OSTEOCHONDRITIS DISSECANS

(contd.)

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Leb (42) and Höftli (43) believe that the condition of osteochondritis dissecans arises in the traumata of everyday life and should be subordinated to the domain of joint fractures. Höftli, however, considers that there may be some abnormality of the bones either in their contour or in their structure and suggests that the fracture may arise through hyperthyroidism, late rickets or an arthritic constitution. Another authority, Kappis (44), in a series of six papers, has suggested that the cause is tangential or rotating force affecting bones which are predisposed to injury by some factor which is unknown.

Such theories appear to have little to commend them for no case has been recorded in association with hyperthyroidism or rickets and, although loose bodies arise in connection with osteoarthritis, it is not the same as the lesion known as osteochondritis dissecans. Of course many cases of osteochondritis dissecans exhibit osteoarthritic changes but they are a sequel and not a predisposing cause of the lesion.

Trauma Initiating a Pathological Process.

The first article on this subject was that by König (3) who wrote on free bodies in the joints in 1888. His contention met with a mixed reception but
the name osteochondritis dissecans with which he labelled the lesion has never been given up although there are few people who believe in a true dissecting osteochondritis at the present time. König believes that the loose body arose by a process of dissection and yet there are many text books which persistently describe the condition as osteochondritis dessicans and some authors have even imagined that the loose bodies show a dessicated appearance although they do not state what they mean by this.

König in his original paper described a series of cases involving the elbow and the knee and was impressed by the long interval between the trauma and the onset of symptoms and also drew attention to the frequency of the absence of a history of trauma. He declared that the teaching on the formation of free joint bodies was not fully settled and asked the questions - can a loose body arise by infraction? and how often is a loose body produced by trauma? He admitted the occurrence of loose bodies due solely to trauma as in the head of the radius but in a review of his case histories found that none of them showed a trauma of sufficient severity to cause the formation of a loose body. He quoted Huerter's work on experiments on the cadaver and disagreed with it in the
light of his own experience. He says - "It succeeds well in one or other place in experiments on the cadaver by twisting out a piece of bone with its corresponding ligament as in the radial or femoral heads or in producing a mark in the joint surface with injury to the overlying structures but I have never succeeded in the least in separating by violence flake-like pieces from the joint surface or related parts." In view of the importance of König's original work I give a summary of his conclusions.

(1) It is comparatively seldom that following an injury pieces of the joint surface are immediately loosened which later on come forth as loose bodies and it is only conceivable in healthy joints following very severe violence.

(2) It is possible to avulse portions of the articular surface by twisting attachments of ligaments or to break off such a whole section of the joint surface as the head of the radius or head of the femur by leverage and severe trauma. However, it is exceedingly unlikely that portions of the articular surface of the end of a long bone such as we have seen and described in the knee and elbow joints should be released by a severe trauma without showing other signs of severe injury to the joint.

(3) It is much more likely that pieces so weighty were originated by a trauma which initiated necrosis itself and a supervening dissecting process or inflammation would lead to their separation.

(4) The most important conclusion I give in the original so that his exact meaning may be
clear:— Es gibt eine spontane osteochondritis dissecans, welche ohne sonstige nennenswerte Schädigung der Gelenke beliebige Stücke der Gelenkoberfläche zur Lösung bringt."

(There is a spontaneous osteochondritis dissecans which, without a definitely known trauma to the joint, causes the separation of considerable portions of the joint surface.) He goes on to say that larger areas may be separated in this way than following a crippling fall. It seems probable that König is right in this contention for Case 1 and Case 22 had very large loosened portions of bone and cartilage and yet there was no history of trauma.

(5) The etiology of the pathological and anatomical processes is for the present still unknown.

This last sentence has stimulated a wealth of investigation and unfortunately it is as true to-day as when enunciated by König nearly fifty years ago.

It is interesting to consider the great discussion which arose over the publication of König's paper because Broca (45) had written almost thirty-five years before on a similar subject and was of the opinion that the loose body was set free by the disturbance of nutrition due to repeated traumata causing necrosis and sequestration of the dead fragment. His work does not appear to have aroused the interest of others, possibly because he had no label to attach to the disease process.

Kragelund (46) in 1887, a year before König's article, had advanced the theory of trauma followed by chronic inflammatory changes and subsequent
sequestration and at a still earlier date a similar theory of trauma followed by necrosis had been advanced by Teale (47). Another paper in support of what I take to be a similar process is the theory of a diaclastic action following trauma advanced by Mouchet and Bruss (48).

Further support for the theory of a dissecting process is found in a paper by Somner (49) in 1923 but he believes that the initial injury is a contusion of the cartilage or of both the cartilage and the subchondral bone and that this inaugurates a dissection of the affected area.

The theories considered in this section may all be regarded as being similar, namely, the occurrence of trauma single or repeated but insufficient to cause immediate separation of the osteocartilaginous fragment and followed by a process of necrosis and later by dissection or sequestration. In regard to those who postulate a chronic infection it may at once be said that there has never been any evidence of this in any of the pathological studies on osteochondritis dissecans.

A grave objection must be raised at once in regard to the process of dissection and that is why should such a process occur in an otherwise normal joint and
with no definite history of trauma and also why is the disease not a common one if the trauma required to inaugurate it is less than that required for other joint lesions which are more common. This at once brings us back to the question of another factor such as hereditary predisposition or a structural anomaly of the joint in association with the dissecting process, a very unlikely possibility.

Again, a sequestrum is separated by fibrous or young granulation tissue not by fibro-cartilage which is usually seen on the surface of the bony cavity.

It is unlikely, therefore, that the truth is to be found in any of these processes.

Primary Vascular Lesions.

A very large amount of research work has been done on the vascular system as a cause of loose body formation and there are so many theories that I have considered it expedient to discuss only those that appear of significance and those that have been based on a careful study of pathological material.

(A) Infarction. Koch(50) in 1879 performed experimental work on the vascular supply and believed that a loose body was caused by the infarction resulting from the blocking of an entire area of capillary
distribution. He was unable to bring forward definite experimental evidence in favour of this theory.

Bergmann(51) in 1927 was inclined to favour the theory of infarction also but had no real evidence in support of this theory.

Konjetzny(52) in 1924 favoured a vascular lesion as the primary cause and found evidence of endarteritis obliterans in the material at his disposal. As far as I can find, this phenomenon has not been described in any other histological studies of osteochondritis dissecans and is certainly not seen in the sections of the complete lesion described below.

(B) Embolism. Buchner and Rieger(27) after showing mathematically that the lesion could not be produced by violence of any description, came to the conclusion that it must be due to embolism and, as there was never any sign of infection, they believed that it must be due to an aseptic embolus, the most likely in their estimation being fat embolism. They had no histological or other evidence to substantiate their theory and it is very unlikely that such a condition should invariably occur in one special site in the knee joint.

Axhausen(53) who has written a large number of papers on the subject of "aseptic necrosis" in bones
and on osteochondritis dissecans, has always believed in the vascular theory of osteochondritis. He has, however, changed his views on the exact method of production of the lesion as they have been modified by the examination of further pathological material. In 1922 he examined microscopically a case of Köhler's disease of the second metatarsal head in which a loose body had become separated. He advanced the theory that it was due to a low grade bacterial embolism of the arterial supply to the head of the metatarsal bone. The infection is so mild that it is easily overcome and so there is no evidence of an acute inflammatory reaction. In 1924 Axhausen altered his bacterial theory to the specific theory of avirulent tuberculous embolism because of the close resemblance to the cone-shaped sequestra seen in tuberculous joints. The site of these sequestra varies considerably, however, and it has not been seen with any great frequency in the site of election in the medial condyle.

All these theories rest of course on the supposition that the area in question is supplied by terminal or end arteries. Great difference of opinion exists in regard to this point and I shall consider it in detail later, after a full discussion of the vascular theories.
The theories of infarction due to vascular occlusion and following embolism depend on the production of a necrotic area of bone which is subsequently cast off as a sequestrum. It is not known whether an area of necrotic cartilage or of bone and cartilage would be separated gradually from the parent bone or not and Pernier(54) designed experiments to try and substantiate or disprove the theory. He operated on a series of seven dogs, inserting into the region of the lateral surface of the medial femoral condyle twelve and five tenths mgm. of radium. The radium was left in for periods varying from eight to twenty-four hours, i.e., dose of 100-300 mgm. hours. Necrosis of cartilage and subchondral bone developed in the medial condyle and also in the upper surface of the tibia in the region of the tibial spine. He examined the joints at intervals from two weeks to nine months after the operation. In none of the seven cases was there the slightest evidence of sequestration of the affected portion of the bone or cartilage. Around the periphery of the necrotic portion (which showed no radiological changes) there was a reaction obviously designed to repair and substitute the necrosed area. Fibrous marrow filled the cancellous spaces and there was some new bone formation about the surfaces of the
trabeculae, while creeping substitution of the necrotic trabeculae had commenced by ingrowing new bone without the presence of lacunar absorption by giant cell osteoclasts.

The area of cartilage was interesting because it was necrotic in the whole of its extent and at the junction of necrotic with living cartilage there was very slight evidence of proliferation and formation of fibrous cartilage which was replacing the necrotic area.

Instead, therefore, of the dead area being cast adrift, reparative changes originated in the living tissue and invaded the dead portion to replace it, though slowly, with new bone.

Experimental evidence is therefore against the theory of aseptic necrosis.

Another factor of importance is the question of the repair of articular cartilage and Fisher(55) has carried out experimental work to try and demonstrate the healing process in the cartilage. Articular cartilage probably obtains its nourishment indirectly from several sources: (a) from the circulus vasculosus to the peripheral part of the cartilage; (b) from the underlying cancellous bone to the deeper strata of the central portion; and (c) from the synovial fluid.
of the joint to the superficial strata of the central portion. This nutritional supply is of great interest because Fisher's work showed not only that repair of articular cartilage can occur but that wounds near the periphery of the cartilage, i.e., in proximity to the vascular synovial fringe, may be partly repaired by actual reformation of cartilage. Wounds of the central portion are repaired by fibrous tissue unless the cut is deep enough to reach the underlying bone, when repair by true cartilage occurs.

This experimental work tends not only to contradict the necrotic cartilage theories but also serves to show that there is no reason why healing of chip fractures should not occur.

(C) Haemophilia. Brailsford (56) who ascribes a common cause to all the varieties of osteochondritis, points out that similar changes occur in the bones in some cases of haemophilia. It is interesting that the knee and elbow joints are also most often affected in haemophilia. In cases in the hip-joint changes comparable to Perthes disease have been described by a large number of authors.

This is of course not the cause of the disease but it is of interest in connection with some of the theories to be described later.
(D) Incomplete or Complete Obstruction to the Vascular Supply (Ischaemia). We have already seen how infarction or necrosis is unlikely to be the etiological factor because of the evidence of Phemister's radium experiments but this group of theories does not depend on necrosis or infarction following vascular obstruction but only on the changes following partial or intermittent lessening of the blood supply which I shall call ischaemia to facilitate the description.

Ludloff(57) in 1908 stated that there was still some doubt on the causation of osteochondritis dissecans and reported three cases of which he said - "I believe they cast a new impetus to the subject and merit a new interesting viewpoint without being able to bring myself to give a universally applicable answer to their meaning. I limit myself absolutely in my statements to the disease of the knee joint and will seek first to provide more considered anatomical details of this joint which are perhaps of importance for the whole question". Ludloff's first case was a bricklayer, aged 27, who had had trouble with the right knee for eight years. There was no history of trauma. An X-ray examination showed a loose body in its bed in the lateral surface of the medial condyle of the femur. Ludloff explored the joint through a Kocher incision.
to obtain a satisfactory view of the femoral condyles and of the menisci. He says - "To my great astonishment and also that of those assisting at the operation, particularly as the X-ray showed a definite lesion, there was not the slightest pathology to be seen in the medial condyle. I looked further in the joint and found nothing. Disappointed because of the fraud of the X-ray, I opened the joint more widely to carry out another investigation of the point in the cartilage which lay 1 cm. medial to the lateral rim of the medial condyle and around the insertion of the posterior cruciate ligament. There an area of cartilage about the size of a pea did not look as blue as the surrounding parts. This rather whitish yellow portion passed gradually into the more bluish cartilage without sharp limits, without any wrinkle, without any fissure and without roughness. I touched this area with the forceps and felt a light ballottement under them. Another examination showed no breach of continuity. After consultation with Professor Kütter I cut around the oval area into healthy tissue, the lateral edge reaching to the insertion of the posterior cruciate ligament. Through this cut in the cartilage I detached the circumscribed area of cartilage with a small hook. A small portion of bone
was attached to the cartilage and it came out of a preformed cavity in the spongiosa of the bone and was attached by some small threads to the base of the bony cavity. After separating these thread-like attachments with the scissors, I obtained a biconvex cartilaginous and bony body. The cartilage was firmly applied to the bony portion. The under-surface of the bone was covered with greyish looking granulations from which two small thread-like stems jutted out. The cavity in the medial condyle was also covered with granulations. With the sharp spoon the granulations were curetted as was the surrounding bone which was very hard and did not bleed. After an area about 1 mm. thick had been removed, the spoon brought out soft bleeding spongiosa. The joint was then closed."

