minutes.

(b) Vomiting and diarrhea (may be bloody).

(2) Stage II.

(a) 6-12 hours.

(b) Latent or improvement.

(3) Stage III.

(a) 18-72 hours.

(b) Cardiovascular collapse.

(c) Coma.

(d) Convulsions.

(e) Coagulation defects.

(f) Hyperpyrexia.

(g) Metabolic acidosis.

(h) Liver failure.

(4) Stage IV.

(a) 4-6 weeks.

(b) Pyloric stenosis and G.I. scarring.

d. Treatment.

(1) Lavage with 5% sodium bicarbonate or emesis if pills still visible on abdominal X rays.

(2) Baseline blood work to include serum Fe.

(3) Chelation only in inpatients (Desferal).

(a) Used in all definitely lethal doses, 300 mg./kg.

(b) Serum Fe TIBC.

(c) Coma, convulsions, and shock.

(d) 40 mg./kg. Desferal 1., to be repeated at expanding time intervals. Do not exceed 1 gm IM initially followed by 500 mg. q.4h. x 2. Never exceed 6 gm in 27 hours.

(e) Urine will turn brown.

(f) Supportive treatment.

CHAPTER 14

NUCLEAR, BIOLOGICAL, CHEMICAL (NBC)

14-1. NUCLEAR. The major problems resulting from nuclear detonation are mass casualties and the destruction of medical care facilities.

a. Of the injured survivors, about one-third of the injuries will be caused by blast effects, one-third by thermal effects (burns), and one-third by both blast and thermal effects. Some in each of these groups will receive radiation from initial radiation and/or radioactive fallout.

(1) Initial treatment for these casualties will be first aid or self aid until they can get to or be brought to a functioning medical care facility.

(2) Once the casualties reach a treatment facility, they must be classified as to the type and urgency of treatment required so appropriate priorities can be established for treatment, evacuation, and hospitalization. This classification is known as triage. Triage is divided into four categories:

(a) Minimal (priority I). Requires only minor treatment, usually on an ambulatory or outpatient basis. This group includes small lacerations and contusions, closed fractures of small bones, second degree burns of less than 20% of the body that are not life threatening, and moderate psychological disorders.

(b) Immediate (priority II). Individuals with life-threatening conditions or moderate injuries that are treatable with a minimum expenditure of time, personnel, and supplies, and who have a good chance of recovery. Conditions include hemorrhage from an accessible site, rapidly correctable mechanical defects (sucking chest wound, respiratory obstruction or distress), severe crushing wounds and incomplete amputations, and open fractures of major bones.

(c) Delayed (priority III). After emergency care, these individuals may have definitive treatment delayed without significant jeopardy to recovery. These include moderate lacerations without bleeding, closed fractures of major bones, noncritical central nervous system (C.N.S.) injuries, and second degree burns between 20 and 40% of the body surface.

(d) Expectant (priority IV). Individuals requiring extensive therapy beyond our means and to the detriment of others. They receive emergency, comfort, and conservative care to the maximum extent possible. Included are critical respiratory and C.N.S. injuries, penetrating abdominal wounds, multiple severe injuries, and severe burns of over 40% of the body surface.

b. Burn and blast injuries are covered in chapter 10.

c. Radiation injuries (acute radiation syndrome) are directly related to the dose (amount) of radiation received. The dose is accumulative.

(1) 50-200 rad. Approximately 6 hours after exposure the individual may have no symptoms to transient mild headaches. There may be

a slight decrease in the ability to conduct normal duties. Less than 5% of individuals in the upper part of the exposure range will require hospitalization. Average hospital stay will be 45-60 days with no deaths.

(2) 200-500 rad. Approximately 4-6 hours after exposure, individuals will experience headaches, malaise, nausea, and vomiting. Symptoms are not relieved by antiemetics in the upper exposure range. Individual can perform routine tasks but any activity requiring moderate to heavy exertion will be hampered for 6-20 hours. After this period, individuals will appear to recover and enter a latent period of 17-21 days. If individual has received 300 rads or more, large quantities of hair will be lost between 12-18 days after exposure. Following the latent stage, symptoms will return, requiring 90% of the personnel to be hospitalized for 60-90 days. Probably less than 5% of those at the lower dose range will die, the percentage increasing toward the upper end of the dose range.

(3) 500-1000 rad. Approximately 1-4 hours after exposure, severe and prolonged nausea and vomiting develop that are difficult to control. Diarrhea and fever develop early in individuals in the upper part of the exposure range. Simple routine tasks can be performed by individuals in the lower dose range. Significant incapacitation is seen in the upper ranges. Initial symptoms last for more than 24 hours, then go into a latent period lasting 7-10 days. Following the latent stage the symptoms return requiring 100% of the individuals to be hospitalized. Of those in the lower range 50% will die, the percentage increasing toward the upper range. All deaths occur within 45 days. The survivors require 90-120 days hospitalization before recovery.

(4) 1000 rad or more. Less than 1 hour after exposure individuals develop severe vomiting, diarrhea, and prostration. There is no latent period. All require hospitalization and die within 30 days.

d. Treatment for radiation exposure includes washing individual thoroughly to remove any radioactive contamination, symptomatic treatment, and prevention of secondary infections.

14-2. BIOLOGICAL WARFARE (BW).

a. Biological agents are divided into two main classes:

(1) Living organisms such as bacteria, viruses, rickettsiae, and fungi.

(2) Poisonous products or toxins produced by living organisms.

b. The most practical method of initiating infection in BW is through the dispersal of agents as minute, airborne particles (aerosols) over a target where they may be inhaled. An aerosol may be effective for some time after delivery, as it will be deposited on clothing, equipment, and soil. When the clothing is used later, or dust is stirred up, personnel may be subject to a "secondary" aerosol.

c. Agents may be able to use portals of entry into the body other than the respiratory tract. Individuals may be infected by ingestion of contaminated food and water or even by direct contact with the skin or mucous membranes through abraided or broken skin.

d. Early warning, immediate detection, and rapid identification of

the agent used in a BW attack are of primary importance.

(1) Early warning can sometimes be supplied by intelligence sources, but early warning is not always available.

(2) Immediate detection can be by seeing a plane spraying or by bombs, shells, or mines producing dense clouds near your area. Immediate detection may not occur; for example, in the case of sabotage or an attack launched a considerable distance upwind from you, the first indication may be the appearance of casualties.

(3) Rapid identification of the biological agent. Due to the concentration and/or portal of entry (respiratory tract), there may be a more rapid onset and wide variances to normal symptoms of even common diseases. This can make diagnosis and treatment extremely difficult. Clinical samples should be collected from the first casualties and sent to the nearest laboratory, if possible.

e. Individual protection prior to and during a BW attack.

(1) Maintain body in the best possible physical condition.

(2) If a BW attack is detected,

(a) Use mask.

(b) Button clothing and tie clothing with string or extra shoelaces at the wrists and ankles. If special protective clothing is available, put it on.

(c) Put on gloves, if available.

(d) While in the contaminated area, practice the procedures outlined above.

(e) Upon leaving the area, decontaminate to the extent the situation permits. If bathing facilities and fresh clothing are available, carefully remove contaminated clothing and thoroughly wash the body and protective mask in soap and water prior to removing the mask. Then don fresh clothing. Give special attention to decontamination and treatment of skin lesions.

f. Group protection. The best protection is a pressurized shelter using filtered, forced air. A building or shelter without this feature provides only limited protection from aerosols. Eventually, microorganisms will penetrate through cracks and constitute a respiratory hazard unless the protective mask is worn. As in the case of individual protective measures, utilization of shelters depends upon early warning.

(1) Protection of food and water depends entirely on following good preventive medicine and veterinary procedures (see chapters 20 and 21). Some biological agents cannot be destroyed by normal water purification techniques. When biological agents are known to have been used, all drinking water must be boiled in addition to normal water treatment measures.

(2) Proper hygiene and sanitation procedures must be used (see chapter 20).

(3) Immunizations must be kept current.

g. Pending identification of the agent, measures should be taken to prevent epidemics as soon as possible after initial exposure. These measures include isolation, quarantine, and restriction of personnel movement. After identification of the agent and if it is not capable of producing an epidemic, these restrictive measures can be relaxed.

14-3. CHEMICAL WARFARE. This section deals mainly with the diagnosis and treatment of specific chemical agents.

a. General considerations.

(1) Chemical casualties who have not been decontaminated may endanger unprotected personnel. Handlers of these patients should wear protective masks, impermeable protective gloves, and chemical protective clothing. If conditions permit, an aid station should be established upwind from the contaminated area. The casualties should be undressed and washed thoroughly, downwind of the aid station, before being brought into the aid station.

(2) Most chemical agents can poison food and water. Suspect food and water must be examined by chemical test procedures, if available. If testing equipment is not available, avoid using the water or food, or get an animal to eat or drink a portion and watch it for at least an hour for adverse reactions. Canned foodstuff is completely protected, but the container might be contaminated and should be washed thoroughly with copious amounts of uncontaminated water. Avoid foodstuff that is not well sealed from vapor and liquid agents.

b. Nerve agents.

(1) Nerve agents are among the deadliest chemical agents. They include (GA) tabun, (GB) sarin, (GO) soman, and VX. They are colorless to light brown liquids, some of which are volatile. They are usually odorless, except for GA which has a faint, sweet fruity odor. Toxic liquids are tasteless. They range from nonpersistent to persistent. Nerve agents may be absorbed through the skin, respiratory tract, gastrointestinal tract, and the eyes. However, significant absorption through the skin takes a period of minutes and prompt decontamination is imperative.

(2) Effects of nerve agents.

Site of Action	Signs and Symptoms Following Local Exposure
Pupils	Constricted (miosis), marked, usually maximal (pinpoint), sometimes unequal.
Ciliary body	Frontal headache, eye pain on focusing, slight dimness of vision, occasional nausea and vomiting.
Conjunctivae	Hyperemia.
Nasal mucous membranes	Rhinorrhea, hyperemia.

Bronchial tree	Tightness in chest, sometimes with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, cough.
	Following Systemic Absorption
Bronchial tree	Tightness in chest, with prolonged wheezing expiration suggestive of bronchoconstriction or increased secretion, dyspnea, slight pain in chest, increased bronchial secretion, cough, pulmonary edema, cyanosis.
Gastrointestinal	Anorexia, nausea, vomiting, abdominal cramps, epigastric and substernal tightness (cardiospasm) with "heartburn" and eructation, diarrhea, tenesmus, involuntary defecation.
Sweat glands	Increased sweating.
Salivary glands	Increased salivation.
Lacrimal glands	Increased lacrimation.
Heart	Slight bradycardia.
Pupils	Slight miosis, occasionally unequal, later maximal miosis (pinpoint).
Ciliary body	Blurring of vision.
Bladder	Frequency, involuntary micturition.
Striated muscle	Easy fatigue, mild weakness, muscular twitching, fasciculations, cramps, generalized weakness, including muscles of respiration, with dyspnea and cyanosis.
Sympathetic ganglia	Pallor, occasional elevation of blood pressure.
Central Nervous System	Giddiness, tension, anxiety, jitteriness, restlessness, emotional lability, excessive dreaming, insomnia, nightmares, headaches, tremor, withdrawal and depression, drowsiness, difficulty concentrating, slowness on recall, confusion, slurred speech, ataxia, generalized weakness, coma, with absence of reflexes, Cheyne-Stokes respirations, convulsions, depression of respiratory and circulatory centers, with dyspnea cyanosis, and fall in blood pressure.

(a) Nerve agents are cumulative in their effect. Daily exposure to concentrations of a nerve agent insufficient to cause symptoms following a single exposure may result in symptoms following several days of exposure.

(b) Suspect nerve agent poisoning if any of the following occurs:

1. A feeling of tightness or constriction in the chest.

2. Unexplained runny nose.

3. Difficulty in breathing, either on inhaling or exhaling.

4. Small, pinpoint-size pupils seen in a mirror or in the eyes of individuals in the vicinity. (On exposure to vapor or aerosol, the pupils become pinpointed immediately. If the nerve agent is absorbed through the skin only or by ingestion of contaminated food or water, the pinpointing will be delayed or even absent.)

5. A drawing, slightly painful sensation in the eyes or unexplained dimness of vision occurring with pinpoint pupils.

(3) Treatment of nerve agent poisoning.

(a) Immediately don the protective mask and hood at the first indication of any chemical attack.

(b) Immediately remove any liquid contamination. (If a drop or a splash of liquid nerve agent gets in the eyes, immediately irrigate the eyes with copious amounts of water).

(c) Administer 2 mg. of atropine as soon as any local or systemic nerve agent symptoms are noted. (Do not give for preventive purposes before exposure to nerve agent.) If the patient has mild symptoms due to nerve agents, the IM injection of 2 mg. atropine should be repeated at 20-minute intervals, 10-minute intervals if moderate to severe symptoms are present, or until signs of atropinization (dry mouth, blurry near vision) are achieved: A mild degree of atropinization should be maintained for at least 24 hours by IM or oral administration of 1-2 mg. of atropine every 1/2 to 4 hours.

