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Da Costa

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## Modern Surgery - Chapter 19. Diseases and Injuries of the Bones and Joints - Diseases of the Bones

John Chalmers Da Costa  
*Jefferson Medical College*

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## XIX. DISEASES AND INJURIES OF BONES AND JOINTS.

### I. DISEASES OF THE BONES.

**Atrophy of bone** is a diminution in the amount of bony matter without change in osseous structure. It arises from want of use (as seen in the wasting of the bone of a stump) or from pressure (as seen in the destruction of the sternum by an aneurysm of the aorta). *Eccentric* atrophy is the thinning of a long bone from within, the outer surface being unchanged. It is usually a senile change. *Concentric* atrophy means a thinning of the outer surface of the shaft, causing a lessened diameter. It is usually linked with eccentric atrophy.

**Hypertrophy of bone** may be due to increased blood-supply (as is seen in chronic epiphyseal inflammation), the bone growing much more than does its fellow. It may arise from excessive use or from strain, as is seen in the increased size of the fibula when the tibia is congenitally absent.

**Tumors of Bone.**—Bones give origin to both innocent and malignant tumors. Myeloid sarcoma takes origin in the endosteum and expands the bone. The fasciculated sarcoma is a periosteal growth. Besides these growths there may develop an osteoma, a chondroma, and secondary deposits of cancer and sarcoma. There is no such thing as primary cancer of bone. A bone may become cystic, and occasionally the cysts are due to hydatids. Gummata are frequently met with.

**Cysts and Cystomata of Bone.**—The majority of bone-cysts are produced by softening of solid neoplasms (sarcoma, myxoma, chondroma). Occasionally "cysts from softening arise in osteomalacia and osteitis deformans" ("An American Text-Book of Pathology"). Hydatid cysts and dermoid cysts are sometimes encountered. A true cystoma of bone, except in one of the jaws, is a surgical rarity. In the maxillary bones dentigerous cysts or cystomata are not very uncommon.

**Actinomycosis of bone** is most usual in the jaw, but may attack the orbit, ribs, sternum, or limbs (see page 221). Actinomycosis of bone may arise secondarily after infection of superficial parts with the ray-fungus. In the jaw the fungus obtains entrance to the interior of the bone through a tooth socket. In some cases of bony actinomycosis the fungus reaches the bone by the blood. Actinomycosis leads to the production of granulation tissue, the bone is expanded and becomes carious, and a quantity of new bone is sometimes produced. In vertebral actinomycosis, although the condition resembles tuberculosis, angular deformity does not occur.

**Tuberculosis of bone** tends especially to appear in the cancellous ends of long bones; a tuberculous area is apt to caseate and destroy large amounts of bone. The bone does not sclerose, but undergoes alterations of an osteoporotic nature (see page 182).

**Osteitis, Periostitis, and Osteoperiostitis.**—**Osteitis, or inflammation of bone,** may be due to traumatism, to a constitutional malady or diathesis, to the extension of inflammation from some other structure, or to infection. In inflammation of bone the exudate and leukocytes pass into the Haversian canals, spaces, and canaliculi. The bone-corpuscles proliferate and the bone undergoes thinning (rarefaction), not because of pressure,

but because of absorption by voracious leukocytes and osteoclasts. This process of rarefaction enlarges all the bony spaces, and by destroying septa throws many of the spaces into one. If the surface of a bone inflames, the periosteum will be separated more or less by the exudation, and the bone will be covered with little pits or erosions made by the leukocytes. Inflamed bone is so soft that it can readily be cut with a knife.

Osteitis may terminate in *resolution* or it may terminate in *sclerosis*, the mass of proliferating cells being converted first into fibrous tissue and next into dense bone which contains very few small cancellous spaces. If the exudation is under the periosteum, the bone will be thickened at this point, bone stalactites marking the points of passage of the vessels. Osteitis may terminate in *suppuration*, this condition being often called *caries*. In tuberculous osteitis caseation of the inflammatory products is very apt to arise (tuberculous or strumous caries). Acute osteitis may terminate in *necrosis*, the inflammatory exudate compressing the vessels in their bony canals, a portion of the bone being in consequence deprived of nutritive material. The portion cut off from nutritive fluid dies *en masse* (necrosis). Osteitis is usually associated with more or less periostitis. A simple acute periostitis without involvement of the bone may arise from traumatism or strain; but in all severe cases of periostitis, in all chronic cases, in all cases due to syphilis, rheumatism, measles, scarlatina, or enteric fever the bone is involved at the same time or subsequently. In syphilitic states gummatous degeneration frequently ensues.

