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Osteochondritis dissecans of the knee joint

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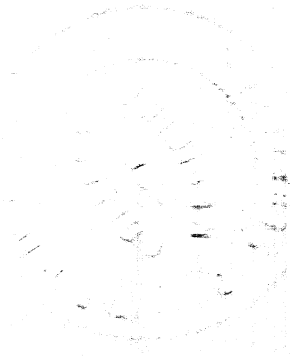


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OSTEOCHONDritis DISSECANS OF THE KNEE JOINT

Rudolph G. Schmidt

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to the

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PREFACE

In presenting the subject of Osteochondritis Dissecans of the Knee Joint as my senior thesis, I have tried to cover a subject of particular interest to myself, having once been a sufferer with this disease entity. The scope of this paper will be limited to a review of the American Literature, the course and treatment of the disease. The technic of operative procedures has intentionally been omitted.

I wish to express my appreciation to Herman F. Johnson, M.D., for the loan of his personal case histories which have been presented in this paper. I also wish to take this opportunity to express my appreciation to Wagoner and Coh, from whose article I have quoted at great length.

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OSTEOCHONDRITIS DISSECANS OF THE KNEE JOINT

Osteochondritis Dissecans is a non-infectious process involving the articular cartilage and the subchondral bone of certain long extremities, which by sequestration from the articular surface produce loose bodies in the joint. This body is originally of an osseocartilaginous composition but its structure subsequently undergoes alteration by action of the fluid of the joint. The knee joint is involved in 85% of the cases according to Conway, in 1937. The mesial half of the articular surface of the internal femoral condyle is the most frequently involved site but the heads of the radius, humerus, lateral head of the femoral condyle, and the ankle may also be sites of the process. Osteochondritis Dissecans occurs most commonly in the young robust youths, most frequently males; probably because of the vigorous sports in which they participate. (Wagoner-1931).

The first record of this condition in the literature is given credit by King (1932) to Ambroise Pare, who in 1558 removed a stone from the knee following which there was complete recovery.

Roswell Parks, in 1891, describes six possible etiological causes in his article on "Loose Bodies

in the Knee". I quote as follows:

1. Hypertrophy and metamorphosis of the synovial fringe. The fringe may hypertrophy and form a small nodule. By some mechanical means the nodule becomes free in the joint.
2. Hypertrophy and metamorphosis of extra synovial tissue around the joint may grow and bulge into the joint, and by the same mechanical means become free in the joint.
3. Periosteal outgrowth--Really amounts to a tumor.
4. Organization of effused fibrin and blood. This was first mentioned by John Hunter.
5. Cartilaginous overgrowth.
6. Detachment by injury of some normal tissue.

This condition was christened by Koenig in 1888, (Phemister 1924). He reported cases with pathological examination and called attention to the frequent absence of history of trauma. His conclusions were that while some of the loose bodies from the articular surfaces are broken off by trauma the majority are broken off by a disease process of unknown nature which implies that the process is inflammatory but bacterial examination was not made, and his gross and microscopic findings fail to show active inflammatory changes either of the synovial lining or of the loose bodies.

Loose bodies in the joints are found in conditions other than that of Osteochondritis Dissecans. Until this distinct disease entity was named by Koenig, in

1888, such bodies were grouped under the common heading of "joint mice". The common causes of joint mice are briefly:

1. Osteochondritis Dissecans.
2. Free bodies of traumatic origin which arise in otherwise normal joints. An example is found in fractures of the semilunar cartilages.
3. Free bodies may appear in joints that are the site of arthritis deformans. These bodies arise by the traumatic or necrotic freeing of articular cartilaginous plaques, hypertrophic bony spurs, bony joint papillae, metaplastic cartilage or hypertrophic fibrinous synovial villi. These bodies are usually present in large numbers and vary greatly in size.
4. The poppyoid bodies present in Charcot's joint.
5. Masses of uric acid salts of various sizes found in gouty joints.
6. Osteolytic sequestrations.
7. Parts of tumors of the capsule in chondromatosis of a joint.
8. Fibrous or lipomatous joint papillae.
9. Parts of hypertrophic synovial villi following

a synovitis.

10. Rice bodies commonly described as the result of tubercular involvement of structures adjacent to the joint.
11. Following hemorrhage into, or inflammation of a joint, masses of fibrin may result and lay free within the joint.
12. Foreign bodies from external sources.

The literature of Osteochondritis Dissecans is divided into two large divisions; those in which trauma is set forth as the chief etiological factor and those in which factors other than trauma are considered as the etiological agent. It is my belief that the history can best be traced by presenting the various theories of etiology in chronological order. In this paper the theories of etiology will be presented in two parts, the two schools of thought being presented separately.

Theories of Etiology With a Traumatic Basis

Monro, in 1726, (Wolback 1928), advanced the theory that joint mice were of traumatic origin. He was supported by Reimer in 1770, and by Haller in 1776. (Wagoner 1931).

In 1848, Rainey (Wolback 1928) observed that

fragments of cartilage and bone, detached as the result of trauma, continue to grow and become sizeable loose bodies.

Brodhurst, in 1861, and Poncet, in 1881, contended that loose bodies were the result of trauma and arthritis deformans. (Wagoner 1931).

Kragelund, in 1884, assumed a trauma of the bone-cartilage area followed by the demarcation and separation of a portion of this area as the result of a chronic inflammatory process. (Wagoner 1931).

In 1887, Wagoner (1931) experimenting on the knees of cadavers corroborated the opinion of Paget by finding that it was impossible to detach a fragment of articular cartilage simply by a blow; an area could be loosened in this manner, but a prying force was necessary to separate it.

Burghard, (1892), found that an oblique blow upon the internal condyle with the knee flexed may with difficulty produce a complete separation of a piece of the articular cartilage.