I quote the operation report of Ludloff's first case in detail because it is on this carefully observed lesion that he bases his theory of the etiology. At a time when the X-ray was only beginning to be used it must have been a grave disappointment to him not to have the findings verified at once at operation and it speaks well for his patience and powers of observation that he was able to bring the case to such a successful conclusion.
Ludloff attaches great significance to the absence of a history of trauma and he too must have had difficulties with the question of "compensation" for he says "The denial of any injury on the part of both patients is evidence of great importance, the more so by present day standards when there is a tendency to recall any injury, however slight, for the sake of canvassing money". He then reviewed the past literature and discovered that every lesion described had been in an exactly similar site in the femur. He therefore examined the circulation of this particular area and gives his findings as follows:

"I have inspected this site (the lower end of the thigh in adolescents) in many skeletons and made the following investigations:- Almost at the site of the intercondylar fossa, where our area lies and where the posterior cruciate ligament is inserted, there is a completely smooth area the size of a bean which in contradistinction to the rest of the intercondylar fossa does not show a single nutrient foramen. The most numerous and largest foramina lie medial and dorsal, behind the insertion of the posterior cruciate ligament. Also under the lower edge of this insertion area are a pair of vascular foramina."
In the knee joints of adolescents we cannot show a predilection for infarction in this site but I have also sought information in the blood supply of this area in children. I find an illustration of an injection of the lower end of the femur in a paper by Lexer(58) in which a small vessel enters this part of the medial condyle and ramifies only in this site."

Ludloff follows this description of the lower end of the femur with a balanced and critical consideration of the likelihood of the process being one of infarction due to tuberculosis or other causes and decides against it because of the difficulty in believing that such a condition would always involve the same area and no case has ever shown signs of tubercle elsewhere.

Ludloff continued his researches on adolescent knee joints and found a small vessel running in the synovia covering the posterior cruciate ligament. This vessel supplies the medial condyle of the femur. There is no similar vessel in the anterior cruciate ligament. He followed this vessel proximally and traced it as it wound out between the two cruciate ligaments to become the arteria genu media. This unpaired artery springs direct from the popliteal, passes through an aperture in the posterior capsule of
the joint and runs to the cruciate ligament. This artery is beautifully seen in the stereoscopic photographs of Hildebrand, Scholz and Wieting, where one can see it to be without anastomosis and ending in the knee joint."

"If now this vessel were injured, a portion of synovia and an area of the medial condyle which this end artery supplies (i.e., around the posterior cruciate ligament) must be affected. Now we ask ourselves how can this vessel be injured. There are two factors which can predispose to it - (1) the passage through the posterior ligaments; (2) the place where the vessels pass between the two cruciate ligaments and pass from behind on to the posterior cruciate ligament. All movements by which the posterior capsule and cruciate ligaments will be tightened must naturally lead to an injury of the vessels either by the twisting violence or through elongation leading to twisting, stretching or compression."

"It is a contribution of factors which brings the vessel into danger - massive stretching through strong internal rotation of the upper part of the leg, when not only will the posterior capsule be greatly tightened and the vessel foramen narrowed but both cruciate ligaments will be stretched over one another. The
artery will be compressed in its passage through the posterior capsule and will eventually be injured by torsion. This supposed mechanism will be produced in the following circumstances: if some one groping his way in the dark were to miss the last step and fall over backwards - thus producing momentarily the hyperextended joint with the whole weight of the body, and if at this moment the tibia were internally rotated we have the combination of movements which injure the vessel."

While not entirely in agreement with Ludloff, I have given an abbreviated translation of his very interesting paper and although many authors have referred to his work they have not considered it in detail and their reasons for discarding it are in many cases inadequate.

Nussbaum(59) was the first to discredit the theory and he proved to his own satisfaction that the arteria genu media was not an end artery. Of course there may be an anastomosis but it is an exceedingly poor one and the blood supply by this means is in all probability inadequate.

I decided to investigate the truth or otherwise of Ludloff's theory and the first question of importance seemed to be to demonstrate the presence or
absence of this artery and to find out if it supplied the bone or not. Accordingly, Cunningham's Text Book of Anatomy was consulted and it was found that the arteria genu media passes directly forwards from the front of the popliteal artery, pierces the central part of the posterior surface of the capsule of the knee joint and enters the intercondyle fossa. It supplies branches to the cruciate ligaments and to the synovial membrane. It does not mention whether the branches enter the bone or not.

To try and establish this fact I explored the knee joints of a series of post mortem cases and on each occasion there was a vessel easily visible to the naked eye, running on the posterior cruciate ligament to the medial condyle of the femur. The posterior cruciate ligament was removed as close to its femoral attachment as possible and transverse sections of the femoral ends of the ligaments were made. In all cases vessels of fairly large calibre were found both on the surface and in the ligament and they were apparently too large to supply such an avascular structure as a ligament. Male and female ligaments were examined at each ten year period and, while there were larger vessels in the ligaments from the male cases than in the female ones, there was no
variation at the different age periods. Figs. 60 and 61 show the average size of the vessels in the male and female subject respectively.

Having established the probability that the vessel was responsible for the blood supply to part of the medial condyle, injection experiments were carried out. A male knee joint was obtained in the post-mortem room and the vessels were injected under pressure with a radiologically opaque solution. Unfortunately the specimen, which was obtained from a fatal accident case, showed trauma to some of the smaller vessels and the escape of the injecting fluid rendered the injection unsatisfactory. The specimen, however, shows the arteria genu media fairly well (Fig. 62). The vessel was followed to the aperture in the posterior surface of the capsule but beyond that the injection was insufficient to make further dissection possible.

A further specimen was obtained and on this occasion the injection was very satisfactory. The arteria genu media is again well shown (Figs. 63 and 64). The vessel was found arising not from the popliteal as the figure suggests, but from the inferior medial geniculate artery. It was followed easily through the posterior capsule of the joint. It then
into two branches, one to the anterior cruciate ligament and a larger one running on the posterior ligament. This vessel was followed running the ligament to the intercondylar fossa, where it divided into numerous branches which passed through a on the lateral surface of the medial condyle to the bone. It is worthy of mention at this point that Ludloff is quite correct in his description of the intercondyloid region of the femur and well shown by Fig. 65, which is a photograph of the lower end of a left femur. It shows the smooth outer the size of a large bean and generalised with vascular foramina in the remaining parts of the intercondyloid notch. The two large foramina for the insertion of the posterior cruciate ligament are clearly visible even in the photograph.

It is unfortunate that up to the present I have been unable to obtain a specimen with the nutrient foramina of the femur and the knee joint intact, owing to difficulty in procuring fresh material which is suitable for injection. As the disease most frequently occurs in young people, before the union of the lower femoral epiphysis with the diaphysis, the surgery would require to be carried out on a femur of similar age, and adult and foetal femora are of
divided into two branches, one to the anterior cruciate ligament and a larger one running on the posterior cruciate ligament. This vessel was followed running along the ligament to the intercondylar fossa, where it divided into numerous branches which passed through foramina on the lateral surface of the medial condyle and so to the bone. It is worthy of mention at this juncture that Ludloff is quite correct in his description of the intercondyloid region of the femur and this is well shown by Fig. 65, which is a photograph of the lower end of a left femur. It shows the smooth area about the size of a large bean and generalised pitting with vascular foramina in the remaining parts of the intercondyloid notch. The two large foramina behind the insertion of the posterior cruciate ligament are clearly visible even in the photograph.

It is unfortunate that up to the present I have been unable to obtain a specimen with the nutrient artery of the femur and the knee joint intact; owing to the difficulty in procuring fresh material which would be suitable for injection. As the disease affects young people most frequently, before the union of the lower femoral epiphysis with the diaphysis, the injection would require to be carried out on a femur of a similar age, and adult and foetal femora are of
no value for the investigation of the peculiarities of vascular supply.

I have already mentioned how the first injection was spoiled by the trauma to the smaller superficial vessels but on dissecting the specimen an interesting appearance was seen in the medial femoral condyle. There was extensive subchondral haemorrhage in the medial rim of the medial femoral condyle and this haemorrhagic area extended also to the cancellous bone of the medial surface of the condyle. The extensive bruising is remarkable when one considers that there was no mark at all on the skin surface. This would appear to support the subchondral fracture theory advanced by Littlejohn and others but I am unable to say whether the injury caused symptoms or not because the patient was rendered unconscious at the time of the accident and never regained consciousness. The appearance of the lower extremity of the femur is shown in Fig. 66.

While the above injection experiments and the sections of the cruciate ligaments do not demonstrate conclusively that Ludloff was correct in his theories, they at least are very good evidence that the blood supply to the lateral part of the medial condyle is carried in whole or in part by the arteria genu media.
The lower end of the femur of a still-born babe in section. The arteries are injected with carmine gelatine. Ossification has commenced in the epiphysis.

**Fig. 67.**

Dissection of the knee-joint of a new-born babe in which the vessels are injected to show the 'vasculosus articularis epiphysium' of William Hunter.

**Fig. 68.**
Harris in his monograph "Bone Growth in Health and Disease" (78) has studied the vascular supply to the lower end of the femur in the foetus and illustrates an injected specimen (Fig. 67) which shows commencing ossification in the epiphysis and its vascular supply. The artery shown in the intercondylar notch is in all probability the vessel shown and followed in my injection experiments. Harris has shown by the injection of carmine gelatine at pressures well within physiological limits that there is no connection between the diaphyseal and epiphyseal vessels. He believes that the vessels in the diaphysis are end arteries and that infarction can occur in the diaphysis, the infarct being wedge-shaped with its base at the epiphyseal cartilage.

Harris is of the opinion that "the epiphyseal cartilage, coinciding as it does with the reflection of the periosteum to form the joint capsule, demarcates one nutritional area, the diaphysis, from the other, which consists of the epiphysis and the joint capsule. As we have seen, the diaphysis is supplied by branches of the nutrient vessel, the terminal vessels being end arteries. The capsule of the joint and the epiphysis are supplied by multiple articular branches given off by any of the vessels
in the vicinity. This arterial system constitutes the 'circulus vasculosus' first described by William Hunter in 1742. The small arteries in the epiphysis are not end arteries and it is this free anastomosis which leads to the important differences in the type of lesion found in the epiphysis as distinct from the diaphysis. The lesion in the shaft or diaphysis tends to be of the character of localized infarction. In the epiphysis it tends to be widespread distraction."
The circulus vasculosus is well seen in Fig. 68, a reproduction of an injection of the vessels of the foetal knee joint illustrated by Harris. It is interesting to see the large number of vessels forming a network on the surface of the posterior cruciate ligament, an appearance which I was unable to obtain even after injection at high pressure.

The view expressed by Harris that there are end arteries in the diaphysis but not in the epiphysis is no new one but I am unable to find any real evidence for this belief. The theory is largely based on clinical and pathological findings and is not substantiated by any absolutely conclusive facts.

We are therefore left with two theories which are directly antagonistic - (1) that the arteria genu media supplies the medial femoral condyle and that it
is an end artery; (2) that branches of any artery in the vicinity of the joint supplies the lower femoral epiphysis and that they are not end arteries. The question at issue is therefore are the vessels to the epiphysis of long bones end arteries? The answer to this question can never be found by working along anatomical lines. The only way to obtain absolute proof is by experiment and it is insufficient to experiment on the bones of growing animals alone because the blood supply to the epiphysis and diaphysis may be changes with the union of the epiphysis. Reference will be made later to work of such a nature which has only recently been carried out.

This subject was pursued further by injecting the femoral artery of a dog's hind limb under pressure. The injection was not entirely successful because the injecting solution was made up more fluid than that previously used in order to try and show the very finest vessels, but the decreased concentration of the mercuric iodide rendered the solution too dilute to case a good shadow with X-rays.

The X-ray films of the injected specimen are of little value because of this insufficiency of opacity of the injecting fluid and are not reproduced. Dissection was carried out and in the dog there are two
to give a positive result. The experiment would require to be carried out by the injection of a particulate emulsion in order to make certain of blockage of the vessels. In any case the changes resulting from the ligation would be so slight that there would be no change in the X-ray appearance. It was decided, however, to preserve the animal for some months, to prove definitely that ligation of the vessel does not produce osteochondritis dissecans.

This general theory of primary vascular lesions has been applied not only to osteochondritis dissecans but to the other varieties of osteochondritis and work of great importance has been carried out but unfortunately some of the evidence is conflicting and the same experimental methods have not always yielded the same results.

Experimental work of great importance from this viewpoint has recently been carried out by Zemansky and Lipmann(61). They investigated the blood supply of the head of the femur to try and ascertain whether some vascular peculiarity might be significant in the etiology of Perthe's disease. Schwartz(62) had declared in 1914 that Perthe's disease was due to the occlusion of vessels in the round ligament but gave no experimental evidence to support his contention.
vessels running along the surface of the posterior cruciate ligament, apparently to end in the lateral surface of the medial condyle, and a good view of the injected vessels was obtained from the anterior aspect of the joint.