**Symptoms of Osteitis and Osteoperiostitis.**—As a chronic process, *osteitis* is most commonly found in the femur. Its history usually exhibits a record of an antecedent injury or chilling of the body. Pain is severe, boring or aching in character, deep-seated, worse at night, and aggravated by a dependent position of the part. The symptoms closely resemble those of periostitis, with which disease it is almost sure to be linked. Tenderness exists on percussion, and sometimes on pressure. Subperiosteal swelling, fusiform in shape, is noted; cutaneous edema and discoloration are observed if a superficial bone is inflamed. In syphilis, atrophic osteitis may attack the cranial bones and produce softening or even perforation, or osteophytic osteitis may arise, exostoses being formed. *Osteoperiostitis* may be acute or chronic, circumscribed, or diffused, and may terminate in resolution, organization, or suppuration. It arises from cold, blows, wounds, strains, the spread of adjacent inflammation, specific febrile maladies, pyogenic infection, syphilis, rheumatism, or tuberculosis. The symptoms are pain (which is worse at night and which is aggravated by motion, pressure, or a dependent position), swelling, edema, and discoloration of the soft parts. Pain in the syphilitic form is not so severe as in other varieties. *Acute necrosis* or *diffuse periostitis*, a septic inflammation of bone and periosteum, is commonest in boys about the age of puberty. It is usually due to cold, a specific fever, or injury, and most often affects the tibia or femur; the symptoms locally are redness, swelling, and severe pain; constitutionally there are rigors, fever, and sometimes convulsions. Necrosis is apt to result. Pyemia is common. In *simple acute periostitis* a swelling is felt upon the osseous surface. The swelling is firmly fixed and is very tender but the bone itself is

not enlarged. There is some local heat, discoloration, often fever, and the patient complains of an aching pain, which is worse at night.

Periostitis due to strain demands some special attention. Sir James Paget, years ago, pointed out that muscular exertion might cause periostitis. C. T. Dent has written a valuable article upon this subject.\*

It is common to hear football players complain of some swelling of the knee-joint. Examination finds tenderness over the tubercle of the tibia with slight swelling of the joint. Dent points out that pain is felt on straightening the leg, not on rotating it. The same observer states that omnibus drivers suffer from periostitis of the fibula, due to pressing forcibly against the foot-board; those who ride may develop periostitis of the adductor insertion (riders' bone); the victims of flat-foot may labor under periostitis of the inner tuberosity of the os calcis; bar-keepers, from working a beer-pump, may get periostitis of the scapula, pain being marked on contracting the biceps; a housemaid may develop periostitis at the points of bony origin of the great pectoral from the chest, the condition being due to sweeping and scrubbing.†

**Treatment of Osteitis and Osteoperiostitis.**—In syphilitic forms the local treatment consists in rest, elevation of the part, the application of iodine and mercurial ointment, and bandaging. Specific treatment is by the stomach or hypodermatically. Operation is rarely justifiable. In other forms, if the case be recent and severe, put the patient to bed, place the limb in a splint and elevate it, employ cold, apply a bandage, and give salines and iodid of potassium internally. Later use ichthyol inunctions locally and apply a hot water-bag. Morphin is administered for pain. If these means fail, order counterirritation by iodine and blue ointment or blisters, and apply heat locally. In severe cases take a tenotome and slit the periosteum subcutaneously to relieve tension; this procedure often quickly relieves the pain. Some cases demand a longitudinal osteotomy, which is performed by taking Hey's saw and dividing the bone longitudinally into the medullary canal. If pus forms, drain at once.

*Diffuse osteoperiostitis* requires early and free incisions, antiseptic irrigation, drainage, rest and elevation of the limb, and strong supporting and stimulating treatment. Amputation is sometimes demanded, as when the patient grows weaker and weaker even after incision, and when a joint is seriously involved. If the necrosis affects the entire shaft, which separates from its epiphyses, and new bone has not yet formed from the periosteum, make a subperiosteal resection of the shaft.

**Chronic periostitis** is usually syphilitic. A *node* is a chronic inflammation of the deep periosteal layers. Nodes occurring early in the secondary stage remain soft and soon pass away under treatment, but those occurring two years or more after infection are apt to cause a bony deposit. A node may soften, leaving a sinus, at the bottom of which is a piece of dead bone. Gumma of the periosteum is one form of node which is apt to produce caries or necrosis.

**Osteoplastic periostitis** accompanies chronic osteitis and causes the deposit of new bone, which undergoes sclerosis. The chief *symptom* is aching pain, which is worse when the patient is warm in bed, and is aggravated by damp and wet. A swelling is found at the seat of pain (often over the tibia

\* Practitioner, Oct., 1897.

† Ibid.

ulna, clavicle, or sternum). The soft parts are uninfamed and move freely unless softening or suppuration has occurred. Tenderness is manifest.

*Treatment of Chronic Periostitis and Osteoplastic Periostitis.*—For the nodes of early syphilis administer mercury by the plan usually followed in secondary syphilis; for the nodes of late syphilis give mercury and large advancing doses of iodid of potassium. Blisters, blue ointment, and iodin are used locally in both forms, and subcutaneous division of the periosteum is of value. If suppuration occurs, incise antiseptically.

**Chronic Abscess of bone, or Brodie's Abscess.**—This condition is usually due to tuberculous infection. It is always chronic, never acute. A very acute inflammation, such as is induced by pyogenic organisms, causes acute necrosis rather than an acute abscess. After typhoid fever an area of suppuration may slowly form in the head of a long bone, due to the action of typhoid bacilli. After a tuberculous abscess forms mixed infection may take place, the seat of abscess being a point of least resistance. Chronic abscess of bone was first described by Sir Benjamin Brodie, and is often called *Brodie's abscess*. It occurs in the cancellous structure of the ends of bones—usually in the head of the tibia, sometimes in the femur (Fig. 143) or humerus. A tuberculous abscess of bone may follow a slight injury, inducing osteitis, which constitutes a point of least resistance. Bacteria lodge and multiply; bone rarefaction leads to the formation of a cavity, the inflammatory products caseate, sometimes suppuration arises, and the surrounding bone thickens and hardens because of growth from the periosteum. The abscess is apt to break into a joint, as the joint-surface is not covered by periosteum and no barrier of bone is there formed. Brodie's abscess may induce necrosis.



