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Non-infectious diseases affecting the epiphyses

Fred F. Hawkins
University of Nebraska Medical Center

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By

Fred B. Hawkins

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In order to obtain an adequate appreciation of the clinical significance of the epiphyses one should reflect that injuries to, or diseases of these centers of ossification are responsible for the majority of deformities and abnormalities of growth of the extremities which arise during the growth period.

The disease factors are many and may be roughly divided into: (a) those in which all epiphyses are affected or subject to the effects of the same generalized deficiency or dyscrasia - examples of this are the hormonal pathologies such as Gigantism, Dwarfism, and Cretinism, and those due to vitamin deficiency, most important of which are rickets and scurvy; and (b) those of localized character, which group includes the neoplastic, neurogenic, infectious and vascular factors.

It is the localized, non-infectious diseases with which we are concerned and in particular that disease called osteochondritis for want of a better name. "Osteochondritis" is used far more loosely than the term "epiphysitis". The latter term assumes that the epiphysis is present and that an inflammatory condition is being manifest. Osteochondritis infers nothing but in itself explains that the inflammation involves both cartilage and bone; the most natural place for such to occur is, of course, the epiphysis.

The pathological process is common to all centers of ossification in the long bones and runs a similar course in all epiphyses, but manifests itself at the age of greatest growth for that particular

epiphysis.

It is perhaps elementary to attempt to describe a typical epiphysis anatomically but it may possibly be of some interest. The epiphysis is made up of six layers, first of which is the articular cartilage which is so frequently involved in osteochondritis dissicans. The second layer is called the bony epiphysis and is in reality the terminal or articular portion of bone. The third and fifth layers are made up of spongy bone and transitional cartilage cells which will be replaced by bone cells of the future. The cartilage plate, or fourth layer, lies between these two sections and is the all important one as regards growth. The end of the shaft or metaphysis is the sixth or final layer.

The earliest description of non-infectious diseases affecting the epiphyses was probably by Vogt in 1869 and next by Muller in 1888. However, to Osgood in 1903 goes the credit for the first published article on an osteochondritic disease. Since then the literature has become voluminous and confusing, mainly because of the failure to recognize and agree upon the etiological factors, the multiplicity of terms used to describe the condition and the scant material available for pathological examination.

March fracture will be included in the discussion of the various diseases. Perhaps this should be considered as an injury to the epiphysis but it is the belief of most investigators that the fracture occurs as a consequence of being primarily a diseased bone. For this reason I feel that it may justly be included in this thesis.

Each particular disease will be considered separately in an attempt to describe it in a complete and concise manner. A general summary of the most noteworthy etiological factors will conclude the discussion.

March Fracture - Pied Force, Deutschlander's Disease, Fussgeschoulst, and Syndesmitis Metatarsus.

March foot is described by D. & M. F. Sloane as being an edematous, painful swelling of the forefoot associated with an insidious and often unrecognized fracture of one of the metatarsals. (92)

The earliest cases were reported by Breithaupt, a German military surgeon, in 1855, who considered the disease to be an inflammatory reaction within the tendon sheaths resulting from trauma. He gave the name Fussgeschoulst to the disease. In 1877, Weisbach, another military surgeon of Germany, described a similar condition which evinced itself two to three days following a route march of a young soldier. He agreed with Breithaupt in that it was traumatic in nature and a direct sequel of untoward strain. He thought, however, that it affected the ligaments rather than the tendons or their sheaths and applied the term "syndesmitis metatarsus." In 1887, Pouzat put forward the hypothesis that the causal factor was to be found in the dorsal fold of the soldier's shoe. One may clearly see that it was not recognized as being a disease of civilian life. Pouzat believed that the first stage was that of simple swelling of the soft tissue but this was followed by periosteal proliferation resulting in definite osteoplastic periostitis. To this condition,

he applied the title "pied force."

In 1888, Poulet, also speaking from experience in the French army, suggested the rheumatic diathesis as the predisposing factor, an hypothesis in keeping with the modern trend of thought.

A review of the whole subject was published in 1897 by Busquet in which he divided the cases into two categories, first, traumatic, and second, diathetic, but even in this latter group he admitted that whereas predisposition, the result of some generalized condition, might exist, the localizing and exciting factors were traumatic.

With the advent of roentgenology much that was previously pure hypothesis was swept away. In an examination of thirty five patients by Stechow in 1897, who was the first to employ radiographs in diagnosis, the lesion behind march foot was proven to be a fracture of one of the metatarsal bones. In the same year Schulte, in reporting a series of fifty-three cases, stated that in all the causal factor was a fracture. In 1899 Thiele reported on seventeen cases which occurred in a single battalion in three and one-half months. Roentgenological examination again showed the presence of a fracture in fifteen of the seventeen cases. He considered the lesion to be the result of indirect force and drew attention to what he claimed was the sequence of events. He claimed that prolonged flexion of the bones lead to an inflammatory reaction within the neck and shaft of the metatarsal, and in those instances where the force continued to act, a fracture occurred.

The etiological background of this condition is by no means definite. Deutschlander in 1921, in his second contribution to study of this subject, suggested a possible bacterial etiology of the condition. He based his views on six cases, all of whom were women. In three cases, the fever was of low grade, and the formation of callus, normally present in three to four weeks, did not appear for nine weeks. He considered that the cause in those three cases was hematogenous periostitis of low grade. Although Deutschlander's name has been given to march foot because of his early work on the subject, research has shown that Breithaupt was the original investigator in this field.

D. and M. F. Sloane in 1936 suggested that the exciting factor was definitely multiple traumata produced by pounding about on flat feet in which the circulation was notoriously poor. Added to this state there is protective spasm of the interossei muscles, as has been reported by Murk Jansen, which further decreases the blood supply to the metatarsals by compressing the perforating vessels. The metatarsals then become brittle and friable and crack under the strain of walking, which, if continued, is accompanied by massive callus formation. In the series of cases which the Sloanes report all had flat feet. This is an especially interesting feature since it was not mentioned as a constant factor in other literature. (92)

Bruce and Watson Jones have suggested the developmental influence of metatarsus atavicus, a congenital shortening of the

first metatarsal so that the longer second metatarsal must assume the role of the first in providing a fulcrum for the take-off in walking. Watson Jones (102) feels that this is especially important in the possibility of recurrence of the fracture.

Myerding and Pollock (73) have suggested that fatigue of the muscles and ligaments, rapid increase of weight, and large badly fitted shoes are also important predisposing factors.

Perhaps the most acceptable and broadest version is that of Zeitlin and Odessky who concluded that the march fracture occurred as a result of overloading a foot already weakened functionally and anatomically. The number of hypotheses advanced to explain this condition indicates the uncertainty which still shrouds its origin. Inflammatory factors doubtless are present in a small proportion of cases. In others, neurogenic factors doubtless play a part, but without doubt in the greater majority of cases the condition is the result of trauma of a greater or lesser degree to feet of which the functional sufficiency is well below par.

Clinically one finds the outstanding sign to be a painful swelling of the forefoot (of a flat footed individual — Sloane) of an individual who walks a great deal. A history of direct trauma is usually lacking. The shaft of the metatarsal is very tender upon direct pressure and upon examination it is found to be either the second, third, or fourth metatarsal, never the first or the fifth. Occasionally two or more may be involved and rare instances have been reported of bilateral march fractures. (92) The condition does not fully incapacitate the patient and he limps about painfully.

Nature tries to protect by the formation of an exhuberant callus. This results in a hard tumor-like mass about the fracture. If first seen at this time, it may be mistaken for a bone tumor.

The diagnosis is often difficult if this condition is not kept in mind as a possibility. Stammers of England (96) says that diagnosis by X-ray is not dependable until late so clinical signs and history, with special concern for occupation, is very important. The history is that of spontaneous discomfort in the forepart of the foot developing within an hour or so into frank pain upon any attempt at weight bearing, local tenderness in the interosseus space followed within a day or so by puffiness of the space which spreads to involve the whole dorsum of the foot burying the extensor tendons, and complete freedom from pain upon resting the foot. With this historical background in addition to the accompanying aforementioned signs and symptoms even in the absence of immediate x-ray findings, the evidence should make one very suspicious of a subperiosteal crack of one of the metatarsals and a radiograph should be repeated each week for three weeks or until definite diagnosis is made.

Mistaken diagnoses are frequent and often disastrous. Watson Jones cites a case in which the wife instituted divorce proceedings when she was told that the lesion on her husband's foot was syphiletic periostitis. Strauss (1932) (98) reported a case in which the patient was advised to have an amputation of the leg for sarcoma. Finally "conservative" removal of the

of the metatarsal was employed and the lesion was found to be a march fracture. Dodd (1933) (28) reported the case of a woman who had a classical Lisfranc amputation of the right foot performed for a march fracture of the second metatarsal which was mistakenly diagnosed as a bone sarcoma.

Treatment resolves itself to that of any other fracture of the metatarsals without displacement. The ideal therapy is that of absolute rest of the foot and heat in the form of infrared or diathermy. Since the injury is not completely incapacitating in many instances, it is necessary to prepare an ambulatory means of fixation. Specially made arch supports are effective as are splints of tongue depressors applied with adhesive tape. Wide toed, well fitted, heavy soled shoes with a square heel and the aid of a crutch or cane enables the patient to get about with minimal discomfort and adequate support.

One of the most widely used and most satisfactory means of fixation in both the physician's and patient's opinion is the light walking plaster which is applied from below the knee to the toes. The light plaster cast holds the metatarsals firmly and prevents too great a degree of proliferative periostitis.