1. Atropine can be given IM, IV, or orally. Atropine given IM requires about 8 minutes before effects are noticed. Given IV, effects begin within 1 minute and reach maximum effect within 6 minutes. Atropine tablets require 20 minutes before effects are felt and 50 minutes before maximum effect takes place.

2. Atropine effects include dryness of the mouth and throat, with slight difficulty in swallowing. Patient may have a feeling of warmth, slight flushing, rapid pulse, some hesitancy of urination, and an occasional desire to belch. Pupils may be dilated slightly but react to light and near vision is blurred. Some individuals may experience mild drowsiness, slowness of memory, and the feeling his body movements are slow. Further doses of 2 mg. of atropine intensify the symptoms and prolong the effects. Effects of one to two 2 mg. injections last 3-5

hours, and the effects of four injections given at close intervals last 6-12 hours.

3. Patients with moderately severe nerve agent symptoms have increased tolerance for atropine, so fairly large doses may be administered before signs of atropinization appear.

(d) Severe nerve agent exposure may rapidly cause unconsciousness, muscular paralysis, and cessation of breathing. If this occurs, artificial respiration is required along with the atropine injections. If the patient is in severe respiratory distress or is convulsing, 4-6 mg. of atropine should be injected IV. If relief does not occur and bronchial secretions and salivation does not decrease, give 2 mg. of atropine q. 3-8 minutes until relief occurs and secretions diminish. In severe nerve agent poisoning the effect of each injection of atropine may be transient, lasting only 3-10 minutes. This requires the patient to be monitored closely and atropine repeated as needed. A mild atropinization should be maintained for at least 48 hours.

(e) Pralidoximine chloride (2-Pam Cl or Protopam Cl) can be used to increase the effectiveness of therapy in nerve agent poisoning. 2-Pam Cl reduces the time during which artificial respiration is required. Dosage: 2-Pam Cl, 1 gm in 100 ml. of sterile water, normal saline, or 5% dextrose and water; IV slowly over 15-30 minutes.

c. Blister agents (vesicants).

(1) Vesicants act on the eyes, lungs, and skin causing burns and blisters. They damage the respiratory tract when inhaled and cause vomiting and diarrhea when absorbed. Most vesicants are insidious in action causing little or no pain at the time of exposure. Lewisite and phosgene oxime cause immediate pain on contact. Vesicants poison food and water and make other supplies dangerous to handle. The severity of a chemical burn is directly related to the concentration of the agent and the duration of contact with the skin.

(2) Mustard (HD). An oily liquid ranging from colorless when pure to dark brown. Mustard is heavier than water, but small droplets float on water surfaces. It is only slightly soluble in water, but freely soluble in fats, oils, gasoline, kerosene, acetone, and alcohol. These solvents do not destroy mustard. Mustard is a persistent agent. It smells like garlic or horseradish. Even very small repeated exposures to mustard are cumulative in effect.

(a) Symptoms.

1. Eye effects. In a single exposure the eye is the most vulnerable. In mild exposure there is a latent period of 4-12 hours followed by tearing and a gritty feeling in the eyes. The conjunctiva and lids become red and edematous. Heavy exposure has a latent period of 1-3 hours followed by severe irritation and lesions. Ischemic necrosis of the conjunctivae, edema, photophobia, and blepharospasm may obstruct vision. Dense corneal opacification with deep ulceration and vascularization may occur.

2. Effects on the skin. Latent period depends on weather conditions. In hot, humid weather latency may be as short as 1 hour; in cool weather after mild vapor exposure, latency may be several

days. Normal latency is 6-12 hours. Initial symptom is erythema, resembling sunburn, followed by multiple pinpoint lesions that enlarge and form the typical blisters. The blisters are usually large, domed, thin walled, superficial, translucent, yellowish, and surrounded by erythema. The blister fluid is clear, thin, and straw colored at first; later it is yellowish and tending to coagulate. Liquid contaminaton of the skin usually results in a ring of vesicles around a gray-white area that does not blister.

3. Respiratory effects. Develop slowly taking several days to reach maximal severity. Symptoms begin with hoarseness (may progress to loss of voice). A cough, which is worse at night, appears early and later becomes productive. Fever, dyspnea, and moist rales may develop into bronchopneumonia.

4. Systemic and gastrointestinal effects. Ingestion of contaminated food or water produces nausea, vomiting, abdominal pain, diarrhea, and prostration. Skin exposure may cause malaise, vomiting, and fever appearing about the same time as the erythema. With severe exposure, symptoms may be so marked as to result in prostration. Severe systemic mustard poisoning may present C.N.S. symptoms such as cerebral depression, bradycardia, and cardiac irregularities.

(b) Treatment of mustard agent.

1. Immediately don protective mask and hood.

2. Immediately remove any liquid contamination. (Speed in decontamination of the eye is absolutely essential. Rinse the eye with copious amounts of water.)

3. After rinsing the eyes, apply a steroid antibiotic eye ointment. Patients with severe photophobia and blepharospasm should have 1 drop of 1% atropic sulfate instilled in the eye t.i.d. The eyes must not be bandaged or the lids allowed to stick together.

4. All blisters should be opened and the fluid drained with care, as the fluid itself may be irritating and cause secondary erythema and blisters. Area should be cleansed with tap water or saline and burn cream applied (10% Sulfamylon burn cream).

5. Respiratory tract injuries are treated symptomatically with steam inhalation.

6. The biggest part of the treatment is symptomatic and preventing or treating secondary infections.

(3) Nitrogen mustard (HN). Oily, colorless, pale yellow liquids; some have a faint fishy odor, while others are odorless.

(a) Effects of HN on the eye. Slight to moderate exposure produces symptoms within 20 minutes that wax and wane until they become persistent about 2 1/2 hours later and reach their maximum in 8-10 hours. Severe exposure causes immediate symptoms that progress for 24 hours. In general the symptoms are the same as mustard, but more severe and requiring intensive and early treatment.

(b) The most specific effect is in the blood and lymph

tissue. .Within 5-10 days after exposure, anemia may develop and W.B.C. can fall to less than 500.

(c) Treatment of HN is generally the same as for mustard, but frequent checks of the hematocrit and W.B.C. are necessary.

(4) Arsenical vesicants. Colorless to brown liquids, soluble in most organic solvents but poorly soluble in water. They are generally more volatile than mustard and have `ruity to geraniumlike odors. Vapors are unlikely to cause significant injuries. Liquids will cause severe burns of the skin and eyes and can gradually penetrate rubber and most impermeable fabrics.

(a) Liquid agent symptoms:

1. Effects on the eye include immediate pain and blepharospasm on contact. Edema follows rapidly, causing the eye to close within an hour. Severe exposure can cause permanent injury or blindness.

2. Effects on the skin are more severe than those from liquid mustard. Stinging pain is usually felt 10 to 20 seconds after contact. The pain increases in severity with penetration and in a few minutes becomes a deep aching pain. About 5 minutes after contact a gray area of dead skin appears resembling that seen in corrosive burns. Erythema resembles that caused by mustard, but is accompanied by more pain. Itching and irritation persist for about 24 hours whether or not a blister develops. Blisters are often well developed in 12 hours and are painful at first (mustard blisters are relatively painless). The pain lessens in 48-72 hours.

3. Respiratory effects are similar to those produced by mustard agent. Systemic absorption of arsenicals causes a change in the capillary permeability. This can permit sufficient fluid loss from the blood stream to cause hemoconcentration, shock, and death. Acute systemic poisoning from large skin burns causes pulmonary edema, diarrhea, restlessness, weakness, subnormal temperature, and low blood pressure.

(b) Treatment. Mask and immediately decontaminate any liquid agent (flush contaminated eyes with copious amounts of water). Treatment for the eyes is mainly symptomatic; atropine sulfate opthalmic ointment or atropine drops should be used in conjunction with an opthalmic antibiotic ointment. BAL ointment should be applied to areas of skin contamination BEFORE any blistering appears and remain on the area for at least 5 minutes. (BAL ointment occasionally causes stinging, itching, or urticarial wheals. Frequent application on the same area of skin causes mild dermatitis.) Treatment of blisters is the same as for mustard agents.

(c) Indication for systemic treatment.

1. Cough with dyspnea and frothy sputum, which may be blood tinged, and other signs of pulmonary edema.

2. Skin contamination the size of the palm of the hand or larger in which there is gray or dead-white blanching of the skin or in which erythema develops over the area within 30 minutes.

(d) Two types of treatment may be used.

1. Local neutralization by liberal application of BAL ointment that must remain on the affected area. Remove any other protective ointment before applying BAL ointment.

2. IM injection of dimercaprol (BAL) 10% solution in oil. For mild to moderate poisoning give 2.5 mg./kg. (1.5 ml./60 kg.) q.4h. x 2 days, then one injection q.12h. the third day, fourth to the tenth day give one injection once or twice a day. For severe poisoning give 3 mg./kg. (1.8 ml./60 kg.) q.4h. x 2 days, third day give one injection q.6h., fourth through fourteenth day one injection twice a day. Up to 5 mg./kg. can be given in severe cases.

Symptoms caused by BAL include dryness of the mouth and throat, mild tearing, slight reddening of the eyes, feeling of constriction in the throat, burning sensation of the lips, generalized muscular aching, abdominal pain, mild restlessness and sweating of the hands, apprehension, mild nausea and vomiting on eating, and a transient rise in blood pressure. Symptoms start 15-30 minutes after injection and last about 30 minutes. Unless they are severe or prolonged, they are not a contraindication for continuing therapy.

(5) Phosgene oxime (CX). A powerful irritant that is especially effective as a liquid. It has a disagreeable penetrating odor and is readily soluble in water.

(a) Phosgene oxime is violently irritating to mucous membranes of the eyes and nose. Even low concentrations can cause tearing. Any exposure to liquid or vapor that produces pain will also produce skin necrosis at the site of contact. The area becomes blanched and is surrounded by an erythematous ring within 30 seconds. This is followed by a wheal within 30 minutes. Within 24 hours the original blanched area acquires a brown pigmentation. An eschar forms at about 1 week and sloughs at about 3 weeks. Itching may be present throughout the entire course of healing. Healing may take 2 months or more.

(b) Decontamination is not effective after pain starts, but the contaminated area should be flushed with copious amounts of water to remove any agent that has not yet reacted with the tissue. Treat as any other ulcerated necrotic skin lesion, plus supportive care, as needed.

(6) Mixtures of blister agents. Arsenical vesicants are often mixed with mustard to confuse and make diagnosis difficult. These mixtures do not produce more severe lesions than either agent alone.

d. Choking agents (lung irritants). Best known of these agents is phosgene, a colorless gas with an odor of new mown hay, grass, or green corn. Phosgene is a nonpersistent agent that is broken down rapidly by water (fog, rain, heavy vegetation).

(1) During and immediately after exposure there is likely to be coughing, choking, a feeling of tightness in the chest, nausea, occasionally vomiting, headache, and tearing. There may be an initial slowing of the pulse followed by an increase. These symptoms may not appear, but if they do, a latent period follows that commonly lasts 2-24 hours but may be shorter. Following the latent period, signs and symptoms of pulmonary edema develop. They start with rapid shallow breathing, painful cough, and cyanosis. Nausea and vomiting may appear. As edema progresses, discomfort, apprehension, and dyspnea increase and frothy

sputum is raised. Rales and rhonchi are heard throughout the chest, and breath sounds are diminished. Patient may develop a shocklike state, with clammy skin, low blood pressure, and feeble rapid heart action.

(2) Protective mask offers adequate protection. Treatment is rest, oxygen therapy, cautious use of IV therapy, codeine for cough control, and antibiotic therapy to prevent secondary infections. Do not use expectorants or atropine. Patients who survive the first 48 hours usually recover.

e. Blood agents (cyanides). Hydrocyanic acid (AC) and cyanogen chloride (CK) are the important agents in this group. AC is a colorless, highly volatile liquid that is highly soluble and stable in water. It has a faint odor like peach kernels or bitter almonds. It is nonpersistent. CK is a colorless, highly volatile liquid that is slightly soluble in water but dissolves readily in organic solvents. It has a pungent, biting odor and is nonpersistent.

(1) Symptoms produced by AC depend upon the concentration of the agent and duration of exposure. Typically, either death occurs rapidly or recovery takes place within a few minutes after removal from the toxic area. Moderate exposure causes vertigo, nausea, and headaches followed by convulsions and coma. Severe exposure causes an increase in the depth of respiration within a few seconds, cessation of regular respiration within 1 minute, occasional shallow gasps, and finally cessation of heart action within a few minutes.