A study of the vessel was made by a posterior approach but the popliteal artery lies so deeply and the arteria genu media is so small in the dog that it was considered impossible to inject emboli of carbon particles into it. Embolic closure of the vessel is the only sure method because it must anastomose with many vessels before it reaches the bone. However, one experiment was carried out. The knee joint of a growing dog had been opened by a vertical incision medial to the patella for the exposure of the medial condyle, as in the experiments subsequently described. On opening the joint two fairly large vessels were observed on the surface of the posterior cruciate ligament and it was decided to ligate and divide these vessels. This procedure was carried out and the joint was closed. The dog was walking on the operated leg on the day following operation. At the time this experiment was carried out I had not read Kistler's paper on infarction in the rabbit's femur by experimental methods and I now realise that it was unlikely
Following Schwartz's paper there has been considerable discussion on the importance of the ligamentum teres in the vascular supply of the femoral head. Kolodny (63) was able to show that the femoral head derives its blood supply from three sources - (1) vessels from the femoral diaphysis; (2) epiphyseal vessels; (3) blood vessels in the ligamentum teres. The diaphyseal vessels are of little importance, for many observers deny their existence and they cannot therefore be responsible for the vascularity of the head. The epiphyseal vessels are of importance in the supply of the periphery of the head and enter it along the rim of the articular surface. The central region and crest of the head of the femur is supplied by the vessels of the round ligament.

This contention has been the subject of much criticism and different observers attach different degrees of importance to the vessels of the ligamentum teres. Moser (64), in an elaborate and careful study, was able to show that the vessels of the round ligament were of great importance in childhood but thereafter they begin to atrophy and are probably of little moment in the adult, but this will be shown later to be an erroneous conclusion.

Chandler and Kreuscher (66) made a careful
examination of a series of round ligaments from the 
hips of persons varying in age from 25 years to 75 
years, but of their total number of 114 ligaments 
they found in fifty cases that there was an artery of 
sufficient size to be of importance in the vascular 
supply of the head of the femur. In many cases they 
were too small to play a major role in the vascular 
supply but in every case there were vessels present. 

Zemansky and Lippmann found that in the rabbit 
there was a large vessel in the round ligament between 
the ages of 2 and 7 weeks and it supplies the crest 
and central zone of the femoral head. After the age 
of seven weeks, however, the femoral head is supplied 
only by the epiphyseal arteries. Accordingly they 
operated on rabbits at the age of two weeks and 
sectioned the ligamentum teres on the one side and, 
for purposes of control, exposed it on the other side 
but did not divide it. The trauma was therefore of 
a similar nature in the two hips. The control hip 
joints showed no changes but on the side with division 
of the round ligament the femoral heads showed changes 
leading to deformity of the femoral head. They there-
fore conclude that Perthe's disease is a maladjustment 
of the delicate physiological balance between the two 
circulations to the femoral head.
Stewart (67) operated on the hip joints of young animals by cutting the ligamentum teres and the vessels of the capsule of the neck and in the majority of cases obtained fairly extensive necrosis of the femoral head. In some cases there was no sign of necrosis and therefore one is forced to the conclusion that in some cases there must be anastomoses between the vessels of the interior of the neck of the femur and those of the capital epiphysis. The anastomotic vessels could only reach the epiphysis in two ways - (1) through the plate of cartilage at the epiphysis (Lezer's theory), or (2) they may pass round the periphery of the cartilage. The presence of such vessels would of course explain the failure to reproduce necrotic changes by ligation of the ligamentum teres (68) as reported by Bonn and Stewart.

More recently Phemister (65) published an excellent paper on aseptic necrosis of the head of the femur. Because of inadequacy of the collateral circulation in the head of the femur, and as its structure is such that injury may damage its vascular supply, a condition of aseptic necrosis may occur in the bone and cartilage and result in delayed union, deformity of the femoral head or poor function. Phemister points out that the head of the femur may receive blood from
three sources - (1) the posterior cervical; (2) the anterior cervical; (3) the ligamentum teres, but suggests that the variable results of injection experiments are probably due to individual differences. The exact nature of the vessels entering the bone does not concern us just now; the important considerations are - (1) Are the vessels to the epiphyses of long bones end arteries? (2) Are the vessels to the metaphysis end arteries? (3) Is there any anastomoses between the epiphyseal and metaphyseal arteries before they give off branches? I believe we are able to answer this question, at any rate for the head and neck of the femur, by a careful study of Phemister's paper.

Phemister studied a series of forty-nine cases of intracapsular fracture of the neck of the femur. The blood supply to the head of the femur in the presence of such an injury can only be by way of the ligamentum teres and through untorn portions of the capsule. The blood supply to the head may be adequate or it may either be partially or completely obliterated, resulting in an aseptic necrosis of the head of the femur. Out of the forty-nine cases, seventeen showed survival of the femoral head with union of the fracture in eight cases and non-union in nine: 32 cases showed partial
or complete necrosis of the head of the bone and union only occurred in four of these cases.

If necrosis of the femoral head should occur and be incomplete, the area remaining alive is usually near the fovea, showing the importance of the vessels of the ligamentum teres or in the inferior surface of the head where the capsule is usually intact. Necrosis can be diagnosed by radiology, because the necrotic area retains normal bone density whereas the living bones show the atrophy of disuse.

The necrotic portion of the head of the femur becomes invaded by connective tissue which may grow out from the margin of a viable portion of the head or may commence around the insertion of the round ligament. Behind this zone of embryonic connective tissue comes a zone of transformation of the dead bone, some of which is absorbed while some has new bone laid down on it until it is replaced by a process of "creeping substitution".

Phemister in his pathological studies of femoral heads following intracapsular fracture of the neck has investigated the behaviour of the articular cartilage, investigations which are significant in regard to osteo-chondritis dissecans. He says, "Articular cartilage very slowly dies when its underlying bone
is necrotic as the great bulk of the nutrition comes from the bone. However, some nutrition comes from the synovial fluid as shown by Nussbaum and cartilage cells are sometimes kept alive by it for long periods. If the underlying dead bone is rapidly absorbed and replaced by new bone the cartilage may be revived and persist." This explanation of the fate of the articular cartilage is a satisfactory one from the viewpoint of those who consider osteochondritis dissecans to be an aseptic necrosis. The antagonists of this contention believe that necrosis will inevitably lead to sequestration but if the cartilage should remain viable for a sufficient length of time to hold the body in its bed, revascularisation and healing could take place.

Two of the cases of aseptic necrosis of the femoral head recorded by Phemister showed osteo-cartilaginous loose bodies and another case showed a very significant X-ray photograph. The case was one of necrosis of the upper part of the femoral head around the fovea, and the X-ray is almost identical with the recorded cases of osteo-chondritis dissecans in the head of the femur. This work of course explains the second case recorded by Stören(11) who was amazed to find exactly comparable X-ray appearances in the hip
joint of two cases, one a familial polyarticular case of osteochondritis dissecans and the other following an intracapsular fracture of the neck of the femur.

Phemister completes his sound paper with a consideration of the circulatory disturbances of a more obscure nature, namely, Perthe's disease, death with collapse and separation, partial or complete, of a portion of the femoral head in adults and osteochondritis dissecans. He arrives at no definite conclusion but suggests that osteochondritis dissecans is closely arried to aseptic necrosis as seen in cases of intracapsular fracture of the femoral neck.

Supported as it is by microscopic and radiological studies of the necrotic heads of femora, this work proves conclusively that (a) the round ligament vessels can be of value in revascularising the femoral head; (b) the vessels in the ligament are not sufficiently large for the supply of the entire head or, failing that, they must be end arteries; (c) aseptic necrosis of large and small areas of bone can occur with and without sequestration; (d) the necrotic areas can become revascularised from the neighbouring viable bone and that healing can occur although it is a very lengthy process. This work also explains why Phemister's own radium experiments (vide supra) were
not followed by sequestration. There must obviously be a second factor in the causation of the sequestration of the necrosed area and this question is considered in detail below.

Ferguson and Howorth (69) in a recent paper believe that Perthe's disease, slipping of the capital epiphysis, chronic degenerative arthritis and osteochondritis are all due to a circulatory disturbance in or adjacent to the femoral head. They then go on to say that for convenience this circulatory disturbance will be referred to as "ischaemia". They give a classification of these ischaemic conditions without any clinical, radiological or ethological grounds for so doing and state that these important considerations will be referred to in subsequent papers. Surely this is putting the cart before the horse, for a classification of such conditions can only be of value when based on histological evidence. However, I give this classification and their views on the etiology of the various conditions.

The above authors believe that the "ischaemia" may be produced by five factors, which they designate Type 1 ischaemia, etc. They are:

1. Inflammation in the hip joint.
2. Inflammation or tumour adjacent to the joint and usually in the region of the femoral neck.
(3) Fracture of the neck of the femur.
(4) Trauma to the femoral head.
(5) Tension of the soft tissues around the neck of the femur.

The age is a factor of great importance and they believe that in the early years of life the femoral head is most vulnerable whereas, after the age of ten, the epiphyseal cartilage is more liable to injury from impairment of the vascular supply. They state that Type 1 and 2 ischaemia may produce coxa plana, slipped epiphysis or degenerative arthritis, depending on the age of onset of the ischaemia. Type 3 ischaemia, i.e. fracture of the femoral neck, may cause coxa plana or a condition which they call coxa magna. Type 4, or trauma to the femoral head, may cause coxa plana or osteochondritis dissecans, etc.

This paper is not only entirely unsupported by pathological observations but we are left completely at a loss to know exactly what the authors mean by the word "ischaemia". In regard to Type 4 (fracture of the femoral neck) the pathological studies advanced by Phemister are much more valuable evidence than a vague theory entirely unsupported by facts.

Experimental work of a very convincing nature, on aseptic infarction of the femur, has recently been performed by Kistler(70) with a view to establish
definitely the vascular arrangements of the bones and, if possible, to correlate the arterial pattern with the various non-specific diseases such as osteochondritis, which he believes to be of vascular origin. He quotes Hirsch and Ryerson (71) who, writing on marked necrosis of the distal epiphysis of the femur, emphasised the long duration and progressive destruction of the bone without any attempt at repair and believed it to be due to vascular occlusion. Metastatic foci of osteomyelitis are often situated in the metaphyseal and subchondral areas and this was believed to be due to the presence of end arteries. Kistler therefore set out to establish if possible the vascular arrangements of bone and his ingenious methods have certainly been of great value to a proper understanding of the blood supply of bone.

The object of Kistler's work was to try and establish beyond any possible doubt (1) the general circulatory pattern of bone; (2) the relative importance for normal nutrition of the various sources of blood; and (3) the presence or absence of end arteries and anastomoses. Because of the great vascularity of bone, experimental division of a vessel does not resemble occlusion of the branches of that vessel by emboli and therefore the experimental work was carried
out by the injection of an emulsion of charcoal particles.

The long bones are supplied with arterial blood from four main sources:—

(1) The principal and accessory nutrient vessels to the diaphysis.

(2) Small metaphyseal vessels piercing the cortex at the ends of the shaft.

(3) Periosteal twigs to the cortex.

(4) Epiphyseal vessels which reach the bone at the attachment of a joint capsule or along such a ligament as the lig. teres to the femoral head.

Following the injection of carbon particles into the nutrient artery of the rabbit's femur, infarction was produced which extended to the epiphyseal cartilages but in the adult rabbit the infarction did not extend further than 6 mm. from the obliterated growth line. The nutrient artery is therefore of more importance in growing bone and because of the infarction produced it can be said that blocking of a vessel will lead to aseptic necrosis. Experiments in regard to the extremities of the bone showed that infarction could also be produced in the epiphyses. These injection experiments show that the vascular system of the rabbit's femur is closed because the charcoal "emboli" were not disseminated in the tissues.

It seems highly probable that if large infarcts
can be produced in this way then smaller ones could arise similarly in the human subject. The problem, however, once more before us is to explain the typical sites of osteochondritis dissecans on the basis of the theory. In the head of the femur and in the medial condyle of the femur there are special arteries which may be of importance but the capitulum of the humerus has no special vessels and I do not like to accept a theory which is not universally applicable.

The theory of aseptic necrosis can be accepted if it is formulated in the following manner. From our experimental evidence we know that aseptic necrosis or infarction can occur even in a tissue so vascular as bone. This necrosis may affect any part of the bone but is commonest at the metaphysis and in the subchondral bone. Should the infarct occur in the shaft or deeply in the bone, there is no reason why vascularization and healing should not result. If, however, the infarct is subchondral in position, the only difference is that it is constantly subjected to trauma with increased production of oedematous granulation tissue leading, finally, to separation of the superficial portion of bone and cartilage as in osteochondritis dissecans. This is Kistler's theory and at first sight it seems an attractive one but
there are several objections to it. In the first place all long bones will presumably be supplied with blood in a similar way; why, therefore, should the medial condyle of the femur be affected in over 70% of cases? If the condition is due to embolism, why are multiple lesions not infinitely more commonly found and, finally, why have emboli never been found in the cases of osteochondritis such as Perthe's disease examined histologically? I must admit that this theory is the nearest to substantiating the clinical and pathological features shown in osteochondritis dissecans.