As suggested by the Sloanes, treatment must be instituted to correct the pes planus deformity when present if recurrence of march fracture is to be prevented.

Osgood-Schlatter's Disease

In 1903 this disease was first described by R. B. Osgood in the Boston Medical and Surgical Journal (77) to be followed within a few months by a report by C. Schlatter in a German Medical Journal. Osgood's original report cited ten cases which affected boys between the ages of thirteen and sixteen years in which the clinical histories were much the same. All of the boys were athletically inclined and well developed muscularly. In running or in some other athletic sport the knee was "strained", in most cases precipitated by a sudden violent extension of the leg. More rarely there was associated a fall on the flexed knee. At the time of the injury there was often slight swelling fairly well localized in the region of the tibial tubercle. There was distinct local tenderness at that point. The ability to use the leg was only slightly diminished and the acute pain was soon replaced by a feeling of weakness upon exertion. Sharp pain was induced by violent extension or extreme flexion of the leg. The condition presented no complete loss of function but a severe handicap to the active, athletic lives which this class of patients wishes to lead. These observations made by Osgood in 1903 were very remarkable and since that time research has led to little progression, especially in the quest for definite etiology.

The ossification of the tibial epiphysis occurs mostly between the twelfth and fifteenth years. Between the first appearance of the bone nucleus and the completion of ossification not more

than three to four months are required. Only a thin cartilage zone remains between the tibial tubercle and the diaphysis up to about the eighteenth year. The epiphysis sends a process over the anterior surface of the head of the tibia while from below an independent nucleus grows against it until at the level of the epiphyseal disc the fusion occurs.

Regarding the etiology, Schlatter, in his work prior to 1903, concluded that there are extrinsic factors which contribute to Osgood-Schlatters disease, for instance, that the ossifying epiphysis is easily exposed to direct or indirect traumatism especially at the point of junction between the upper and lower epiphyses. Thompson reports a periostitis at the point of insertion of the ligamentum patellae caused by a pull of the quadriceps. Keinbock, upon the basis of his radiological findings, concludes at least for some of the cases that there exists an active destruction of the bone of the tuberosity together with an inflammatory reactive swelling, or in other words an osteochondritis. Ebbinghaus considers the condition to be an osteochondritis dissicans.

Bergeman pointed out that aside from the inflammatory changes, pathological changes of ossification analogous to those seen in late rickets are to be found. Similarly, Fromme in a comprehensive clinical review found that in a number of cases with late rickets the patients showed signs of Osgood-Schlatters disease, deducing from it that this disease occurs in the majority of patients

afflicted with late rickets. Hinrichs, in agreement, points out that if severe changes are seen in long bones in regards to ossification and growth in a person afflicted with late rickets such would also have to be expected to occur in the tibial epiphysis where a comparatively thin bone plate rests upon a thick layer of cartilage. The pull of the quadriceps tendon may easily lift the process off its cartilage base and cause a kinking of the base of the epiphysis. Severe signs of late rickets existed in all the cases which were studied by him. He concludes that Osgood-Schlatters disease is merely a symptom of late rickets. Conversely, Grossman (48) reported a series of cases in which there were no signs or symptoms of rickets.

Cole (22) offers a plausible explanation of the condition. The disease manifests itself at puberty, the period of very rapid linear growth, and it is known that about two-thirds of the length of the lower extremity comes from the epiphyses about the knee. The rate of muscular linear growth depends upon the rate of growth of the bones. The patellar tendon either does not lengthen or does so an infinitesmal length during this period. Thus we have a very powerful muscle arising over a wide area inserting into the tibial tubercle by a narrow tendon under great physiological strain. Under the irritation of this stress and repeated minor injuries the tendon becomes swollen which impairs its blood supply and that of the tibial tubercle which receives a major part of its circulation from the tendon and the soft tissue about it. The process

may go on to aseptic necrosis of the beak of the tibial epiphysis or may result in a more simple tendonitis and peritendonitis. It should be remembered that this disease is not an avulsion fracture of the tibial tubercle nor a separation of the tibial epiphysis.

This view presented by Cole is in keeping with that of the modern trend of thought but it is quite evident from the preceding material that the etiology of the condition has been in question since its first observation and is yet a fruitful field for research.

Usually there is a definite history of trauma immediately preceding the disability which may be either direct or indirect through striking the knee or through a sudden violent strain of the quadriceps muscle in an attempt to bring the leg into complete forcible extension. The direct fibers of the patellar ligament are attached to the beaklike projection of the tibial epiphysis, and in addition, radiating fibers are traced in a fan shaped distribution to either side attached to the tibial diaphysis. From this fan shaped distribution of the patellar tendon fibers it can be understood why the force applied does not produce a total inability of extension of the injured leg. In all cases there is some power of extension following such an injury, though this ability is greatly reduced and is extremely painful. There is usually a marked yet localized swelling which is attributed to the increase of bursal fluid pro-

duced by trauma to the bursa lying between the ligament and the tubercle plus infiltration resulting from the tearing of the ligamentous fibers and direct tissue damage. One should remember in comparing the patient's knees that Osgood-Schlatters disease may be bilateral and that a deformity affecting both should not be considered normal. Further symptoms of importance to the physician in diagnosis are localized tenderness over the lower end of the patellar tendon and its insertion, limp, and increased pain upon passive flexion of the leg on the thigh and active extension of the leg.

Sutra and Pomerans of New York (99) reported their roentgenological findings in 106 cases of Osgood-Schlatters disease, the ages of which ranged from nine to eighteen years, the majority being thirteen years of age. They found that thickening of the patellar tendon at its insertion was a fairly constand, if not invariable, sign of the disease. In fact, such a swelling was occasionally observed roentgenographically even prior to the finding clinically of a mass of soft tissue in the region of the tubercle. Next in importance was the presence of irregular calcific spicules radiating from the anterior surface of the tubercle. As the lesion progresses, they may extend for some distance into the patellar ligament. Furthermore, small or large fragments of bone avulsed from the anterior surface of the tubercle were also to be found in the patellar ligament. Islands of bone were seen in the patellar ligament at a

considerable distance above the lowest point of the tibial tubercle. These may have formed there or may have been fragments avulsed from the tubercle. In most of the cases the epiphyseal plate between the tubercle and the tibial metaphysis was not unusually widened. In some, the distal tip of the tubercle pointed toward the region of the knee joint.

Most cases can be treated conservatively by plaster cylinders as advocated by Whitman (103), Lovett (69), Jones (56), Mercer (70), and others or by adhesive strapping as suggested by Moschocowitz (72). All cases seen late when symptoms are subsiding should be treated in this way. If adhesive strapping is instituted, bed rest for ten days to two weeks is of definite advantage in healing. Cole advises splitting of the patellar ligament to relieve pressure within and thus help to restore circulation to the tibial tubercle. This treatment is employed in the cases seen early and which are very aggravating to the patient.

Walter Mercer, in treating the disease conservatively, uses a plaster cylinder and recumbency for a short period followed by the Jones knee cage, with a stop lock to prevent undue flexion, and early weight bearing. More than a minimal amount of flexion is not permitted and violent exercise is not allowed for four months.

Bosworth advocates pegging of the tubercle with autogenous bone grafts and weight bearing within two weeks following operation (8). Some of his patients have been allowed to bear

weight within six days. He feels that loss of pain and tenderness is very rapid following this type of treatment.

Oglive Will was perhaps the first to employ internal fixation for this condition. In mistaking Osgood-Schlatters disease for a fracture of the patella he operated a boy. When he discovered his error, he thrust his drill through the tubercle into the shaft and obtained early and astounding results. The drill was removed in three weeks and the patient began active symptomless motion. Will, of course, recommended surgical therapy.

Since Bosworth began internal fixation employing bone pegs, various and sundry objects have been used for that purpose. Recent ingenious operators have felt that the principle behind the rapid cure of the condition lay in the re-establishment of an adequate circulation by perforation of the tubercle and shaft by the pegs or steel pins. They have perforated the epiphysis with numerous small drill holes and they too have reported the maximum of success.

Whatever form of treatment is followed, quadriceps exercises should be practiced during the period of recovery. After removal of the splint, baking, massage, and active motion should be instituted but violent exercise should be restricted for four to six months.

Kohler's Disease of the Tarsal Scaphoid

Kohler's disease is a peculiar condition in which the tarsal scaphoid is abnormal in size, shape, and density. It was described by Kohler of Weisbaden in German literature in 1908. Tarsal scaphoiditis is a self-limiting disease, the final result of which is complete restoration of the bone. Years later the only roentgenographic evidence of the old disease may be facetting of the articular surfaces due to pressure from the adjacent bones during the decalcified stage.

The disease occurs between the ages of three to ten years and is much more common in boys in the ratio of about five to one girl.

The etiology here again is of questionable origin and as obscure today as it was when first described by Kohler in 1908. Some believe it to be a congenital anomaly pathologically manifest during the years it is first subjected to trauma, stress and strain. Others feel that it is due to infected emboli and still others assume it to be of entirely traumatic origin. Lewin (67) expresses it as being a compression osteochondritis.

Karp (57) made a study of fifty normal children equally divided as to sex and had them x-rayed every year until they were six years old. He found that this bone appears much earlier in females and is well developed in most girls by two or two and one-half years. On the other hand, boys are much later, and they are three and one-half to four before their scaphoids are at the same stage. These

retarded scaphoids are very often irregular in outline, of increased density, and often flattened. His conclusions are in agreement with the statement by Hauser (50), that a bone, late in ossifying and subject to strain and repeated traumata, is very apt to develop a circulatory disturbance.