(2) Symptoms of CK are immediate intense irritation of the nose, throat, and eyes, with coughing, tightness in the chest, and tearing. The patient may become dizzy and increasingly dyspneic. Unconsciousness is followed by failing respiration and death within a few minutes. Convulsions, retching, and involuntary urination and defecation may occur. If effects are not fatal, signs and symptoms of pulmonary edema may develop: persistent cough with frothy sputum, rales in the chest, severe dyspnea, and marked cyanosis.

(3) Mask immediately. Crush 2 ampules of amyl nitrite and insert into the region of the eyelenses of the mask. Repeat every 4-5 minutes until a total of 8 ampules have been used or normal respiration has resumed. Give artificial respiration if patient is not breathing. Second step in emergency treatment is IV administration of 10 ml. of 3% sodium nitrite over a 1-minute period plus 50 ml. of a 25% solution of sodium thiosulfate given slowly IV. Further treatment is symptomatic. Recovery from AC or CK may disclose residual C.N.S. damage with irrationality, altered reflexes, and unsteady gait that may last for weeks, months, or be permanent.

f. Incapacitating agents. Agents producing a temporary disabling condition that persists for hours to days after exposure to the agent has ceased.

(1) Signs and symptoms produced by incapacitating agents.

Signs and symptoms	Possible etiology
Restlessness, dizziness, or giddiness; failure to obey orders, confusion, erratic behavior, stumbling or staggering, vomiting.	Anticholinergics, indoles, cannabinols. Anxiety reaction. Other intoxications (e.g., alcohol, bromides, barbiturates, lead).
Dryness of mouth, tachycardia at rest, elevated temperature, flushing of face; blurred vision, pupillary dilation; slurred or nonsensical speech, hallucinatory behavior, disrobing, mumbling and picking behavior, stupor and coma.	Anticholinergics (e.g., BZ).
Inappropriate smiling or laughing, irrational fear, distractability, difficulty expressing self, perceptual distortions; labile increase in pupil size, heart rate, B.P.; stomach cramps and vomiting may occur.	Indoles (e.g., LSD). (Schizophrenic psychosis may mimic in some respects.)
Euphoric, relaxed, unconcerned daydreaming attitude, easy laughter; hypotension and dizziness on sudden standing.	Cannabinols (e.g., marihuana).
Tremor, clinging or pleading, crying; clear answers, decrease in disturbance with reassurance; history of nervousness or immaturity, phobias.	Anxiety reaction.

(2) General treatment consists of close observation, restraint and confinement as required, supportive care with fluids, and appropriate clothing. Underlying medical problems should be treated as needed. If the specific agent can be identified, treat appropriately.

g. Vomiting agents. Produce a strong pepperlike irritation in the upper respiratory tract with irritation and tearing of the eyes. Principal agents of this group are DA, DM, and DC that are usually dispersed by heat as fine particulate smoke. When concentrated, DM smoke is canary yellow,

DA and DC smokes are white. All are colorless when diluted with air.

(1) Vomiting agents produce a feeling of pain and fullness in the nose and sinuses accompanied by severe headache, intense burning in the throat, tightness and pain in the chest, irritation and tearing of the eyes, uncontrollable coughing, violent and persistent sneezing, runny nose, and ropy saliva flow from the mouth. Nausea and vomiting are prominent and mental depression may occur. Onset of symptoms may be delayed several minutes after exposure. Mild exposure symptoms resemble those of a severe cold.

(2) Most individuals recover promptly after removal from the contaminated area. The few that don't can receive symptomatic relief by inhaling chloroform vapors either directly from a bottle or by pouring a few drops into the cupped palms and breathing. Chloroform is inhaled until the symptoms or irritation subside and is repeated when the symptoms become severe again. Do not use to the point of anesthesia. Aspirin may be given to relieve the headache and general discomfort.

h. Irritant agents (CS, CN, CA). CS has a pungent pepperlike odor. It is faster acting, about 10 times more potent, and less toxic than CN. CN has an apple blossom odor, and CA has a sour fruit odor.

(1) With CS there is marked burning pain and tearing of the eyes, runny nose, coughing, and dyspnea. Following heavy exposure there may be nausea and vomiting. Warm moist skin, especially on the face, neck, ears, and body folds, is susceptible to irritation by CS. CS causes a stinging burning sensation even at moderately low concentration. Higher concentrations may cause an irritant dermatitis with edema and (rarely) blisters. An increase in the stinging is usually noted upon leaving the contaminated area, but usually subsides in 5-10 minutes. CN and CA cause basically the same reaction as CS, but require a higher concentration and are more toxic.

(2) When it is safe to do so, remove mask and blot eyes. Do not rub the eyes. Flush the eyes with copious amounts of water. To prevent skin reaction, rinse the body with water or 5 or 10% sodium bicarbonate in water. Delayed erythema (irritant dermatitis) may be treated with a bland shake lotion. Most persons affected by irritant agents require no medical treatment. Severe reactions of the eyes or skin may take days or weeks to heal depending on their severity.

CHAPTER 15

SHOCK

15-1. SHOCK. A breakdown of effective circulation at the cellular level and/or failure of the peripheral circulatory system. Failure causes tissue perfusion to become inadaquate to feed the body cells.

15-2. CAUSES. Different types of shock result from different kinds of failure in the circulatory system.

a. Hypovolemic shock (peripheral resistance). Caused by hemorrhage, burns (loss of plasma), and/or decreased body water and electrolytes (vomiting and bowel obstruction or diarrhea).

b. Cardiogenic shock (resistance to heart muscle, pump failure). Caused by myocardial infarction, cardiac arrhythmias, and congestive heart failure. Pump failure of the heart causes a reduction in blood flow and then blood backs up behind the heart, causing an increase in venous pressure.

c. Neurogenic shock. Caused by spinal injuries, spinal anesthesia, trauma, manipulation of fractures, and some head wounds. There is a failure of arterial resistance with a pooling of blood in dilated capillary vessels. Cardiac activity increases in an attempt to increase the blood volume to preserve capillary pressure.

d. Septic shock. Caused by wound infection, peritonitis, meningitis, etc. Septic shock is usually caused by gram-negative bacteria causing a septicemia (invasion of the blood by pathogenic bacteria or their toxins). Hypovolemia develops as a result of pooling of blood in the capillaries and a loss of fluid from the vascular space as a result of a generalized increase in capillary permeability. There is also a possibility of a direct toxic effect on the heart with depressed cardiac function. Peripheral resistance is usually decreased but can increase as shock worsens.

e. Anaphylactic shock. Acute, often explosive, systemic reaction characterized by urticaria, respiratory distress, vascular collapse, and occasionally vomiting, cramps, diarrhea.

(1) Signs and symptoms. Usually occurs in 1-15 minutes; patient becomes agitated, uneasy, and flushed. Palpitations, paresthesia, pruritus, throbbing in the ears, coughing, sneezing and difficulty in breathing, followed by dizziness, disorientation, collapse, coma, and death.

(2) Treatment.

(a) Epinephrine solution, 1:1,000, 0.4-1 ml., IV stat. Repeat every 5-10 min p.r.n.

(b) Recumbent position, elevate legs, establish airway (tracheostomy if necessary).

(c) Diphenhydramine HCl, 5-20 mg. IV p.r.n.

(d) Aminophylline solution, 250-500 mg. IV slowly for severe asthma without shock.

(e) IV fluids to correct hypovolemia if present.

(f) Hydrocortisone sodium succinate, 100-250 mg. IV over 30 seconds for hypotension control if needed.

(g) +for injected antigen (e.g., vaccination) a constricting band (rarely a tourniquet) should be applied proximal to the injection site. An additional 0.1-0.2 ml. epinephrine (1:1,000) may be injected into the site to reduce systemic absorption.

(h) +oxygen should be utilized if available at 4-6 liters/minute.

(i) Definitive care p.r.n. and continue observation for 24 hours.

15-3. SIGNS AND SYMPTOMS OF SHOCK.

a. Shock chart.

Degree of Shock	Blood Volume Loss	B.P. (approx)	Pulse	Temp	Color	Circu-lation	Thirst	Mental State
Mild	Up to 20%	Up to 20% Increase	Normal	Cool	Pale	Slowing	Normal	Clear Distinct
Moderate	20-40%	Decrease 20-40%	In-creased	Cool	Pale	Slowing	Definite	Clear With Apathy
Severe	40% or More	Decreased Below 40%	Weak to Absent	Cold	Ashen to Cya-notic	Very Sluggish	Severe	Apathetic to Comatose

b. The patient appears anxious and looks tired. Later he appears apathetic or exhausted. If bleeding continues, the patient will go into a coma and die.

c. The skin usually feels cool, is pale and mottled, and nail beds blanch easily.

d. The pulse and blood pressure are not totally accurate.

(1) Decreased blood pressure is always significant.

(2) In a healthy adult, blood pressure may remain normal until large volumes of blood are lost.

(3) Respirations, heart beat, and pulse are usually increased, but this increase may not occur in the prone position. If the patient is in shock and you sit him up, the systolic blood pressure will show a decrease of up to 15 mm. and you will observe an increase of 15 beats or more in the pulse.

15-4. TREATMENT.

a. Hemorrhagic shock - low peripheral vein pressure. You can

expect early collapse of the usual IV routes; venous cutdown may be indicated.

b. Primary therapy for hypovolemic and hemorrhagic shock.

(1) Standard IV fluids listed in order of effectiveness.

(a) Whole blood - administer with crystalloid solutions.

(b) Plasma - administer with crystalloid solutions.

(c) Serum albumin - administer with crystalloid solutions.

(d) Dextran - should administer with crystalloid solutions.

(e) Lactated Ringer's solution (crystalloid).

(f) Normal saline (crystalloid).

(g) D5W - use alone only if nothing else is available (crystalloid).

(2) To insure adequate IV fluids you should monitor the urinary output.

(3) Keep patient warm and dry and place in the shock position unless contraindicated, e.g., head wounds, chest wounds.

(4) Analgesics such as morphine should be given for pain as necessary.

(5) Broad spectrum antibiotic treatment should be started as soon as possible as a prophylaxis for large wounds or burns.

CHAPTER 16

EMERGENCY WAR SURGERY

16-1. PRIORITIES OF TREATMENT.

a. The following priorities for surgical intervention are recommended. Injuries are dealt with on an individual basis.

b. Follow the rule of ABC.

A - Airway. Insure it is clear.

B - Breathing. Insure patient is able to breath, e.g., no sucking chest wounds, etc.

C - Circulation. Insure heart is beating and there is adequate circulating blood volume.

c. First priority.

(1) Asphyxia, respiratory obstruction from mechanical causes, sucking chest wounds, tension pneumothorax, and maxillofacial wounds in which asphyxia exists or is likely to develop.

(2) Shock caused by major external hemorrhage, major internal hemorrhage, visceral injuries, massive muscle damage, major fractures, multiple wounds, and severe burns over 20% of the body.

d. Second priority.

(1) Visceral injuries, including perforations of the gastrointestinal tract, wounds of the biliary and pancreatic systems, wounds of the genitourinary tract, and thoracic wounds without asphyxia.

(2) Vascular injuries requiring repair. All injuries in which the use of a tourniquet is necessary fall into this group.

(3) Closed cerebral injuries with increasing loss of conciousness.

(4) Burns of 20% of certain locations; hands, feet, genitalia, and perineum.

e. Third priority.

(1) Brain and spinal injuries in which decompression is required.

(2) Soft-tissue wounds in which debridement is necessary but in which muscle damage is less than major.

(3) Lesser fractures and dislocations.

(4) Injuries of the eye.

(5) Maxillofacial injuries without asphyxia.

(6) Burns of other locations under 20%.

16-2. SOFT TISSUE INJURIES.

a. In the following surgical procedures we will assume that the medic knows how to prepare a patient for surgery and set up for sterile procedures.

b. The primary objective in the treatment of soft tissue injuries is localization or isolation of the deleterious effects of the injury. To best accomplish this objective, remove all foreign substances and devitalized tissue and maintain an adequate blood supply to the injured part. This can be achieved by a two-step procedure.

(1) Step one is a thorough debridement of the injured area, accomplished as early as possible after the injury (when delay is unavoidable, systemic antibiotics should be started). The wound is left open, with few exceptions, to granulate.

(2) Step two is a delayed primary closure (DPC) within 4-10 days after injury. The wound must be kept clean during this time and antibiotics are usualy indicated. The indication for a DPC is the clean appearance of the wound during this time.

c. Antibiotic wound therapy. Should be started prior to debridement.

(1) Penicillin (aqueous) - 10 million units IV q.8h. x 3 days then reevaluate.

(2) Kanamycin - 500 mg. IM q.12h. x 3 days then reevaluate.

(3) Tetanus toxoid - 0.5 cc. IM or SQ once (test for allergy only if not previously immunized).

d. Wound debridement.

(1) An incision is made in the skin and fascia long enough to give good exposure. Good exposure is required for accurate evaluation. Incisions are made over both the entry and exit wound along the longitudinal axis of extremities (S-shaped crossing joint creases). Avoid making an incision over superficial bones. When excising skin only, cut 2-3 cm. from the wound edge.