Fig. 143.—Abscess in the great trochanter ("American Text-book of Surgery").

**Symptoms.**—The symptoms are like those of osteoperiostitis, only they are localized and persistent. These symptoms are thickening of the bone and soft parts, edema and discoloration of the skin over the seat of trouble, tenderness, constant pain (subject to violent exacerbations, worse at night when warm in bed, and made worse by motion, pressure, or a dependent position), and attack after attack of synovitis in the nearest joint. Irregular fever and sweats may be noted.

**Treatment.**—In treating bone-abscess, trephine the bone at the point of greatest tenderness, and if the abscess is missed, follow the advice of Holmes and perforate the wall of bone with the trephine, opening in several directions to discover the tuberculous matter or pus. It is often easy to open into the abscess with a chisel or gouge. After opening the cavity scrape its walls thoroughly dry with gauze, touch with pure carbolic acid, and pack with iodoform gauze. If the abscess opens into a joint, trephine the bone and open, irrigate, and drain the joint.

**Caries** was a term used formerly to signify suppuration or molecular death of bone. In some cases caries means suppurative osteitis; in others, tuberculous osteitis; in still others, gummatous osteitis. Typhoid fever is occasionally followed by a carious condition of bone. Osteitis is apt to become purulent when the bone is exposed to the air, when rest is not secured, when the health of the individual is below normal, when a foreign body such as a bullet is in the bone, or when tubercle or syphilis exists. The term is rarely used to-day except loosely, and then usually to signify tuberculous disease of bone. When caries arises, the softened and granulating bone breaks down and is discharged through a sinus. After drainage is secured organization, sclerosis, and healing may result. In these cases new bone may form, and a cure result.

Tuberculous or strumous caries, a condition produced by the caseation of the products of a tuberculous osteitis, shows no tendency to self-cure, no organization or sclerosis takes place, and no new bone forms unless an operation is performed. The interior of bones, especially of the carpus and tarsus, is entirely softened and destroyed, and thin shells only are left.

*Caries necrotica* is a condition in which small but visible portions of soft and dead bone are cast off; *caries sicca* is molecular death of bone without suppuration.

The caseating masses in tuberculous caries contain the tubercle bacillus. If a tuberculous collection is evacuated and infection with pus organisms occurs, genuine suppuration takes place, and constitutional infection causes septic fever, and may cause death. Purulent osteitis may affect any part of any bone; but caseous osteitis (tuberculous caries) tends to arise especially in cancellous structures (heads of long bones, vertebral bodies, ribs and sternum, and bones of the carpus and tarsus). Tuberculous osteitis of the shaft of a long bone occasionally, but rarely, arises. Tuberculous osteitis is apt to cause tuberculous disease in an adjacent joint. Tuberculous osteitis may be followed by the formation of a cold abscess.

**Symptoms.**—In the beginning the evidences of caries are usually those of osteitis, but the first sign noted may be a fluctuating swelling due to pus or to caseated tubercle. After a time, at any rate, a fluctuating swelling is discovered. If not opened, the softened mass breaks externally, voids its contents, and leaves a sinus from which flows caseated matter which after a time becomes thin, reddish, and irritating to the skin, contains small portions of gritty bone, and has a foul smell. The opening of the sinus fills up with edematous granulations. A probe carried to the bottom of the sinus finds bone which is sieve-like (worm-eaten), and which on being struck gives a muffled note rather than the clear, sharp note of necrosis; the bone is rough, is bared, and is so soft that the probe can usually be stuck into it. In old cases of caries amyloid disease may arise.

**Treatment.**—If syphilis exists, give iodid of potassium in advancing doses and a mild mercurial course. If tubercle exists, give iodid of iron, arsenic, cod-liver oil, and nourishing foods, and recommend a change of air. Locally, in all cases, insist on rest and at once secure drainage, enlarging the opening, if necessary, and inserting a tube, and even making additional openings; syringe often with antiseptic fluids and dress antiseptically. If the case is seen before spontaneous evacuation has occurred, open under strict antiseptic

precautions. When a chronic sinus exists there arises the question of operation. Incomplete operations are worse than useless, for they may be followed by diffuse tuberculosis or pyemia. If the gouge is used, try to remove *all* carious bone. The diseased bone is white, crumbles, and does not bleed; the non-carious bone is pink and vascular. Scrape away all granulations; swab the cavity with pure carbolic acid and pack it with iodoform gauze. Instead of gouging away bone, there may be used the actual cautery, sulphuric acid, or hydrochloric acid. In severe cases excision is required, and in some rare cases amputation may be necessary. Caries of the spine is considered under Diseases of the Spine.