Pain, exaggerated upon weight bearing, with localized tenderness and thickening over the dorsum of the foot are the major symptoms. The patient limps and bears his weight on the lateral border of the foot to relieve the affected side. A few cases have been reported in which there were no symptoms, having been found only by accident in roentgenograms taken for some other complaint. An interesting feature is that during the height of the symptoms none or slight bone changes are observed while later, when the deformity by x-ray is marked, there are no symptoms.

The diagnosis is easily confirmed by x-rays which, if taken early in the course of the disease, reveal a relative increase in the general density. This gradually increases, apparently at the expense of general decalcification of the other tarsal bones which, after a period of six to eight weeks, have little detail of cancellous structure but have a fine pencilled outline of their peripheries.

The scaphoid at this stage has maintained its normal outline. Gradually during the next few weeks, it becomes compressed from front to back and becomes extremely dense so that in the lateral view it may look like a silver half dollar. Microscopic sections of

such bones show necrotic changes and a replacement of the marrow by fibrous tissue. Three to four or even more years may ensue before the normal structure is restored on the x-ray film.

The treatment as suggested by Lewin is the initial application of a plaster cast to hold the foot in slight varus and the use of crutches to prevent, or a walking iron to permit, weight bearing. After a few weeks the ankle and metatarsal regions are strapped with adhesive tape and the patient should wear a high laced shoe with a Thomas heel and felt pads to relieve the strain on the longitudinal arch structures. Later foot baths, flat foot exercises, gentle massage and contrast baths are prescribed. Lewin further advises a thorough investigation of all possible foci of infection, especially the tonsils and sinuses of this young age group.

Harp disagrees with this form of treatment and in his series of forty-five cases he found that plaster strapping, hot socks, caliper splints, plaster casts, and so forth, did not influence the course, duration, or end result of the disease.

In adults, the disease is quite different in its course. It runs a much more chronic course and leads to a crippling disease. An oblique splitting of the scaphoid occurs with displacement of the two fragments and this results in a severe osteo-arthritis. Operative intervention is usually necessary to remove the fragments which are displaced and to fuse the bones at this joint.

Freiberg's Infraction of the Metatarsal Head - Kohler's Disease, Juvenile Deforming Metatarsophalangeal Osteochondritis, Metatarsal Epiphysitis, Kohler II.

No mention of this lesion as a clinical entity had been made until it was described by Dr. Albert Freiberg (40) of Cincinnati in 1913 in a paper read before the Southern Surgery and Gynecology Association. He saw his first case in 1903 but his paper reporting six cases did not appear until August 1914 in that issue of Surgery, Gynecology and Obstetrics. A. Kohler of Wiesbaden described the condition in 1915 and in 1920 he reported five cases.

Frieberg's description of his first case, which he considered typical of the condition, very clearly defined the disease and is briefly as follows: "The first patient in whom I recognized the disease was a girl of sixteen. She had been suffering pain in the ball of her foot for six months. The pain was precisely like that which we so often encounter in the metatarsophalangeal region in connection with static incompetence of the foot. At the time I examined the patient she complained of pain in weight bearing only. In attempting any unusual exertion, as in walking considerable distances, she was compelled to limp and the pain became severe.

The patient was quite sure that the condition dated from and was due to a game of tennis in which she "stubbed" her foot. The pain was severe at that time and it was considered to be a sprain and she was able to be about the next day. My examination disclosed a well formed and apparently strong foot. The metatarsophalangeal

articulation of the second toe was thickened and very sensitive upon palpation and pressure. Passive movement of the joint was painful and accomplished by slight grating. The x-ray showed quite clearly that the distal end of the second metatarsal had been crushed in, causing the articular surface to lose its curved outline. There was apparently a small loose body in the joint about two m.m. in diameter.

The treatment consisted in applying a felt pad to the plantar surface of the foot by means of adhesive plaster so that the anterior end was placed just back of the injured joint. A steel plate was inserted between the layers of the sole of the boot in order to deprive the foot of the motion in the metatarsophalangeal joints. (A practice I have since discarded.) The patient was able to walk painlessly without the pad within six weeks and has had no further trouble with the foot."

All writers substantiated Frieberg's observation in that the disease belongs typically to the young, two-thirds of the cases occurring between ten and eighteen years of age predominantly in females, four to one. Kohler's series of cases ranged from ten to forty-eight years of age but he noted that in persons beyond fifteen to eighteen years of age the disease was very probably not of recent development but originated during the growth period and was accompanied by associated changes in the sense of arthritis deformans. The number of cases reported up to date indicates that the disease is not especially rare; in any case, it is encountered more frequently

than the disease that is peculiar to the tarsal scaphoid. In nine cases out of ten, the second metatarsal is involved; one time in ten, the third alone or together with the second. In a few isolated instances both feet were involved.

Anatomically, the metatarsal bones are each ossified from two centers; one for the body and one for the head of the second, third and fourth metatarsals. The first has one center for the body and one for its base. Ossification begins in the center of the body about the ninth week and extends toward each extremity. The center of the base of the first metatarsal appears about the third year, the centers for the heads of the other bones between the fifth and the eighth years. They join the bodies between the eighteenth and the twentieth years. The circulation to the metatarsophalangeal region is supplied by the dorsal and plantar metatarsal arteries.

The etiology of this condition is as in the other osteochondritides but fortunately there is some agreement between investigators. Freiberg attributed the disease to trauma and its manifestations. Kohler found that about one patient in ten gave a history of definite trauma. Certainly a single traumatism cannot alone be the cause. It is much more likely that a minimal mechanical insult, perhaps repeated, is necessary to bring about the clinical manifestation. Against traumatic etiology there speaks further the slow beginning and the occasional bilateral occurrence of the disease as well as the histological and roentgenological findings. The points of principal supports of the arch upon standing on both feet or in

walking, according to the investigations of Beely, are the heel and the distal ends of the second and third metatarsals. If the foot is hanging, or if it rests only lightly on its sole, then the distal ends of the first and fifth metatarsals are supports, but if a considerable pressure is placed upon the anterior transverse arch, the arch gives and the distal ends of the second, third, and fourth metatarsals become burdened. In case of flat or splay-foot, the second assumes very much of the burden. In keeping with this, flat-foot appears in the history of many of the patients, and Sonntag found in twelve cases critically described and examined the arch to be normal in only three and flat or splay-feet in nine. However, in studying seventy cases, Cahen and Brach found flat feet in only about twenty-five per cent. In two or three cases, going barefoot was blamed for the condition. In going barefoot one naturally loses the support of the shoe. If there is added a weakness of the connective tissue, which leads to a sinking of the anterior transverse arch, especially the second metatarsal, the first metatarsal gives medially and when the foot is pronated the weight is principally thrown upon the second. Three of the cases reported by Kohler showed a bowing of the metatarsal upward.

Muller says that the bone is simply reacting to mechanical insults on the same principle that it forms callus after fracture. It is pathological only in that the tolerance of the bony tissue to the influences in question is disturbed. Furthermore, the fashionable women's shoes, with their high heels which throw their weight on the

forepart of the foot are responsible at least in part for splay-foot so far as etiology goes, and this is in keeping with the fact that the great majority of patients are females. Histologic and roentgenologic findings speak against the inflammatory origin of the disease. Tuberculosis, syphilis and osteomyelitis could not be found, but in four of Alberti's six cases, tuberculosis was found elsewhere in the body. Fromme defends his theory that the condition is an osteochondritis findings its origin in late rickets. He also regards Legg-Perthes disease of the hip, Schlatter's disease of the tibial tuberosity and tarsal scaphoiditis all as manifestations of the same thing. The thickened and softened cartilage of rickets or late rickets is especially susceptible to traumatic injury. The theory has much in favor but in almost all of the patients studied by Kohler there were no manifestations of rickets. Yet, late rickets is much more frequent than generally appreciated and clinical signs may be absent.

As for this being an inanition-osteopathy, most cases are found in well nourished individuals. Of the few microscopic studies, there are some descriptions by Axhausen, Kappis, Herzog and Sonntag of peculiar findings in fair agreement in the form of wedge shaped necroses of the epiphyses from interruption of nutrition. In 1905 Konig emphasized that in relation to operative cases of osteochondritis ossificans, that there must in addition to trauma be some other cause operating in an unknown manner to explain the formation of a sequestrum in a joint lined with

cartilage. Thus it is believed that the wedge shaped necroses of the epiphyses is caused by a mechanical occlusion of the epiphyseal end arteries which may be initiated by bacterial emboli, aseptic emboli, spastic constriction of the vessels or by closure through spasm of the musculature.

At the 47th Congress of the German Surgical Society in 1923 in Berlin Aschoff of Freiberg stated that this disease is probably the same process as that involved in Legg-Perthes, the essential feature being bone necrosis which, in the hip, leads to a reforming of the whole head of the bone without the cartilage being destroyed. Vascular disturbance, he felt, doubtless play a role in the development of the disease. Axhausen is of the opinion that these need not always be embolic in origin. Traumatic strains which lead to an occlusion of the vessels and other factors can cause the disease. Aschoff does not consider as possible a direct transition from this disease into arthritis deformans.

Kohler concludes that there are anatomic-physiologic reasons for the localization of the disease in the second or third metatarsophalangeal articulations. The second metatarsal is the one most exposed to weight bearing and various insults as is indicated by its being much the most frequently involved in the march fracture of soldiers. This factor is not in itself sufficient for the production of the disease. There must be another pathological factor, a certain debility of the skeletal system, perhaps less of

an infectious nature than of a toxic, toxic infectious, or hormonic nature or a dyscrasia or diathesis through which the general resistance of the organism is lowered to become manifest at the point of greatest strain. There may be a predisposition in the sense of a constitutional anomaly, and it is not entirely disproved that heridity plays a part. Possibly slight repeated or even more considerable over-strains lead to a form of osteochondritis.