(2) Skin, fascia, and muscle should be separated to give adequate exposure. Muscles should be separated into their groups and each muscle group debrided separately.

(3) Distinguishing tissue viability. Use the four Cs: color, consistency, contractility, and circulation; color being the least desirable.

	Viable	Dead or Dying
Color	Bright reddish brown	Dark, cyanotic
Consistency	Springy	Mushy

Contractility	Contracts when pinched or cut	Does not contract when pinched or cut
Circulation	Bleeds when cut	Does not bleed when cut

(4) Steps of debridement. All devitalized muscle must be removed; if not, the chance of infection is greater. It is better to take good muscle tissue and have some deformity than to leave devitalized muscle and have infection. The preferred method of debridement is to cut along one side of a muscle group in strips or in blocks and not piecemeal or in small bunches.

(a) Remove all blood clots, foreign material, and debris from the wound during exploration of the wound with a gloved finger.

(b) Vital structures like major nerves and blood vessels must be protected from damage.

(c) All procedures must be carried out gently with precision and skill.

(d) Major blood vessels must be repaired promptly.

(e) All foreign bodies must be removed, including small detached bone fragments, but time should not be wasted looking for elusive metallic fragments that would require more extensive dissection.

(f) Tendons usually do not require extensive debridement. Trim loose frayed edges and ends. Repair should not be performed during initial treatment.

(g) Hemostasis must be precise.

(h) Repeated irrigations of the wound with physiologic salt solution during the operation will keep the wound clean and free of foreign material. This step cannot be overemphasized.

(i) When debridement is complete, all blood vessels, nerves, and tendons should be covered with soft tissue to prevent drying and maceration.

(j) Joint synovium should be closed or at least the joint capsule. The skin and subcutaneous tissue is left open in any case.

(k) Dependent drainage of deep wounds must be employed.

(l) Liberal fasciotomy of an extremity is often an additional precaution that allows for postoperative swelling. Use when the five Ps are present distal to an injury or wound (pain, pallor, pulselessness, puffiness, and paresthesia).

(m) DO NOT dress the wound with an occlusive dressing, but place a few wide strips of fine-mesh gauze between the walls of the wound; place fluffed gauze in the pocket that is formed, then dress the wound to protect but not constrict.

(n) All wounds will be left open with the exception of

wounds of the face, sucking chest wounds, head wounds, wounds of the joint capsule or synovial membrane, and wounds of the peritoneum.

(o) Immobilization and correct positioning of the injured part promotes healing, and these measures should be used even if no fracture is present.

16-3. VASCULAR INJURIES. Although a vascular injury is extremely serious, you must consider the equipment available, other injuries to the patient, and other casualties.

a. Accurate diagnosis of a vascular injury may not be possible until exploration is undertaken, but the following signs and symptoms may be used as evidence of arterial damage:

(1) Extremity may be pale, waxy, mottled, cyanotic, and cold.

(2) Pulse may be absent, but the presence of a pulse does not rule out arterial injury.

(3) Analgesia, loss of voluntary motion of extremity, muscle spasm or contracture may be present.

(4) External hemorrhage, like bright red spurting blood, may or may not be present.

(5) The affected limb may be larger than the intact limb.

b. There is no set time when a vascular injury must be repaired to insure saving the limb, but the longer the time lag, the greater the failure rate. The best results are obtained within 6 to 10 hours of the injury.

c. You probably will not be able to undertake major vascular repairs, but you should have the equipment to handle arterial lacerations caused by low velocity missiles or sharp instruments.

(1) Clamps of a noncrushing type should be applied to the injured artery, the first proximal to the injury and the second distal.

(2) Keep the artery moist with a saline solution.

(3) All debridement accomplished by the standard technique should be completed before arterial repair is begun.

(4) Run a Fogarty balloon catheter distally in the artery to determine the patency. This will also clear any distal thrombus.

(5) Use a continuous suture of 5-0 or 6-0 synthetic suture with a fine curved, noncutting swaged needle.

(6) Release the clamps and observe for leaks.

(7) Dress the wound as a soft tissue injury.

(8) Keep the extremity at heart level.

(9) Begin active muscle exercises while patient is still in bed.

d. When the muscle tissue is of questionable viability after arterial continuity has been restored, the patient is observed for:

(1) A decrease in urinary output.

(2) Increasing pain toxicity, confusion, and fever.

(3) Increase in pulse rate.

(4) Evidence of clostridial myositis.

e. If this evidence is present, excision of necrotic muscle tissue or early amputation may be called for. It is usually safe to hold off on amputation for up to 5 days until a line of demarcation is established.

16-4. BONE AND JOINT INJURIES.

a. For all open bone and joint injuries, the following principles apply:

(1) Initial determination of the extent of the wound and the structures involved.

(2) Generous extensile incision and removal of foreign material, debridement, and removal of small bone chips.

(3) Arthrotomy.

(4) Vascular repair and fasciotomy.

(5) Wound is left open for delayed primary closure.

(6) Bulky nonocclusive dressing and immobilization of fractures, nonfractures, and joint injuries.

(7) Documentation of everything observed.

b. War wound therapy is indicated in all open bone or joint injuries.

16-5. PERIPHERAL NERVE INJURIES.

a. The field medic does not have the equipment or the expertise to perform nerve repair. Nor is it really necessary.

b. Closed nerve injuries are never surgically explored. Open injuries of nerves are handled as any other soft-tissue injury with the nerve left intact and covered with muscle tissue to prevent exposure.

16-6. AMPUTATIONS.

a. Amputations are performed to save life and are done at the lowest level possible. All attempts should be made to save the knee and elbow joints even if this means having a short stump.

b. Indications for amputation are:

(1) Massive gas gangrene (clostridial myositis).

(2) Overwhelming local infection that endangers life despite antibiotic therapy and surgical measures.

(3) Established death of a limb.

(4) Massive injuries in which structures of a limb are obviously nonviable.

(5) Secondary hemorrhage in the presence of severe infection.

(6) Extremities with severe involvement of skin, muscle, and bone with anesthetic terminus and irreparable nerve damage.

c. Under combat conditions the most acceptable type of amputation is the open circular technique.

(1) A circumferential incision is made through the skin and deep fascia at the lowest viable level. This layer is allowed to retract.

(2) The muscle bundles are exposed and then divided circumferentially at the new level of the skin edge. The muscle bundles will retract promptly exposing the bone.

(3) Upward pressure is placed on the proximal muscle stump and the bone is then transected at a still higher level. The surgical wound will have the appearance of an inverted cone.

(4) Blood vessels are isolated, clamped, and ligated as they are encountered. Bone wax is applied to the open end of the bone to prevent oozing.

(5) Major nerves are transected at the highest level possible.

(6) Never close an amputation primarily.

(7) Cold injuries are not indications for emergency amputation. Wait until the edges demarcate.

d. A layer of fine mesh gauze is placed over the wound and the recess is packed loosely with fluffed gauze. A stockinette is then applied over the stump securing the stockinette above the stump using liquid adhesive. The stump is then wrapped with ace wraps using compression decreasing proximally and 5 to 6 pounds of traction is applied. Continued traction will result in secondary skin closure over the stump.

16-7. REGIONAL INJURIES.

a. Craniocerebral injuries. Serious injuries to the head require more extensive surgery than can be done in the field. There are some expedient measures, however, that can be taken to give the patient a chance. These are:

(1) Prophylactic antibiotic therapy.

(2) Grossly devitalized and contaminated soft tissue and bone should be removed, along with any foreign material, visible on inspection, superficial to the dura. The dura should not be attacked.

(3) Gently irrigate the wound with physiologic salt solution and ligate all bleeding vessels. Gelfoam can be used to control oozing.

(4) If possible, the scalp wound should be loosely approximated to provide temporary coverage.

(5) Sterile petroleum-impregnated gauze should be laid over the wound. A thick gauze dressing should be placed over that and held in place by a bandage.

(6) High priority should be given for evacuation.

(7) Mark the medical record prominently and call attention to the incompleteness of treatment.

b. Spinal cord injuries.

(1) The primary aim of early surgical treatment of open spinal cord injuries is the prevention of localized or general infection including meningeal infections.

(2) The patient is placed on a frame made with two stretchers. He is sandwiched face down between the stretchers with holes cut out of the stretchers to expose the injury, the genitals, and the face.

(3) General debridement is then performed, with special care given to isolating the spinal wound from an abdominal wound when present.

(4) If dura is visualized and appears lacerated, place gelfoam over area and close overlying muscles and skin with sutures.

(5) Prophylactic antibiotic therapy should be initiated.

c. Maxillofacial injuries.

(1) The primary concern in facial injuries is the maintenance of a patent airway.

(2) Once an airway is opened, minimal debridement is performed and the wound is closed primarily.

(3) Prophylactic antibiotic therapy should be initiated.

(4) Fractures are handled in the best way possible. The main thing is to immobilize the fracture.

d. Eye injuries.

(1) Conjunctival foreign body.

(a) Pull eyelid away from eye.

(b) Pass a sterile wet cotton applicator across the conjunctival surface. Touching the object with the wet applicator makes it stick to the applicator.

(2) Corneal foreign body.

(a) Place a fluorescein strip in the corner of the eye, then examine the cornea with the aid of a magnifying device and strong illumination.

(b) Remove the foreign body with a sterile wet cotton applicator.

(c) Apply an antibiotic opthalmic ointment.

(d) Reexamine the eye for secondary infections 24 hours later.

(3) Lacerations of eyelid.

(a) Lid laceration not involving the lid margin can be sutured like any other laceration.

(b) If the lid margin is lacerated, the patient should be evacuated for specialized care to prevent permanent notching.

(4) Laceration of conjunctiva.

(a) Superficial lacerations of the conjunctiva do not require sutures.

(b) Apply broad-spectrum antibiotic opthalmic ointment until the laceration heals.

(5) Deep laceration or puncture wounds of the eye, foreign bodies that can't be removed, or vitreous hemorrhage (blood in the vitreous body may obscure a retinal detachment).

(a) Apply anesthetic drops to the eye.

(b) Bandage both eyes lightly and cover injured eye with an eye shield.

(c) Evacuate as soon as possible.

e. Ear injuries.

(1) You are limited to surgical repair of the external ear.

(2) Perform minimal debridement.

(3) Close lacerations in layers, being careful to realign the cartilage.

(4) Initiate prophylactic antibiotic therapy.

f. Neck injuries. Wounds of the neck are very serious and usually complicated.

(1) Establish and maintain a patent airway.

(2) Carefully debride the wound.

(3) Initiate prophylactic antibiotic therapy.

g. Chest injuries.

(1) The treatment of chest wounds is based upon the following special principles of management.

(a) Normal pleural and pericardial pressures must be maintained.

(b) The pleural space must be kept empty.

(c) The bronchial tree must be kept clean.

(d) Ventilation sufficient for adequate oxygenation and removal of carbon dioxide must be assured.

(e) The amount of hemorrhage must be estimated and blood replaced as necessary.

(2) Pneumothorax.

(a) Seal the wound(s) airtight.

(b) Place a chest tube anteriorly in the second interspace.

(c) Hook the chest tube into a closed drainage system with a waterseal.

(3) Hemothorax.

(a) Seal the wound(s) airtight.

(b) Place a chest tube through the chest wall in the midaxillary line for the removal of blood and fluid.

(4) Cardiac tamponade (fluid buildup in the pericardial sack causing muffled heart sounds and added pressure on the heart).

(a) Pericardium must be aspirated.

1. Insert cardiac needle in the angle between the xiphisternum and the costal margin.

2. Pass the needle upward and backward at a 45-degree angle into the pericardium.

3. Remove only enough fluid to improve the patient's blood pressure and pulse.

(b) Continue to monitor the patient's heart sounds, pulse, and blood pressure.

(5) Severe flail chest.

(a) Immediately intubate.

(b) Place a chest tube in the same way as with pneumothorax.

(c) Initiate positive pressure breathing.

(d) For lesser degrees of flail chest, strap the affected side with a firm dressing.

h. Abdominal injuries. The only abdominal wound we will discuss is evisceration.

(1) Stabilize the patient.

(2) Initiate prophylactic antibiotic therapy.

(3) Remove bowels from abdominal cavity and check for nicks and cuts.

(4) Suture or tag any nicks or cuts.

(5) Irrigate the abdominal cavity with sterile saline solution and remove all foreign material.

(6) Replace all good bowel into the abdominal cavity leaving the sutured and/or tagged bowel outside.

(7) Close the abdominal cavity partially and in layers leaving the tagged and sutured bowels outside on a sterile dressing to drain.

(8) Keep the patient NPO.

(9) Evacuate as soon as possible.