Vascular Causes due to some other factor and therefore summarised as Secondary Vascular Lesions.

This section comprises the work of three separate observers or groups of observers and all believe that the disease is the result of hyperaemia and not of ischaemia as in the previous group.

Bentzon (72) believes that the condition is the result of trauma affecting the periarticular vasomotor nerves with a consequent hyperaemia. He tried to reproduce this experimentally by the injection of alcohol to block the nerves and claims by this method to have produced changes in the femoral head analogous
to Perthe's disease.

Sommer (49) published a series of twelve cases with a definite history of trauma in seven and believed that the disease arose through repeated traumata causing a localized paralysis of the vessels in the traumatized area.

Leriche and Policard (104) in their book, "The Physiology of Bone" have advanced an entirely new theory on the ossification of bone. They show that bone formation always occurs in young mesenchymal tissue which they call an ossifiable medium. The only factors required to determine bone as the ultimate destiny of this mesenchyme is an excess of calcium and an adequate blood supply. This theory has the great advantage that it offers a plausible theory for heterotopic bone formation because bone may occur in any tissue which is capable of de-differentiation into primitive mesenchyme. According to Leriche and Policard the osteoblast is merely a connective tissue cell adapted by its environment and is not responsible for bone formation which can occur in the absence of osteoblasts.

Greig (105) in this country has championed the theory of Leriche and Policard and has demonstrated by means of specimens from large numbers of diseases that
the theory is capable of an almost universal application. Watson Jones and Roberts (107) have also advocated the adoption of the theory and, along with Dick and Illingworth (108) they believe that its greatest importance lies in the great importance given to the changes in bone resulting from vascular disturbances. Leriche and Policard have supplied excellent evidence for their conclusions in regard to circulatory changes and their views are excellently summarised by Jones and Roberts:—

(1) There is normally a balance between the calcium content and the vascularity of mesenchymatous tissues—(a) In the case of bone—

<table>
<thead>
<tr>
<th>Normal circulation</th>
<th>normal calcification.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased blood supply</td>
<td>decalcification.</td>
</tr>
<tr>
<td>Decreased blood supply</td>
<td>increased calcification.</td>
</tr>
<tr>
<td>Blood supply cut off</td>
<td>unchanged calcification.</td>
</tr>
</tbody>
</table>

(b) In any mesenchymatous tissue of low metabolism: Decreased blood supply — Pathological (fibrosis of trauma or calcification or infection).

(2) Fibroblasts + excess of calcium + adequate blood supply = bone.

From this theory, therefore, the radiologist must interpret decalcification as hyperaemia, increased calcification as ischaemia and, if one area
retains normal calcification, he will have to understand from that that the fragment is avascular.

Recently interest has been aroused in a condition of post-traumatic osteodystrophy at joints, which was originally described by Sudeck in 1900 and received considerable attention through the work of Leriche. Middleton and Bruce (106) in an excellent review of the condition, state that it is within the bounds of human certainty that this form of osteoporosis results from or is accompanied by hyperaemia. Leriche believes that the hyperaemia is produced by trauma through a local autonomic axon reflex.

Middleton and Bruce report cases which have been operated on by periarterial sympathectomy and ganglionectomy and state that the immediate results of the two operative procedures are alike. They therefore advance three important conclusions:

(1) "The fact that recovery from pain and recovery of movement can take place while the osteoporosis remains unaltered, means that the disability does not in any way arise from the hyperaemia and the rarefaction of the bones. The osteoporosis is an accompanying manifestation and helpful in diagnosis, but nothing more. It need not continue to fill the clinical picture to the virtual exclusion of the other
features of the condition."

(2) "The fact that recovery is claimed to occur as completely after the admittedly very temporary hyperaemia of a parietal sympathectomy as after a ganglionectomy, can have only one explanation. Sympathectomy does not cause recovery from post-traumatic osteodystrophy by virtue of division of vasoconstrictor fibres and resulting hyperaemia."

(3) "The relief of pain after operation is almost immediate. The patient, whose wrist was very painful before entering the theatre, awakens from an anaesthetic practically free from his pain. There can, we think, be only one explanation of such a phenomenon. The operative procedure has succeeded in dividing the afferent tracts by which the patient's painful sensations were carried."

From these conclusions Middleton and Bruce believe that the painful sensations carried in the afferent sympathetic fibres cause a reflex efferent impulse causing vasodilatation and therefore hyperaemia and osteoporosis.

Having surveyed the evidence that hyperaemia and other vascular lesions have a profound influence on the calcification of bone, let us return to study the conclusions of Leriche and Policard in regard to
osteochoondritis. They refer to post-traumatic osteo-
dystrophy in the following way: "The affected bones
are rarefied. We know that there is no rarefaction
without hyperaemia and that hyperaemia can be accom-
panied by it. Now, on the other hand, all peripheral
nerve traumatism is accompanied by a more or less
lasting disturbance in equilibrium which begins with
vasoconstriction and quickly turns into a vasodilata-
tion, which the oscillometer clearly shows. This is
what we call reflexes of the traumatised axone.
Albert has shown that experimentation brings about
this state in conditions which permit of no other
interpretation. In this connection one can imagine
the following explanation, peripheral hyperaemia by
reflex of the axone and consecutive bone resorption."
(A view exactly parallel with that held by Middleton
and Bruce.)

Leriche and Policard then go on to what is for us
the most significant part of their work. "Here it
may be properly asked whether a parallel mechanism is
not the origin of certain singular syndromes which have
been elevated to the dignity of specific bone diseases,
such as the tarsal scaphoiditis of Köhler, the traumatic
malacia of the semilunar of Kienböck, the osteochond-
ritis of the hip, Calvé-Waldenström-Legg-Perthes'
disease, and the vertebral deformities of Kimmel-Verneuil disease. It is very probable that still other isolated localisations of this bone rarefaction of traumatic vasomotor origin will be found, and that other specific diseases will yet be diseases of X. or Y. " (In this connection it is interesting that osteochondritis of the sesamoid bone of the first metatarsal was described in 1933 by Kimmelstiel, Kronser and Richter (109).) "One can say that from now on all will have the same character, an insignificant trauma or one distant in origin, an evolution which is slow, progressive, apyretic, accompanied by slight pains and resulting in final spontaneous curability, with at times definite bone deformity, and absence of all signs of inflammation. Up to the present pathology has not taken account of the reflexes of the traumatised axone. Now that the intimate relation which unites vasodilatation and bone resorption is known, it is permissible to believe that the syndromes which they bring about should be frequent."

To my mind this is a most attractive theory for it explains the absence of symptoms, the slow apyretic course of the lesion and also allows of its occurrences in any site. While it seems to be attractive in relation to osteochondritis as a whole, it seems
difficult to credit that there should be such a localised reflex change which would be responsible for the small lesions of osteochondritis dissecans.

We do not, however, need to accept this theory just as it stands for there does not require to be any reflex disturbance of the autonomic system to cause hyperaemia. Any inflammatory focus in the vicinity will cause decalcification as in the cases of hyperaemic dislocation of the atlas reported by Watson Jones and others.

Watson Jones, writing on calcification and ossification, makes the following statement in regard to Köhler's disease of the second metatarsal head. Köhler's disease of the metatarsal is exactly analogous to Kienböck's disease of the semilunar. There is evidence of impairment of the blood supply not only in the increased density of the bone, but in the fragmentation and distortion of the metatarsal head which results in arthritic changes in the metatarso-phalangeal joint. Excision of the head of the bone relieves the symptoms, and histological examination of the bone shows definite evidence of fibrosis and necrosis, confirming the fact that impairment of blood supply is the underlying cause. The factors responsible for the interference with blood supply are not yet clearly
established, although the frequent association of the condition with congenital anomalies of the first metatarsal which increase the weight bearing strain on the affected one, would point to repeated trauma." I shall consider this theory in detail after the pathology of the disease has been fully examined.

Neilsen\(^{(11)}\) writing on osteochondritis capituli humeri, presents an exhaustive clinical study of 168 elbow joints affected by osteochondritis. The lesion was bilateral in thirty-five cases. The incidence was predominantly in male patients and occurred much more frequently in manual workers. He points out that the disease affects almost entirely two homologous joints, the knee and the elbow, and in these joints two special areas are involved, the capitulum of the humerus and the lateral surface of the medial condyle. He believes that its manifestations are peculiar to it and show no connection with the pathological changes that occur in joints as a result of frank trauma. Neilsen found a strong hereditary tendency in his large series of cases and believes the condition to be essentially a constitutional defect. Trauma he states is not a sufficient cause but trauma can cause in a hereditarily predisposed field an aseptic necrosis comparable to that seen in Köhler's, Kienböck's and
Perthe's diseases. It differs from these only in that the necrosis is limited sharply to a narrow portion of the cartilage and it shares with them the fact that it is difficult to explain how the onset of a quiet, aseptic necrosis occurs. He finishes his paper with the conclusion that "None of the present theories fit the facts of the case and probably an entirely new pathological process must be sought".

As my arguments for and against the theory of hyperaemia as the causal factor are based on histological evidence I propose to defer its discussion to a later section following a description of the pathology.

Infection.

As is only to be expected, there have been attempts on many occasions to place osteochondritis in general on an infective basis and at the outset I may say that, although organisms have been cultivated from the lesions of the various types of osteochondritis, the negative findings far outnumber the positive ones.

Phemister, Brunschwig and Day (73) were successful in recovering streptococci and culturing them from the diseased marrow in two cases of Kienbock's disease, one case of Köhler's disease and one of Perthe's
disease. The intravenous injection of these and other pyogenic organisms did not reproduce the disease. Kidner (74) isolated staphylococci from scrapings of tissue from the femoral head in a case of Perthe's disease.

Knaggs (75) is of the opinion that the initial lesion is a periostitis due to avirulent staphylococci and he is supported in this contention by Platt (76), who considers osteochondritis as a low grade infective process.

In a consideration of Köhler's tarsal scaphoiditis Greenwood (77) placed it on an infective basis because on one occasion he was able to aspirate pneumococci and to culture them. This is of great interest to me because on one occasion an apparently typical case of tarsal scaphoiditis was admitted to the ward in which I was a house surgeon. There was an abscess over the scaphoid and incision and drainage led to amelioration of both the symptoms and the X-ray changes and a staphylococcus was isolated on culture from the pus.

In spite of these definite bacteriological findings, the entire absence of an inflammatory process, as shown in the pathological section, is sufficient I believe to negative infection as a possible cause of
osteochondritis. It is also well known that such cases never show any sign of systemic disturbance, as they would almost certainly do in the presence of a bone infection.

Localised Osteo-arthritis.

In view of the almost inevitable sequelae of osteoarthritis following an untreated case of osteochondritis dissecans, it is not surprising that some of the older writers particularly were inclined to favour osteoarthritis as the primary cause of the disease.

Laennec (79) Humphrey (80) and Moulonquet (81) have all written papers advancing osteoarthritis as the cause of osteochondritis dissecans. It seems unlikely that osteo-arthritis should remain so localised to one particular area of the joint, particularly as this area very often escapes in well-marked cases of osteoarthritis. The loose body also does not show any sign of osteoarthritis but is obviously in its early stages, at least, a portion of a normal articular surface.

Barth (25), as previously mentioned, believes that the localised osteo-arthritis follows on trauma, which may be produced either by direct or indirect violence.
to the joint.

As the typical X-ray appearances of osteochondritis dissecans have been observed in many joints which do not otherwise show any abnormality, it seems very unlikely that osteoarthritis is a factor in the etiology. There is of course no doubt that if osteochondritis is not treated early it is followed by osteo-arthritis.

Skeletal Dystrophies.

(A) Chondro-Osteo-Dystrophy. This condition may be confused with osteochondritis because the X-ray appearances of some stages of osteochondritis are very similar to those seen in this disease. The X-ray shows multiple irregular ossific nuclei in the epiphyses and in the extremities of the diaphysis. The condition must be associated with softening of the bones because pressure deformities arise. The active stage of the dystrophy ceases when fusion of the epiphysis and diaphysis occurs but severe cases are followed by considerable dwarfing of the skeleton.

This dystrophy is unlikely to be confused with osteochondritis dissecans but may be confused with osteochondritis in other sites. The changes seen in the X-ray are, however, usually bilateral and are of equal age on the two sides. The spine is usually
involved and very frequently the affection is generalised.

Silfverskiöld(82) and Martin and Roseler(83) have published cases which, if the joints were reviewed singly, might quite well be diagnosed as osteochondritis. We must of course remember that translucent areas in the bone may be due to different pathological factors, for instance they may represent unossified cartilage or they may represent fibrous tissue replacement of the bone. It is therefore possible for different pathological entities in bone to show similar X-ray appearances.

Metabolic Disorders.

Calvé(84) in 1910 suggested that as many cases of osteochondritis showed signs of healed rickets on X-ray examination, rickets might be the factor which disturbed the nutrition of the bone and so brought about osteochondritis. If this were so it seems strange that osteochondritis should be comparatively uncommon while rickets, in spite of preventive medical treatment, is still quite a common disease.