Pathologically there have been various findings reported which are more or less detailed. There were found necrosis, at times fibrous marrow, other times tissue resembling granulation tissue, thickened cortices and well preserved cartilage. Fromme found subchondral callus-like tissue with fibrillary connective tissue, fibrous cartilage, hyaline cartilage and osteoid substance. Axhausen observed a massive subchondral focus with bone and marrow necrosis and calcification surrounded by a resorption zone of thick connective tissue free of leucocytes. The cartilage was preserved and thickened, somewhat necrotic in its deepest parts. There was villous hyperplasia of the synovia and lipping as in arthritis deformans. There were found no signs of rickets, tuberculosis, syphilis, osteomyelitis or the like. In Cahen-Brach's article a report by Goldschmidt was presented in which he again found a rounded focus in the head of the metatarsal in which the bony trabeculae were fragmented or entirely absent. In their place was a connective tissue focus rich in cells. At the edge the connective

tissue extended into the marrow. Beneath the articular cartilage the trabeculae showed no particular change.

Objectively the patient presents a slight swelling of the soft parts of the foot, more marked on the dorsum in the vicinity of the lesion, with tenderness on pressure on the dorsum or on the sole or both. Limitation of motion is universally present. Pain and a marked limp on weight bearing are perhaps the most common presenting symptoms. In the acute phase, pain may even be present while the foot is at rest. Attempts to flex the toes in a plantar direction by passive motion causes severe pain. At times the enlarged head of the involved metatarsal bone is palpable.

In the early stages very little change is seen in the x-ray. However, there may be a slight decrease in the calcium deposit of the head of the affected metatarsal bone. The structures are altered in the following manner:

1. The shadow of the articular surface of the proximal phalanx in plantar-dorsal exposures loses its perfectly circular form and becomes irregular.
2. The joint space in almost all cases is broader than normal. This is remarkable because in all arthritides except in cases with effusion there is never a broadening of the space but instead an actual narrowing. Not only is there a broadening but this broadening is very irregular so that the fibular half is often double that of the tibial half.

3. The articular surface of the head of the metatarsal loses its normal roundness, in early cases it is only more or less flattened but in older cases it shows irregular knobs and defects.

4. In advanced cases of long standing, on the fibular aspect and even deep in the soft parts are occasional shadows of density equal to bone varying from the size of a pin head to that of a pea, always circular in nature.

5. The head of the metatarsal is shortened in its distal third as if the cap had been driven in. This naturally makes the whole metatarsal appear shorter.

6. In all frank cases, the head is increased in circumference and the constriction at the base of the head is lost. There is thickening which not only involves the medulla but the cortex as well.

The treatment consists of absolute relief of the affected foot from weight bearing by means of crutches and a block under the heel and sole of the shoe of the opposite foot. The foot should be immobilized in a plaster cast with a small beveled felt pad just back of the metatarsal heads. As the cast sets, pressure is applied laterally and upward to build up the metatarsal arch (70). Cod liver oil internally is of value. Fresh air, good hygiene and nutritious food hasten recovery. (104)

When all symptoms have disappeared, a proper shoe is pre-

scribed. This should be a straight last, round toe, medium shank shoe into which is inserted a felt for the transverse arch. This may be applied by means of glue and tacks directly into the shoe or sewed to the bottom of a leather insole. A metatarsal bar or anterior heel may be used and the entire leather heel should be removed and a low rubber heel applied. Physiotherapy, heliotherapy, hyperemia and hydrotherapy are indicated after the cast is removed. Baking and gentle massage, passive and active movements with contrast baths hasten recovery. Special exercises for the metatarsal arch are performed over a round door stop screwed to a board. The patient sits with the door stop exerting pressure back of the metatarsal head, the toes are forcibly flexed and extended. Picking up various sized marbles from the floor using the bare toes and pulling objects along the floor with the toes are auxillary exercises.

In cases in which loose bodies are evident arthrotomy and surgical removal of the particles are necessary.

In chronic cases which do not respond to conservative therapy, it is occasionally necessary to resect the metatarsal and its corresponding phalanx.

Sever's Disease or Apophysitis of the Os Calcis

"Inflammation of the cap-like epiphysis at the posterior portion of the os calcis is called apophysitis." This term was used by Sever (88) who first described the disease in the New York Medical Journal, May 18, 1912, at which time he reported five cases. Myerding and Stuck (75) in reviewing the literature in 1934 found only forty cases reported. To this they added twenty-one cases which had been seen at the Mayo Clinic. Doubtless, there have been many more cases seen which have gone unrecorded or possibly undiagnosed and still others not severe enough to bring the patient in for treatment. At any rate, the disease is of greater incidence than statistics would indicate.

Epiphysitis of the heel occurs most commonly between the ages of eight and seventeen, or during the period of greatest growth. Boys are more often affected than girls in the ratio of six to one. The epiphysis of the os calcis may develop by one or two centers of ossification; it is stated by most authors that the centers of ossification appear at the ninth year on the average and that the epiphysis is united to the body of the os calcis either before puberty or soon after. In Sever's opinion the epiphyseal ossification begins well before the ninth year, often as early as the seventh. He further pointed out that the children who are large for their age will always show greater and earlier epiphyseal development than the underdeveloped children or even normal sized children of the same age. In the twenty-one cases reported by Myerding and Stuck it was found that the

average age of onset of the disease was ten and two-tenths years, the ages of the patients ranging from seven and one-half to seventeen years.

The etiological factors described for the other diseases hold true here as well. The epiphysis of the heel is a "pressure epiphysis" and is subject to direct trauma. In this respect, it is similar to the epiphyses of the femoral head or vertebral bodies. Furthermore, as in the epiphyses of the tibial tubercle or olecranon this epiphysis is also a "traction epiphyses" and is subject to the strong pull of the attached muscles. Consequently the heel is unique in being subject to both direct and indirect trauma, and it is this traumatic factor which probably gives rise to the disease. Lewin (66) is in agreement with this but adds the theory that metabolic and circulatory alterations are undoubtedly very important in their relation to trauma.

The onset of the disease is almost always insidious and a limp is usually the first apparent symptom which may or may not be accompanied by pain. There may be a history of injury or trauma but this is by no means constant. The child may have been running on hard pavements, wearing sandals or tennis shoes which offer little protection to the calcaneal epiphysis. Often the patient is well overweight for his years, very active and strong physically. Pain is usually dull and localized to theaffected area, less marked while wearing shoes and more pronounced when climbing stairs. An examination usually reveals a slight pronation of the foot but this factor

is in no way pathognomic. There is a moderately tender area over the posterior portion of the os calcis, deeply situated and localized in front of the tendo Achilles on either side. There is invariably moderate thickening about the whole posterior portion of the os calcis with some tenderness and partial obliteration of the hollows on either side of the tendo Achilles. The motions of the foot are all somewhat limited, especially in full dorsal flexion, and any movement which tends to put a strain on the tendo Achilles causes pain. There is pain and tenderness on weight bearing when the heel is put to the floor but less so when walking on the toes with the heel elevated.

The disease resembles somewhat the condition known as achillo-bursitis, an inflammation of the bursa between the tendo Achilles and the os calcis, but is more extensive and more deeply seated. Tenosynovitis must also be considered in diagnosis being characterized by pain referred to the tendon and by palpable crepitus on movement. The x-ray is negative. Bursitis involving bursa between the tendo Achilles and the skin is a superficial inflammation, usually the result of pressure of the shoe and is easily determined. Calcaneal spur is rare in early adolesence and is usually found on the inferior internal aspect of the os calcis. The area of sensitiveness should determine the diagnosis. The inflammation is associated with the attachment of the plantar fascia instead of the tendo Achilles. Tuberculosis of the os calcis is usually in the anterior portion of the body and not in the posterior

region. Other evidence of tuberculosis are absent in apophysitis and the roentgenogram will aid very materially in differentiation. A pyogenic infection would produce more marked inflammatory reaction with destructive osteitis. The painful heel of arthritis occurs later in life, is aggravated by focal infection, other joints may be involved, the tenderness is most often at the attachment of the plantar ligaments, and roentgenograms may show narrowing of the articular spaces and bony proliferation.

The most convincing diagnostic feature of all is the characteristic roentgenographic change that occurs with the described symptoms and signs. In the lateral view of the foot, the epiphysis of the heel is seen to be fluffy, moth eaten, somewhat flattened or partially fragmented according to the stage of the disease. Furthermore, there is usually considerable irregularity of the adjacent posterior portion of the os calcis and a punched out appearance in this region due to the alternation of areas of rarefaction with areas of increased density. Finally, the epiphyseal line typically appears cloudy and abnormally irregular. These changes, if in variable degrees, should assure a fairly positive diagnosis.

Of the pathologic changes that take place all are dependent directly or indirectly upon the disturbance in circulation. Leriche and Policard's hypothesis regarding this was that the injury was in the nature of an axon reflex trauma to nerves, leading to vaso-dilatation and a more or less permanent vascular imbalance.

This, in turn, produced rarefaction of the bone and local tenderness. Afterward local edema resulted which stimulated increased calcification, so clearly seen in the x-ray. Bentzon investigated the arterial supply to the os calcis in this condition and also concluded that there was a "paralytic hyperimia" of the heel following trauma to the nerves that led to a pathologic callus formation. Others believed that the distrubed blood supply followed a partial separation of the epiphysis from muscular pull or from direct injury to the epiphyseal cartilage. In any event, all symptoms disappear at time of union of the epiphysis to the os calcis and the consequent restoration of an optimum blood supply.