Lane, (1893), emphasized the fact that during flexion considerable portions of the articular surfaces are exposed to trauma. Since this area is practically devoid of nerve supply he suggests that

even minor trauma, applied at the right angle might produce these fractures.

Experimentally on animals, in 1896, Hildebrand (Wagoner 1931) was unable to obtain permanent free bodies by surgical detachment of fragments of cartilage or cartilage and bone.

Preiser, 1898, considered Osteochondritis Dissecans to be the result of static imperfection. (Brackett 1917). Harris, in 1901, makes the statement that almost no one who reports a case who does not reach the conclusion that Koenig was wrong.

Boerner, in 1903, (Wagoner 1931) considered the production of loose bodies upon purely a mechanical basis.

While assisting with an operation, Codman (1903) noted that the bodies had the same general configurations as did scars often found on the articular surface. Further studies done on cadavers convinced him that this condition was the result of trauma. He also drew the conclusion that two injuries were necessary to produce a free body, one injury to produce the fracture, and one to free the fragment.

Cornil and Coydray, in 1905, (Wagoner 1931) found that bodies of traumatic origin produced through ex-

periment in animals became united to the articular extremity of the bone or to the synovial membrane of the joint, thus upholding the work of Hildebrand in 1896.

Conflicting reports, however, came from Riman in the same year (Wagoner 1931), who following experiments on both dogs and goats stated "there is neither a secondary nor a primary Osteochondritis Dissecans." True free joint bodies arise only upon a traumatic basis.

Ludloff, in 1908, (Wagoner 1931) advanced the theory that loose bodies were the result of injury to the arteria genu media at a point where the artery perforates the capsule, the resulting infarct leading to necrosis of the area of bone supplied by this artery. The necrotic bone gradually separating because of insufficient nourishment. It has, however, been shown that this is not an end artery. There is some contention, however, that the collateral circulation in the articular cartilage is not a functional collateral circulation.

Axhauser, in 1914, (Wagoner 1931) assumed that violence played a slightly different role. As a result of the impaction from the forces of the blow, injury

occurred to the vessels. This would lead to necrosis of the bone supplied by the damaged vessels. Instead of the creeping substitution which would take place in quiet necrosis, Axhauser believes, that a zone of absorption is established resulting in a gradual separation and eventual extrusion into the joint.

Friedrich, in 1913, and Bernard, in 1925, (Wagoner 1931) both favored the theory of traumatism.

Fisher, in 1920, stated that detachment could occur by indirect injury through tension upon the posterior ligament of the knee joint, which is attached immediately adjacent to the articular margin and is powerfully reinforced by the tendons of the gastrocnemius and semimembranosus. To strengthen his argument for trauma, Fisher chiseled off a small piece of articular cartilage, together with a portion of the underlying bone from the femoral condyle of a rabbit. "The portion was completely detached and pushed up into the suprapatellar recess of the joint", and then closed. Five weeks later the loose body was removed and examined microscopically. His findings did not uphold the findings which would be expected in a quiet necrosis as explained by Paget.

Kappis, in 1920, (Wagoner 1931) pointed out that in

the knee and elbow which are the common seat of loose bodies, tangential and rotating forces may act on the convex surface of the condyle causing a fissure and partially or completely detaching a portion of the articular end. To explain the traumatic origin in the absence of a history of serious injury he assumes that the articular cartilage has little or no nerve supply and the underlying spongy bone is extremely insensitive. This would permit the occurrence of serious injury with little or no serious pain.

Phemister, in 1920, believed Osteochondritis Dissecans in all cases to be a fracture caused by mechanical relationships or slight unnoticeable trauma which results in either complete or partial separation of the loose bodies.

Hellstrom, in 1922, (Wagoner 1931) first expounded the subchondral fracture theory. The failure of firm healing of the fracture completes the clinical picture. He states that in giving legal opinion, the disease must be regarded as an occupational disease produced by trauma.

Experimentally, in 1923, Freiberg, by means of x-ray studies of five cases and the knees of a number of cadavers found that it was quite easy to make the

tibial tubercle impinge upon the posterior crucial ligament when the knee was flexed and the tibia rotated outward. In cases where the tubercle was long, the impingement occurred much sooner and it seemed more easily conceivable that it might take place with enough force to damage a small vessel, but he is more inclined to look upon this condition purely from the traumatic origin.

In 1923, Burckhardt (Wagoner 1931) experimented on cadavers and showed that injury of the median condyle takes place from pressure on the patella. By calculation according to the laws of mechanics it was shown that sufficient force could be derived from contraction of the quadriceps extensor muscle to produce a fracture.

Schmidt, in 1924, (Wagoner 1931) demonstrated on the cadaver that at the level of the condyles the cartilage in the joint was more easily injured by a tangential than by a vertical force. By varying the position of the patella when the leg was internally or externally rotated, he found that the patella came in contact with the external condyle when rotated outwardly, and with the internal condyle when the leg is rotated inwardly. He claimed that condylar fragments are broken off by impacts between the patella and the condyles following

sudden rotations of the leg in flexion.

Phemister, in 1924, was unable to create loose bodies in the joints of dogs, either experimentally with force, or with the use of radium. He found that bone chipped off and then reinserted in its bed became reattached or was absorbed and disappeared in a couple of months.

Hauptli, also in 1924, (Wagoner 1931) stated that Osteochondritis Dissecans was purely traumatic, complete or incomplete detachment of cartilage, or bone and cartilage in the joint space. He found that if the bits are incompletely broken off, the process of healing in the fragment leads to rudimentary callous formation and now and then to consolidation. The movement and trauma of daily life is sufficient to prevent this consolidation and cause detachment into the knee joint. He also believes that there is a predisposition to the disease, probably due to special bone configuration in the joint, hyperthyroidism or hypothyroidism, late rickets, or most of all by an arthritic constitution.

Leb, also in 1924, (Wagoner 1931) concluded that since no chronic inflammatory process could be demonstrated, this theory should be discarded. He believed the disease could be explained completely by joint

fractures.

