THE ROLE OF CAPSULAR CHANGES IN OSTEOARTHRITIS OF THE HIP JOINT

G. C. LLOYD-ROBERTS, LONDON, ENGLAND*

From the Institute of Orthopaedics, Royal National Orthopaedic Hospital

Osteoarthritis is characterised by degenerative changes in the articular cartilage and the bones of the affected joints. These abnormalities are apparent in radiographs and museum specimens. The surgeon who operates upon these joints is, in addition, aware of the striking changes that occur in the capsular and synovial tissues in advanced cases. In this paper I will describe these soft-tissue changes, and then discuss their etiology and their influence upon the clinical manifestations and course of osteoarthritis of the hip. The hip has been chosen because osteoarthritis in this joint frequently demands operation employing exposures that allow inspection and biopsy of any part of the joint.

MATERIAL

The following observations are based upon an operative and histological study of twenty-five osteoarthritic hips subjected to arthroplasty or arthrodesis. In no case did infective, rheumatoid or ischaemic factors determine the onset or complicate the condition. The youngest patient was aged forty-nine and the oldest seventy-three.

The structure and functional anatomy of the normal hip joint were studied by dissection and microscopic examination in seven fresh cadavers, from subjects ranging in age from birth to sixty-five.

THE NATURE AND CAUSE OF THE PATHOLOGICAL CHANGES IN THE SYNOVIAL MEMBRANE AND CAPSULE

The entire synovial membrane of an osteoarthritic hip is congested and unduly villous for the patient’s age, especially in certain areas. The lowest part of the joint is most involved, the villi being large and grouped in bunches upon a thickened base. Where the articular cartilage has suffered most a ring of hyperplastic villi may also encircle the head. The villi in the more congested areas are frequently engorged and appear blue, whereas in other, less vascular, areas they may be discrete, firm and white. Intra-articular adhesions are often conspicuous but may easily be overlooked if the external layer of the joint capsule is forcibly separated by a bone lever from the synovial layer investing the femoral neck before the areas of the capsular reflection are inspected, for it is within these synovial pockets that they occur most often, and also between the margin of the head and the surrounding lining of the articular capsule. In patients in whom movement is almost lost, and in whom deformity rather than pain predominates, these adhesions may fill the joint to such an extent that some difficulty may be encountered in defining its cavity, the capsule surrounding the neck like a tight collar. More typically, however, the capsule is thickened and shortened and lacks its normal pliability. Sometimes fibrosis spreads to the adjacent muscles, especially the short rotators, so that these adhere to the outer surface of the capsule and can be reflected only by sharp dissection. Calcification is occasionally present, more particularly on the upper surface of the capsule.

Histological examination of congested areas of synovial tissue, selected at operation, discloses hyperplasia with formation of villi and multiplication of surface cells. The presence

* Nuffield Fellow in Orthopaedic Surgery; Senior Registrar, Orthopaedic Department, St George’s Hospital; Clinical Research Assistant, Institute of Orthopaedics.
Osteoarthritic hip. Synovial membrane and capsule from inferior part of joint. Note the diffuse fibrosis which obscures the junction between synovial membrane and capsule ($\times 64$).

Contracted capsule. Synovial membrane and capsule from behind elbow joint. The elbow was fixed in extension after a brachial plexus injury nine months before. It could not be flexed until the capsule was divided. The histological appearance is normal ($\times 64$).
of haemosiderin in some specimens, below the surface cells of the villi and in collections in the subsynovial tissue, suggests that haemorrhage may occur into the joint cavity. Perivascular lymphoid aggregations are commonly present and should not be regarded as a rheumatoid manifestation. Metaplasia leading to the formation of cartilage or bone was seen in only two of the twenty-five cases. The pale pedunculated villi, so frequently seen at operation and superficially resembling cartilage, were found to consist merely of connective tissue—less vascular and more fibrotic than usual, and covered by a single layer of synovial cells.

A further striking abnormality is a progressive fibrosis of both the synovial membrane and the capsule. In advanced cases the joint lining becomes almost completely replaced by this scar tissue. Figure 1 illustrates an example of advanced fibrosis which has obliterated the normally conspicuous boundary between subsynovial tissue and joint capsule shown in Figure 2.