The prognosis for this condition is always good. The course of the disease is comparatively short varying from a few weeks to several months. It may, however, recur as a result of overactivity or trauma. Spontaneous cure may be expected, even in untreated cases, when consolidation occurs between the os calcis and the epiphysis.

The treatment of this condition is relatively simple, the desired effects being to relieve the tendo Achilles of strain and to prevent weightbearing on the os calcis. Local heat and massage to the feet in the interval of acute pain may relieve the pain somewhat. The most efficacious treatment for the less severe cases consists of elevation of the heels to relieve tension on the Achilles tendon. These lifts should always be made of rubber. Heel pads made of soft sponge or felt, to be worn in the shoes, are also of benefit and avoidance of any strenuous exercise or exertion is indicated until

the acute phase of the condition is over. In the more severe or resistant cases the most satisfactory treatment consists of the application of a plaster cast extending from the toes to just above the knee in such a manner as to hold the foot in slight equinus and the knee in slight flexion, thus relaxing the pull of the gastrocnemius. Two crutches and a two inch block under the heel and sole of the opposite shoe aid in walking. The cast should be removed in two weeks and another reapplied extending to the mid-calf holding the foot at a right angle in neither varus nor valgus. At the end of four more weeks, this cast should be removed and a high lace shoe with an elevated heel worn. Weight bearing with the aid of crutches should be carried out for another two weeks during which time diathermy, baking and contrast baths should be employed. (70)

During the course of treatment emphasis should be placed upon adequate sunlight and nutritious food. If there are any glandular disturbances or focal infections proper therapy should be instituted. Cod liver oil is prescribed by some authors.

Keinboch's Disease - Chronic Osteitis of the Semilunar Bone, Traumatic Osteoporosis of the Semilunar Bone

Keinboch's Disease is the name given by Kellogg Speed (94) to a lesion characterized by fracture of the semilunar bone, usually comminuted, associated with partial absorption of its fragments. A distinguishing feature of this disease is the absence of history of trauma severe enough to account for the roentgenographic appearance. The condition was first described by Peste in 1843 but was not widely recognized until Keinboch, A german radiologist, described it in 1910. Prior to his report, Finsterer had listed all such lesions as fracture of the semilunar. Saupe in 1923 collected fifty reported cases of Keinboch's disease. He concluded from his study that the condition occurred about seven times as often in males as in females. The right wrist is involved about two and one-half times as often as the left due to the predominence of right-handedness. The lesion was found to occur for the most part between thirty and fifty years of age, a few occurring earlier and later.

Here we find a quite definite etiological factor, namely trauma; rarely, however, severe enough to cause sudden acute disability. Keinboch carried out a careful examination of 1400 wrist joints and concluded that the disease can occur without trauma severe enough to cause the patient to cease work at once. The pathology probably depends on interference with the blood supply which reaches the bone by way of the ligaments. Speed suggests that when a slight trauma is received the ligaments may be torn with consequent inter-

ruption of the nourishment to the bone. Later absorption of the diseased bone begins. The semilunar is more subject to this condition because it lacks the broad vascular attachment to the ligaments common to all the other carpal bones. Over two-thirds of its surface is covered by joint cartilage which is devoid of blood supply.

Roentgenographically, there may be no early change but as the condition progresses characteristic alterations of the affected bone occur. The semilunar becomes flattened and may be abnormally dense. In some cases irregular areas of rarefaction are noticeable suggesting fragmentation. Often in adjacent bones changes suggestive of an early hypertrophic arthritis may develop.

In the typical case there is pain in the region of the semilunar bone, usually of months duration. The pain is made more intense by motion, oftimes making complete flexion of the wrist impossible. There is swelling of the wrist, usually of mild or moderate degree, especially on the dorsum and the volar sides. Sharply localized tenderness to pressure over the bone is a very important sign. Occasionally there is a bony protrusion immediately below the distal articular surface of the radius. Finisterer states that two signs, if present, are almost pathognomic. They are:- (1.) "The head of the third metacarpal on the same level as those of the second and fourth, while normally it is more prominent." This is, of course, due to partial absorption of the semilunar. (2.) Pain localized over the semilunar bone on tapping the head of the third metacarpal with the fist closed and the hand in ulnar adduction."

Occasionally, the condition is confused with sprain or chronic arthritis because of the comparative mildness of the symptoms and of the frequent absence of definite traumatic history.

Treatment of the condition if recognized early is principally non-operative. The wrist is immobilized in a slightly cocked-up position for two months. Speed extends the third finger by straps or a spring apparatus which is attached to the splint. If recovery does not take place or fragmentation has occurred or if arthritic changes have developed in the old cases operative interference is indicated. The whole bone is removed and post operative x-ray films are taken to be certain that no fragments have been overlooked. The hand is dressed in a bandage which effects a slight cock-up position for one week. After this, physiotherapy is begun, consisting of diathermy, baking and massage. Active motion is started early.

Vertebral Epiphysitis,- Scheurmann's Disease, Osteochondritis Deformans Dorsalis, Osteochondritis Dorsalis Juvenilis.

Scheurmann first described this disease under the name "Osteochondritis Dorsalis Juvenilis" in 1921 in German literature. In 1924 Andre Delahaye described it under the same title in France. Prior to this time in discussing anteroposterior deformities of this age, occurring without definite etiology, most authors were content to assume a state of lowered resistance of the spine against functional requirements which were accentuated during the critical growth period, and in addition, some inherent weakness or insufficiency of the spine. (Schanz)

However, when it was observed that individuals afflicted with the anteroposterior deformity were frequently of athletic stature, possessing strong musculature and durable skeletal structure, the mechanical explanation of the deformity became unsatisfactory and search was made for other causes.

In 1916, Virchow described what he called a wearing off of the anterior edges of the vertebral bodies. He found this condition to be prevalent among among the Australian negroes who since early in their lives were in the habit of carrying heavy weights upon their shoulders and pullin carts with the trunk flexed forward. This observation suggested an early involvement of the epiphyses and a disturbance in their normal ossification.

Under normal conditions the ossification of the vertebral bodies occurs from three bony centers, one in the body and two in

the arches. These gradually increase in size until the arches and the body unites somewhere between the third and eighth year of life. After this, the body depends upon further lengthening thru the ossification of the epiphyses. At about the fourteenth year there appears, according to Kohler (twelve years--Buchman (15)), at the upper and lower anterior edges of the vertebral bodies a small triangular x-ray shadow which represents the earliest signs of the epiphyseal ring. Later as they become elongated they appear as definite rings, thick peripherally and thin centrally. Thus at twelve or fourteen years of age the vertebra in a lateral film shows a bony diaphysis with upper and lower epiphyseal cartilages beyond which are the bony epiphyses. These rings remain separated from the body until the time of fusion, which Schultress describes as taking place at the twenty-fifth year, others say the twenty-second to the twenty-fourth year. At that time the length of the vertebrae is normally established.

Under certain conditions during the period from eight to twenty-four years the normal process of ossification becomes disturbed. Scheurmann in his report described the vertibral bodies as being lower in front and the epiphyseal discs as being broad and irregular. The peripheral placques or epiphyses become enlarged and motheaten and the vertibral borders are irregular and indistinct while the intervertebral spaces become cloudy in appearance. He concluded from his studies that the primary effect was disturbance of the epiphyseal growth. In later stages he found that the epiphyseal ring again assumed more

definite contours and that its irregular fragmentation disappeared and it again became solid. Since the process followed the course of Legg-Calve-Perthes disease of the hip, Kohler's disease of the tarsal scaphoid and Freiberg's disease of the metatarsal, he felt that it was of the same nature and applied the term "Osteochondritis Deformans Juvenilis."

This disease occurs, as mentioned previously, during the period of growth from eight to twenty-five years and according to Nathan and Kuhns (76) occurs more in females in the ratio of two to one. According to Mau and Edelstein, 1927, there are manifest pathologically, as can be demonstrated by x-ray, three stages. They are:

1. The stage of irritation, or florid stage - During this period the vertibral body appears wedged with its apex in front. There is some mottling of the upper and lower ring epiphyses and the upper and lower surfaces of the vertebral body appear fuzzy and uneven. The intervertebral space is cloudy and diminished. In a large number of cases Schmorl's bodies or islands may appear, these bodies being areas of decreased density at about the middle of the vertebra in lateral view, and in reality are intrusions of the nuclear substance of the disc into the body of the vertebra.
2. Destructive Stage - At this period the epiphysis becomes fragmented and there is often disappearance of the upper

and lower anterior corners of the body.

3. Reparative Stage - Characteristically the density returns to the epiphyses and definition to the upper and lower anterior margins of the bodies. In the final adult picture the epiphyses are fused but appear in some cases to be irregular and sclerotic. The Schmorl's bodies, once present, remain present throughout. The body still remains wedged, this condition being permanent.