Other metabolic disorders mentioned by Brailsford(85) are scurvy and renal rickets, but there appears to be no foundation on which to base the
theory that either of these diseases are responsible for the production of osteochondritis.

**Endocrine Imbalance.**

It is well known that the endocrine glands have a powerful influence over the development of bone and it is therefore not surprising that impairment of their function has been suggested as a cause of osteochondritis. Unfortunately, apart from the parathyroids, there is little exact knowledge of the relationship of the endocrine glands to ossification and bone growth.

Berard and Novel (86) published radiographs of the pelvis of a cretin showing deformity and fragmentation of the heads of the femora very like the changes seen in Perthe's disease. It is, however, a commonplace that in cases of hypothyroidism in the child (i.e. cretinism) the development of the epiphyses is abnormal. Instead of there being one bony nucleus in the epiphysis, multiple ossific centres appear and ossification is delayed. This gives the appearance, radiologically, of fragmentation and as parts of the epiphysis are stronger than others, it responds irregularly to the stress of weight bearing. The epiphysis therefore becomes irregularly flettened and
this, along with the appearance of fragmentation, leads to an erroneous diagnosis of osteochondritis. Wakeley(87) reported a case of bilateral Perthe’s disease in a child with bilateral undescended testes. It is of course not definitely known what part the testes play in the regulation of ossification but it seems unlikely that there is anything further than coincidence in this case.

Miscellaneous Theories of the Etiology.

König’s(3) theory of a spontaneous osteochondritis dissecans has already been referred to and several other theories of a similar nature have been advanced from time to time. König’s original paper was published in 1888 but it is interesting to find that several other papers of a similar nature had been published before that date.

Paget(88 & 89), eighteen years before König’s article, had written on the condition and was of the opinion that it was due to a quiet necrosis, while Broca(80) was in all probability of a similar opinion although he called the process a dry sequestration. Klein(91) as early as 1864 had written on a process in the knee joint which he called a spontaneous demarcation of a portion of the lower end of the
femur. Poulet and Vaillard\(^{(92)}\) in 1885 believed that the lesion was produced by a process of spontaneous necrosis followed by secondary loosening of the devitalised portion of bone.

All these theories are based on the fact that the authors realised that they were dealing with a disease entity which they could not explain by trauma, infection - acute or chronic - or any other pathological process of which they were aware. They were well acquainted with the sequestration seen in osteomyelitis and it was only natural that they should consider osteochondritis dissecans as a similar process which they called spontaneous, or referred to it as a quiet necrosis in view of the absence of the signs of infection.

Preisser\(^{(93)}\) believed that the loose body was the result of the trauma subsequent to a static imperfection of the joint, while John Hunter\(^{(94)}\) believed that loose bodies followed the organisation and calcification of blood which had been extravasated into the joint cavity. These two theories need no discussion as they are obviously impossible in the light cast by more recent work on the subject of osteochondritis dissecans.

Wolbach and Allison\(^{(95)}\), while conducting a post-mortem examination on a case of diabetes mellitus,
noticed a swelling of the knee joint and, on opening the joint, discovered a typical lesion of osteochondritis dissecans in the lateral surface of the medial femoral condyle. The lesion was removed entire and submitted to histological examination. The loose body was still incompletely detached from the bony cavity and was divided into two almost equal portions by a transverse depression. They believe that this buckling of the fragment is due to mechanical effects owing to the loss of support of the deeper parts of the bone.

When a cut was made into the bone of the condyle to remove the specimen for examination it revealed almost immediately beneath the crater floor a bone cyst about 1.5 cm. by 2.5 cm. The cyst was separated from the floor of the crater by a thin layer of bone cancellous/about 3 mm. thick. Two smaller cysts were also found close to the crater and histologically the three "cysts" showed the same structure. They were areas devoid of bone trabeculae and without specialised boundary zones. The bulk of the tissue in these regions was a loose textured, myxomatous-like connective tissue enclosing small numbers of fat cells. In places there were dense fibrous tissue bands traversing these regions. At the peripheries there were
compact fibrous tissue or merely the normal fat-laden cells of the marrow." "Blood vessels were numerous in these 'cysts' except in central regions in which the tissue was compact. Here there were few blood vessels and large extents of tissue had none."

Wolback and Allison (95) declare that "The histological observations have not yielded any clue to the processes antecedent to the separation of the fragments. Neither is there any explanation of the origin of the cysts". They conclude that the separation was the effect of mechanical pressure on a portion of the articular cartilage with underlying cancellous bone bridging a cyst - the pressure probably being intermittent from weight bearing or movements of the joint.

They conclude their paper with the following statement - "There are two essentials to our explanation: one a sufficient loss in the condyle of cancellous bone to weaken materially the support of the articular cartilage as a whole; the other, the pressure of a 'cyst' immediately below the articular cartilage in a position subject to vertical and horizontal stresses. The rarity of typical instances of osteochondritis dissecans of the femur suggests that our explanation, even if dependent on the
fortuitous existence and distribution of 'cysts' may apply to other examples."

I shall reserve my criticisms of this theory and consider it in detail in the section on the morbid anatomy and histology of osteochondritis dissecans. It is, however, unlikely that this is the true cause of loose body formation for such cysts must arise in other bones where the overlying bone and cartilage would be subjected to stresses and strains of a similar nature, and yet I cannot find any case recorded in the condyles or lower surface of the tibia. So far as I can ascertain from the literature on the subject, the only sites affected, apart from the lower end of the femur, have been the capitellum of the humerus, the head of the femur and the astragalus, with doubtful cases in the shoulder.

Fromme(96) believes that loose bodies are detached by the development of a zone of transformation previously described by Looser(97) at the points of greatest strain in nutritional diseases of bone. X-rays of these lines of transformation usually show a transverse area of translucency in the shaft of a long bone which resembles an ununited fracture.

Phemister reports histologically on a zone excised from the femur to correct deformity and he believes
that the condition is nothing more than ununited fracture. It is also significant that up to the present time no case showing Looser's lines has been complicated by loose body production and no case of osteochondritis dissecans has even shown the slightest sign of such a zone of transformation.

From the summary of the theories given above it will be seen that a large amount of work has been performed on the causation of osteochondritis but that there is still little unanimity in regard to it. It is significant that Cotton (98), writing as recently as 1922, stated that he had no clear idea of the process and Colvin (99) in 1923 was also unable to come to a decision but paid tribute to the work of König. The greatest objection to most of the theories is that they tend to consider only the lesions in the medial condyle of the femur, e.g., the theory of impaction of the tibial spine, the theory of obliteration of the arteria genu media, traction on the posterior cruciate ligament, etc., whereas the disease is found in other sites. To my mind a theory of the etiology must be applicable to the disease in any situation and must explain why similar findings occur in cases without any history of injury as in those on a definite traumatic basis. If we regard all varieties of
osteochondritis as similar lesions, the theory must also explain the causation of all the typical osteochondritis dissecans.

In order to try and evolve such a universally applicable theory I have decided to consider the pathological features of osteochondritis dissecans by the study of a section of a complete lesion and of the loose bodies, and of cases of Köhler's disease and of Preiser's disease of the navicular. Having reviewed the macroscopic and microscopic findings it may be possible to uphold or discredit the various theories and to advance a theory which would explain all varieties of the disease.
Résumé of the Theories of Etiology of Köhler's disease of the second metatarsal head.

Since Köhler and Freiberg published their first descriptions of the disease a large number of papers have been published, many of which are based on a histological examination of one or two specimens. In my opinion it would serve no useful purpose to examine these theories in detail because the same theories reviewed above in regard to the causation of osteochondritis dissecans have all been advanced to account for this disease.

In brief, the following factors have been blamed for the production of the lesion:-

(1) A single trauma.
(2) A single trauma initiating an unknown pathological process.
(3) Repeated traumata due to faulty weight bearing.
(4) Embolism.
(5) Vascular lesions causing aseptic necrosis.
(6) Tuberculosis.
(7) Osteomyelitis.
(8) Rickets.

Many of the authors have declared that the disease is of the same nature as the other osteochondritides but the greater number have made no pathological studies to support their views.
I propose to divide this section into five parts, comprising the macroscopic and microscopic features of (1) the loose bodies removed from the knee joints of P.B., Case 1, and W.S., Case 22: (2) the complete lesion of osteochondritis dissecans obtained at operation in the case of P.R.? Case 23: (3) three specimens of metatarsal heads excised for Köhler's disease: (4) the loose body removed from the metatarsophalangeal joint of a case of Köhler's disease: (5) the navicular excised because of pain due to Preissner's disease. In addition I shall refer briefly to the characteristics of the single cartilaginous body from the elbow of F.K., Case 33. After the specimens have been examined in detail we shall be in a position to discuss the following aspects of the problem:-

(1) Are osteochondritis dissecans, infraction of the second metatarsal head and Preissner's disease of the navicular due to the same pathological process?

(2) Is it possible to reconcile the histological changes with the X-ray findings?

(3) Is there a theory capable of universal application to the disease in any situation?

(4) Has this theory been proved experimentally or, failing this, is there any method of establishing its value in the experimental animal.

With this brief review of the aims of this
section of my paper I shall pass on to consider the features of the loose bodies obtained from cases of osteochondritis dissecans. It must be remembered that the loose bodies are essentially the end result of the disease process and even a careful survey of their structure is unlikely to throw light on the factor responsible for their separation.

The Loose Bodies of Osteochondritis Dissecans.

Macroscopic Features.

The naked eye appearances of these osteocartilaginous loose bodies vary with the time that has elapsed from their separation into the joint. It is now a well known fact that loose bodies continue to grow by virtue of the proliferation of the cartilage they contain even after complete severing of their connections to the parent bone. For this reason Timbrall Fisher has adopted the following classification, which is supported by histological investigations:

(1) Recent detachments: living bone and cartilage.

(2) Bodies whose sojourn in the joint has been longer and showing proliferation of the articular cartilage. (The classical type of loose body.)

(3) Marked proliferation of cartilage.

(4) Excessive proliferation of cartilage.
 Feeble cartilage proliferation.

Formation of new bone in the loose body, either from the formation of adhesions or from incomplete detachment.

Loose bodies which have been recently detached from the articular surface vary greatly in size and show two surfaces of totally different characteristics. The superficial surface of the loose body is smooth and glistening, of a bluish-gray colour and is composed of apparently normal articular cartilage, whereas the deep surface is irregular and is made up of cancellous bone. The edges of the loose body tend to be irregular and this has led Timbrell Fisher and other workers to favour the fracture theory.

Following the complete separation of the loose body into the joint the bone, deprived of its vascular supply, undergoes necrosis but the cartilage is able to proliferate as it is sufficiently nourished at the edges of the loose body at least by the synovial fluid. The loose body therefore becomes more regular in shape and its borders become smooth and rounded. This cartilage proliferation tends to spread so that it gradually covers the rough bony surface of the loose body. In some cases, however, the bone may also continue to live.

I have never seen loose bodies with a marked
Fig. 69.
degree of proliferation but Timbrell Fisher illustrates a case in which the cartilage proliferation had been so exuberant that it had almost completely obliterated the original articular cartilage, and this may go on to such an extreme degree that a cartilaginous 'tumour' is formed with the old articular surface in its centre.

Fisher believes that feeble proliferation of articular cartilage is a rare condition as he only saw it in one case but it will be seen later that this proliferation has not been a marked feature in my series of cases.

Fisher also records the formation of new bone in the loose bodies, a feature no other author has observed but it is to be borne in mind that all these cases were examples either of incomplete detachment or secondary attachment to the synovial membrane resulting in an adequate blood supply.

**Loose Bodies from Case 1: P.B.**

The two loose bodies removed from the medial condyle of the femur are illustrated in Fig. 69. The larger body measured 3.75 cm. x 2.2 cm., and was irregular in shape with rough irregular edges. Its superficial surface was composed of apparently normal articular cartilage while its base was rough and
showed the appearance of cancellous bone. The smaller loose body was exactly similar. The larger loose body, probably due to the pressure of weight bearing on its centre, which was unsupported, was buckled and gave way through this area when in the decalcifying solution.

The smaller loose body was decalcified in Perenney's fluid and sections were cut transversely and stained in the ordinary way with haematoxylin and eosin. The sections show an osteocartilaginous loose body with living bone and cartilage. The bony trabeculae are of smaller size than normal, as shown by the increase in size of the cancellous spaces which are filled with a fairly vascular fibrous tissue. The articular surface of the loose body is covered with articular cartilage, which is slightly increased in thickness but shows no gross change except at the edge of the loose body. Fig. 70 is a low power view of the loose body showing very clearly the cartilaginous surface, the bony portion made up of living bony trabeculae with an increase in the size of the inter-trabecular spaces. The surface of separation is also well shown and presents a striking claim for the discarding of the theory of traumatic separation. It is smooth and for the most part is composed of the ends of
bony trabeculae covered over with a thin layer of fibrous tissue. At its borders there is a rounding off of the edge of the loose body and this is produced by the proliferation of the cells of the articular cartilage forming a fibro-cartilaginous layer over the bony surface. This growth by multiplication of the cartilage cells is shown in Fig. 71, which is a greater magnification of the area marked x in the low power view of the body.