**Necrosis** is the death of visible portions of bone from circulatory impediment. It is analogous to gangrene. One cause of necrosis is traumatism (such as the tearing off of periosteum) which deprives the bone of blood. Inflammation of the periosteum further lessens the nutrition. Acute inflammation in bone causes necrosis, the excessive exudation in the canals and spaces occluding the blood-vessels by pressure. The occlusion of vessels by septic thrombi may lead to necrosis, or the direct action of toxins may first inflame and finally destroy a portion of the bone. A thin shell of bone only may necrose from periosteal separation, or an entire shaft may die from acute pyogenic osteomyelitis or diffuse infective periostitis. Osteomyelitis is the most usual cause of necrosis. Necrosis is most frequently met with in the diaphyses of the long bones, caries in the cancellous tissue of bones. The ribs may become carious, but very rarely become necrotic. A sequestrum may form in a vertebral body, in the carpus, or in the tarsus, but rarely does; hence, we conclude that sequestra do not often result from tuberculous osteitis. A fragment of dead bone is a foreign body; the healthy bone adjacent to it inflames and softens; granulations form, and this line of granulation, like the line of demarcation of gangrene, separates the dead part from the living, the white dead bone being surrounded by the red zone of granulation tissue. A bit of dead bone is called a "sequestrum," and Nature tries to cast it off. A superficial sequestrum is known as an "exfoliation."

Nature's method of casting off a sequestrum is as follows: suppuration takes place at the line of demarcation, osteitis extends for a considerable distance around this line, the periosteum shares in the inflammation, and new bone forms. A cavity is thus made within by suppuration, and a box or case forms without by ossification, the now entirely loosened sequestrum being so encased that it cannot escape. The pus finds its way through the new bone, and there is presented the condition so often seen by the surgeon—namely, a case of new bone known as the "involucrum," a cavity containing pus and the dead fragment or sequestrum, and a discharging sinus or "cloaca"

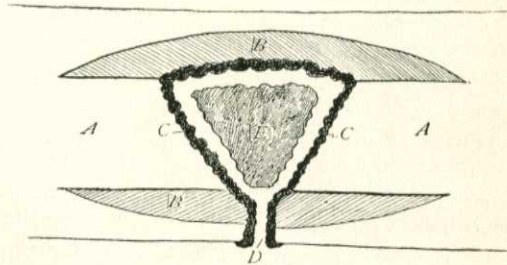


Fig. 144.—Diagram illustrating the formation of a sequestrum: *A*, Sound bone; *B*, new bone; *C*, granulations lining involucrum; *D*, cloaca; *E*, sequestrum.

(Fig. 144). Nature may eventually get rid of the fragment, but the surgeon should not wait for the completion of this slow process.

When a portion of the bone surrounding the medullary canal dies, the condition is called "central necrosis." In some rare cases necrosis occurs without apparent suppuration, a painless swelling of bone simulating sarcoma. This condition is known as *quiet necrosis*, and has been described by Sir James Paget and Mr. Marrant Baker. Mercury is an occasional cause of necrosis. The fumes of phosphorus may cause necrosis of the lower jaw in those with decayed teeth. Necrosis may be produced also by frost-bites and burns. Many fevers (measles, typhoid, scarlet fever, etc.) are occasionally followed by necrosis. Syphilis and tuberculosis are occasional causes.

**Symptoms.**—The symptoms of necrosis are at first those of osteitis or osteomyelitis. The abscess, when formed, opens of itself or is opened by the surgeon, and a sinus or sinuses form in the soft parts as happens in caries. A probe introduced into the sinus strikes upon hard bone with a clear, ringing note, and often finds a sinus or sinuses in the bone. In superficial necrosis the discharge is slight and the probe shows the limitations of the disease. In extensive necrosis the discharge is profuse, much new bone forms, several sinuses appear far apart, and the probe must pass through a considerable thickness of new bone before it finds the bit of dead bone. The surgeon should not operate until the dead bone is separated from the living by a line of demarcation, and until the sequestrum is loose. In youth dead bone loosens quickly, but in old age slowly. An exfoliation becomes loose sooner than the sequestrum of central necrosis. In diffuse periostitis the necrosed shaft loosens quickly. Necrosed portions of the upper extremity loosen more rapidly than those of the lower. In a young adult two or three months will be required to loosen a necrosed fragment in the lower extremity, and from six weeks to two months in the upper. A loose sequestrum may be moved by the probe, and when struck gives a hollow note. In protracted cases of necrosis there is always danger that amyloid disease may arise.

Quiet necrosis is a rare condition which has led to some deplorable but pardonable mistakes, because it resembles ossifying sarcoma. It follows injury, particularly fracture. The bone enlarges greatly. There is little or no pain and no fever. The diagnosis can only be made by exploratory incision, and it may even be necessary to remove portions for microscopic study before a conclusion can be reached.

*Postfebrile necrosis* is most usually met with after typhoid fever. The bacilli of typhoid cause osteomyelitis, and this is followed by necrosis. Scarlet fever, measles, and other febrile processes may also induce necrosis. It is certain that bacilli accumulate in the bones during typhoid fever. They may promptly induce disease; they may remain for long periods apparently inactive and finally pass away; or after a slight strain or injury these organisms may induce bone disease months or even years after the primary infection. *Typhoid bone disease* is often multiple, many bones being involved successively.\* Not unusually after typhoid fever muscle strain causes periostitis and osteitis, and at such a point necrosis may occur. Either exfoliation or central necrosis may follow typhoid fever. The tibia is involved more often than other bones.