The question of etiology again presents itself as a stumbling block in what would otherwise be a fairly well defined disease entity. Most of the obervers agree with Scheurmann who ranks the disturbance along with Kohler's disease and Perthes disease, that is, a primary interference of the ossification process in the epiphyses. Mercer and others regard the disease as a sequel to disproportion between the capacity of the spine and the load it has to bear. Theories as to the factors bringing about this disproportion are many and varied. Axhausen suggests the circulatory influence which he has described in most of the other epiphyseal diseases. Infection has also been mentioned but is not highly regarded as a etiologic factor by most recent investigators. Endocrine dysfunction may possibly influence the condition but Nathan and Kuhns have definitely shown that it is not a constant feature. Congenital disc prolapse is an interesting theory but of little value in the quest for the true origin of this process. It is supposed that the vertebral bodies are brought nearer together as a result of the prolapse, and with the posterior articulation acting as

a hinge, rotary movement occurs so that the anterior edges of the bodies are swung closer together. The "water cistern" effect is lost and pressure disturbances on the anterior part of the body are sufficient to cause wedging of the body. Lambrinudi has shown that when the hamstring muscles are shortened it results in increased flexion at the thoraco-lumbar junction on stooping. As a result of this, aided by minor trauma, hemorrhage occurs beneath the cartilage plates. The cartilage is then apt to fragment and the disc tends to prolapse. This theory is significant perhaps in that it refers to the traumatic influence. Schmorl attributes the condition of adolescent kyphosis to a primary disturbance of the discs. Possibly the truth may lie somewhere between the two latter theories and it is an adolescent kyphosis due to sub minimal trauma in those individuals who have a congenital tendency toward nuclear herniation.

The disease is self limited like the other osteochondritic affections and appears about the time of puberty in well developed and well nourished children. The onset is usually associated with a sensation of fatigue and backache and pain in the limbs. There is often tenderness over the spinous processes, especially in the dorsal region. The lumbar bodies are occasionally tender upon abdominal palpation. The epiphyses of the iliac crests and those of the extremities may likewise be painful to pressure. Deformity develops secondarily and it may be a kyphosis or a scoliosis, usually and typically the former. Frequently the condition is entirely asymptomatic or the symptoms may be so slight they attract little or no attention. The deformity is

the disturbing factor and it is because of the deformity that the doctor's aid is sought.

Since nothing is known definitely regarding the etiology, the treatment can only be symptomatic. It is the opinion of Nathan and Kuhns that the treatment selected must depend upon the severity of the disease. In the early stages, when the involved portion of the spine can be straightened by recumbency, exercises to correct the kyphosis, good food and hygiene will prevent deformity in a small percentage of the cases. Usually this therapy is not adequate and more vigorous treatment is necessary. During the early stage the deformity can be corrected to a large extent while the patient is ambulatory by use of a well fitting plaster jacket applied with the thoracic spine in as much extension as possible. New jackets are applied frequently as further correction takes place. Later, exercises are taken to improve posture.

Prolonged bed rest in a plaster shell or on a hyperextended Bradford frame is indicated for any patient who is in poor health or rapidly growing. In the more severe forms of the disease recumbency prevents greater deformity but is disappointing, as are all other forms of therapy, in that correction cannot be accomplished.

After fusion of the epiphyses to the vertebrae, the changes remain practically constant throughout life but there is a tendency toward hypertrophic arthritis to occur at an earlier age than normally seen.

Calve's Disease or Osteochondritis of the Vertebral Body

Calve (18) in 1925 reported two cases which presented the symptoms of tuberculosis of the spine and were diagnosed as Pott's disease. These patients were observed during the entire course of the disease and after three or four years were cured without any deformity. It was decided that these cases were osteochondritis and not tuberculosis as first suspected because the tuberculin tests were consistently negative, only one vertebra was involved, the inter-vertebral discs remained intact and the bone appeared denser. The disease was described by Buchman in 1927 (16) and his findings were in agreement with those at Calve. In a more recent article by Calve in 1928, he reviewed the known and reported instances of this affliction and found that there were only eight cases on record. Mitchell (68), in 1932, reported a case observed in the Willis E. Campbell Clinic which raised the total to eight. Since this time, the literature has been very sparse and one may safely infer that the condition is rare and very subject to misdiagnosis.

The disease involves the centrum of the vertebra occurring during the first decade of life. For this reason, it cannot be considered to be an epiphysitis because the epiphyses of the vertebral bodies, according to Calve, do not appear until the eleventh to the fifteenth years. The age of onset is the period of most active growth for the area involved. This is during the first few years of life in the case of the vertebral bodies. It has been observed that males and females are affected in about the same ratio. The symptoms of pain

and stiffness in the back are moderate or slight for the extent of the pathology as seen by x-ray. Referred symptoms such as limp or pain in the knee are features not uncommon to the condition. There may be a deformity, such as a hyphosis or a scoliosis, depending upon the secondary factors of stress and strain. By x-ray we see an irregularity of the vertebral outline, there may be flattening or wedging. The discs are wider as the body becomes narrower and the vertebral outlines become dense and sclerotic as regeneration takes place. The vertebra becomes widened in the transverse diameter and narrowed in its vertical diameter.

The etiology is obscure but it is accepted by most men today that there exists a greater static demand than there is a static capacity of the newly formed bone. This is the explanation offered by Whitman (103) and he believes that the process goes on to aseptic necrosis as in other osteochondritic diseases. Calve believed that it was due to infection while Axhausen postulated emboli. Murk Jansen stated that stasis of the blood and lymph causes the bones to become brittle and Buckman offered the explanation that the physiological weakness of the bone incident to rapid growth is the most common causative factor. Late rickets have been suggested as the etiology but clinical and roentgenological studies have failed to substantiate this assumption. In addition, it has been found that the blood calcium and phosphorus have been within the limits of normal.

The treatment instituted by Calve was essentially that

for Pott's disease because he actually felt that the condition was tuberculosis of the spine. The patients, as reported, responded spectacularly and went on to complete cure. Today, the treatment is with due regard for individual variation a plaster spica or recumbency in hyper-extension on a Bradford frame during the acute stage. Later a body jacket or brace is applied until the bone shows complete regeneration. Violent exercise and manual labor are restricted during the period of treatment and undue flexion of the spine prevented until complete recovery is made. Obvious foci of infection should be cleared up. Local heat in the form of infra-red or diathermy in addition to massage are especially helpful during the convalescence. Little must be said regarding the necessity of good hygiene, nutritious food and sunlight for these growing children.

Osteochondritis Dissicans

Wagoner and Cohn (99) have defined osteochondritis dissicans as "a non-infectious process involving the articular cartilage and the subchondral bone of certain of the long bones of the extremities which by sequestration from the articular surface usually produces a single foreign body or more rarely two in a contiguous joint. The body is usually of an osteocartilagineous composition but its structure subsequently undergoes alteration by the fluid of the joints."

Munro in 1738 first described this condition, but Konig in 1887 wrote the original article and in 1905 gave it the name by which we know it now. In Freiberg's (41) article in the Journal of Bone and Joint Surgery quoted Konig's original article which was, as he pointed out, a remarkably accurate description considering that at the time the x-ray was not available and the opening of joints was a very much more serious matter than at the present stage of joint surgery. Briefly, Konig's description was as follows: "Without any injury, there separates from the joint ends fragments of varying size in consequence of a process as yet unexplained. Their bony surface becomes covered with a dense connective tissue containing cartilage cells here and there. In the same manner the defect in the bone becomes covered over. In some cases a smaller body composed entirely of bone and smooth with the appearance of necrotic bone lay under a larger piece perhaps two cm. in diameter. These pieces often fitted almost exactly in the corresponding bone defect, seeming at times somewhat too large because the pits in the bone had become filled in. Aside from this,

together with a fluid effusion and slight villous hypertrophy, these joints looked perfectly sound and they remained so after the removal of the loose bodies."

It was not until 1910 that the subject appeared for the first time in English literature, this being a paper written by Freiberg and Woolley (39). Since that time, numerous articles have appeared including those of Brackett and Hall (10), Bernstein (6), Balensweig (3), Ridlon (87), Richards (85), and others. Recently the literature has become voluminous regarding this condition.

Here, as in other lesions of this type, the etiology has confounded the investigators since its earliest description. Trauma as a causative agent was the most obvious explanation and consequently the first. Munro, 1726, advanced the theory that loose bodies within the joint were of traumatic origin. In this belief he was supported by Rieman, 1770, and Haller in 1776. Other workers who were followers of the traumatic theory were Rainey (1848), Bradhurst (1861), Poncet (1881), Kragelund (1884) and Bunghard in 1892.

Preiser (1898) considered osteochondritis dissicans to be the result of static imperfection. Ludloff in 1908 created a new variation in the trend of thinking in his theory regarding the circulatory influence. He said "the loose bodies arising from the lateral surface of the mesial condyle result from injury to the arteria genu media at the point where it perforates the posterior capsule of the joint and before it enters the bone at the seat of

insertion of the posterior crucial ligament. The resulting circulatory disturbance leads to necrosis of the area of bone supplied by the injured vessel. The necrotic bone is gradually separated as the result of insufficient nutrition. Axhausen (1914) indicated that the vascular system might play a slightly different role than that described by Ludloff. He advanced the idea that as a result of impaction from the opposing articular surface, the blood vessels to the part are damaged, either with or without partial fracture, according to the severity of the violence. This leads to necrosis of the area supplied by the damaged vessels and as a result there forms a zone of absorption resulting in a gradual separation and eventual extrusion of the dead portion of the articular surface into the joint. In further consideration of the role of the vascular system, Axhausen (1924) stated that the primary lesion was a necrosis of the epiphysis which is followed by a proliferation of the surrounding tissues that penetrate the dead bone and may end with complete reorganization. The regeneration is interrupted by compression fracture of the diseased epiphysis, which is frequently erroneously considered as the beginning of the disease. The fracture is limited everywhere by dead tissue, and therefore healing is impossible. The bone ends rub against each other and produce "bone flour". Instead of comparatively quick substitution, a slow reparation sets in. The cartilage is not yet severely changed, but the necrotic part of the epiphysis begins to be demarcated from the remaining bone by a layer of granulation tissue or of older connective

tissue. A slight injury may free the necrotic part in the joint. As the primary cause of the necrosis he suggests the possibility of tortion of blood vessels but believes that benign infected emboli are more probable. A year later he stated that the cause of epiphyseal necrosis is not as yet proven.