Von Dittrich, in 1925, (Wagoner 1931) thought that Osteochondritis Dissecans was the result of mechanical and functional traumatic influence.

Balensweig, in 1925, believed that Osteochondritis was the result of an osteochondral fracture of the external condyle of the femur as a result of cross strain, aided by the pull of the anterior crucial ligament.

Wolback and Allison, in 1928, described the condition as the effect of mechanical pressure on a portion of the articular cartilage with underlying cancellous bone bridging a cyst. In order to have a working theory on this basis, there must be sufficient loss in the condyle of the cancellous bone to weaken the support of the articular cartilage in a position to produce strain upon its covering.

Richards (1928) described a case with bilateral involvement in which he found, by means of the x-ray, that the mesial tubercle of the tibial spine was elongated. He therefore suggested a purely local traumatic process.

Krida, in 1930, stated that he had never operated on a case in which he could hold any other etiological factor except trauma. He states, however, that Dr. Milch,

his associate, believes Osteochondritis Dissecans to be a group of conditions in which trauma is only one phase.

That Osteochondritis Dissecans was the result of a subchondral fracture, was the belief of Littlejohn in 1933. In fact he believed that because of a similarity of history, signs and symptoms, x-ray appearances and course, all osteochondritides may be explained on that basis. He assumed that the cartilage is insensitive and that the underlying bone, which is devoid of periosteum has only a very meager nerve supply. By directing a blow on the patella, the force would be transmitted through the articular cartilage which acts as an elastic sponge, split off from the underlying bone of the condyle; a chip much the same as a blacksmith by pressure with a piece of wood takes flake off his flint. The elasticity of the cartilage prevents it from splitting with the bone, and thus a subchondral fracture is born. Because of the relative small amount of pain present, the joint would soon be in use again. Because of rocking of the fragment in its bed as the result of weight bearing and movements, non-union would most likely occur. Non-union implies a fibrocartilaginous covering for the adjacent bone

surfaces, and this is usually found.

Fairbanks (1933) draws certain conclusions following a thorough study of the literature and his own private cases: The typical lesion of Osteochondritis Dissecans is a fracture and nothing else for the following reasons:

1. It most frequently occurs in adolescents and young adults indulging in vigorous pastimes.
2. Typical lesions are seen in radiograms and revealed by operation after definite trauma, which in some cases is quite recent.
3. A lesion at the typical site may involve the cartilage only, the detached fragment consisting of normal articular cartilage. In such cases there is a definite history of trauma.
4. There is an entire absence of any inflammation, both macroscopically or microscopically, in or about the lesions.
5. The naked eye appearances, when operation is performed early, offer nothing but a simple recent fracture. When sufficient time has elapsed for changes to occur, they are only those which we should expect as the result of an effort on

the part of tissues to repair the damage. Precisely similar changes are occasionally found on the more exposed parts of the femoral articular surface, where the traumatic origin of the lesion is never disputed.

6. When the detached fragment is suspended by a vascular pedicle, the bone in it is not dead and is not a sequestrum, so why should it be exfoliated.
7. To explain the occurrence of the lesion in both knees or the knees of more than one member of a family, it is easier to accept the presence of anatomical peculiarities which favor exceptional local trauma than the suggestion of embolism, damage to blood vessels, or indeed, any other theory.

He concludes that violent rotation inwards of the tibia, driving the tibial spine against the inner condyle is responsible for the lesion in most cases.

Three distinct incidents of trauma with the production of three bodies was reported by Monteith in 1934. He found at operation that three bodies were present, and was of the opinion that one was produced with each trauma.

Conway, likewise, in 1934, states that the most logical explanation seems to be that which allows for a preliminary trauma to a non-sensitive articular surface with subsequent injury to a functional end artery. Following the vessel damage and thrombosis, a localized area of necrosis results with sequestration from the articular surface.

Brailsford (1935) was of the opinion that the loose body is the result of sequestration of a small fragment of subchondral bone. This separation is probably due to trauma and subsequent local vascular disturbances.

Lesions of the carpal scapoid and the palmer surface of the semilunar have been described by Funsten and Kinser (1936). They uphold that trauma is the primary cause followed by a localized area of aseptic necrosis stimulating an infarct.

Outland, in 1936, reported a case in which there was a defect found at operation in the articular cartilages of both condyles following one specific blow.

By means of x-ray, Miller, in 1936, followed a case of Osteochondritis Dissecans of the elbow through the development of the loose body. He is of the opinion that Axhauser's theory can most readily be applied to this condition.

Galloway and Macey, in 1937, reported a case of bilateral involvement with bilateral dislocation of the patellae. The first history of trauma goes back to the age of six years on the right knee, and fifteen years on the left knee. This leads to the belief that this condition is one of trauma superimposed upon some predisposing anatomical inadequacy, probably rickets.

Theories of Etiology With a Non-Traumatic Basis

Perhaps one of the first theories not supporting the theory of trauma was sponsored by the father of surgery, John Hunter. He believed that the formation of cartilaginous bodies in joints was due to extravasated blood which assumed the nature of the parts into which it was effused (1793).

Rainey and Solly, in 1848, concluded that the glands of the synovial membrane, instead of secreting synovial fluid, under some unknown influence produced cartilage instead, which later became converted into imperfectly formed bone.

Rokitansky, in 1851, stated that "bodies composed of bone and cartilage might arise from the articular serosa, representing an excessive development and ossification of isolated nodules of cartilage". (Wolback

and Allison 1928).

Paget, in 1870, made the statement that "these loose bodies are sequestra, exfoliated after necrosis of injured portions of cartilage, exfoliated without acute inflammation.

Kock, in 1879, (Wagoner 1931) performing experiments on embolic necrosis of the bone proved to his own satisfaction that loose bodies were the result of the obstruction of the entire capillary area of nutrition.