In addition the synovial membrane contains debris which appears to be related to the destruction of the cartilage and bone of the degenerated articular surfaces. This extraneous material is found either on the surface or just beneath the lining cells. Histologically it resembles bone or cartilage. The cartilaginous fragments, however, do not show the staining reactions of normal cartilage but those of the degenerated cartilage of the joint surface from which they have presumably been separated. Figure 3 illustrates the articular cartilage from a case of moderately advanced osteoarthritis. The projections of degenerate cartilage have but a tenuous connection with the deeper layers and are obviously liable to become dislodged. Later the bone becomes vulnerable to the same process of attrition.

Once free in the joint cavity these fragments must either persist as loose bodies or adhere to the synovial membrane, for the synovial fluid cannot dissolve them. Experimental studies of other particles introduced into a joint show that they are engulfed by the synovial membrane and are dealt with subsequently in a manner dependent upon their size and nature. Particles larger than 0.1 μ cannot be carried away unchanged from the membrane (Adkins and Davies 1940); they either remain permanently, if non-digestible, or they are removed by phagocytosis. The particles of cartilage and bone are too large to be carried away unchanged from beneath the synovial lining but they may undergo digestion in situ (Figs. 4 to 8).

In sections stained by haematoxylin and eosin, the cartilage fragments differ from normal articular cartilage in that there is no sign of the familiar blue metachromatic reaction and the chondrocytes are scanty and atypical. To recognise the relationship it must be realised that these are fragments of degenerated cartilage whose structure and staining reactions are abnormal even before they are dislodged, and which undergo still further changes during their subsequent digestion by the synovial membrane.

The main features can, however, be seen in these preparations. The fragments of cartilage lie on or beneath the surface layer and do not appear to pass deeper during their absorption. They have an eosinophilic reaction which becomes less intense as disintegration proceeds and which is accompanied by an increase in the visible fibrillar network (Fig. 4). Atypical cartilage cells or their remains may sometimes be seen in the larger and less disorganised specimens (Fig. 5). Giant cells are occasionally found nearby. The fragments are clearly demarcated from the surrounding synovial tissue (Fig. 6). Their partial dislocation from their enclosing cells during the process of histological preparation often emphasises this point (Fig. 4). Typically, however, they are small and lie with their long axis parallel to the synovial surface where they can be readily overlooked (Fig. 7).

The cartilaginous nature of some of these fragments is indicated by the slight degree of metachromasia that can be demonstrated by means of the Eosin Azur technique and by a positive periodic acid Schiff reaction. The staining reaction of the cartilaginous debris resembles that of degenerate cartilage from the articular surfaces of osteoarthritic joints rather than that of normal articular cartilage. The fragments show the same reactions with collagen stains as does degenerate articular cartilage in osteoarthritis. Special stains for fibrin prevent confusion between the fragments and the masses of fibrin frequently seen in osteoarthritic joints. Furthermore, examination of the fibrillar structure of these particles by the polarisation method of Price (1952) shows the collagen fibrils to be arranged in a manner similar to that seen in degenerate articular cartilage.
Articular cartilage from osteoarthritic hip. Note the fragmentation and loss of metachromatic staining in the superficial part of the articular cartilage (× 100).

Osteoarthritic hip. A group of cartilage fragments lying beneath the synovial surface. Note their fibrillar appearance and the partial dislocation which has occurred during preparation (× 280).
The role of capsular changes in osteoarthritis of the hip joint

**Fig. 5**
Osteoarthritic hip. An exceptionally large fragment of cartilage lying below the synovial surface. The remains of cartilage cell nuclei can be seen (× 85).

**Fig. 6**
Osteoarthritic hip. Large cartilage fragment with small bone spicule lying superficial to one edge (× 200).
Osteoarthritic hip. This field shows the typical histological appearance of the cartilage fragments. The space between the main fragments probably contained a fragment which was dislodged during preparation (× 280).