At no part is there the slightest sign of new bone formation. This is the type of loose body referred to as the 'classical' variety in Timbrell Fisher's paper.

A portion of the wall of the bony crater was also obtained from this case but its important features will be discussed with the appearances of the complete lesion from another case in a later section.

Loose Bodies from Case 22.

This was of larger size than either of the loose bodies previously described but was also incompletely detached at the time of operation. Its naked eye appearances were different from those of the other loose bodies in that the deep surface showed only a small area of cancellous bone towards the site of attachment to the floor of the crater, whereas the
remainder of this surface was smooth and apparently composed of cartilage (Fig. 72). The loose body was of much greater length than those in the previous case but it is interesting that it also showed at almost its central point an area of cracking or buckling, suggestive of inability to stand the strain of weight bearing owing to the absence of underlying support. This is the fact to which Woolbach and Allison attached so much significance and one of their main reasons for blaming a subjacent bone cyst for producing osteochondritis dissecans.

Sections of the loose body after decalcification and stained by ordinary methods (Fig. 73) showed the presence of only a small amount of cancellous bone which, although not dead bone, has the appearance of bone about to undergo necrosis. The bone cells are shrunken and stain poorly and in some cases are missing from the lacunae of the bone trabeculae. The cartilage shows a considerable increase in thickness due to proliferation of its cells. The surface of separation is smooth and regular and, except at the point where the loose body was still attached to the crater, shows a covering of fibrous tissue laid down in lines parallel to the long axis of the loose body.

All that an examination of the loose bodies
reveals is — (1) that the loose body continues to live and that it may increase in size owing to proliferation of its cartilage cells. (2) The surface of separation even in cases of recent detachment or incomplete detachment shows a smooth covering of cellular fibrous tissue. It does not suggest a fracture surface.

It is useless, therefore, to theorise on the etiology of the disease when the theory can only be based on the examination of its end products, the loose bodies. Surely it may be accepted from this examination, however, that trauma is not the cause. If the loose body were the result of an ununited fracture there would never be living bone in the loose body unless it had rapidly gained an attachment to the synovial membrane.

Complete lesion of Osteochondritis Dissecaus from P. R.: Case 23.

Naked eye appearances.

The specimen is composed of a small portion of the medial condyle of the femur and presents two main surfaces. The superficial surface is composed of articular cartilage which ends in a crater in which a loose body covered with articular cartilage is lying. The loose body does not completely fill the area of
the cavity in the cartilaginous surface.

The deep portion of the specimen is composed of cancellous bone which shows a recently cut surface. The loose body is mobile but apparently is still attached to the floor of the bony crater. The specimen was decalcified and sections were then cut to include the loose body and the walls of the cavity in the condyle. Ordinary haematoxylin and eosin staining was employed but sections stained with Azan were also prepared to demonstrate the bone and cartilage in sharp contrast.

**Histopathology.**

The morbid histology of this specimen is of great importance as it is apparently the first specimen to be obtained in an otherwise normal joint. Therefore illustrate a low power view of the specimen in Fig. 74, which can be used for key purposes in referring to the other figures of certain areas under greater magnification.

The loose body is lying incompletely filling a crater in the cancellous bone of the medial femoral condyle. On its superficial surface the loose body is covered with articular cartilage which shows some increase in thickness in comparison with the articular cartilage of the edges of the bony cavity. There is
proliferation of the cartilage cells exactly comparable to that shown in an earlier specimen.

The surface of separation is smooth and regular in outline and is covered over by a thin layer of fibrous tissue in the central portion and shows at its edges a degree of cartilage proliferation.

The bone of this loose body is dead. There can be no other interpretation of the changes seen in Fig. 75, which is a low power view of the osseous tissue of the loose body. It shows bony trabeculae with lacunae but a complete absence of bone corpuscles while the cancellous spaces show only débris with no sign of definite marrow or fibrous tissue.

From our previous consideration of loose bodies this, therefore, was apparently not of very recent detachment from the cavity.

The surface of separation of the Bony Cavity.

As we have seen above that the loose body had been free for some time, we should expect some degree of repair in the walls of the bony crater. This supposition is confirmed on microscopical examination of the area where the surface of separation abuts on the articular cartilage of the condyle. Here we find once more a proliferation of cartilage leading to the production of a layer of fibro-cartilage which is creeping
along the line of separation. The proliferation of the cartilage cells is shown in Fig. 76, which is a higher magnification of the area marked A on the low power view of the specimen.

The floor of the cavity presents other features of great interest to our present study. The bony trabeculae are in many places becoming eroded with a consequent widening of the inter-trabecular spaces and this is well shown in Fig. 77, which is taken at a point B in the key specimen. This shows the decrease in size of the bone trabeculae with the presence of numerous giant cells or osteoclasts along their surface and the filling up of the marrow spaces with an extremely vascular and cellular young fibrous tissue. In addition, along the floor of the crater there is evidence of intense activity with great increase in the vascularity and the production of fibrous tissue filling the inter-trabecular spaces and associated with bone resorption even in the absence of osteoclasts.

The area of osteoclastic bone resorption is limited to the central area of the crater surface and this is well shown in Fig. 78, a high power view. A very interesting picture which I have not previously observed in any section is seen in close association
with this zone of osteoclastic activity. This is illustrated in a higher magnification at the point marked c in the low power figure (fig. 79). Here the bone trabeculae are shown becoming less definite in size and shape and losing their staining reaction.

In the middle of the trabeculae islands of proliferating cartilage cells can be seen. This would appear to support the theory of Leriche and Policard that all derivatives of mesenchyme are capable of being dedifferentiated to be differentiated into another tissue derived from mesoblast.

There are obvious fallacies in regard to the interpretation of the findings in this specimen. In the first place to preserve the function of the joint it was impossible to remove more than a mere shaving of the cancellous bone forming the floor of the cavity so that we do not know what changes may have been present in the bone of the femoral condyle. Secondly, the changes observed are capable of two main and totally distinct interpretations. The changes of bone resorption and fibrous tissue proliferation with increased vascularity are the factors responsible for (a) the separation of the loose body; (b) the repair of the defect in the condyle. I believe that they are to be regarded as the causal agent in the
of the lesion - a view which is supported by changes described in the cases of Köhler's in a later section.

Floor of the Cavity in P.B.: Case 1. This section was taken from the wall of the cavity in the medial femoral condyle and was cut at angles to the articular surface of the condyle, thus articular cartilage and the bone forming the floor of the cavity.

Microscopic Appearances. The microscopic appearances are in diametric contrast to the prevailing nature of the floor of such cavities. The surface is usually described as being avascular and lined with old fibrous tissue or fibrosis. Here, however, the universal feature of section is the presence of intense hyperaemia with proliferation of a young oedematous fibrous tissue and absorption of living bone trabeculae. However, no osteoclasts to be seen in this section. Hyperaemia is well shown in Fig. 80.
production of the lesion - a view which is supported by the changes described in the cases of Köhler's disease in a later section.

The Floor of the Cavity in P.B.: Case 1.

This section was taken from the wall of the cavity in the medial femoral condyle and was cut at right angles to the articular surface of the condyle to include articular cartilage and the bone forming the floor of the cavity.

Microscopic Appearances. The microscopic features are in diametric contrast to the prevailing opinions of the nature of the floor of such cavities. The crater surface is usually described as being avascular and lined with old fibrous tissue or fibro-cartilage. Here, however, the universal feature of the section is the presence of intense hyperaemia with the proliferation of a young oedematous fibrous tissue and the absorption of living bone trabeculae. There are, however, no osteoclasts to be seen in this section. The hyperaemia is well shown in Fig. 80.
Pathology of Köhler's Disease of the Second Metatarsal Bone.

The past views on the pathology of this disease have been excellently summarised by Köhler in his book on Roentgenology. The changes occurring may conveniently be classified into:

1. Changes affecting the capsule of the metatarseo-phalangeal joint.
2. Changes in the synovial membrane.
3. Changes in the shape of the metatarsal head.
5. Changes in the shaft of the metatarsal.
6. Changes in structure of the bones.

Naked eye appearances in Mrs M.S.: Case 1.

At operation the metatarsophalangeal joint was found grossly swollen but the capsule was not opened. There was marked thickening of the capsular ligaments, especially on the plantar surface of the joint, and considerable difficulty was encountered in freeing the capsule in this area. The specimen consisted of the head and distal third of the shaft of the metatarsal bone, the metatarsophalangeal joint and the base of the first phalanx. A small window was cut in the ligaments on the dorsal surface of the metatarsal-
phalangeal joint and two loose bodies could be seen still attached to the metatarsal head. There was a definite increase in the size of the joint cavity and a corresponding increase in the amount of synovial fluid which, however, was of normal colour and consistency. The synovial membrane was thickened, especially on the plantar aspect of the joint and one or two hypertrophied fringes were visible. There was no change in the synovial membrane suggesting an inflammatory process.

The specimen was then divided into two equal parts, a dorsal and a plantar, and the appearance of these two portions is well shown in Figs. 81 and 82. They show the grossly deformed metatarsal head with two loose bodies, one apparently firmly attached and the other almost completely separated. A third loose body was actually present but it became detached during the cutting of the specimen. The base of the phalanx also showed deformity and irregularity of its surface but the changes were not so marked as in the metatarsal head. The opening of the joint on section brought the condition of the ligaments more clearly to view and on the fibular side of the articulation there was a bony particle about the size of an orange seed lying embedded in the ligament. This portion of bone
was almost circular in outline and corresponds very closely to Köhler's description of similar bodies varying in size from that of a pin-head to that of a lentil but always circular.

On the opposite side the metatarso-phalangeal joint was opened and the ligaments were considerably thickened. The grossly deformed, flattened metatarsal head was excised and the specimen was divided so that sections at right angles to the articular surface could be prepared. The first specimen was sectioned in the same way and it was found possible to obtain a section of the metatarsal head with the loose bodies, the base of the phalanx and the joint. In this slide we have a complete lesion and its appearance under low magnification is shown in Fig. 83. This figure is of insufficient magnification to show detail but is intended to serve as a key to explain the microphotographs which follow. It also confirms the naked eye description of the joint but does not reveal any ossification in the capsule. This bony nodule in the ligaments of the joint was apparent in a section which did not include the loose bodies and it will be shown in a later illustration.

The metatarsal head from the opposite foot is illustrated under a low magnification in Fig. 84, which
is also meant to serve as a plan for facilitating the microscopic description of the specimen.

Morbid Histology.

Köhler reviewed the microscopic findings in one metatarsal bone from this disease and was of the opinion that the only divergence from the normal was a new formation of connective tissue, with plentiful cells of the embryonic type, filling the marrow cavity of the bone. He could find no sign of necrosis, inflammation, embolism or tuberculosis, all of which had been described in earlier papers on the pathology.

Axhausen reported on two cases of Köhler's disease in which his attention had been drawn to wedge-shaped areas of subchondral bone necrosis with necrotic lamellae of spongy bone and necrotic marrow. The condition of the fibrous tissue was interesting for in some areas it was avascular and sclerotic and in other areas was richly cellular and extremely vascular. In this area of vascular granulation tissue there was evidence of lacunar absorption in some cases, with the presence of osteoclasts. The cartilage was thickened over the diseased area of the bone.

From these appearances Axhausen formulated the
theory that the disease was inaugurated by embolism of an end artery with the formation of an infarct in the bone. The changes seen with proliferation of the fibrous tissue and absorption of bone he believed to be evidence of repair which would gradually extend to and revascularise the necrosed area. This reparative process would lead to softening of the underlying bone and the trauma of weight bearing would cause the deformity of the metatarsal head.

Axhausen's conclusions have found support in the work of Cahen-Brach, Holst and Chandrikoff and Heitzmann and Engel, but Kappis, on the evidence of his own histological studies, was unable to accept them. Kappis found that the bone had undergone absorption under the articular cartilage at certain limited and flattened areas. "The region between the cartilage of the joint surface and the bone was partially occupied by small cavities, but principally by a tissue composed partly of pure fibrous tissue of varying cellular richness and partly of chondroid tissue and one or two osseous remnants. Necrotic bone could not be seen anywhere. Signs of inflammation, embolism, thrombosis, tumour and osteitis fibrosa were entirely absent".

Engelbe believes that the primary factor is the
conversion of the bone marrow into a more vascular young fibrous marrow, causing the normal physiology of the bone to be deranged with the result that it becomes very liable to trauma.

After this brief review of the prevailing opinions on the pathology let us study the sections we have obtained.

A glance at the low power view of the metatarsophalangeal joint shown in Fig. 83 will show that the bony trabeculae of the metatarsal head do not run in a regular manner. There is an area in the centre of the metatarsal head composed of coarser trabeculae with a more densely staining tissue in their meshes than in other parts of the bone. On either side of this area the bone is spongy and is composed of firm trabeculae with no obvious tissue in their marrow spaces.

Histological examination of the completely separated loose body shows that it is composed of living cartilage and the body still partly attached to the bone has a similar structure. The articular cartilage is greatly thickened and irregular in its extent.