Konig, in 1887, described Osteochondritis Dissecans as a separate entity. He concluded that it was a blasting of a piece of the surface of the joint, which resulted in the severance of a piece of the joint surface without otherwise damaging the joint. He did not deny that trauma did have some part, however, he thought that it served to merely accentuate the damage already present. Konig, however, was convinced that the true etiology was obscure.

Roswell Parks, in 1891, described the following as possibilities other than trauma:

1. Hypertrophy and metamorphosis of the synovial fringe. The fringe may hypertrophy and form a small nodule. By some mechanical means the nodule becomes free in the joint.

2. Hypertrophy and metamorphosis of the extra-synovial tissue. The tissue around the joint grows and bulges into the joint and by the same mechanical means becomes free in the joint.
3. Periosteal outgrowth or in reality a tumor growth.
4. Organization of effused fibrin and blood as assumed by John Hunter.
5. Cartilaginous overgrowth.

Preiser, in 1898, presented the static imperfection theory. He states that improper weight bearing from flat feet and other causes may produce this condition. In the hip, the superior portion of the acetabulum or the superior weight bearing portion of the head of the femur may be affected. (Wyckoff 1938).

In 1903, Codman stated that the prevalent theory was that loose bodies of the joints were concretions comparable with the formation of biliary and cystic calculi. The supposed nucleus was a clot of fibrin, a piece of cartilage or a bit of torn fringe. Codman, however, believed trauma to be the true etiology.

Henderson, in 1916, suggested some metabolic condition. He based his opinion on the fact that people are often seen with brittle finger nails, brittle hair,

brittle bones. He, therefore, thought that it was conceivable to have a brittleness of the articulating surfaces. This combined with the great strain thrown on the internal crucial ligament when the knee is extended, and the constant tugging of this ligament might have some effect upon the cartilage.

Axhausen, who at first was a strong advocate of the damage to blood vessels theory, suggested in 1924, that this might be due to embolism. He attributes the lesions to embolic infarction. He suggested attenuated tubercle bacillus, or some bacteria of greatly reduced virulence be deposited, which would lead to closure of an epiphyseal end artery. Since the bacteria are of low virulence they are immediately conquered and the necrosis remains aseptic. Paitre and de Bouguet, also supporting this theory, suggested a "special fragility" of the epiphysis. (Fairbanks 1933).

Bernstein (1925) reported three cases in one family in which the condition occurred bilaterally. Upon this he bases the opinion that there must be some familial predisposition to the disease upon which trauma is superimposed.

Reiger, in 1920, suggested fat embolism and reported that he found evidence to support it. However, no one

has ever been able to reduplicate his findings. (Fairbanks 1933).

Knaggs in his conception of "the quiet necrosis of Paget" believed that the initial lesion was one of low grade periostitis due probably to staphylococcus, because of its low virulence the infection does not spread deep in the bone, but is speedily limited. Granulation tissue developing in the deeper structures causes a compressing of the blood vessels with subsequent necrosis. (Fairbanks 1933).

Archer and Peterson (1930) are of the opinion that Osteochondritis Dissecans is but one phase of the deforming, non-infectious disease of adolescence, examples of which are Legge-Perthes disease, Kohlor's disease, and sliding epiphysis.

Wagoner and Cohn, in 1931, reported five cases of Osteochondritis Dissecans in members of one family. On this basis they conclude that heredity is a factor in the etiology.

Ghormley of the Mayo Clinic, in 1932, supported the theory of Wolback and Allison, in which cystic degeneration was advanced. He, however, also thinks that there may be some relationship between these conditions and the disturbances of epiphyseal growth.

Metabolic upset was advanced by Stevenson and Henderson in 1937. They reported a case in which they were able to demonstrate decalcification of other bones as well as the affected bone. Blood studies showed a normal calcium, but urine studies showed a decreased calcium output. This together with the experimental work of Mirvish and Bosman upon the effect of gonad extracts on the blood calcium, lead to the theory that Osteochondritis Dissecans was in reality a temporary hypergonadism. The gonad activity causing a constant lowering of the blood calcium with consequent withdrawal of lime from the bone to maintain the blood level. The normal food calcium supply is either deficient in the circumstances or is insufficiently absorbed and utilized. The result of this process is decalcification. Recurrent trauma is believed to be responsible for the dislodgement of the bodies.

That Osteochondritis Dissecans definitely emulates Legge-Perthes disease was concluded by Balensweig, in 1938. He reported a case involving the entire articulating surface of the distal end of the femur. He states that "Osteochondritis Dissecans may be looked upon as resembling in great part Osteochondritis of the first decade of life, in which, as a result of faulty nutri-

tion and permanent damage to the underlying vascular structure, a loose segment of bone is thrown off to produce a resemblance to the subchondral fracture. In other words Osteochondritis Dissecans may be a late stage of certain forms of Osteochondritis Juvenalis.

Case Reports

Case 1. S. S. A 51 year old female. Six years ago following a twisting fall on the right knee, the knee was twisted medially and bent medially. Pain on the medial and anterior side of the knee. The knee had locked on several occasions. An elastic knee bandage was constantly worn. Walking caused pain in anterior medial aspect of knee. Physical examination showed no swelling or increased fluid. The knee was painful to motion and tender over the medial semilunar cartilage. On flexion there was definite grating. Patient was not operated.

Case 2. D. H. A 34 year old male. Had cartilages removed in 1932. Was well until 3, 1937, at that time following exposure to cold he woke in the morning with pain, stiffness and swelling in the

knee. Improved when not on feet. Any further attempts to walk brought increased pain and swelling. X-ray, AP and lateral showed irregular fragments of bone separating from the medial condyle of the femur measuring about 15 millimeters in diameter and 3 to 5 millimeters in thickness. Arthrotomy was done, followed by recovery.