CHAPTER 17

ANESTHESIA

17-1. CHOICE OF ANESTHESIA.

a. In a general hospital 70-75 percent of surgery is performed under general anesthesia and the remainder under regional or local anesthesia. Operating outside a hospital these percentages should be turned around.

b. General anesthesia carries a risk with it no matter how simple the surgical procedure. Local anesthesia is often preferable to general anesthesia for the following reasons: The technique is simple and minimal equipment is required; there is less bleeding, nausea, and vomiting and less disturbance to body functions; it can be used when general anesthesia is contraindicated (e.g., recent ingestion of food by the patient); less postoperative observation and patient care are required; and there is a much lower incidence of pulmonary complications.

c. Regional anesthesia (regional block) produces complete sensory block; it prevents nerve impulses from passing by injecting the anesthetic solution around the nerve trunk at a distance from the area to be anesthetized. Regional blocks can be used almost anywhere in the body, but we will confine the blocks to dental and the upper extremities.

17-2. LOCAL ANESTHETICS.

a. Doses of local anesthetics for topical use:

Drug	Concentration	Duration	Maximal Dose
Cocaine	4%	30 min	200 mg.
Lidocaine (Xylocaine)	2-4%	15 min	200 mg.
Tetracaine (Pontocaine)	0.5%	45 min	50 mg.
Benzocaine	2-10%	Several hours	

b. Doses of local anesthetics for infiltration and nerve blocks:

Drug	Concentration	Duration	Maximal Dose
Procaine (Novocain)	2-4%	1/2 hr	1,000 mg.
Lidocaine (Xylocaine)	1-2%	1-2 hr	500 mg.
Mepivacaine (Carbocaine)	1-2%	1-2 hr	500 mg.
Tetracaine (Pontocaine)	0.1-0.25%	2-3 hr	75 mg.
Chloroprocaine (Nesacaine)	1-2%		1,000 mg.
Piperocaine (Metycaine)	1-2%		750 mg.
Hexylcaine (Cyclaine)	1-2%		500 mg.
Prilocaine (Citanest)	1-2%		500 mg.
Bupivacaine (Marcaine)	0.5%	5-7 hr	200 mg.
Etidocaine (Duranest)	0.5-1%	4-6 hr	300 mg.

c. Local anesthetic drugs (except cocaine) dilate the blood vessels, causing an increased rate of absorption and decreased duration of anesthetic action. A vasoconstrictor drug (epinephrine) may be added to injectable local anesthetic solutions to prolong and increase the anesthetic effect. Epinephrine counteracts the depressing action of local anesthetics on the heart and circulation. Epinephrine is used in concentrations of 1:100,000 (1 mg./100 ml.) or 1:200,000 (1 mg./200 ml.).

Stronger solutions should not be used because they may cause tissue damage due to ischemia.

Contraindications to adding epinephrine to local anesthetics are:

(1) Patients with history of hypertension, thyrotoxicosis, diabetes, or heart disease.

(2) Surgery on fingers or toes because severe vasospasm and ischemia of the extremities may occur.

d. Local anesthetics are used either topically or by infiltrating (injecting) the anesthesia directly around the area of surgery.

17-3. REGIONAL NERVE BLOCKS.

a. Nerve blocks are extremely effective, but in order to succeed with a nerve block you must know the anatomy of the area you want to block.

b. Premedication should be given before a nerve block is performed. Often premedication will make a block successful, especially if the patient interprets touch and motion as pain.

(1) 100 mg. (1 1/2 gr.) of phenobarbital by mouth can be given 1 1/2 to 2 hours before the block is performed.

(2) 50 to 100 mg. of phenobarbital can be given IV just before the block is performed.

(3) An alternate would be 1/8 to 1/4 gr. morphine prior to the block.

c. Axillary block of the brachial plexus.

(1) Indications. Surgery or setting fractures of the arm, forearm, and hand.

(2) Contraindications. Local infection or inflammation of the axillary nodes.

(3) Axillary blocks are used because of ease and accuracy of placement of the needle as well as the minimal incidence of complications. Additional advantages are that the axillary block can be repeated if necessary during the course of a lengthy operation and it is easily applied to a child or a somewhat uncooperative patient.

(4) The brachial plexus, axillary artery, and axillary vein are enclosed in a neurovascular compartment in the axilla. Solution injected into this sheath is limited and can spread only up or down, parallel to the neurovascular bundle.

(5) Technique. The axilla should be shaved and the arm abducted 90 degrees with the forearm flexed at a right angle and lying flat on a table. A pneumatic tourniquet (B.P. cuff) is placed just below the axilla to direct the local anesthetic toward the supraclavicular region.

The tourniquet is removed after the injection is completed. Standing at the patient's side, palpate the axillary artery as high as

possible and fix it with your index finger against the humerus. Using a 23-gage needle (or smaller; larger needles can cause hematomas if the artery is punctured), raise a skin wheal. Insert the needle at a 45-degree angle in the direction of the artery until pulsations of the axillary artery are transmitted to the needle.

This is most often preceded by a palpable click as the needle penetrates the deep fascia forming the axillary sheath. If there is paresthesia radiating down the arm to the fingers or if you aspirate blood, you are in the right area. If you aspirate blood, withdraw the needle slowly until the aspiration of blood stops. In either case you can then inject 30-40 ml. of 1% lidocaine with 1:200,000 epinephrine.

The intercostobrachial nerve that innervates the skin of the upper half of the medial and posterior side of the arm is sometimes missed but can be anesthetized by a subcutaneous injection of 3 ml. of 1% lidocaine and 1:200,000 epinephrine over the axillary artery.

AXILLARY BLOCK OF THE BRACHIAL PLEXUS.

d. Nerve block of the wrist.

(1) Indications. Surgery or setting fractures of the hand or fingers.

(2) There are three major nerves that innervate the hand and fingers: the radial nerve, the median nerve, and the ulnar nerve. To completely block the hand and fingers, all three nerves must be blocked.

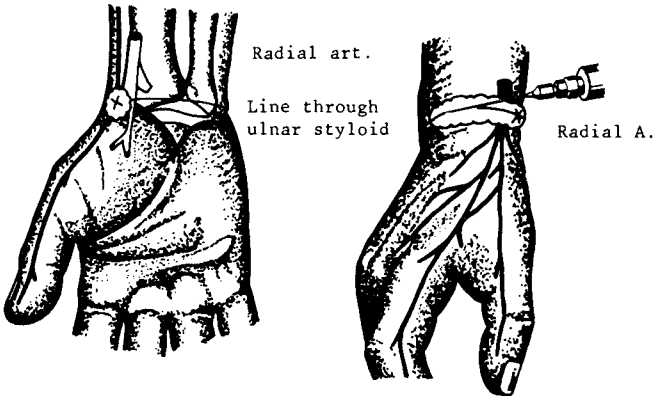
(3) For these blocks use the principle "No paresthesia no anesthesia."

(4) No more than 50 mg. of anesthesia should be used for the entire block.

(a) The radial nerve innervates the thumb and the back of the first three fingers. It is anesthetized by subcutaneous infiltration at the dorsolateral aspect of the wrist, using slow careful movement of the needle to insure an even distribution of the anesthetic solution.

1. Technique. Form a skin wheal, using a 22-gage needle (or smaller) at the point shown in the drawing. Working through the skin wheal with the syringe parallel to the nerve, elicit paresthesia (an electric shocklike sensation) in the thumb and the back of the first three fingers. When paresthesia is achieved, aspirate to insure you are not in a blood vessel, then inject 10 ml. of 1% lidocaine in a ring fashion. Begin lateral to the radial artery and extend the ring to the center of the back of the hand using slow, careful movement to insure even distribution. No

more than 20 cc. of 1% lidocaine should be used for the entire procedure.

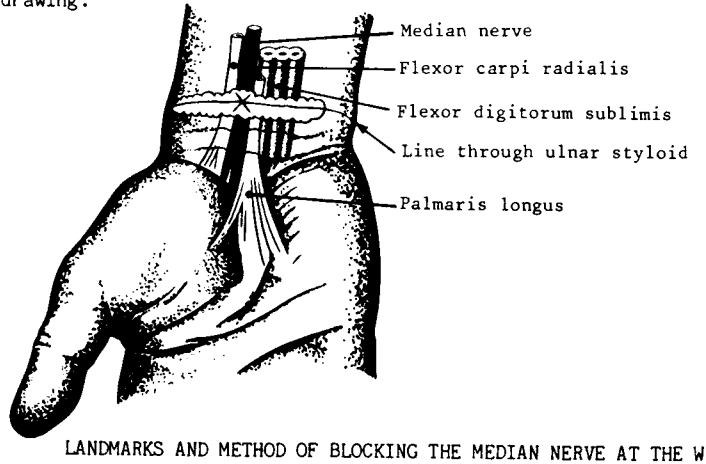


LANDMARKS AND METHODS OF BLOCKING THE RADIAL NERVE AT THE WRIST.

2. Complications. IV injection and/or hematoma of the joint.

(b) Median nerve innervates the palm of the hand, the index finger, the middle finger, and the radial side of the ring finger.

1. Technique. Locate the palmaris longus ligament and form a skin wheal, using a 22-gage needle (or smaller), just to the radial side of the palmaris longus. Working through the skin wheal, attempt to elicit paresthesia in the palm of the hand and fingers. When paresthesia is achieved, aspirate, then inject 5 ml. of 1% lidocaine. Then begin at the wheal and using slow, careful movements to insure even distribution, inject in a line right and left of the median nerve as depicted in the drawing.



LANDMARKS AND METHOD OF BLOCKING THE MEDIAN NERVE AT THE WRIST.

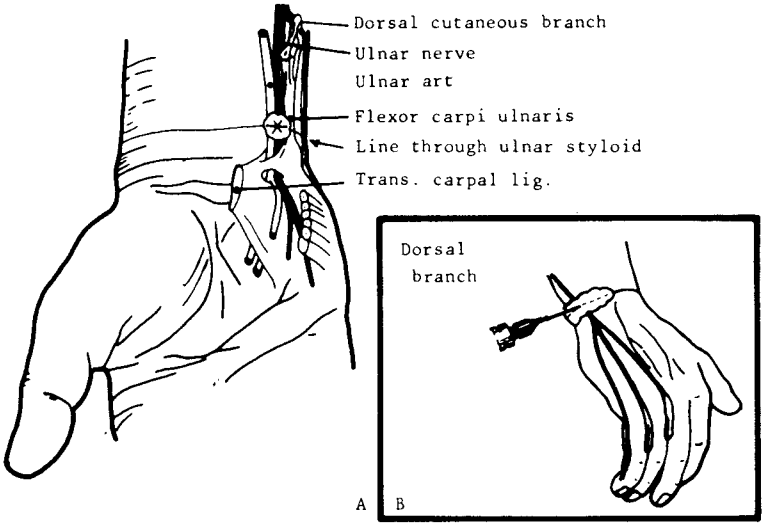
The injection to the right and left might not be necessary, but

occasionally there are collateral nerves that have moved down into the palm, and this will anesthetize them also.

2. Complications. IV injection and/or hematoma.

(c) Ulnar nerve innervates the ulnar side of the ring finger, the little finger, the ulnar side of the palm, and the back of the little and ring fingers.

1. Technique. Locate the flexor carpi ulnaris (see drawing) by palpation on a line through the ulnar styloid. Using a 22-gage needle, raise a skin wheal just lateral to the flexor carpi ulnaris. Working through the skin wheal, introduce the needle in the direction of and parallel to the nerve. After achieving paresthesia and aspiration, inject 5 ml. of 1% lidocaine with 1:200,000 epinephrine.



LANDMARKS AND METHODS OF BLOCKING THE ULNAR NERVE AT THE WRIST.

A. Volar Branch B. Dorsal Branch.

Once this is done begin at the skin wheal and extend the anesthesia dorsally in a ring fashion to the center of the back of the hand, using slow careful movement to insure even distribution of the anesthesia in the subcutaneous layer.

2. Complications. IV injection and/or hematoma.

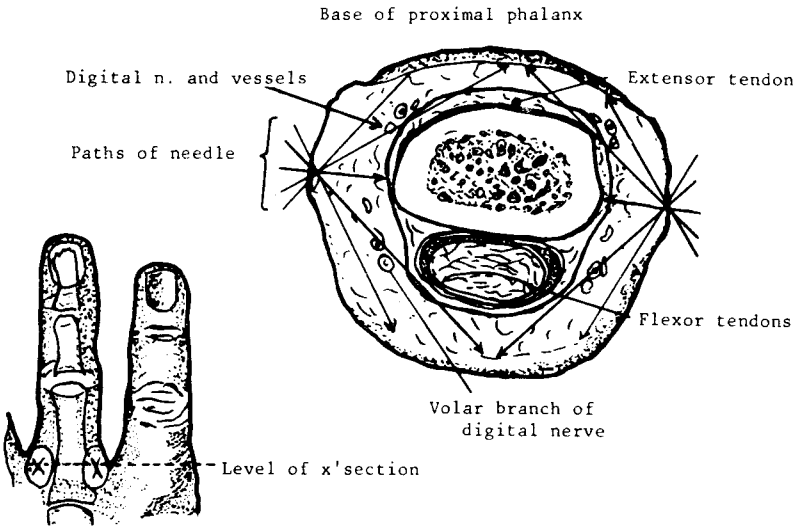
e. Digital block (fingers & toes).