Osteoarthritic hip. Two bone fragments lie a short distance below the synovial surface. One of them contains a lacuna (× 350).
Bony fragments are more easily recognised as such. They are usually smaller than the cartilaginous fragments and lie at a short distance from the surface (Fig. 8). Unless decalcified they appear in sections as dark brown objects exhibiting optical activity when viewed with polarised light. They become black when stained by the Von Kossa method for the demonstration of bone salts.

I believe that the evidence that has been presented allows us to assume, with some confidence, that these fragments are composed of abnormal articular cartilage and bone. It has been assumed that the source of this debris is the articulating surfaces, whereas it might be suggested that it arises within the synovial membrane by metaplasia, a feature of osteoarthritis which has been frequently described. But metaplastic cartilage and bone formations differ in almost every respect from the appearance of the debris that I have described, for metaplasia is a vital process in which the connective tissue of the centre of the villus merges imperceptibly with an area of either metachromatic fibrocartilage, calcified cartilage or bone (Fig. 9), whereas the debris occurs just below the surface, is often clearly demarcated from the surrounding tissue and responds poorly to metachromatic stains (Table I). This distinction was emphasised by Horwitz (1948), who described similar bone

Fig. 9
Synovial membrane in Charcot’s osteoarthropathy. There is diffuse metaplasia of the synovial connective tissue with formation of cartilage and calcified cartilage within the deeper layers of the synovial membrane (x 75).
and cartilage fragments in the soft tissues of neuropathic knee joints in addition to areas of metaplasia. He pointed out that these fragments also occurred in two out of a control group of twelve osteoarthritic joints. We have had the opportunity to study one syphilitic neuropathy of the hip joint in which only minor bone changes had occurred. In addition to the marked amount of joint debris present the synovial tissues in this case also showed advanced cartilaginous metaplasia (Fig. 9). This contrasts with our findings in twenty-five cases of uncomplicated osteoarthritis in which metaplasia was found in only two. It seems possible that the emphasis which has been laid on this change in many accounts of osteoarthritis may therefore have arisen from a study of Charcot’s joints (Nichols and Richardson 1909).

### TABLE 1

**Contrasting Features of Synovial Metaplasia and Articular Debris within the Synovial Membrane**

<table>
<thead>
<tr>
<th>Position</th>
<th>Metaplasia</th>
<th>Articular debris</th>
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<tbody>
<tr>
<td></td>
<td>Within the central connective tissue of the synovial membrane</td>
<td>Upon or just below the surface epithelium</td>
</tr>
<tr>
<td>Relationship to surrounding tissue</td>
<td>Cartilage, calcified cartilage and bone merges with connective tissue</td>
<td>Clearly demarcated from surroundings, often dislocated during histological preparation</td>
</tr>
<tr>
<td>Staining reactions</td>
<td>Metachromatic staining reactions as in normal cartilage</td>
<td>Only a slight degree of metachromatic reaction (similar to degenerate cartilage)</td>
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Histological examination of the twenty-five cases that form the basis of this study has disclosed debris in twenty-three. In most cases the debris contained both cartilage and bone, but in five only bone fragments were found. In some they were visible in the first histological section examined, but in others multiple sections were required to demonstrate their presence. Both the cartilaginous and bony fragments are found more commonly where there is hyperplasia of the surface cells and villous proliferation of the synovial membrane. For this purpose it is important to take the specimens from areas showing the greatest amount of synovial congestion and hyperplasia, which as already mentioned are usually to be found below the neck of the femur. Synovial membrane from this area, behind more often than in front of the transverse plane, contained the greatest amount of debris when several specimens from different parts of the joint cavity were examined. Gravity may be the factor which directs the fragments to the synovial recesses of this part of the joint.

I was unable to demonstrate these particles in two of the joints examined. In one of these the capsule and synovial layers were so welded together by scar tissue that little of the synovial lining could be recognised with certainty. No explanation can be offered to account for the apparent absence of debris in the other case.