Case 3. A. J. S. A 58 year old male. Kicked on outer aspect of knee. Patient fell to the ground with the left leg bent under the right. The patella was dislocated medially. Patient was treated by immobilization for five weeks, following which, attempts at walking produced severe pain. X-ray showed an area of decreased density in the intercondylar space. Operation-Arthrotomy, medial patellar incision was made. The patella being retracted laterally. A soft whitish area of cartilage on the articular cartilage was seen. Cartilage was removed down to the normal bone. Checkup showed an approximate disability of 25%.

Case 4. A. F. A 17 year old male. The right knee locked suddenly three days previous. Locking

persisted. History of painful creaking of knee for some time although never disabled before this time. Operation showed an almond sized calcareous body attached by a pedicle which was removed from the intercondylar space. Internal cartilage also found relaxed, and was excised. Good recovery.

Case 5. C. K. An 11 year old male. Complains of pain on medial aspect of knee when walking and running. Patient had had flat feet since birth which had improved with treatment. Physical examination showed severely pronated feet, otherwise negative. X-rayed on suspicion. AP view showed Osteochondritis Dissecans very clearly. Condition explained, and warned to be careful. Checkup to follow later.

Case 6. L. D. While playing basket ball two months ago patient received a twisting injury to the right knee. Was forced to discontinue play. Later patient noticed a slip of the knee with each step. Sense of bone rubbing together was also complained of. Locking had occurred only once. The knee was twisted and the knee unlocked. Pain had continued but was not as

severe as previously. Occasional swelling had occurred. Patient had been unable to work since the injury. Physical examination showed slight effusion. Upon flexion of the knee, there was definite sensation of bone slipping. X-ray--typical of Osteochondritis Dissecans. Three small calcareous bodies were visualized. Operation Arthrotomy of the area found it to be attached to the articular surface of the femur by a pedicle. Bodies removed. Complete recovery.

Case 7. A. D. M. A 23 year old male. Patient complained of locking of the left knee on several occasions the past three years. Gives a history of several minor injuries. Physical examination showed marked weakness. There was a defect in the central portion of the quadriceps muscle about six inches above the knee. There was demonstrable weakness upon extension. Operation-- The internal condylar margin of the articular cartilage of the femur showed irregular proliferation with panus formation. Four bodies were removed. Recovery was satisfactory.

Case 8. E. A. A 22 year old male. About three years

ago patient developed pain in the right knee. The knee had never locked. There was no history of trauma. The knee gave way suddenly when the pain struck. This usually occurred in partial flexion. The knee was bothersome in changeable weather. The physical examination was negative. X-ray showed possible early Osteochondritis Dissecans. Patient was again x-rayed six months later at which time the lesion was very clearly visualized. On operation there were no loose bodies. However, there was an area seen about the size of a quarter in which the cartilage had a different color. The osteochondritic area was incised and lifted out. Recovery was satisfactory.

About two years later this same patient came in complaining of pain and a grinding sensation in the left knee. Again no history of trauma was obtained. X-rays at that time were negative. At the same time the patient complained of pain and popping of the right shoulder when the arm was raised over the patients head. X-ray and physical examination were both negative. Patient had not returned for a checkup.

Case 9. F. G. A 20 year old male. Patient complained of pain in the right knee upon exertion. There was only history of minor trauma. Patient stated that the knee never locked, but that it occasionally seemed to slip following which there was usually some effusion. The knee was more painful after a days work. Physical examination revealed only moderate tenderness over the medial condyle. X-ray revealed a typical area of Osteochondritis Dissecans. Operation--the area of Osteochondritis Dissecans was enucleated following which there was complete recovery.

Case 10. K. E. A 21 year old male. Patient states that he suffered a twisting injury to the knee. The knee had not locked but was painful to full extension. Physical examination showed that the patient walked with a limp. Complete extension was painful. There was tenderness over the medial condyle. X-ray showed typical area of Osteochondritis Dissecans. Operation with removal of the bodies was followed by complete recovery.

Case 11. L. A. Patient a 33 year old female. Patient

stated that she suffered a twisting injury to the right knee about 13 years ago. Since that time she had no definite constant symptoms until about 18 months ago when she developed a sensation of giving way and weakness. She developed a limp about one year ago and for the last three months she had to go about on crutches. Physical examination was essentially negative. X-ray showed an almond shaped mass in the intercondylar space. Operation--removal of a pedunculated mass of cartilage. An area was seen on the medial condyle which had become filled with granulation tissue. Recovery was satisfactory.

Case 12. R. S. Patient a 24 year old male complained of vague pain on the medial side of the knee with sensations of slipping. Hard to take steps and to walk far. History of trauma on four occasions. Has been operated for deranged cartilage. Knee had never locked. When knee gave way it seemed to slip medially. There was always effusion following this sensation of giving way. X-ray showed a lesion typical of Osteochondritis Dissecans with one fragment

in the posterior joint cavity and one in the anterior pouch. Operation Arthrotomy -- four loose bodies removed. Typical Osteochondritis Dissecans bed on the medial condyle of the femur. Recovery was satisfactory.

Symptoms

Symptoms of Osteochondritis Dissecans are by no means constant. The typical symptom of a loose body in the joint is that of locking. There is a momentary sudden pain, lasting only a few minutes, the joint remaining fixed in one position until unlocked. This is usually followed by a transient effusion into the joint with moderate synovitis.

This, however, is not a typical chain of events in our small series of cases just presented. The most common symptom is pain. This pain is most often classified as a dull ache usually located over the medial condyle. This pain is more noticeable after walking stairs, running or walking. This pain was present in nearly 100% of the cases.

Typical locking occurred in only three cases or in 25% of our series. However, a rather typical slipping is complained of in about 40% of the cases. This slip-

ping is described as a sensation of giving way which causes intense momentary pain. There is no characteristic locking, or at least the locking does not persist. This is usually followed by effusion into the joint with tenderness and stiffness.