Konjestzny, (1924), found an obvious endarteritis obliterans and suggested this as a possible cause of the necrosis.

Barth, 1896, advanced the idea that the loose fragments were due to a pull of the posterior cruciate ligament. This has been proven experimentally to be impossible and doesn't explain the patellar fragments or pieces originating from the opposite femoral condyle.

Knaggs, 1926, suggested a new conception regarding the etiology of this type of necrosis without suppuration. He believes that the intiial lesion is a periostitis due to a micro-organic infection of a very mild degree, probably staphlococcic in type. He felt that the infection invaded the surface of the bone but because of its negligible virulence was quickly limited and failed to penetrate deeply into the compact bone. Granulation tissue developing under the periosteum and in the Haversion canals of the superficial layers caused compression of the vessels and interfered with the blood supply of the compact bone in the deeper parts. By itself, this would not be sufficient to cause death if the blood supply to the interior of the bone were adequate. If, however, the nutrient canal is similarly invaded compression of

the artery will likewise diminish the supply of blood to the medulla and render the circulation unequal to the demands placed upon it. Thus, the compact bone being gradually deprived of its blood supply passes into a state of necrosis.

Bernstein, Blanca, Richards, Walter and other have reported cases in which the disease was bilateral and Bernstein and Wagoner and Cohn have reported cases in which more than one member of a family were involved. From this fact, Wagoner and Cahn have suggested that heridity is very possibly an etiologic factor.

Of the involved joints, the knee joint comprises over eighty five per cent of the reported cases. The elbow is next in order followed by the hip and ankle. Some isolated cases have been reported in the metacarpo-phalangeal articulations. In the knee the most common sites are the medial condyle, then the lateral condyle and last the posterior articulating surface of the patella. In the ankle joint it is almost always seen on the supero-medial border of the astragalus.

Conway (24) describes the pathological process in three more or less distinct stages. In the first stage there may be only a fairly well demarcated prominence of the articular surface with the cartilage covering this elevation of a slightly different color than the rest of the cartilaginous surface. If this prominence is excised at this time an early excavation of the cancellous sub-chondral bone may be observed. Usually the fragment is quite easily

removed at this time. The second stage in the process is one in which the fragment has become more distinctly separated and lies within the excavated area in the articular surface, being held by a mere shred or more firmly by adhesions. The fragment is easily removable at this time; in fact, it may be merely lifted out of its bed. Surrounding the excavation the articular cartilage is of peculiar appearance appearing almost buff colored in contrast to the normal cartilage. In addition, the cartilage is not firmly attached to the underlying cancellous bone, but is easily removable. The ease with which it may be lifted off gives it an appearance of having been dissected off and, hence, the name "osteochondritis dissicans" which was first applied by Konig in 1887. The third stage is merely a completion of the first two and is characterized by complete sequestration of the fragment from its normal situation in the articular surface of the epiphysis into the joint cavity. The fragment may remain free in the joint being bathed by the synovial fluid and become lamellated in structure, or it may become affixed to the synovial membrane. The excavation gradually becomes filled with granulation tissue and later by fibrocartilage and becomes shallower and its outline less pronounced.

After the fragment has been freed from the articular surface and lies within the joint all the bone, which previously had a source of blood supply through the fibrous adhesions, becomes necrotic. The fibrocartilage and the fibrous tissue along the surface of separation receive sufficent nutrition from the synovial fluid and

proliferate, thereby causing a slow but steady increase in the size of the loose body. Due to gradual absorption and proliferation, it is found in those specimens which have been separated for years that the original constituents of the fragment have completely disappeared.

Clinically it is found that a history of definite direct trauma is rare. However, it must be remembered that a subchondral fracture can be caused by several repeated subminimal injuries and then become aggravated by weight bearing and exercises. Such a fracture in itself is painless as there are no nerve endings in the articular cartilage. For this reason, the "acute" stage goes unnoticed. However, if the involved extremity could be immobilized during the active stage of the disease the fragment might possibly reunite. Subjective symptoms arise only when the pathological process is reaching its conclusion and the fragment begins to free itself or completely disloges itself. The earliest symptoms may be aching after excercise or tenderness on pressure over the joint and possibly some degree of muscle spasm. Locking of the joint, a very characteristic symptom, traumatic synovitis and the attendant joint disability all follow in order with the wandering of the loose fragment.

Diagnosis depends upon the clinical and radiographic evidence. The x-ray is diagnostic in all stages of the disease. The lesion is sharply defined and punched out and has well marked borders. The osteochondritic focus is denser than the underlying bone and rests directly upon it. Later when the fragment has

has become freed it will appear as a loose body in the joint unless it becomes decalcified in the synovial fluid.

Hellstrom advises operation despite the absence of symptoms after the diagnosis has been made, as he believes that if such treatment is not employed osteo-arthritis will develop. In Fairbank's (35) opinion, however, this late complication cannot be prevented even by early operation.

In the presence of characteristic symptoms an arthrotomy is indicated. If, when the joint is opened, the articular surface is found to be unbroken but the site of the lesion clearly indicated by the change in color and countour of the surface and it appears to be positively diseased, it should be removed complete with its necrotic bone base. If the fragment is free in the joint space it should obviously be removed.

The immediate prognosis and the prognosis for some years to come are undoubtedly good but the remote prognosis is less favorable in view of the rather high incidence of osteo-arthritis.

Osteochondritis Dissecans of the Hip

Osteochondritis dissecans of the hip joint deserves a few remarks by itself in view of the recent interest in its occurrence.

It was first mentioned as a true hip disease in 1925 by Haenisch. Richards (85) in 1928 in discussing the roentgenolographic picture of osteochondritis of the hip included in his paper the case report of a young man suffering from this involvement of the hip. Richard's patient was also the first to be subjected to surgical intervention, apparently with a favorable result. During 1929, more cases were reported by Bergmann, Goldau and Lange to be followed in 1930 by Gold who reported two cases. In 1931 Mangin reviewed the literature and concluded that Konig's disease actually exists in the hip joint.

The most recent literature lists only fourteen cases which have been reported, of which only two were females. Without doubt, this disease occurs more frequently than indicated by the literature but due to failure in recognizing and reporting the condition it appears to be decidedly rare. It attacks the femoral head usually on the supero-lateral aspect and never involves the whole of the caput. It has been reported bilateral in thirty per cent of the cases.

Symptoms are pain in the hip, mechanical in nature, which is aggravated by motion and weight bearing and relieved by rest. Occasionally there may be some slight degree of "locking" of the joint. Spasm of the muscles about the hip joint is common. The gait is

usually of the coxalgic type and mild atrophy of the gluteals gradually develops.

In this disease there is no flattening of the head nor any changes in the neck by x-ray. Free separation of the fragment is almost impossible because of the anatomic arrangement of the joint.

As in osteochondritis dissecans of the knee, arthrotomy is the only procedure by which relief is gained. Much controversial material against operation has been written with the following apparently the major objections:

(a) The hip must be dislocated, injuring or tearing the ligamentum teres.

(b) There is little proof that the hip will not be painful even following operation.

(c) Removal of the loose body leaves a depressed articular surface at this point which may lead to traumatic arthritis.

However, arthrotomy, with removal of the loose body, does offer a chance for complete cure and does give considerable immediate relief to those suffering from osteochondritis of the hip.

Osteochondritis of the Patella

Both primary and secondary osteochondritis of the patella are rare diseases. The primary type, involving the main center of ossification was first described by Kohler in 1908. It is more common about the age of six years at which time the primary ossification center is rapidly growing.

Sindig Larssen in 1921 presented two cases which had never before been described in the literature and in the following year Johansson reported four cases of the same lesion which now bears the name of these two men. It is not infrequent that the patella has more than one center of ossification, and it is this anomalous epiphysis which is affected in Larsson-Johansson's disease. The accessory center is usually at the outer margin of the patella at its inferior pole or along its lateral aspect. It may be mistaken for a fracture and is most often bilateral. The age group for this disease is ten to fourteen years of age.

The etiology is considered the same as that in Osgood-Schlatter's Disease.

Treatment of either type of osteochondritis of the patella consists of physiological rest in a plaster cylinder or adhesive strapping for a few weeks depending upon the severity of the disease. This should be followed by massage and increased active motion. Recovery clinically and by x-ray should be complete after six months.

Legg-Calve'-Perthes' Disease or Osteochondritis Deformans Juveniles

Between 1909 and 1910 several authors independently described this disease and each one offered a different opinion as to its etiology. Waldenstrom (101), who claims to have written the first description, claimed it was a benign form of tuberculosis; Legg in 1909 believed it was due to a staphylococcus infection; (63) Calve' in 1910 ascribed it to late rickets and Perthes in the same year reported it as a septic osteomyelitis. All these men were correct as far as the description of the disease went, but none were right with regard to the etiology, and this is still an unsolved problem today.

There are several synonyms for this disease, the most frequently used being Perthes disease, Osteochondritis Deformans Juvenilis, and Coxa Plana. The latter term was given to the condition by Waldenstrom in 1920 because, while it takes no account of the etiology, it does describe the appearance of the deformity, the flattened epiphysis. Many look upon this flattening as a late stage of the disease but Waldenstrom feels that it is the principal sign of the pathological process in its very earliest stages. Gill (43) and others say there is no flattening if weight bearing is not allowed.