(1) No premedication is necessary.

(2) Do not use vasoconstrictor agents (epinephrine).

(3) Do not exceed 8 cc. of anesthesia per digit.

(a) Technique. Raise a skin wheal on the dorsolateral sides of the digit at the interdigital folds (see drawing).



LANDMARKS AND METHOD OF BLOCKING DIGITS.

Working through the wheals in a fan-shaped method, place a ring of local anesthetic around the digit (see drawing). Massage the digit where the anesthesia was deposited to facilitate spread of the solution.

(b) Repeat the block if analgesia is inadequate.

(c) Complications. IV injection and/or hematoma.

17-4. GENERAL ANESTHESIA.

a. Ideally, the patient should have a complete physical and history done at least a day before surgery. Special examinations and laboratory work should be done as needed. The patient should have a good night's sleep and be placed N.P.O. 6-8 hours prior to surgery. The operation should be explained to the patient to help calm his fears. Finally, the patient should receive the proper premedication.

b. Premedication.

(1) Produces psychic sedation (relaxes and calms the patient, making administration of anesthesia easier).

(2) Reduces metabolic rate and decreases reflex irritability.

(3) Reduces quantity of anesthetic drug necessary.

(4) Minimizes or abolishes secretions of saliva and mucus.

(5) There are a wide range of sedatives, narcotic analgesics,

tranquilizers, and belladonna compounds (atropine and scopolamine) that can be used as preanesthesia medication. You must determine which is best for your situation. A good example is:

(a) Adult premedication.

1. Place patient N.P.O. 6-8 hours preoperatively.

2. 100 mg. pentobarbital P.O. at bedtime.

3. 100 mg. pentobarbital IM 2 hours preoperatively.

4. 0.5 mg. atropine SQ 1 hour preoperatively.

(b) Child premedication.

Age	Weight			Pentobarbital or Secobarbital	Morphine	Atropine or Scopolamine
Newborn	7	lb	3.2 kg.	----	----	0.1 mg.
6 months	16	lb	7.3 kg.	----	----	0.1 mg.
1 year	22	lb	10 kg.	35 mg.	1 mg.	0.2 mg.
2 years	26.5	lb	12 kg.	50 mg.	1.2 mg.	0.3 mg.
4 years	33	lb	15 kg.	65 mg.	1.5 mg.	0.3 mg.
6 years	44	lb	20 kg.	75 mg.	2 mg.	0.4 mg.
8 years	55	lb	25 kg.	90 mg.	2.5 mg.	0.4 mg.
10 years	66	lb	30 kg.	100 mg.	3 mg.	0.4 mg.
12 years	88	lb	40 kg.	100 mg.	4 mg.	0.4 mg.

1. N.P.O. 6-8 hours preoperatively.

2. _____ mg. pentobarbital, IM 2 hours preoperatively.

3. _____ mg. atropine, SQ 1 hour preoperatively.

c. Ether anesthesia. Used for all types of surgery, particularly that requiring muscle relaxation. It is probably the safest of the inhalation anesthesias. Induction of ether anesthesia is prolonged because of its irritating effects. To shorten the induction period the patient can be preanesthetized ("knocked down") with a nonirritating rapid-acting drug such as sodium pentothal.

(1) Advantages.

(a) Reliable signs of anesthesia depth.

(b) Stimulation of respiration.

(c) Bronchodilation.

(d) Does not depress circulation.

(e) Good muscle relaxation.

(f) Relatively nontoxic and safe. (Death rate is lower than any other anesthesia.)

(2) Disadvantages.

(a) Prolonged induction and recovery.

(b) Irritating action causes secretions of mucus from upper airway.

(c) Emetic action is dangerous in patients with full stomach. (Aspiration pneumonia.)

d. Sodium pentothal. Used for minor procedures of approximately 30 minutes or less. Can also be used as a "knock down" for ether anesthesia.

(1) Technique. Slowly inject the drug through an existing IV. Do not exceed 2 cc. in the first 15 seconds; then stop and wait (patient will be narcotized in 30-40 seconds). Having the patient count will let you know how it is affecting him. Slowly inject 1/2 to 1 cc. from time to time as required.

(2) Disadvantage.

(a) The anesthesia is noncontrollable.

(b) Laryngeal spasm may develop.

(c) The necessary effective dose is difficult to estimate.

(d) A severe respiratory depression ensues.

(e) Pentothal is a barbiturate that does not possess any analgesic properties.

(f) The muscular relaxation is not satisfactory.

(3) Signs of anesthesia. No reliable signs of anesthesia exist. The anesthetist must attempt to maintain the patient between the zones of decreased reflex activity and respiratory and circulatory failure.

(4) Complications.

(a) Respiratory failure.

(b) Hypotension.

(c) Laryngeal spasm.

(d) Slough of skin. Solutions of the sodium salts of barbiturates are alkaline and cause damage to tissues in event of seepage.

(e) Phlebothrombosis.

(f) Prolonged somnolence.

(g) Operations of undetermined length. Large amounts of the drug may be necessary to complete the operation. This causes a marked depression of respiration and circulation from cumulative effects.

(h) Shock from trauma or hemorrhage. Irreversible

respiratory failure or enhancement of hypotension may occur.

(5) Precautions.

(a) The limit should be approximately 1 gram of the drug for an adult.

(b) Be positive that the drug is completely dissolved and that the solution is clear before performing venipuncture. Undissolved particles act as foreign bodies in the solution and may cause "reactions."

(c) Inject the solution slowly. Do not inject more than 6 cc. of a 2-1/2% solution at any one time at the onset.

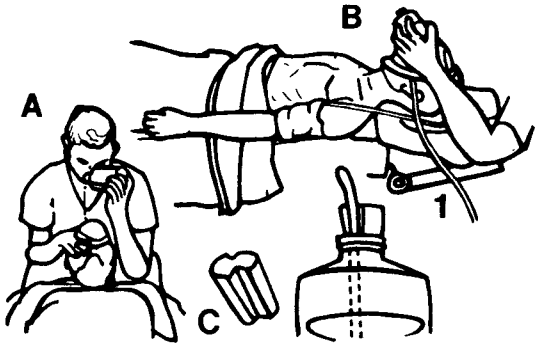
e. Open drop method of inhalation anesthesia. The simplest method requiring the least equipment.

(1) Make a wire frame in a cup shape to fit over the nose and mouth. Pad and tape the bottom edges to make a better fit and protect the patient's face. Place one or two unfolded 4 X 4s over the top to completely cover the frame and tape the edges down. Cut two notches in the cork going into the ether container, one for air to enter the container and one for the wick that will drop the anesthesia on the mask.

(2) By regulating the amount of anesthesia dripped on the mask, you can regulate the depth of anesthesia.

(3) Oxygen can be fed into the mask by running a small tube under the edge of the mask; however, the higher the flow of oxygen the lower the concentration of anesthesia.

(4) Condensation of water vapors on the gauze interferes with vaporization of the anesthesia. The colder the gauze the more rapid the condensation of moisture in the expired air. Replace the gauze as required to correct the impaired vaporization.



TECHNIQUE OF "OPEN DROP" ANESTHESIA. A - Method of supporting the head with the arms. B - Lateral view showing support of head and insufflation of oxygen (1) beneath the mask. C - Cork cut and wick in place to drip ether.

f. Stages and signs of anesthesia. Anesthesia is divided into four

stages, and the third or surgical stage is subdivided into four planes.

STAGE I - ANALGESIA

STAGE II - DELIRIUM

STAGE III - SURGICAL

Plane 1

Plane 2

Plane 3

Plane 4

STAGE IV - RESPIRATORY PARALYSIS

(1) STAGE I. The Stage of Analgesia is that period from the beginning of induction to the loss of consciousness.

(a) Analgesia is the loss of the sense of pain without the loss of consciousness or sense of touch. Pain sense is progressively depressed during this stage. The point of pain abolition is known as total analgesia, and the approach to total analgesia is relative analgesia. The danger and difficulty in achieving and maintaining total analgesia is its proximity to the second stage. It is sometimes attempted, however, in dentistry, obstetrics, and to a small extent in minor surgery.

(b) False anesthesia as a manifestation of hysteria may occur in this stage. The signs may indicate quiet surgical anesthesia, i.e., regular rhythmic respiration with apparent loss of eyelid reflex. Starting the preparation or the operation at this point may precipitate fatal ventricular fibrillation. The only way of differentiating between false anesthesia and true surgical anesthesia is the length of time elapsed since induction. Three or four minutes may be long enough to reach Stage III with cyclopropane and pentothal, but it is not when using ether. When in doubt, wait much longer than you would otherwise wait before starting a procedure.

(c) "Brain anesthesia" is another phenomenon that may be encountered. The brain has a very rich blood supply and high partial pressure of the agent may cause the brain to become saturated with the first few respirations giving the appearance of surgical anesthesia. Subsequently, the agent will diffuse out of the brain almost as rapidly as it diffused in, and the signs will become a more accurate index.

(2) STAGE II. This is the Stage of Delirium or excitement stage and represents the period of the earliest loss of consciousness. The hazards of the second stage are physical injury and ventricular fibrillation.

(a) The higher cerebral or voluntary control centers are abolished, leaving the secondary centers free. Response to stimulation or to dreams the patient may be having is exaggerated and is frequently expressed in violent physical activity. This violence may be initiated by external stimulation such as instruments being knocked together, talking, moving the patient or the operating table, etc. The excitement will be

more pronounced in patients who are afraid. (Who isn't afraid when coming to the operating room?)

(b) The stages of anesthesia are the same whether the patient is going to sleep or waking up. Emergence delirium is less frequently violent than that of induction because of several factors. First, external stimulation is seldom as great; and second, postoperative surgical depression limits the amount of activity. Remember that nonshocking procedures done under short-acting drugs leave the patient potentially as active during emergence as during induction.

(c) Ventricular fibrillation occurs most frequently in the group from 5 to 35 years of age, but may occur at any age. This danger also increases in proportion to the degree of fear--probably due to increased adrenalin output accompanying fear.

(d) Management of the second stage consists of:

1. Reducing nervous irritability by use of adequate premedication.

2. Proper restraint of the patient.

3. Avoidance of external stimuli.

4. Rapid, smooth induction.

(3) STAGE III. The Surgical Stage.

(a) Plane 1. Entrance into Plane 1 is marked by the appearance of full, rhythmic, and mechanical respiration. The tidal volume and respiratory rate are increased depending on the efficiency of respiration in the preceding stages. If the first and second stages have been uneventful, the increase in volume is about 25% above normal. If the course has been stormy with consequent carbon dioxide retention, the volume may be twice normal. If hyperpnea has preceded this stage, it is possible that the respiratory volume may be below normal. Within a minute or so, however, the balance between the respiratory threshold and the carbon dioxide tension should be established at a moderate hyperpnea.

1. Inspiration is shorter and slightly quicker than expiration, and there is a pause at the end of expiration.

2. Premedication has a direct bearing on the rate and volume throughout the anesthesia. Other things being equal, the minute volume is decreased in proportion to the threshold elevation by the preanesthetic drug.

3. Response to reflex stimulation is still present and minute volume is also directly proportionate to the amount of stimulation from the operative field.

(b) Plane 2. The tidal volume is usually somewhat decreased while the rate may be either decreased or increased. As the rate increases, the pause at the end of expiration becomes shorter so that both phases are more nearly equal in length. The response to reflex stimulation from the operative site is somewhat less than in Plane 1.

(c) Plane 3. Entrance into this plane is marked by beginning paralysis of the intercostal muscles. The first evidence of this paralysis is a delayed thoracic inspiratory effort. That is, the diaphragm begins its excursion before the intercostals cause thoracic expansion. This phenomenon can be felt before it is visible. The pause between inspiration and expiration becomes progressively longer at the expense of inspiration. Inspiration becomes a quick jerky movement. The progressive intercostal paralysis gradually decreases the tidal volume with progression downward.

The lower border of Plane 3 is marked by the completion of intercostal paralysis. The bony thorax is stationary, and there is decided retraction of the intercostal spaces with each inspiration. It should be remembered that intercostal retraction also occurs with respiratory obstruction.

(d) Plane 4. At this point the diaphragmatic excursion is greater than at any other period of anesthesia. The resistance to its descent, which is normally provided by the expansion of the bony thorax, is absent; therefore, it becomes a quick jerky movement. This movement, long before intercostal paralysis is complete, is annoying to the surgeon working in the abdomen. He may complain that the patient is "pushing" and may attempt, to alleviate the condition by deepening the anesthesia, resulting in aggravation on the part of the surgeon and consternation on the part of the anesthetist when the patient suddenly stops breathing.

Diaphragmatic paralysis begins, and there is a marked decrease in tidal volume, progressing rapidly as diaphragmatic paralysis increases. Inspiration becomes more shallow and gasping, and the rhythm is very irregular.