\* Keen's "Surgical Complications of Typhoid Fever."



**Treatment.**—An exfoliation should be removed as soon as it becomes loose, the seat of trouble should be touched with pure carbolic acid, and packing of iodoform gauze should be inserted. The treatment of central necrosis comprises free incisions for drainage, antiseptic dressing, frequent cleansing, rest, nourishing food, stimulants, and tonics. When the sequestrum becomes loose, the involucrum should be broken through with the chisel, gouge, and rongeur. The dead bone should be removed and the cavity scraped, irrigated with hot salt solution, dried, painted with pure carbolic acid, and packed with iodoform gauze. This operation is known as "sequestrotomy." The simple removal of a sequestrum—*i. e.*, the operation of sequestrotomy—often fails to effect a cure. "The involucrum always contains pyogenic germs that may live in its small foramina and crevices almost indefinitely. For this reason, and on account of the denseness of bony structure, it is well-nigh impossible to disinfect it" (Dr. J. Shelton Horsley, in the "Medical Record," Oct. 20, 1900). Because of the difficulty of curing a case when an involucrum has formed, Dr. Cushing, of Baltimore, has warmly advocated early operation in osteomyelitis; that is, operation before an involucrum has formed, and when the osteoblasts of the periosteum are extremely active. He points out that if an involucrum has formed, the sequestrum and involucrum should be removed after stripping the periosteum from this region. If the periosteum is found not to be infected, it may be stitched together at the gap where the bone has been removed, so that a periosteal cord exists between the two ends of the bone; and the soft parts above this may be closed. If the periosteum is found to be infected, we agree with Cushing that the cavity should be packed with gauze. The cavity that is left by the removal of a sequestrum and the chiseling of the walls of the involucrum, if large, may be filled by various methods, more or less satisfactory.

The surgeon may try to fill it by taking flaps of skin and fastening them to the bottom with nails (Neuber's operation), by breaking the edges of the involucrum and turning them in, or by inserting bone-chips. Bone-chips are obtained from the compact part of the tibia or femur of an ox, and are decalcified by being placed for a couple of weeks in a 10 per cent. aqueous solution of hydrochloric acid (which is renewed every day); they are well washed in a weak alkali and then in water, are cut into strips, are soaked for two days in a 1 : 1000 solution of corrosive sublimate, and are kept until needed in a saturated ethereal solution of iodoform. The cavity is made sterile and is well dusted with iodoform, the bone-chips are dried and inserted into the cavity, a capillary drain is employed, the periosteum is stitched over the opening, and the soft parts are sutured; but if this cannot be done, iodoform packing is used to keep the chips in place. This method we owe to the genius of Senn. Attempts have been made to fill bone-cavities with gutta-percha, plaster of Paris, etc. Schleich uses formalin-gelatin to fill bone-cavities. The difficulty is to completely aseptinize the walls of the cavity. Dressman has advised for this purpose the use of boiling oil, but it is apt to cause superficial necrosis. In some cases the cavity has been healed by the insertion of a Thiersch skin-graft. This method has been advocated by J. P. Lord ("Jour. Am. Med. Assoc.," May 31, 1902). Many attempts have been made to fill the defect by bone-grafting. The first case of satisfactory transplanting from the lower animals with the leaving of a vascular attachment was reported by

A. W. Morton in "American Medicine," July 12, 1902. The patient suffered from a compound comminuted fracture of both bones of the right leg. The fracture in the fibula united, but the tibia underwent necrosis, and it was necessary to remove five inches of the lower end of the bone. Some days later, the periosteum was raised from the ends of the bone and these ends were freshened. The left leg of a dog was amputated just above the tarsus, the bones being sawed so that the ulna was one inch longer than the radius. The lower end was partly bared of periosteum, and the ulna of the dog was forced into the cavity of the tibia of the man, and wired to that bone with silver wire. The incision in the man's leg was then sutured, and powerful tendons in each leg of the dog were divided. Each of the dog's other legs was wrapped separately in a plaster of Paris bandage, and the entire animal and the leg of the man were then put up in a plaster of Paris dressing. Five weeks later the cast was removed, and the bones were sawed and placed in contact with the astragalus. Union took place, and the man was fortunate enough to obtain a useful leg. In some cases of widespread necrosis due to diffuse infective osteoperiostitis or to osteomyelitis extensive resection, or even amputation, may be necessary.

**Acute osteomyelitis** is an acute and diffuse inflammation of the bone-marrow due to pyogenic organisms. Infection from staphylococci may be limited to a portion of one bone. Streptococcus infection causes widespread involvement of a bone or of several bones. Acute osteomyelitis may be due to mixed infection with bacilli of typhoid and pyogenic organisms, or bacilli of tubercle and pyogenic organisms, a typhoid process or a tuberculous process serving to establish a point of least resistance. The gonococcus and the pneumococcus occasionally produce acute osteomyelitis. In a case of gonorrhoeal arthritis in which I resected the wrist-joint cultures of gonococci were obtained from the interior of the bone removed.

The pyogenic organisms may gain entrance directly by way of a wound (a gunshot-wound, a compound fracture, an amputation). The causative organisms may reach the bone by way of the blood, having entered the blood originally through the lymphatic system or from a focus of suppuration in the skin, the subcutaneous tissue, or a deeper part.