For the sake of convenience, the cases may be divided into True and False Perthes' disease. The latter are those which follow such conditions as Congenital Dislocation of the Hip. The former, it is felt, develops in a hip which was normal before hand.

The pathological process is the same in both cases.

The disease occurs in the age group of three to eleven and in males about eighty-five to ninety per cent, just the reverse of the sex incidence in congenital dislocation of the hip. There may or may not be a history of trauma.

Pain is the first symptom and is more commonly felt in the knee rather than the hip. Limp occurs early but along with the pain will subside with rest in bed and relief from weight bearing. Limited motion, particularly rotatory, a thickening of the hip joint region, and slight atrophy of the thigh are the main findings clinically. Bilateral disease is about as common as bilateral slipping of the upper femoral epiphyses.

The etiological factors most frequently considered are constitutional, hormonal, developmental, infections, traumatic, and circulatory. No one factor seems to be solely responsible for the massive subchondral bone and marrow necrosis which is the main pathological finding. If it is due to a constitutional or endocrine disturbance alone, it seems queer that this single particular area is selected for this disease. The developmental factors involves changes in the circulatory system. No growths on culture have been consistently found in any of these cases and bone abscess is never seen.

Thirteen patients with Perthes' have come to autopsy and have been recorded in the literature. The findings have been essentially the same in all,namely, massive subchondral bone and marrow

necrosis extending from the surface cartilage to the epiphyseal plate, extensive granulation tissue, intact epiphyseal cartilage, edema and hemorrhage of the round ligament with obliterative thickenings of the blood vessels. In children who have been autopsied without Perthes' disease, the vessels in the round ligaments have appeared normal or have shown some early obliterative changes, but much less marked than those in Perthes. With a diminished or absent blood supply through this source, the epiphysis depends on the two small vessels reaching it through the periosteum. No vessels enter the head through the cartilage plate. Thus the nutritional demand is greater than the physiological supply and aseptic necrosis results. What causes this diminished supply through the round ligament or periosteal vessels is the question that has not as yet been answered. Zemansky (109) does not believe that it is the same in all cases, but trauma is by far the more common agent. Only slight injury may upset the balance. Some anomaly may be present, but anomaly or normal, one need not postulate complete ischemia, but merely a quantitative diminution of the blood supply.

The most striking uniform feature is early necrosis in the metaphysis of the neck. The areas of necrosis differ in size, shape, number and locality. They more commonly appear at the outer margin or in the center of the neck. Soon they fuse or coalesce and form a band like area across the entire metaphysis. Waldenstrom's theory is that the changes take place in the head first, and that

as a result of the flattening of the epiphysis the distance between it, and the bottom of the acetabulum is greater than normal from the very beginning of the disease. This distance is further increased by the partial subluxation which occurs, as the head becomes changed. But this subluxation is due to flattening of the head by weight bearing, which does not happen if the hip is well protected early in the disease. Thus, it would seem that this is rather a late manifestation of disease.

With the neck involved the subsequent changes are in the head. The first area of necrosis in the head overlies the first area in the metaphysis. It is difficult to escape the conclusion that the degeneration of the head is a result of necrosis in the neck. The process goes on to decalcification, fragmentation, and changes in the size and shape of the epiphysis. It may appear almost completely destroyed. The period of degeneration and disintegration lasts about one year and a half. It may be well advanced by the time the first symptoms present themselves. Lack of any proper treatment definitely prolongs this period.

Lewis (68), after reviewing many films, states the x-ray findings follow closely the pathological process. The articular cartilage remains intact and there is no absence or diminution of the joint space. If the condition is not treated, flattening and deformity results, due to necrosis far in excess of the reparative process.

According to Ferguson and Howorth (36), the earliest

changes seen in the disease is a distended capsule and swelling of soft parts. Of course, this is not diagnostic of Perthes' disease. Then the joint space seems to become wider especially in the inferior part, and there is slight flattening of the crest of the head. This fits in with the picture give by Waldenstrom.

Legg (64) describes two types of heads, the cap - and the mushroom. The cap type practically always mushrooms and migrates well out toward the greater trochanter. After fragmentation has taken place it begins to fill in with new cortical bone, and ultimately becomes solid but remains flattened. The prognosis is poorer, and the course of the disease may be four to five years. The course of the mushroom type, on the other hand, is distinctly different from the cap type. It does not show as much atrophy nor does it fragment as often. The migration of the epiphysis is rarely as much as the other type and therefore is less limited in abduction when healed. The end result is reached considerably sooner and is much better.

The change from degeneration to regeneration occurs rather abruptly, as can be seen in serial x-rays. The healing process starts in the neck and in the location which showed the first necrosis. The time for complete regeneration takes two to three years. This period is naturally delayed by improper treatment. All sorts of deformities may result from poor treatment, while correct treatment instituted early should result in a neck,

head, and acetabulum of normal size, shape and density.

Legg, in 1927, discussing the treatment, said:- "While a process suggesting weakness of bone structure is going on, it is theoretically sound to allow no weight bearing, but in practice relief from weight bearing in no way affects the end result."

Sundt, about the same time remarked, "The treatment directed to the elimination of weight bearing has not proved influential on the train of morbid changes, but its application is indicated during the stage of prominent symptoms."

The concepts of the best treatment have changed during the past few years. In the early thirties, Danforth (27) advocated bed rest with traction for the first few months. He reviewed several cases which were originally thought to be Tuberculosis, but which now appear to be Perthes' disease. These cases had all been treated with traction and/or casts, and their end results were far superior to those treated by allowing weight bearing. This is now the accepted method of treatment. Buch's Extension should be used until the acute symptoms have subsided. Pike (83) then applies a Wu splint which is worn night and day except for the periods of time that the patient is receiving active exercises, stretching and massage to maintain muscle tone and increase joint function. Ambulatory non weight bearing splints do not seem to be satisfactory because they allow a minimum of weight bearing which is detrimental to a good end result.

The prognosis of Perthes disease depends on the age of the patient, the duration of the disease before treatment, the type of head, and the efficiency of the treatment.

No weight bearing should be allowed until reparative process is so advanced that the head can support without damage the articular cartilage. Casts should not be applied because of the possibility of limitation of motion. Drilling does not seem to help and is definitely to be condemned because of the fact that it closes the epiphyseal line.

The general well being should not be overlooked. Tuberculosis tests should be carried out as well as sedimentation rate. All foci of infection should be removed. Endocrine disturbances should be corrected as far as possible.

SUMMARY

Perthes disease is by far the most common of these diseases with Osgood-Schlatter's disease, Osteochondritis Dissecans, Freiberg's Infraction, Kohler's Tarsal Scaphoiditis, Scheurman's Vertebral Epiphysitis and Keinboch's disease following in that order. The other conditions are rare.

That all of the separately described diseases are manifestations of a systemic affection is now generally agreed upon. Hartley states, "It is interesting to note that each lesion is associated with a definite age period and in each, the age period is that in which the affected bone nucleus normally is actively developing."

Christie is of the opinion that no epiphysis in the body is immune to the disease and that it is the same pathological entity modified only by the particular location.

It is definitely agreed by all men that tuberculosis and syphilis are not responsible for any of these lesions. Late rickets has been suggested as a cause of this disturbance but clinically and roentgenographically there is no evidence whatsoever of rickets. All laboratory tests on blood calcium, phosporus and phosphatase have been normal.

The theory of endocrine dyscrasia is purely an assumption, for the great majority of cases are healthy active children who do not present any evidence of endocrine dysfunction. However, in a few cases, it is known that there exists a decreased or very low

metabolic rate which means a diminished circulation to all parts. Combine this with repeated minor injuries or a developmental anomaly and the balance between the nutritional demand and the physiological supply is upset. The endocrine theory cannot be presented as the etiological factor, as the only one in all cases, but it may well be a predisposing or contributory cause in a few cases.

The infectious theory has only in its favor the observation that several surgeons at operation have obtained positive cultures. It is quite likely that the positive culture have been incidental or due to contamination. Furthermore, none of the conditions have been known to suppurate, and there is a complete absence of inflammatory reaction.

Axhausen's theory of aseptic embolism, necrosis, and minute compression fractures resulting from slight traumata has been discounted by many writers. It seems inconceivable that emboli should always occur at given age periods in given locations. Just why embolism should occur in the spines of otherwise healthy individuals first during the first few years of life and then after a quiescent period recur during the second period of rapid growth is difficult to explain on this hypothesis. Similarly it does not explain the bilateral cases, nor the changes occurring in the femoral neck along with the changes in the head of the femur.

Trauma seems to play an important part in the causation of these disturbances. Mau has pointed out that rapidly growing

bone cells are physiologically weak. If at the time of rapid growth a static imbalance occurs either from an increased stress or strain, or a decreased capacity to withstand stress or strain, a derangement of the normal process of growth occurs, giving rise to compression fractures, irregularities of growth and vascular changes. Furthermore, the various diseases occur most frequently in the lower extremity and at the various epiphyses most subject to stress and strain.

Though it must be admitted that the etiological and pathogenic concepts of osteochondritis are still somewhat indefinite, yet one is forced to admit that trauma, in the form of increased stress or strain, is in all probability a very important factor.

To sum it up briefly, aseptic necrosis occurs because the nutritional supply is not equal to the physiological demand, and this is produced by internal or external trauma, endocrine dysfunction, developmental anomalies, rapidity of growth, or any combination of the above factors.

Osteochondritis dissecans may be a late result of this aseptic necrosis and various writers believe in a few cases it is. However, we are led to believe today that most cases occur in an otherwise normal joint and that they are small fractures which, aggravated by weight bearing and motion, never unite. Internal trauma is by far the most common causative agent producing these fractures.

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