In two cases pain on walking or weight bearing was so intense that the patient had to go about on crutches. One of these cases was, no doubt, a fracture of the chondrium. The pain began immediately following a traumatic injury and was not relieved by rest and immobilization. The other case was one in which there was a history of trauma thirteen years previously. Until eighteen months ago there were no symptoms. At that time she developed a limp which has progressively become worse, so that the last three months she has had to go about on crutches.

King, in 1932, divided the cases of Osteochondritis Dissecans into three general groups as far as symptoms are concerned:

Group I. This group of symptoms included the extremely painful swollen tender joints, which locked in 15 or 20 to 45 degrees flexion. These symptoms usually began suddenly, a few days previous, subsequent

to a twist or some other minor injury. There is usually no previous history of disability.

Group II. In this group he included the asymptomatic or slumbering cases of Osteochondritis Dissecans. These cases are usually discovered quite by accident, by means of the x-ray.

Group III. Here he classes those chronically troubled with the joint for 2 or 3 years. Complaint is usually of soreness or definite pain on weight bearing, associated with swelling. "Giving way" and stiffness were also common symptoms.

Physical Signs

Physical signs are not reliable. If the patient is seen during the acute state or rather immediately following injury there may be painful effusion into the joint. Usually there is pain over the medial condyle to pressure. Crepitus or grating can occasionally be felt on movement of the joint. Rarely can the joint mouse be palpated. Those cases in which locking occurs it is sometimes possible to palpate the body which will disappear when the

locking is relieved.

X-Ray

X-ray findings in Osteochondritis Dissecans is usually characteristic. Positive diagnosis can usually be made, however, cases in which the bodies are composed of cartilage it is occasionally difficult to visualize the individual bodies.

The importance of x-ray studies was emphasized by Ridlon (1913), Lamson (1921), Richards (1928) and Miller in 1936.

The knee joint may best be visualized in the AP view. It may also be visualized in the lateral view and if so, the additional information provided is of such importance as to make a study in both positions essential.

The defect is seen as a sharply defined shallow depression, irregularly circular in outline and containing in its center a button of bone, the density of which may vary from that of a fully separated sequestrum to one differing so slightly from the normal bone about it as to entirely escape notice. The defect is usually seen upon the outer side of the inner condyle of the femur, immediately opposite the mesial tubercle of the tibial spine. (Richards 1928).

Various degrees of development may be observed from

those cases in which the process is fully established to those which are examined during the early stage of the disease. Miller, in 1936, followed a case of Osteochondritis Dissecans of the elbow through the development of the loose body. From his observations, he concludes, Axhausen's theory can most readily be applied to the condition.

In the fully developed stage the button of bone has the density of a sequestration lying in a sharply defined niche or recess just large enough to contain it. At this stage the loose body is often actually what it appears to be, namely a completely separated sequestrum, requiring merely a force under the proper conditions to dislodge it into the joint. In the earlier stages, the button of bone is still attached to the condyle, either by a broad base or by a pedicle and its density will be more or less determined by the degree of this attachment. If the degree of separation is slight, it may be that the variation in density between the button and the normal bone of the condyle is so slight as to be easily overlooked. In such a case the only indication of its presence may be an indistinct line of demarcation forming an irregularly circular outline upon the articular surface of the condyle in this particular location. If

this line can be established beyond question, it may be considered of diagnostic importance.

Pathology

Macroscopically, Conway (1937) described, three definite stages in the development of loose bodies as he found it at operation.

The first stage, very early, shows a fairly well demarcated prominence of the articular cartilage covering the area, of a different color than the rest of the cartilaginous surface. When this area was incised, it was rather easy to separate and a beginning excavation of the cancellous subchondral portion of the articular end of the bone was observed.

The second stage showed that the fragment had become distinctly separated and was lying within the excavated area of the articular surface. Occasionally the fragment was still attached by means of a pedicle. The fragment was easily removeable. At this stage the fragment showed a distinct color change, having an ivory cast in contrast to the normal. In addition the cartilage was not firmly attached to the underlying bone and could be easily removed for a varying distance from the sequestered focus.

The case with which the process could be lifted off gives it the actual appearance of having been dissected off.

The third stage was merely a completion of the first two stages, and was characterized by the complete sequestration of the fragment from its place on the articular surface into the joint cavity.

The character of the synovial membrane lining the joint depends upon the amount of irritation that it has been subjected to from the loose bodies, and also the amount of hemarthrosis present, and the length of time this irritation is present. Changes in the synovial membrane represent all the stages between simple edema of the synovial papillae to a pronounced hypertrophy of the individual, and multiple single papillae which have been in direct contact with the loose body. The extent of this traumatic hypertrophic synovitis is in direct proportion with the amount of irritation and the length of time it is present. There is a striking subsistence of symptoms within a very short time following an arthrotomy.

The pathology seems to be quite constant in Osteochondritis Dissecans. Microscopically there is no indication that this is an inflammatory process.

It is interesting to note just what is the fate of these loose bodies in the knee. Ely, in 1915, reported a case in which there was a history of only one injury. At operation he found the body to be thicker than the place of origin, indicating that growth had taken place.

Fisher, in 1920, chiseled a small piece of articular cartilage together with a small portion of underlying bone from the femoral condyle of a rabbit. The portion was completely detached and pushed up into the suprapatellar recess of the joint. Five weeks later the loose body was removed. Microscopically the findings were almost identical with those found in Osteochondritis Dissecans.

Rombold found no evidence of degeneration of the hyaline cartilage. The bone trabeculae were of normal form but no stained cells were seen. The intratrabecular tissue was without blood vessels. Osteoblasts were seen lining several of the trabeculae. In some places the intratrabecular tissue was replaced by fibrocartilage. The fibrocartilage was connected with, and seemed to originate from the hyaline cartilage covering the articular surface of the specimen.