On observing the area immediately below the completely separated loose body with a higher magnification, i.e., at a point A in Fig. 83, we find a state
of affairs which supports the findings of Kappis rather than those of Axhausen. The whole field (Fig. 85) is occupied by large numbers of distended blood vessels in a young embryonic type of fibrous tissue which can be seen in many cases in close association with bone lamellae which are undergoing absorption. The absorption in many areas is extremely well marked, the lamellae becoming greatly separated leaving large inter-lamellar spaces filled with vascular fibrous tissue. In no area is there any sign of the presence of the osteoclastic giant cell, showing that bone resorption can take place without its activity. An area of absorption or osteoporosis is shown in Fig. 86 which is a higher magnification at B in the low power photograph. Similar changes are occurring in the base of the phalanx but are not nearly so far advanced.

The last point of interest in this specimen is the small nodule of bone in the ligaments on the lateral side of the joint. The ligaments are thickened but do not show any other change apart from increased vascularity. The nodule is composed of living bone which does not show the changes of absorption but gives the impression from the small size of the trabeculae that it is young, recently formed bone (Fig. 87).
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The metatarsal head from the left foot of the same patient shows an exaggerated degree of the same process seen in the metatarso-phalangeal joint of the opposite side. The specimen shows extensive areas of fibrous replacement of the pre-existing bone and this change is seen to greatest advantage in the subchondral area of the bone. In a section taken at point A in the low power photograph, higher magnification (Fig. 88) shows the absorption of the subchondral bone and its replacement by fibrous tissue. Other areas in the subchondral zone show marked hyperaemia, with dilated blood vessels and decalcification and absorption of the bone trabeculae without the aid of osteoclasts as shown in Fig. 89. Overlying the hyperaemic areas there is a great increase in the thickness of the articular cartilage which presents an irregular superficial surface. The localisation of this hyperaemic process to the subchondral tissue is shown in Fig. 90 where, included in one microscopic field, are cartilage with an underlying area of hyperaemia and osteoporosis.

The changes seen in these two specimens, which were obtained from a case of fairly long standing, are difficult to interpret. As far as I am aware there is no means of differentiating newly formed bone from bone undergoing resorption and the changes observed may be the evidence of the repair of the lesion. The osteoporotic areas might conceivably be the areas of newly formed
bone trabeculae. We shall see, however, that the microscopic appearances are comparable to those seen in the next specimen, which was obtained from a very early case of Köhler's disease. It seems reasonable, therefore, to interpret the findings in the following way.—

The lesion is characterised by (1) subchondral hyperaemia leading to the production of young, vascular, fibrous tissue and bone resorption; (2) growth in thickness of the articular cartilage due to the improvement in blood supply; (3) owing to the hyperaemic osteoporosis, the bone in the subchondral area becomes softened and deformity of the head follows weight bearing. (4) In some cases the vascular fibrous tissue demarcates certain areas of the subchondral bone and the portions superficial to them may become loosened and set free as loose bodies. The loose bodies may be composed of cartilage and bone. (5) In other cases the loose bodies may be formed by the absence of support for the articular cartilage and subchondral bone produced by the hyperaemia.

Histopathology of an early case of Köhler's disease.

The point of great interest in this specimen is the observation of an area at one point of the
articulur surface which is apparently in process of demarcation. Fig. 91 is a low power view of the entire metatarsal head and the line of cleavage in the subchondral tissue can be seen between the points A and B.

A higher magnification along this line ought, therefore to be of significance to our study of the problem. We find in such a field (Figs. 92 & 93) evidence in support of our contentions because the line of cleavage runs through an area of subchondral bone which shows hyperaemia, bone resorption, and marked proliferation of embryonic fibrous tissue. The changes are exactly similar along the whole extent of the line of cleavage and at some areas the formation of a fibro-cartilaginous layer from the fibrous tissue can be seen. This is apparently to repair the raw surface which would be left after the separation of the loose body.

In brief, therefore, it may be said that the changes are exactly parallel to those found in the whole section of the lesion of osteochondritis dissecans from the knee joint. Support for this opinion may be sought in the structure of the loose body removed from a case of Köhler's disease previously described. If the loose body is of a similar nature to those found
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the knee joint. Support for this opinion may be
sought in the structure of the loose body removed from
a case of Köhler's disease previously described. If
the loose body is of a similar nature to those found
in osteochondritis dissecans, it will be additional evidence that the two diseases are essentially the same.

**Macroscopic and Microscopic Features of Loose Body from a Case of Köhler's disease of the second metatarsal head.**

The loose body presents two main surfaces, the one smooth and glistening and apparently composed of articular cartilage, the other rough and made up of cancellous bone. Over the roughened surfaces there is an apparent growth of tissue, particularly at the edges just as in the loose bodies previously described.

A low power view of a section of this free body shows a similar appearance to those previously illustrated. The body is composed of cancellous bone in the central area, with a covering of articular cartilage showing some proliferation at the edges. The bone is normal cancellous bone, showing in its spaces a very vascular marrow tissue which is more fibrous than normal. The surface of separation is composed of cancellous bone which has been covered over with a layer of fibrous tissue and fibro-cartilage is apparent in some areas. The underlying bone contains in its cancellous spaces a vascular fibrous tissue.
The loose body is illustrated in Fig. 94 and the surface of separation is seen under greater magnification in Fig. 95.

From the description, therefore, the loose body might quite easily be one that had been separated from a case of osteochondritis dissecans.

An unusual specimen is shown in Fig. 96, which is a low magnification of a section through a second metatarsal head. This portion of bone was excised from a patient who had an abnormally long second metatarsal bone and, following a debilitating illness, there was superimposed, on an already potentially weak foot, a loss of muscle power. There was marked falling of the anterior arch so that the second metatarsal head became the point of maximum weight bearing. When she reported to hospital the skin of the sole of the foot had almost given way over the second metatarsal head and the bone was therefore removed. It shows the normal shape of a metatarsal head in sharp contrast with the sections from the cases of Köhler's disease. The interesting feature, however, is the small size of the bony trabeculae and the large size of the cancellous spaces. There is no increase in the thickness of the cortex of the shaft. Examination with a higher magnification shows increased
vascularity of the marrow with the proliferation of vascular connective tissue and bone absorption. This in the young subject to my mind would have resulted in the changes of Köhler's disease.

Preissner's Disease of the Navicular.

The changes seen in the sections of the navicular bone from the case of Preissner's disease are not typical of osteochondritis as shown by the sections examined above. Along the line of the fracture the bone is necrotic and is becoming replaced through the agency of vascular fibrous tissue. On either side of the cyst the changes are similar and show nothing more than an ununited fracture. It is my contention, therefore, that Preissner's disease is not a true osteochondritis but is an ununited fracture of the navicular. Failure of union may result from one of two causes - (1) Inadequate immobilisation; (2) necrosis of one of the bony fragments. Union of the navicular will occur provided the fracture is kept sufficiently immobilised for an adequate period. It is of no value to have a fixed time for the splinting in Plaster of Paris of such cases. Each case must be judged on its merits and only when radiological healing has occurred should the plaster be discontinued.
In some cases the blood supply of the proximal fragment of the bone is cut off (as shown by its normal radiological density when all the other bones show the atrophy of disuse) and healing can only occur when this area has been revascularised from the distal fragment. Such cases are of course exactly comparable to the cases of necrosis in the femoral head recorded by Phemister.

Loose Body from the Elbow Joint of F.K.: Case 33.

This loose body differs from those of osteochondritis dessicans in that its surfaces are all smooth and glistening and macroscopically composed of cartilage (Fig. 97). The loose body on section is composed almost entirely of cartilage with a thin fibrous covering. In the centre there are areas of a more hyaline type, with early calcification but no sign of ossification.

There is no doubt that this type of body is derived from the proliferation of cartilage cells in the synovial membrane and is not a detachment from the articular surface.
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Are Osteochondritis dissecans, the Osteochondritides (as exemplified by Köhler’s disease of the second metatarsal head) and Preissner’s disease of the Navicular due to the same pathological process?

As mentioned above I am of the opinion, on histological grounds, that Preissner’s disease of the navicular is nothing more than a non-union of a fracture which has been untreated or badly treated.

The histological features of Köhler’s disease and of the lesions of osteochondritis dissecans are strictly comparable for in both we find—
(1) Hyperaemia.
(2) Fibrous replacement of the marrow.
(3) Bone resorption.
(4) Proliferation of cartilage.
(5) Loose body formation.

The bone in the loose bodies in both diseases is living cancellous bone which has been separated by exactly the same pathological process. Even in the early stages of separation there is no question of the bone being necrotic as is suggested by the theories advanced by Axhausen and others who believe that embolism and infarction are the primary factors in the separation of the loose bodies. There is no evidence on microscopic examination to support the theory of a
fracture either of recent origin or showing non-union.

There can be no doubt, therefore, that the two conditions are analogous and I see no reason why the other lesions known as osteochondritis should not be of a similar nature, modified by the site of the lesion and the different stresses and strains to which different situations are liable. For instance, Osgood Schlatter's disease causes an apparent partial separation of the lower part of the tibial tubercle, which heals in the course of time. Now the condition, to my way of thinking at least, is kept from healing by the stress and strain of the pull of the quadriceps tendon. If this were so, then a fixation operation should lead to greater rapidity of healing and this theory is supported by the recent work of Bosworth who secured healing in resistant cases by the insertion of an autogenous bone peg.

As I have no specimens of the other osteochondritis for histological examination, it is impossible to be definite that they are all essentially the same but I propose to try and correlate the histological findings with the X-ray appearances and, having done that, to compare the X-ray features of osteochondritis in the diverse situations in which it is found.
Are the X-ray features comparable to the histological changes?

From our brief consideration of the theory of Leriche and Policard and Watson Jones' deductions therefrom, we know that hyperaemia results in decalcification and therefore we see osteoporosis on X-ray examination. Similarly we know that necrotic bone casts a denser shadow than normal, particularly if the surrounding bones show atrophy.

In osteochondritis dissecans in the case of P.R., Case 23, the X-ray examination showed a loose body of a slightly increased opacity separated from the bone of the medial condyle of the femur by a comparatively wide area of translucency. On examination of the specimen we find that the loose body is virtually in contact with the floor of the crater. This anomaly is easily explained when we remember that the floor of the crater is formed by hyperaemic decalcified bone and this is the reason for the apparently wide interval between the body and the condyle as the hyperaemic area is decalcified and therefore radiotranslucent.

In the same way the irregularity of the hyperaemia explains the irregularity of the surface of separation on the side of the parent bone.

In the case of Köhler's disease the great increase
in the thickness of the articular cartilage explains the great increase in the joint space as seen on X-ray examination. Similarly it was pointed out that the periphery of the head showed the greatest bone absorption and this is supported by the decreased opacity of the X-ray shadow in these areas.

It is therefore possible by a careful study of X-ray films to forecast accurately the histological changes which are present, e.g., we can say from an X-ray photograph that the bone is dead or possibly that it is osteoporotic, and we can then picture in our minds dead bone or bone absorption and, as we have seen, in all probability hyperaemia. Let us therefore compare the X-ray findings in the various lesions grouped under the generic term of osteochondritis.

Comparison of the Changes seen by X-ray examination in the various lesions of Osteochondritis.

In the lesions of osteochondritis the X-ray reveals the feature known as fragmentation in which islands of sclerosed bone alternate with islands of osteoporosis. We have therefore to consider that these appearances are due to dead bone and areas of hyperaemia. Now from the recent work on infarction of the rabbit's femur by carbon emboli, we find that
an exactly comparable condition can be produced in the experimental animal. We may say, therefore, that the changes occurring in Perthe's disease, for example, are in all probability of an embolic nature. Again in Köhler's tarsal scaphoiditis the bone is shown to be sclerotic and of much greater density than the other bones of the foot. We must interpret this as death of the bone and the prolonged healing process is due to the delay in the revascularisation of the part. These two examples will serve to illustrate the pathology as interpreted from the X-ray findings.

In Köhler's disease, however, we find that the first change is the appearance of islands of radiotranslucency. There is no real sclerosis and the primary change is in all probability a hyperaemia and not an ischaemia.

The classification of the diseases labelled osteochondritis has largely been based on X-ray evidence and on the clinical course of the disease, and there have never been definite pathological studies worthy of mention in the great majority of the lesions. From the radiologist's point of view I should therefore say that osteochondritis dissecans and Köhler's disease of the second metatarsal head were strictly analogous but that the other lesions suggest a different etiology.
This is however a hypothetical question because we do not know how far advanced the lesion is before it shows evidence of its presence on X-ray examination. In some sites there may be no symptoms in the early stages and by the time advice is sought we may be dealing with a late stage of the disease. From these considerations, therefore, the X-ray, though valuable in diagnosis, is of no value in the estimation of the underlying pathological process.

We may say, therefore, that from the X-ray findings we are not entitled to decide whether all forms of osteochondritis are due to the same pathological process.

Is there a Theory capable of explaining Osteochondritis dissecans in any site?