Pus organisms may pass into the blood from the tonsils or respiratory organs (Kraske); the intestinal canal (Kocher); the genito-urinary tract; or from excoriations, bruises, or small wounds in the skin (Warren). Certain fevers strongly predispose to the disease by preparing the soil as it were for the growth of pyogenic bacteria. Typhus fever, smallpox, malarial fever, scarlet fever, measles, and diphtheria lessen the vital resistance of bone-marrow. Typhoid fever is not unusually followed by a chronic osteomyelitis, due solely to typhoid bacilli. If mixed infection with pus organisms occurs, acute osteomyelitis arises. Vital resistance of marrow is lessened by exhausting diseases, overexertion, unhealthy and especially putrid food. When organisms gain entrance directly by a wound (as in a compound fracture), the endosteum, the medulla, and the cancellous tissue inflame and suppurate, and the entire length and thickness of the bone may be involved. The periosteum becomes infiltrated, detached from the bone, and retracted from the edges of the wound in the bone. The soft tissues around the bone may inflame, suppurate, or slough. More or less necrosis inevitably occurs.

Acute osteomyelitis without a wound is often called acute epiphysitis or acute infantile arthritis. This condition is most common in infants or children of one or two years of age, but occasionally arises in older children (from ten to fourteen years) or even in adults. It is most common during the period of active growth of bone. It is frequently preceded by one of the predisposing causes before mentioned. In many cases a strain or bruise is followed by pyogenic infection, because the damaged tissue extends a hospitable welcome to micro-organisms which are traveling in the body-fluids and pass through the injured area. In some cases chilling of the surface of the body is a predisposing cause. In others no predisposing cause is discoverable.

The compact bone suffers secondarily, but is never attacked primarily. New tissue is more susceptible to infection than old tissue, and the disease, as a rule, begins near the epiphyseal line, where new bone is being formed. This point was spoken of by Ollier as "the zone of election of pathological processes." Warren points out that in a growing bone near the epiphyseal cartilage there exists a newly formed spongy tissue, very vascular and connected with the cartilage by a spongy layer of tissue, which is not yet bone, but which does not possess a cartilaginous structure. It is in this portion of the skeleton that the most active changes take place during the period of growth. The medullary substance is very vascular at this point; it is red and without fatty tissue. It communicates with the medullary canal and with the periosteum by a number of vascular channels. The epiphyseal cartilage itself is intimately blended with the periosteum. The diaphyseal side of the cartilage produces much more bone than is found in the epiphyseal margin. There is also an active growth of bone in the periosteum, and it is in these regions and in the medullary canal that the inflammatory process originates.\* The lower end of the femur and the upper end of the tibia are the regions most commonly attacked; but the upper end of the femur and the lower end of the tibia may suffer, and other bones may be attacked, especially the humerus, radius, ulna, and inferior maxilla. The adjacent joint not unusually becomes involved. Though the inflammation begins in the spongy tissue or medulla, it passes to the canals and spaces of the compact bone. The inflammatory exudate in the canals compresses the vessels and cuts off nutrition from certain areas. Suppuration begins, clots form in the medulla from thrombophlebitis, and the clots in the vessels of the Haversian canals become septic. A small sequestrum forms at the seat of origin of the disease, and the pus about the sequestrum is apt to empty into the medullary canal, causing diffuse osteomyelitis, or into the adjacent joint, causing suppurative inflammation of the articulation.

Marked constitutional symptoms arise from absorption of toxins (suppurative fever), and sometimes true septic infection or even pyemia arises.

Very extensive necrosis may follow osteomyelitis if the patient recovers.

**Symptoms.**—Osteomyelitis secondary to a wound may occur in a person of any age. If a wound exists,—for instance, a compound fracture,—the diagnosis is evident. The constitutional symptoms of septic absorption are positive: there is a profuse, offensive, purulent discharge containing bone-fragments and tissue-sloughs; the periosteum is red, thick, and separated; there are swelling over the bone, great tenderness, and violent boring, gnawing,

\*Warren's "Surgical Pathology."

or aching pain. Osteomyelitis occurring without a wound, the condition known as acute epiphysitis, occurs in the young, and particularly in children under three years of age.

The symptoms of acute epiphysitis usually come on suddenly and especially at night, and the attack may be so acute as to cause death by systemic poisoning before a diagnosis is arrived at. The disease is generally ushered in by a chill, which is followed by septic febrile temperature. The history will sometimes contain the statement that a blow had been received, that a febrile process had existed, or that the patient had been suddenly chilled after having been overheated (sitting in a draft or in a cellar on a hot day, possibly swimming when very warm, etc.). There is violent aching pain in the bone and acute tenderness near the joint; the soft parts, which at first are healthy in appearance, after a time discolor, swell, and present distended veins, and may become glossy and edematous because pus is gathered below. An abscess sometimes reaches the surface and may break spontaneously. The neighboring joint swells, and may become filled with pus; the periosteum and the shaft are involved for a considerable distance; each epiphysis may become affected, the shaft between being comparatively uninvolved, and the epiphyses may separate, displacement and shortening taking place. This disease is often mistaken for rheumatism because of the joint-swelling, occasionally for typhoid fever because of the fever, and in some cases for erysipelas because of the redness of the skin. It gives a very grave prognosis. Sometimes an epiphysitis shows milder symptoms and is slower in progress (subacute). These cases are very often mistaken for rheumatism. But in rheumatism the joint is the part involved from the beginning, while in epiphysitis the joint is involved secondarily after obvious evidence of inflammation well clear of the articulation. Further, the symptoms of rheumatism will be rapidly improved by the use of the alkalies or the salicylates.