(4) STAGE IV. Complete diaphragmatic paralysis or cessation of respiration marks the entrance into Stage IV, and death ensues within 1 to 5 minutes unless corrective measures are instituted immediately.

(5) Eye signs. It is the degree of activity of the motor muscles of the eyeball that serves as an anesthetic guide, not the type of activity. Preanesthetic medication may retard eyeball activity, but it does not destroy its value as an anesthetic guide. The protruding eyeball is usually not as active as the normal one.

(a) Stage I. Eyeball activity is normal or under voluntary control.

(b) Stage II. The motor muscles undergo a period of excitation activity. The mechanism is not completely understood, but it is believed that it is the result of the impulses "running riot" in the central nervous system. These "berserk impulses" result in the stimulation of first one nucleus then another. This is manifested in the hyperactivity of the eyeball in Stage II.

(c) Stage III.

1. Plane 1. The hyperactivity is still present upon entrance into Plane $\overline{1}$ and decreases progressively as anesthesia is carried downward. This may be explained on the basis of progressive depression of the central nervous system with consequent diminution of stimulation of the various centers.

2. Plane 2. Complete cessation of activity marks entrance into Plane 2. From this point on down, the eyes should be fixed concentrically because the muscles are paralyzed and flaccid. Eccentric fixation should suggest the possibility of hypoxia.

(d) Reflexes.

1. Conjunctiva palpebral. Present in Stages I and II. Absent in Stage III, Plane 1. The eyelids close when the tip of the finger brushes the margin of the upper lid.

2. Lid. Present in Stages I and II. Absent in Stage III, Plane 1. Tested by gently raising the upper eyelid with the finger. If the reflex is present, the lid will attempt to close either at once or after a few seconds' exposure. If it is absent, no effort to close the lid will occur.

(6) Muscular reflexes.

(a) Vomiting occurs at the extreme lower border of Stage II.

(b) Swallowing occurs at the extreme upper border of Plane 1.

(c) Jaw sign is tested by pushing the jaw forward and up from the angle. In light anesthesia there will be a marked change in respiration in response. The response will not be marked in mid Plane 2, and in lower Plane 2 there should be no change in respiration.

(d) Pharyngeal reflex is abolished in mid Plane 1.

(e) Laryngeal reflex response to direct stimulation is abolished at the junction of Plane 1 and Plane 2. The response to reflex stimulation is abolished in mid Plane 2.

(f) Reflex response to skin traumatism is usually abolished by anesthesia in the upper half of Plane 1.

(g) Skeletal muscles are relaxed in the following order:

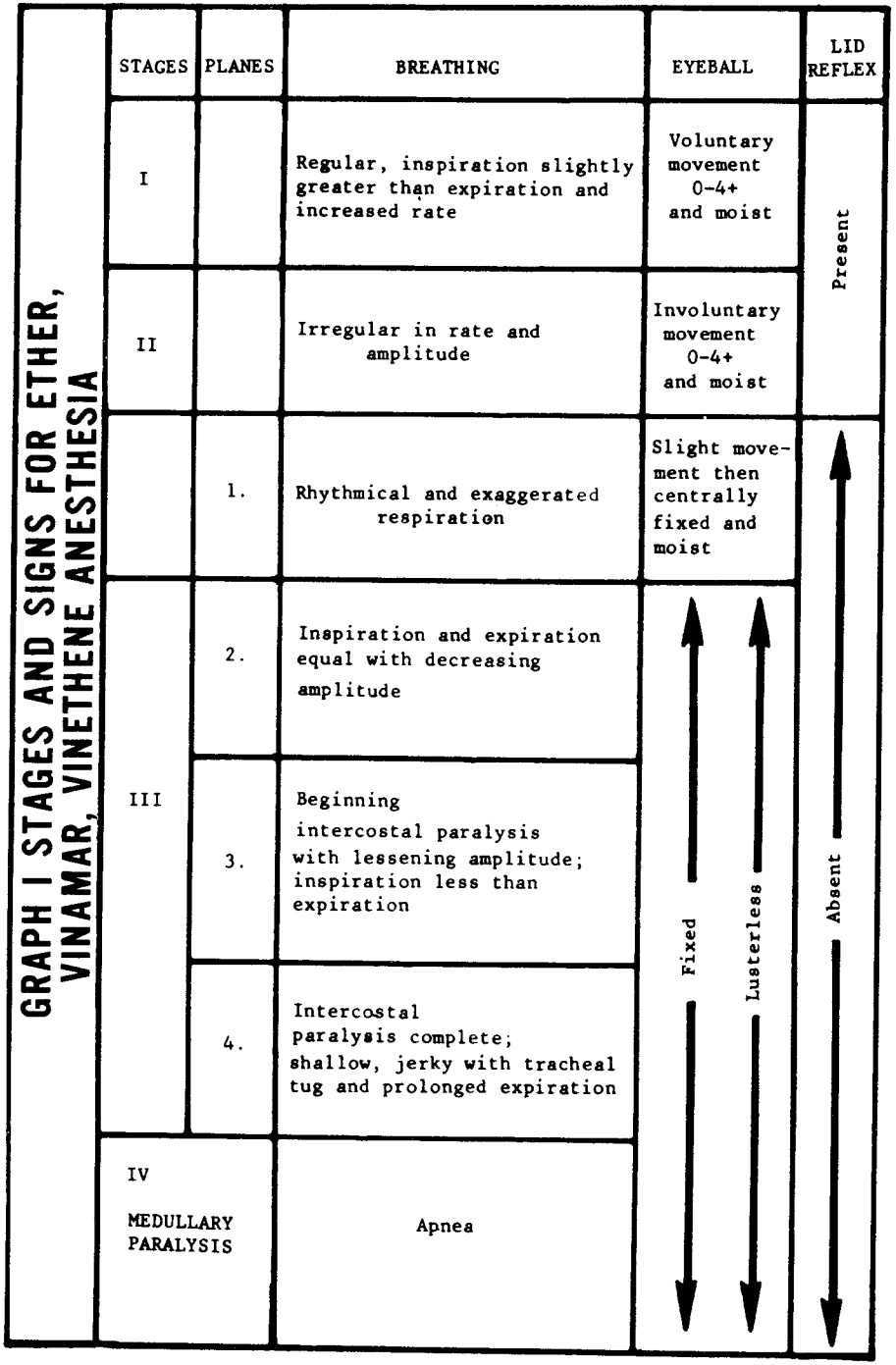
1. Small - Plane 1

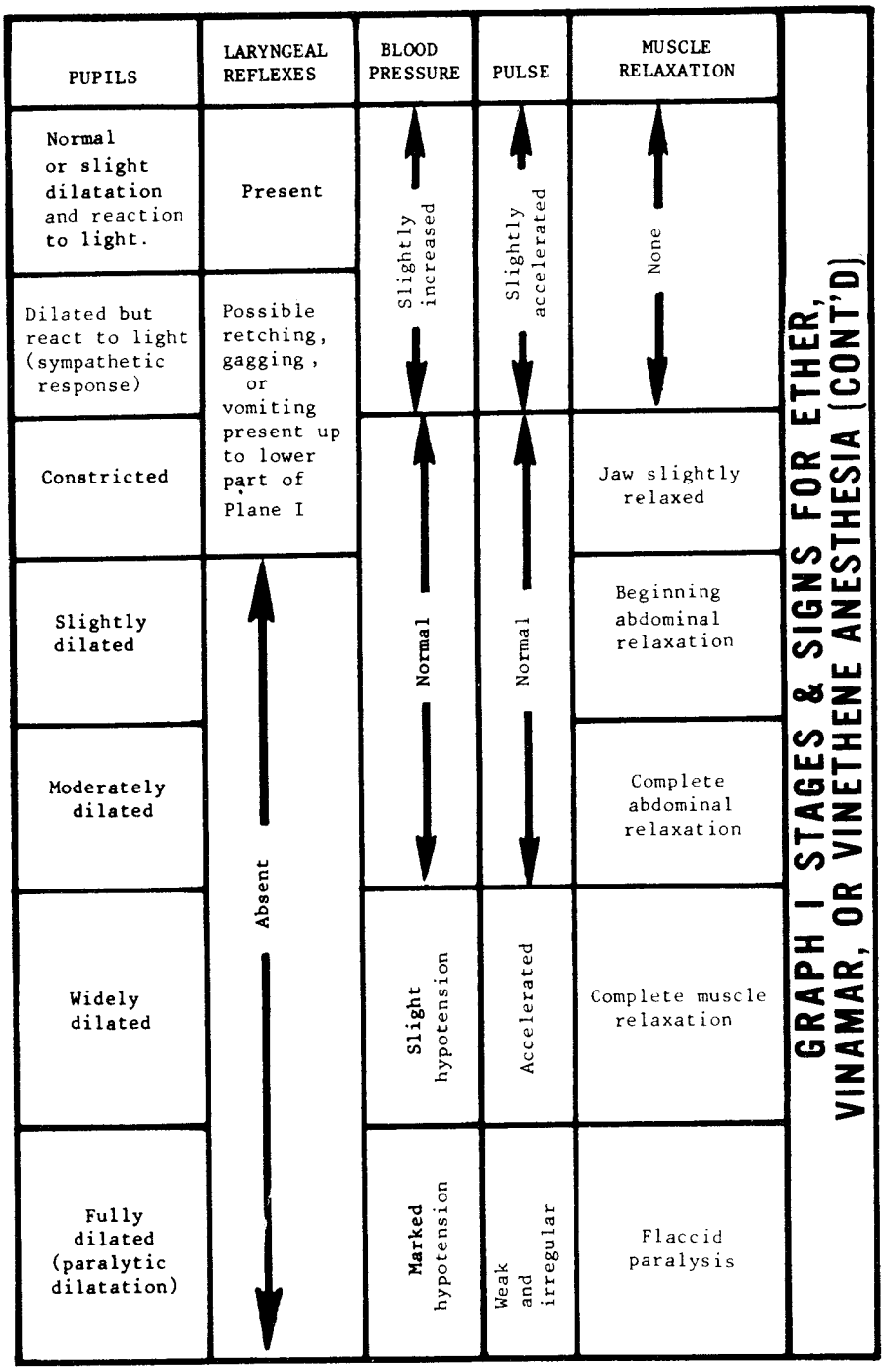
2. Large - Plane 2

3. Abdominal - Mid Plane 2

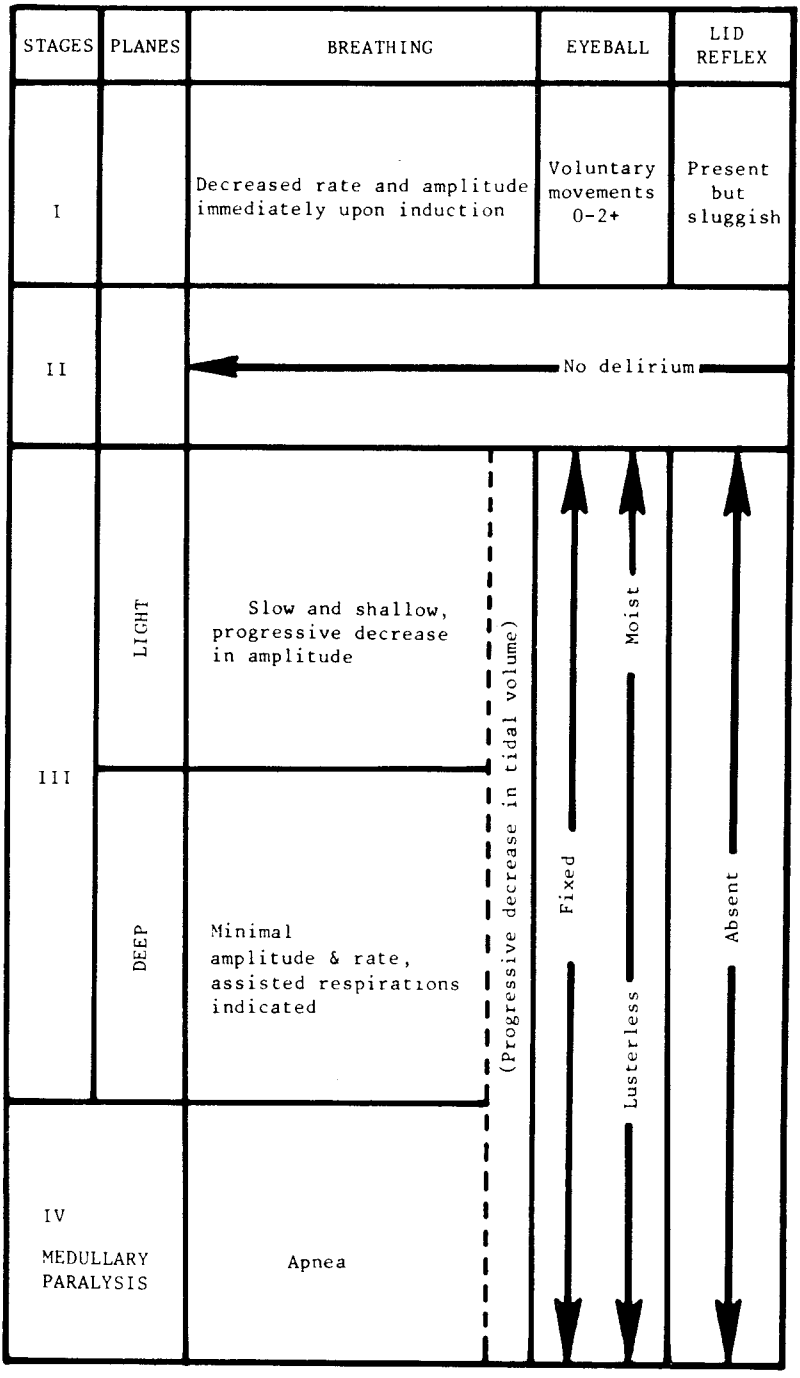
4. Diaphragm - Plane 4

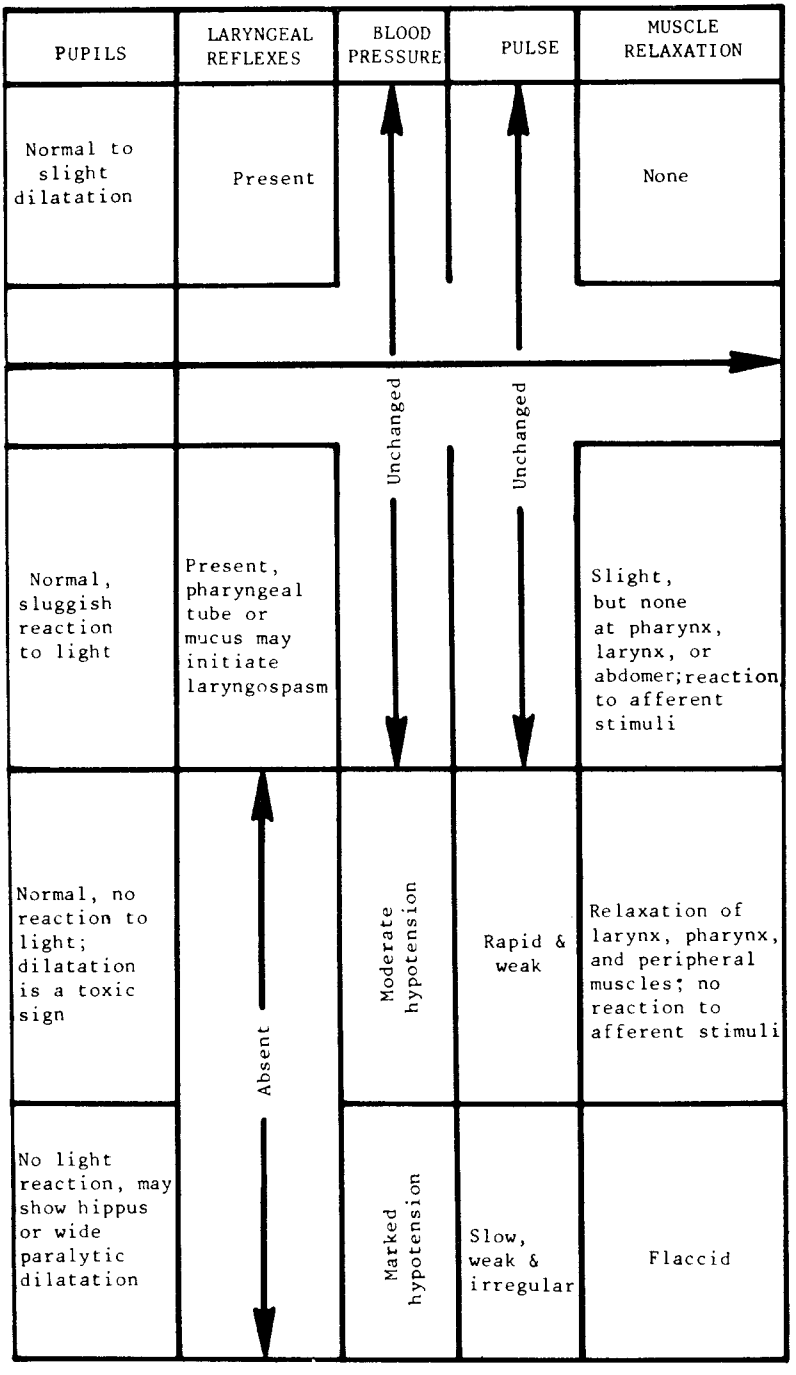
(h) Smooth muscle tone (intestinal tract and blood vessels) is lost in lower Plane 3.


GRAPH I STAGES AND SIGNS FOR ETHER, VINAMAR, VINETHENE ANESTHESIA
STAGES
PLANES
BREATHING
EYEBALL
LID REFLEX
I
Regular, inspiration slightly greater than expiration and increased rate
Voluntary movement 0-4+ and moist
Present
II
Irregular in rate and amplitude
Involuntary movement 0-4+ and moist
1.
Rhythmical and exaggerated respiration
Slight movement then centrally fixed and moist
III
2.
Inspiration and expiration equal with decreasing amplitude
3.
Beginning intercostal paralysis with lessening amplitude; inspiration less than expiration
4.
Intercostal paralysis complete; shallow, jerky with tracheal tug and prolonged expiration
Fixed
Lusterless
Absent
IV
MEDULLARY PARALYSIS
Apnea

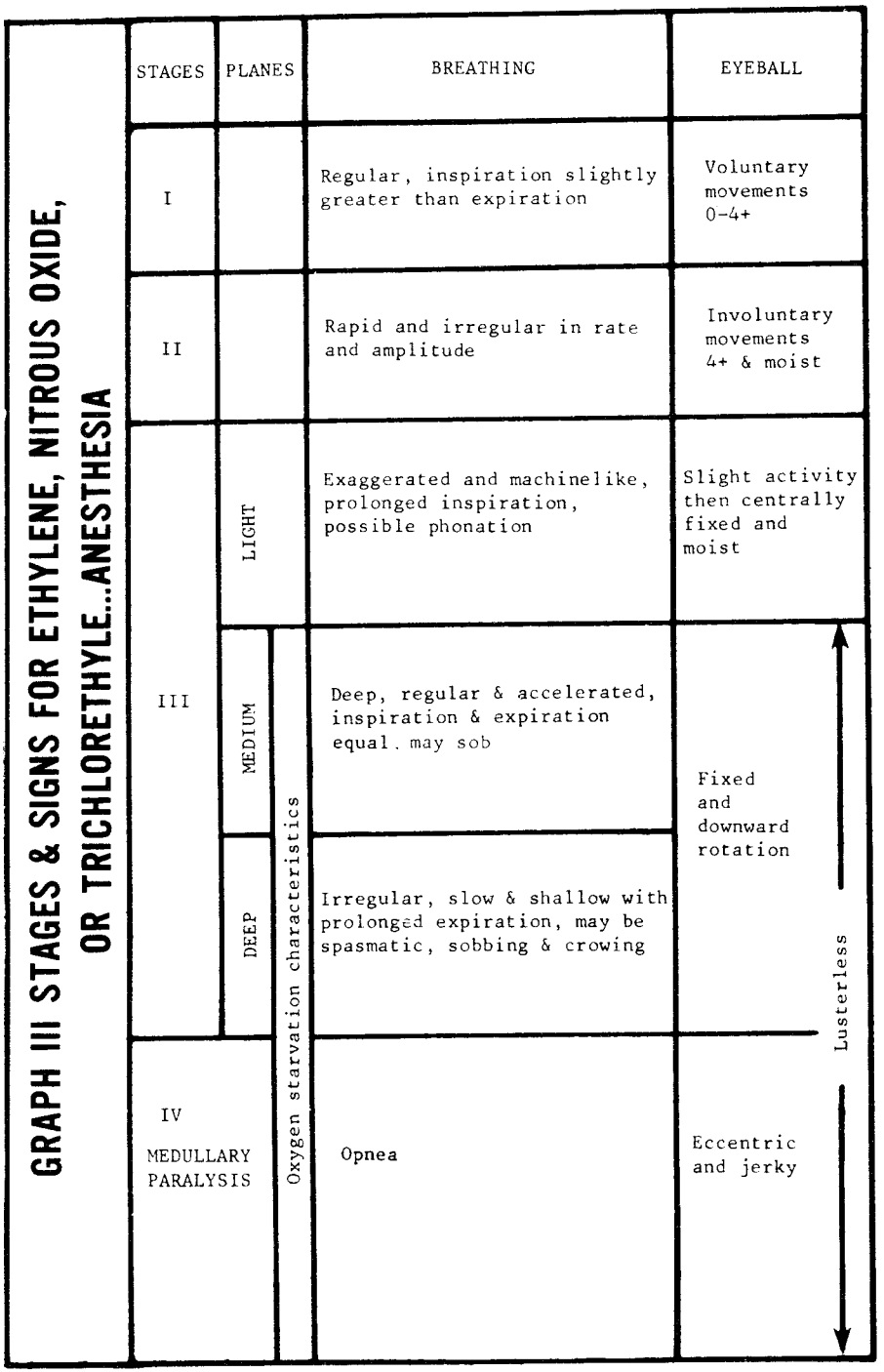

PUPILS
LARYNGEAL REFLEXES
BLOOD PRESSURE
PULSE
MUSCLE RELAXATION
Normal or slight dilatation and reaction to light.
Present
Slightly increased
Slightly accelerated
None
Dilated but react to light (sympathetic response)
Possible retching, gagging, or vomiting present up to lower part of Plane I
Constricted
Jaw slightly relaxed
Slightly dilated
Normal
Normal
Beginning abdominal relaxation
Moderately dilated
Complete abdominal relaxation
Absent
Widely dilated
Slight hypotension
Accelerated
Complete muscle relaxation
Fully dilated (paralytic dilatation)
Marked hypotension
Weak and irregular
Flaccid paralysis
GRAPH I STAGES & SIGNS FOR ETHER, VINAMAR, OR VINETHENE ANESTHESIA (CONT'D)

GRAPH II STAGES + SIGNS FOR SODIUM PENTOTHAL





GRAPH II STAGES + SIGNS FOR SODIUM PENTOTHAL


GRAPH III STAGES & SIGNS FOR ETHYLENE, NITROUS OXIDE, OR TRICHLORETHYLE...ANESTHESIA
STAGES
PLANES
BREATHING
EYEBALL
I
Regular, inspiration slightly greater than expiration
Voluntary movements 0-4+
II
Rapid and irregular in rate and amplitude
Involuntary movements 4+ & moist
III
LIGHT
Exaggerated and machinelike, prolonged inspiration, possible phonation
Slight activity then centrally fixed and moist
MEDIUM
Deep, regular & accelerated, inspiration & expiration equal, may sob
Fixed and downward rotation
DEEP
Irregular, slow & shallow with prolonged expiration, may be spasmatic, sobbing & crowing
IV
MEDULLARY PARALYSIS
Oxygen starvation characteristics
Opnea
Eccentric and jerky
Lusterless

GRAPH III STAGES & SIGNS FOR ETHYLENE, NITROUS OXIDE, OR TRICHLORETHYLE...ANESTHESIA (CONT'D)

LID REFLEX	PUPILS	LARYNGEAL REFLEXES	BLOOD PRESS	PULSE
Reflex present	Constricted or dilated	Present	Normal	Normal
Reflex present, lids - resistant	Dilated (sympathetic response)	Swallowing with tendency to retching gagging, or vomiting	Unchanged	Slightly accelerated
Reflex - faintly present & lids slightly resistant	Small and sluggish to light			
Reflex - absent, lids open	Small to moderate, NO light reaction	Absent	Slight hypertension	Increasing rate
Reflex absent and lids open and stiff	Dilated (danger sign)	Absent reflex with tendency to gasping, retching and vomiting	Marked hypertension	Weak and irregular
	Maximum dilatation, may become irregular			

GRAPH III STAGES & SIGNS FOR ETHYLENE, NITROUS OXIDE, OR TRICHLORETHYLE...ANESTHESIA (CONT'D)

MUSCLE RELAXATION	CENTRAL NERVOUS DEPRESSION (EEG) cps = cycles per sec. mvs = microvolts
None	Intermediate frequency, 8-13 cycles per sec; low voltage, less than 50 microvolts
Exaggerated reflex and rigidity	Low frequency, 4-6 cps; low voltages, less than 50 microvolts
None	Mixed wave forms 4-6 cps, and 40-70 mvs, superimposed upon slow frequency 2-3 cps, 100-150 microvolts
Slight	
Absent with rigidity & spasm	Continuation of mixed wave patterns with burst suppression, rhythmicity is lost; frequencies are relatively slow 3-5 cps, but the amplitude remains high, over 200 mvs
Spastic	