From our study of a series of cases we know that osteochondritis may be found in either condyle of the femur, in the capitellum of the humerus and in the talus, while other cases have been reported in the hip and other joints. It is therefore of no value to postulate such theories as the impaction of the tibial spine, the pull of the cruciate ligament or torsion of the arteria genu media, etc. We must have a theory which will allow for the development of osteochondritis
dissecans in any site. To my mind there is only one possible theory which is concerned with vascular changes in the bone.

From our histological study of the cases of Köhler's disease and of the complete lesion of osteochondritis dissecans we find that the predominant feature is hyperaemia and decalcification. What we have to decide in, is this the primary manifestation of the disease or is it an attempt at revascularisation of necrotic bone as has been suggested by the protagonists of the embolic theory. In the material at my disposal I have observed no sign of bone necrosis except in completely detached loose bodies and have therefore no hesitation in saying that the hyperaemia is the primary factor in the etiology. How does the increased vascularity arise?

When we examine cases of Köhler's disease of the second metatarsal head we invariably find that the lesion is found in those patients who show some deformity of the fore foot. These deformities, in brief, are any lesion which will throw the main point of weight bearing on to the second metatarsal head instead of the first. This is found in abnormally short first metatarsals or in abnormally long second metatarsal bones or in the condition of metatarsus
primus varus.

If the weight is borne on the second metatarsal head it is of course liable to repeated injury as it is not built on the stout lines of the first metatarsal. The altered weight bearing is shown by the alteration in the shaft of the bone and the adaptation for weight bearing is characterised by the thickening of the cortex, a condition known as Deutschlander's disease. The repeated trauma is sufficient to cause hyperaemia in the actively growing epiphysis of the head of the bone. As we have seen above, hyperaemia leads to decalcification and therefore to softening of the bone. Once the bone has become softened, the persistence of the abnormal weight bearing will cause deformity of the head of the bone. The cartilage and the subchondral bone have a relatively poor blood supply in comparison with the deeper cancellous bone and therefore do not share in the hyperaemia and subsequent softening. They lose the support normally afforded them owing to the softening of the underlying bone and therefore may give way through the strain of weight bearing. Once this has happened, two further processes may occur - (1) increase of the deformity of the head of the bone and (2) the unsupported portion may be thrown off as a loose body.
This theory would explain the presence or absence of the loose bodies in cases of Köhler's disease. We have still to consider, however, the sex incidence of the disease. Deformities of the fore foot are certainly more common in the female and the wearing of high heels throws the weight forward on to the front part of the foot and therefore increases the trauma to the second metatarsal head.

The theory of traumatic hyperaemia is supported by the X-rays of a case (Fig. 49) in which there are small areas of translucency. The mere cessation of the trauma of weight bearing was sufficient to stop the hyperaemia and lead to recalcification as shown in Fig. 50.

We have also to try and justify the theory in regard to age incidence of Köhler's disease. The disease occurs principally in young girls before the union of the epiphysis and diaphysis of the metatarsal bone, i.e., before the age of eighteen. It occurs therefore at the time when the epiphysis is growing actively and the more newly formed bone is more liable to the effects of the changes in blood supply. If the repeated trauma should begin in an adult bone, the changes are more liable to be of a diffuse nature due to the difference in the vascular supply and in the
age of the bone. This is supported by the appearances shown in the adult metatarsal head in Fig. 95.

We find also in the cases of osteochondritis dissecans that, although there is not a universal history of injury, the disease is more common in those who are subject to repeated trauma. In miners their work entails constant kneeling and therefore traction on the femoral condyle which, in a young epiphysis, would be sufficient to cause hyperaemia. No decalcification would be seen on X-ray examination because the bulk of the femoral condyles prevents a small decalcified area from showing. I have proved this experimentally for, on boring a hole transversely in the lower end of the dog's femur, X-ray examination shows no lesion if carried out in the antero-posterior plane, but if carried out in the axis of the bore-hole it is shown as a decalcified area.

Just as in the metatarsal head, the cartilage and subchondral bone do not share in the pathology and two courses are open for the process - (1) healing and (2) the extrusion of a loose body. Healing may occur if the exciting cause is removed and no lesion would ever become apparent on the X-ray examination. Just as in the metatarsal head, a loose body may be formed. The fact that the body is, at any rate early
in its evolution, composed of living bone is confirmed by Littlejohn's cases showing rapid healing by preventing the trauma of weight bearing.

When considering this theory I was struck by seeing compressed air drill workers engaged in breaking up the roadway and immediately thought, here is repeated trauma to the arms by the concussion of the powerful drill. Such intermittent trauma should cause osteochondritis dissecans if the theory were correct. A survey of the literature found that the German Government in 1929 ordered an investigation into the diseases of compressed air drill workers. Paul Rostock (112) reviews the diseases produced by the rhythmical concussion of the compressed air drill. He points out that the nature and method of recoil in a joint depends on its anatomical form as well as on its position at the moment of impact. He divides the lesions quite generally into two types.

(1) Involving the joint capsule and muscle attachments;

(2) Involving the articular surfaces and underlying bones.

In the case of the muscles and joint capsules the partial separation of their attachments by the great force of the impact leads to calcification of their insertions, e.g., in the brachialis. In joints such
as those of the hand, where the vibration cannot be compensated for by muscular action, the bones show partial necrosis and osteochondritis dissecans. Similarly, the changes of osteochondritis dissecans are commonly found in the capitellum of the humerus due to the trauma from the repeated impaction of the radial head on the articular surface.

This is, therefore, striking evidence of the application of the theory. It is impossible to speak for the other lesions of osteochondritis without submitting early cases to histological examination.

With this conception of the etiology and pathology of the lesion we have to consider the question - what is the rational treatment of the disease.

**TREATMENT.**

We know that the disease is not embolic in nature and that if given a proper opportunity, healing can and will occur. The treatment therefore, to my mind, should be on the following lines.

(1) In early cases of osteochondritis dissecans in which the body is still in situ - conservative treatment.
(2) In cases with slight extrusion of the loose body - conservative treatment.

By conservative treatment I mean the prevention of the repeated trauma, i.e., weight bearing, which may be obtained by the wearing of a knee cage to limit extension of the knee or, in the case of the capitellum of the humerus, the arm should be put at rest in a sling.

(3) When the loose body has become free in the joint cavity, the joint should be explored and the loose bodies should be removed. All loose tissue should be removed from the floor of the crater to guard against recurrence.

(4) If conservative treatment fails in Types 2 and 3, operation should be advised to prevent the onset of osteo-arthritic changes.

In the case of Köhler's disease the treatment should also be conservative. The weight should be borne further back on the foot by the wearing of a metatarsal pad or a metatarsal bar may be worn on the sole of the shoe. A low heel is of course an advantage. If conservative treatment fails and pain is severe, it may be necessary to excise the affected metatarsal head.
EXPERIMENTAL WORK.

It is easy to theorise but it is hard to prove the correctness of a theory. It is obviously impossible to produce repeated concussional trauma in the experimental animal and some other means therefore of producing hyperaemia had to be sought. In this connection I must refer to the work of Tammann (113) performed on the knee joints of young dogs in 1933.

Tammann reviews briefly the various etiological theories with which he is familiar and refers to the repeated failure to produce loose bodies by detaching portions of the articular surface. He quotes the experimental work of Axhausen, who tried to reproduce the disease by the electrolytic destruction of circumscribed areas of the articular cartilage. Mannheim repeated these experiments and they were able to obtain a process which was not said to be osteochondritis dissecans but at least had a resemblance to it.

Here is a justifiable criticism of this technique for we have seen that the cartilage is not the tissue which is injured first. Cases have been found at operation in which the cartilage has been intact although the body has been quite loosely attached to
the wall of the cavity. The primary lesion appears in the bone of the condyle close to the subchondral layer of bone and therefore experimental work must aim at producing a lesion in this situation.

Because of the inaccuracy of Axhausen's experimental technique, and as failure had always followed other methods, Tammann decided to perform a series of experiments on the knee joints of growing dogs. Bergmann, Walter and Lobeck had failed to produce loose bodies by injecting the arteries of the knee joints with silver iodide, silver powder, Indian ink, charcoal and cultures of fungus. These experiments produced more or less widespread necroses but the necrotic area was never cast off. Tammann therefore tried to produce an injury in the subchondral region of the lower femoral epiphysis and his technique was as follows:–

The knee joint of a growing dog was opened and the medial femoral condyle was exposed. A drill hole was bored in the epiphysis to end subchondrally under the joint surface of the medial condyle or in the region of the intercondylar fossa. A small insulated ball electrode was passed along the drill hole until the electrode lay in the subchondral region. The subchondral bone in the region of the electrode was coagulated until the cartilage overlying the electrode
showed a very slight change of colour. The success of the experiment depended on the coagulation occurring in the subchondral bone. Tammann was able in this way to demonstrate changes strictly comparable macroscopically with osteochondritis dissecans but was unable to obtain X-ray evidence of the lesions in the living animal.

While acknowledging the importance of Tammann's work I have one or two criticisms to offer. In the first place he produced injury to the articular cartilage although he strongly denies that this is so. The mere fact that the cartilage changed colour is proof of its injury. Secondly, he produced necrosis of bone whereas we know that bone necrosis does not occur until the loose body has severed its connections. Lastly, the process may have simply been the removal of dead tissue by vascular granulation tissue or it may have resulted from the mere mechanical failure of support due to the removal of the cancellous bone.

I decided therefore to repeat Tammann's work and to modify the technique to try and produce hyperaemia in the subchondral region of the lower femoral epiphysis. The following experiments have therefore been carried out.
(1) In this case it was decided to try and avoid opening the joint and to drill from the inner side of the femur and to coagulate an area of bone very slightly with a fine cautery point. X-ray examination showed that the bore hole was placed rather too far from the subchondral tissue and it was therefore considered advisable to open the joint and perform the subsequent experiments under full vision.

(2) The right knee joint of a growing dog was opened by a straight incision medial to the patella. It had been my intention to perform an experiment on the subchondral bone but as the vessels in the posterior cruciate ligament were well shown they were ligated and divided.

(3 & 4) The knee joints were opened similarly in a growing dog and in an adult dog. In both cases a hole was bored through the medial condyle so that it lay about 2 mm. above the subchondral tissue. With the object of producing irritation and hyperaemia, powdered silica was introduced into the bore hole and the hole was sealed over with Horsley's wax.

(5) An exactly similar experiment to the above was carried out but no silica was introduced into the bore hole so that this animal (a growing dog) could
serve as a control.

(6) In a growing dog a similar bed was prepared in the medial femoral condyle but charcoal was introduced instead of silica.

Owing to the delay in the granting of the certificates to operate on dogs and keep them alive, it is too early yet to give the results of the experimental work which is being continued. Dog 1 was killed and no change was found, the bore hole having completely healed. This would point to the conclusion that Tammann obtained his results because of the destruction of the bone and cartilage.

X-ray examination of the other dogs shows no definite sign of osteochondritis dissecans a week after the operation, but the first silica experiment shows changes in the medial femoral condyle which are regarded as hopeful. It is interesting that the bore holes in the silica experiments appear larger on X-ray examination than the others and, as the same drill was employed, this must be due to hyperaemia. In spite of the opening of the knee joints all the dogs were able to walk normally on the day following operation and all wounds healed by first intention.
The results following the reduction of a sample from a series of appearances showed:

The reductions in the bone were observed in the observed face.

As possible, it is possible that the bone, and that the vascular and decalcified.

In great detail, Infirmary and X-ray, full use was made of work in Mercer, the results have generated from the...
The progress of this series of animals will be followed by weekly X-ray examination and if results of a satisfactory nature are obtained, a further series will be operated on. No X-ray change was apparent in the case of ligature of the vessels.

The X-ray of dog 3, after operation, is reproduced in Fig. 98, where decalcification can be seen in the medial condyle and a suggestive opacity can be observed overlying the medial femoral articular surface.

As far as I can ascertain, this is the only possible method of producing a localised hyperaemia in bone and I hope to be able to prove in this way that the cause of osteochondritis dissecans is a vascular lesion comprising hyperaemia and subsequent decalcification.

In conclusion, I should like to acknowledge the great debt I owe to the Senior Surgeons of the Royal Infirmary for permission to use their case records and X-rays; to Professor John Fraser for giving me full use of his private cases and for allowing me to work in the Clinical Surgery laboratory. Mr Walter Mercer, Mr T. McW. Millar and Mr D. S. Middleton have generously placed their cases and the specimens from them at my disposal. I must also express my
thanks to Mr D. B. Smith of the Clinical Surgery Department for his patience and skill in the reproduction of X-rays and the preparation of the microphotographs.

SUMMARY and CONCLUSIONS.

(1) A series of thirty cases of osteochondritis dissecans is reviewed.

(2) The etiology of osteochondritis dissecans and Köhler's disease of the second metatarsal head is the same.

(3) The factors in the production of the loose bodies are -

   (a) repeated trauma causing

   (b) hyperaemia and decalcification in the cancellous bone underlying the subchondral layer of bone.

(4) Healing may readily occur and conservative treatment should be advised instead of immediate operation, in early cases.
I.

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