Littlejohn, in 1933, found that the fibrocartilage may be irregularly calcified. Apparently after separa-

tion of the articular bone from its blood supply, it will die except for a small portion which may obtain sufficient nutrition from the synovial fluid to survive. The growth of the loose body in the cavity of the joint, however, is due to proliferation of the fibrocartilage which derives its nutrition from the synovial fluid, and which may undergo several forms of growth. The bed from which the body originates fills and sometimes overfills with fibrocartilage.

In section, the bone will be found to be for the most part dead, although not uncommonly a small portion may be found to be alive. The bed is lined by fibrocartilage, the growth in thickness of which appears in some cases to have determined the freeing of the body into the joint cavity. In no case was there any evidence of recent fracture or any inflammatory process.

In few cases fine fibrils of connective tissue may connect the body with its bed and in these a larger proportion of the bone cells may survive, deriving some blood supply through these connections. More frequently there may be a vascular hinge formed from the synovial sheath of the posterior crucial ligament, and by its blood supply the bone fragment may survive or even grow.

In cases detected early and opened before the body is extruded into the joint there is no apparent pathology. On closer inspection a slight difference of color is noticed over the area. The cartilage seems also to have a slight increased sponginess. Incision of the cartilage will disclose the bony fragment lying in its bed.

Wyckoff (1937) likewise finds that the findings are quite uniform. The cartilage cells usually show signs of proliferation and the external bone cells are living while those in the center usually show signs of degeneration.

Treatment

The treatment of Osteochondritis Dissecans is Arthroscopy with the removal of the joint bodies. In cases where the fragment lies in the excavation and is not completely detached the piece should be removed. Some men advocate the curetting of the crater, but it is likely that the cavity will eventually fill in with fibrocartilage as is found in cases in which the body has been free in the joint for considerable lengths of time. In some cases it is usually advisable to remove the undermined portion.

The optimum time for operation is before the fragment has been completely set free in the joint. Since the joint changes, namely the synovitis, varies with relation to the amount of irritation and the length of time over which it has been applied, it can be seen that if the fragment is removed before it becomes free in the joint the irritation would naturally be lessened with consequent reduced function.

The technic of arthrotomy will not be discussed in this paper. However, it is worthy of mention that due to improved asepsis and technic the mortality from this operation has been reduced to a negligible figure. This is interesting in the light of statistics offered by Larry, in 1860. Of 170 operations performed 127 operations were successful, 33 died, and 20 were failures. Nine years later, Benndorff collected 169 cases, 109 of which were successful, 46 died and 14 were failures. Muller, in 1886, gathered 190 cases, 96% of which were successful and 4% died. Woodward up to 1889 found 104 cases with 6 bad results, 2 amputations and one death. Benj. Bell, in 1787, while speaking of those bodies in the knee that are not freely moveable, said, "In this case I would advise amputation of the limb". This remedy is no doubt severe, but it is less painful and less

hazardous than excision of any of these concretions that have been attached to the capsular ligament.

The period of convalescence of these cases is usually entirely uneventful. Motion is usually started about the 12th or 14th day. Weight bearing is usually safe after about 3 or 4 weeks, and at the end of 8 to 10 weeks the patient has usually ended his period of disability.

It has been found that physiotherapy and massage do much to relieve the residual synovitis and thereby the period of disability is markedly reduced.

In those cases in which it is not advisable to operate or if for some reason operation is refused, much can be done to relieve the symptoms by continuous physiotherapy and massage.

The immediate prognosis is very good. However, it must not be forgotten that in later life the possibility of "rheumatic" aches and pains are greatly increased. The possibility of a recurrence is small after the patient has past the adolescent age. In the series of cases presented only one case was out of the adolescent age. In that case there was no doubt that the injury was that of a fracture of the articular surface purely on a traumatic basis. Once the patient is safely through this period, there is no danger of recurrence except

from some traumatic accident severe enough to cause a fracture as was mentioned above.

Patient	Sex	Age	Site of Involvement	Number of Fragments	History of Trauma	Immediate Disability	History of Locking	Xray	Associated Conditions
S.S	F	51			Yes	Yes	Yes	Pos.	Negative
D.H.	M	34	Medial Condyle	1	no	Yes	No	Pos.	Negative
A.J.S.	M	58	Medial Condyle	1	Yes	Yes	No	Pos.	Negative
P.F.	M	17	Intercondylar Space	1	No	Yes	Yes	Pos.	Negative
C.K.	M	11			No	No	No	Pos.	Congenital Flat Feet
L.D.	M	20	Medial Condyle	3	Yes	Yes	Yes	Pos.	Negative
A.D.M.	M	23	Medial Condyle	4	Yes	No	Yes	Pos.	Negative
E.A.	M	22	Intercondylar Space	1	No	No	No	Pos.	Possible Left knee and shoulder involvement
F.G.	M	20	Interconylar Space	1	Yes	No	No	Pos.	Negative
K.E.	M	21	Intercondylar Space	2	Yes	Yes	No	Pos.	Negative
L.A.	F	33	Medial Condyle	1	Yes	No	No	Pos.	Negative
R.S.	M	24	Medial Condyle	4	Yes	No	No	Pos.	Negative

Following a review of the literature and the study of individual cases let us pass to a discussion of possible theories of etiology. In the series of cases presented trauma of some degree was found to be present in 75% of the cases. With such a percentage, trauma can not be overlooked as at least a part of the picture.

Trauma with injury to blood vessels with the formation of an infarct has been presented as a possible theory. It has been shown that anatomically the arteries to the articular surfaces are not end arteries. It has, however, not been shown that the anatomical collateral circulation present is a functional collateral circulation. The shape of the bodies is in general against that of an infarct. Comparison here must be drawn to the infarct found in tuberculosis in which there is a definite characteristic wedge shaped cavity which is not present in this condition. For the same reason emboli of either bacterial or fatty origin must be more or less disregarded.