**Treatment.**—If a wound exists, apply a tourniquet, sterilize the parts, enlarge the wound, expose and curet the medullary cavity, remove loose fragments of bone, irrigate the medullary cavity with a hot solution of corrosive sublimate or hot salt solution, scrape it with bits of gauze held in the bite of a forceps, paint with pure carbolic acid, pack lightly with iodoform gauze, dress with hot antiseptic fomentations, and secure rest for the parts by splints and bandages. The constitutional treatment is the same as that for septicemia. Acute osteomyelitis without a wound is a most serious condition, rapidly progressive, apt to be quickly fatal, and requiring prompt and radical treatment. In treating it do not wait for fluctuation, but incise at once; break through the bone at one or more points with a gouge or chisel; chisel away the diseased bone, and if necessary curet the medullary canal; irrigate with hot corrosive sublimate solutions or hot salt solution; swab with pure carbolic acid; use iodoform plentifully; pack with iodoform gauze; dress with hot antiseptic fomentations; drain the joint if it is involved; employ rest, anodynes, strong supporting treatment, and other remedies advised in septicemia. Remove dead bone subsequently when it becomes loose. Amputation may be required in either form of the disease.

**Chronic osteomyelitis** is usually linked with osteitis. It may eventuate in osteosclerosis with filling up of the medullary canal, in limited suppuration, in caseation of the cancellous tissue (Brodie's abscess), or in

necrosis. A tuberculous inflammation is one form of chronic osteomyelitis. Syphilis, typhoid fever, etc., may cause it, and it can be caused by glanders, leprosy, and actinomycosis.

The typhoid bacillus is pyogenic. Fränkel taught this some years ago, and Keen seems to prove it in his work on the surgery of typhoid fever. Osteomyelitis due purely to typhoid bacilli is chronic. When the medulla contains typhoid bacilli pus infection is apt to take place, and if such a mixed infection arises acute osteomyelitis develops.

In chronic osteomyelitis there are pain, tenderness, and swelling, but no marked constitutional symptoms. In some cases the real trouble is not identified until an abscess forms (see Necrosis).

**Treatment.**—If an abscess exists, at once evacuate it by incising the soft parts and chiseling the bone. Do not wait for an involucrum to form, but incise and disinfect promptly, and drain. If dead bone is present it must be removed.

**Osteomalacia, or Mollities Ossium.**—In this disease the bones are partly decalcified, and consequently soften and bend. Masses of new uncalcified bone-tissue are formed. Many bones are usually involved, but the head is not obviously affected. It is commoner beyond than before middle age, though it may occur in infancy; it is more frequently met with in women than in men, and pregnancy seems to bear more than a casual relation to its production. In osteomalacia the medulla increases in bulk and becomes more fatty, and the osseous matter is absorbed gradually, first from the cancellous tissue and then from the compact tissue. Some observers believe that this curious condition is due to lactic acid in the blood, an abnormal amount of acid having been produced and absorbed because of disorder of the primary assimilation. Volkmann asserts that some inflammatory condition disturbs the blood-supply of the medulla, and von Recklinghausen asserts that arterial hyperemia is responsible.

**Symptoms.**—The symptoms of osteomalacia are as follows: many points of pain which are often thought to be due to rheumatism; deformities from twisting and bending of bone; and a large excess of calcium salts in the urine. Fractures occur from very slight force. In the majority of cases the disease is not cured, but grows progressively worse until the patient dies, after many years, from exhaustion. In some cases the process is arrested and the osteoid tissue is calcified.

**Treatment.**—In treating osteomalacia in women insist that pregnancy must not occur. Put braces and supports upon distorted limbs to prevent fracture. Advise hygienic surroundings and nourishing food, and insist on the value of fresh air. Among the medicines that can be used may be mentioned cod-liver oil, lime salts, preparations of phosphorus, and bone-marrow. In women the removal of the ovaries sometimes produces cure. It has been asserted that the production of anesthesia by means of chloroform may be of benefit.

**Acromegaly.**—This is a disease which causes progressive and often great enlargement of both the bones and soft parts of the extremities, which enlargement is symmetrical. The cranium is triangular in shape, with the base below. The lower jaw projects in advance of the upper jaw, the nose becomes prominent and thick, the supra-orbital ridges are accentuated, and

the costal cartilages and inner ends of the clavicles become protuberant. Later the larynx, ribs, shoulder-blades, and vertebræ become involved, and the back becomes markedly humped (cervicodorsal hump). The hands and feet are affected in advanced cases. As a rule, the thyroid gland is enlarged, and a post-mortem examination may detect an enlarged pituitary gland. Severe and uncontrollable headache is sometimes a distressing feature of the disease. Treatment is futile. The disease slowly but surely causes death.