Pathological studies fail to disclose any evidence of an inflammatory process. This would cast into disregard inflammatory processes either as the result of bacteria or following trauma. A bacterial infectious process would also be unlikely to occur in cases such as those

which are normally affected by this disease, namely the young, robust adolescent youth. It is unlikely that the cystic degeneration found by Wolback and Allison is a constant phase. It has never been possible to demonstrate this, and no other similar report has appeared in the literature.

This brings us then to the question of a subchondral fracture with nonunion. It must be admitted that the pathological findings in this condition are strikingly constant. The detached surface is always covered with fibrocartilage. This finding is consistent with nonunion. It is conceivable that trauma could produce such an injury. There are no associated joint changes to indicate any other process. It has been pointed out that articular surface and subchondral bone is relatively insensitive, and it is possible that severe trauma or injury would not cause symptoms in any proportion to the damage done. How is the fragment set loose in the joint? Littlejohn has gone to great length to show that due to the relative insensitivity of the area, the joint would no doubt be used immediately, provided that in the initial damage the cartilage was left unbroken over the site of the subchondral fracture. In this case there would be no effusion of blood or roughened surfaces to

cause irritation for the setting up of a synovitis. Continued trauma of every day life such as walking would cause rocking of the fragment in its bed. This would be sufficient to cause nonunion. Fibrocartilage fills in the bed and as it gradually grows thicker it pushes the fragment out where only a minor injury or jolt would be necessary to pry it out of its cavity. This has been upheld by pathological studies and Conway describes three definite stages in the development of a loose body as he has found it in his series of cases.

The question might well be brought up as to how this trauma must be applied to produce such a fracture. There have been many possible avenues of force suggested. Since this lesion is most commonly found in the knee, the knee will be used as the example. Force applied on the patella through the range of motion of the knee would in itself cover considerable articular surface. It has also been shown that when the knee is flexed there is a portion of the articular surface which is left unprotected. Then to, there is the damage which might be done by the tibial spine in violent twisting. The site of this lesion in Osteochondritis Dissecans, however, is quite uniformly characteristic. In Charcot's joint of the knee, chips are not infrequently broken off as a result of excessive

trauma consequent to ataxia and to the loss of the protective sense of deep pain. In this condition, however, they never originate from the lateral surface of the mesial condyle or from the inferior surface of the patella, but nearly always from the margins of the tuberosities of the tibia. This is quite contrary to the expected findings.

Given that trauma may cause a subchondral fracture, does it not seem peculiar that this should be an entity with such a characteristic age grouping? True, the young robust adolescent youth does participate in strenuous games, but, are there not laboring conditions which are engaged in everyday during which minor trauma is a routine occurrence, but in which this condition does not develop? I am inclined to believe that we are dealing with two distinctly different conditions. In one we have a subchondral fracture as the result of one severe trauma; which may occur at any age; while in the other we have a subchondral fracture superimposed upon some condition which would predispose the individual of adolescent age to this entity. In the series of cases reported 10 cases fell within the usual age group of 17 to 24; one case fell in the group of subchondral fracture as the result of severe trauma, while the other fell

into a group which Preiser would call static imperfection, that is with an associated condition such as congenital flat feet.

Case 8 presents an interesting problem, with no history of trauma, an Osteochondritis Dissecans of the right knee developed. This was relieved by arthrotomy. Two years later he again returns complaining of similar pain in the left knee. X-rays, however, were negative, and the patient was not operated. At the date of this writing the patient has not returned for a checkup. Does this patient fit into the metabolic upset described by Stevenson and Henderson which they attributed to a temporary hypergonadism. It would have been interesting to have had calcium determinations on both the urine and blood. Would the urinary excretion of calcium have been below normal? Would his bone have shown the general decalcification described by Stevenson and Henderson.

Case 5, likewise, is an interesting problem. At the age of eleven, a male boy with severely pronated feet develops a lesion typical of Osteochondritis Dissecans. Was the Osteochondritis Dissecans due to alteration of the weight bearing lines or was it a case of trauma pure and simple, or was it trauma superimposed upon some growth disturbance?

In the series of cases presented there were no cases in which more than one member in a family were afflicted. It is conceivable that some variation in either the blood supply to the subchondral area or in the anatomical structure of the articular surface would predispose members of a family to the same disease entity.

Of particular interest to myself, is of course, my own case, which is found as case 12. In my case there is definite history of trauma on four different occasions. The first trauma was a blow to the lateral side of the knee while the knee was fully extended. Following the injury there was immediate pain which persisted for a short time followed by some transient tenderness and stiffness. This was seemingly without residual consequences. About one year later the knee suddenly "gave way" while descending a flight of steps. The knee was not locked, but there was great pain present when walking. About one month later a twisting injury was received. After this injury it was necessary to keep the knee wrapped at all times because of a constant feeling of slipping in the joint, associated with pain. Diagnosis of Deranged Semilunar Cartilage was made and operation performed. At operation a small split was found in the medial cartilage. This was removed and the joint closed.

Recovery from the operation was without incident. However, following this operation there was still a sensation of occasional slipping. About 3 years later still another occasion of trauma following which disability was increased. It was following this injury that the diagnosis of Osteochondritis Dissecans was made and operation performed with relief of symptoms. When in this chain of events did the Osteochondritis Dissecans develop? Was it present at the time of the first operation?

There is nothing in my past development of any diseases which would indicate any late stage of a juvenile affliction. There is also no familial history of bone disease.

It is my opinion that this case could be entirely explained on a traumatic basis. However, the question in my mind as to why this disease entity should appear so often in the young adult is as yet unanswered.

There is no doubt that trauma contributes greatly to the development of Osteochondritis Dissecans. The symptoms, physical examination, x-ray, and pathological findings are consistent, and treatment by means of arthrotomy is satisfactory. However, additional studies are necessary to definitely establish the true etiological agent, and the role of trauma as a part of this etiological agent.

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