**Leontiasis Ossium (Virchow's Disease).**—This is a symmetrical hypertrophy limited to the facial and cranial bones, and which begins, as a rule, in the superior maxillæ. The hypertrophy progressively increases, causes difficulty of mastication, and is accompanied by headache. It produces distinct deformity of the jaw like a tumor, whereas acromegaly enlarges all of the proportions of a bone (Fig. 145). It may produce blindness, new bone pressing upon the optic nerves. Treatment is not satisfactory, as a rule. Recently Horsley has obtained amelioration by operating and removing masses of bone.

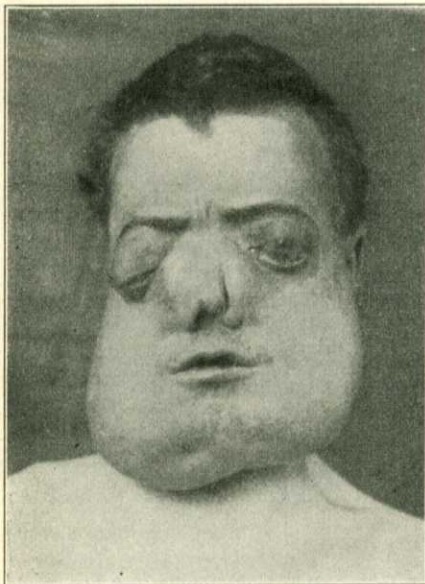


Fig. 145.—Leontiasis ossium.

**Ostitis Deformans (Paget's Disease).**—This disease was first described by Paget in 1877, and in the neighborhood of 100 cases have been reported. Packard and Steele ("Amer. Jour. of Med. Sciences," Nov., 1901) point out that many of the reported cases are not genuine instances of the disease, some being ordinary osseous tumors, others being cases of enlargement after fracture, and still others being instances of mollities ossium. They think that 67 of the reported

cases are genuine instances of the disease. In this disease great quantities of new bone are formed, but calcification does not occur. The material undergoes absorption, and the medullary substance of the bone becomes extremely vascular and filled with white blood-cells, and also with giant-cells. The fact that the new bone does not calcify leads to various deformities of the long bones, on account of the weight of the body; but fracture is not particularly apt to occur. Numbers of bones may be decidedly thickened. The underlying cause of this curious condition is entirely unknown, but it is assumed to be trophic. It is claimed that it has occasionally arisen after an injury to a long bone, and has been excited into activity by heat and cold. It is extremely rare before the age of forty, and usually begins between forty and fifty. The enlargement of the bones may be first detected in the cranium, but is more often first seen in some other bone

—for instance, the clavicle, the tibia, the spine, or the radius. In fact, in some cases the bones of the head do not enlarge at all; but, taking all the reported cases, the skull is affected more frequently than any of the other bones. In some cases, the enlargement of the bones seems to be symmetrical; in others, it is not. In the disease known as leontiasis ossium, the chief enlargement is manifested in the face; in Paget's disease there is no enlargement of the bones of the face, or else these bones are trivially involved. Packard and Steele point out that the diagnosis is extremely difficult when but a single bone is involved; but that if two or more bones are involved, we should think of Paget's disease as the condition, especially if we are able to exclude syphilis, cancer, and sarcoma. In mollities ossium the head is not involved at all; and there is not nearly so much thickening of the bone. The two authors before quoted show that in acromegaly the cranium is a triangle with its base below the lower jaw, the orbital arches being chiefly involved; but that in Paget's disease the involvement is chiefly of the calvarium. In this curious malady there may or may not be pain. The patient actually diminishes in height. The chest becomes deformed. There is angular curvature in the dorsocervical region. The lower extremities are usually bent; and the pelvis, as a general thing, is broadened. In the 67 cases collected by Packard and Steele, 3 suffered with cancer and 5 with sarcoma.

**Treatment.**—Treatment is practically useless. No known expedient diminishes the size of the bones, although iodid of potassium is said occasionally to mitigate the pain.

## 2. FRACTURES.

**Definition.**—A fracture is a solution, by sudden force, of the continuity of a bone or of a cartilage. Clinically, under this head are placed epiphyseal separations and the tearing apart of ribs and their cartilages.

**Varieties of Fractures.**—The varieties of fractures are as follows:

*Simple fracture* is a subcutaneous fracture, or one in which there is no

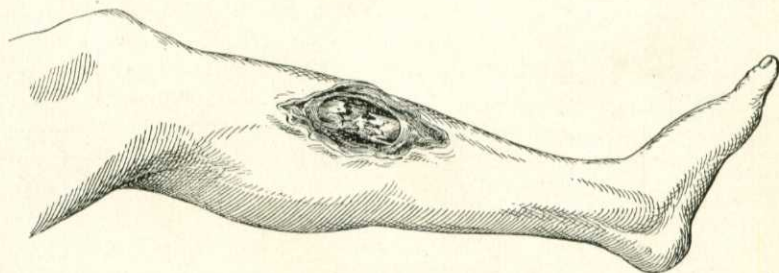


Fig. 146.—Fracture of the leg complicated with wound and comminution of the bone.

wound extending from the surface to the seat of bone-injury. This corresponds to a contusion of the soft parts.

*Compound fracture* is an open fracture, or one in which an open wound extends from the surface to the seat of bone-injury or in which a wound opens up a passage from the fracture to the surface. This corresponds to a contused or lacerated wound of the soft parts (Fig. 146). The opening may be through