### The Relationship between Joint Debris and Capsular Fibrosis

It must now be considered whether a relationship exists between joint debris and capsular fibrosis. Observation at operation suggests that there is a relationship between the degree of joint destruction and the extent of the capsular change. This impression accords with the descriptions of the morbid anatomy of the disease; thickening of the capsule is associated with relatively advanced cartilage degeneration (Nichols and Richardson 1909, Collins 1949). On the other hand we have observed synovial hyperplasia in the joint of a young man showing evidence of very early osteoarthritis due to coxa vara. Synovial hyperplasia occurs readily in response to blood in the synovial cavity, and Key (1929, 1932) has shown, experimentally and in a study of haemophilic arthritis, that sustained or repeated synovial hyperplasia...
**Fig. 10**
Synovial membrane of rabbit four weeks after injection of cartilage fragments. Two of the fragments are seen just below the surface layer, the cells of which show some hyperplasia (× 225).

**Fig. 11**
Synovial membrane of rabbit four weeks after injection of cartilage fragments. This field is adjacent to that shown in Figure 10. Note the generalised synovial hyperplasia and the fibroblastic response in the deeper layers (× 112).
leads to subsynovial fibrosis. The presence of haemosiderin, however, is not an outstanding feature of osteoarthritis and it seems unlikely that all the villous proliferation and fibrosis could be a reaction to haemorrhage. Similar changes have, however, been demonstrated by Key after the injection of Indian ink. Hultén and Gellerstedt (1940) were the first to observe that a cartilage-like substance appeared beneath the synovial surface in osteoarthritis. They introduced fragmentated articular cartilage into the joints of animals and demonstrated a progressive fibrosis in response to repeated injections. Their experiments also showed that fresh autogenous and homogenous cartilage fragments were engulfed by the synovial membrane and remained beneath its surface, undergoing disintegration and digestion within a few days.

In collaboration with Dr H. A. Sissons, I have undertaken a similar experimental study to confirm this work and to investigate the fate of fresh homogenous cartilaginous and bony particles injected into the knee joints of rabbits. A single injection of finely ground material was made into the knee joints of eight young rabbits—bone on the one side and cartilage on the other. There was no untoward reaction or sepsis. The results (in contrast to those of Hultén and Gellerstedt) indicated that a fibroblastic reaction occurs in response to only one injection, and that debris and consequent synovial hyperplasia persist for at least four weeks. Figures 10 and 11 show specimens removed from a rabbit killed four weeks after the injection of cartilage particles. Figure 10 shows two fragments lying in a position like that observed in the human material, and Figure 11 is an adjacent low-power field which demonstrates the synovial hyperplasia and the fibrosis in the subsynovial layer. A similar result was obtained from the injection of bony particles.

The rate of removal of debris from a joint must be further considered. It is doubtful whether the debris disappears rapidly in osteoarthritis, for the recognition of the fragments would then be extremely unlikely. Their presence suggests either that the synovial membrane becomes progressively less efficient as a scavenger, or that degenerate articular cartilage, and especially the spicules of bone, are more resistant to the process than the healthy articular cartilage used in the experiments. Persistence of the debris would serve to sustain the synovial hyperplasia and thereby to increase the consequent fibrosis.

In summary, it is concluded—on the basis of observations at operation, microscopic study and animal experiment—that joint debris not only accompanies capsular fibrosis but also plays a part in its production.

THE RELATIONSHIP OF THE SYNDOVL AND CAPSULAR CHANGES TO THE CLINICAL FEATURES

Surgeons have long suspected that the capsule contributes to both the symptoms and the signs of osteoarthritis and have consequently excised it, in whole or part, in the course of operations upon the hip joint. Some, especially Gade (1947), have credited it with a dominant role. An attempt will be made here to correlate the clinical manifestations of pain, deformity and selective loss of movement with the observed pathological abnormalities in the joint capsule.

It has been found that three factors—deformed bone, fibrotic muscle and contracted capsule—all play a part in maintaining such deformity as persists when the patient is examined under general anaesthesia.

The bony element is usually considered to be late and relatively unimportant, being dependent upon contact between large marginal osteophytes and on gross changes of contour such as may occur when osteoarthritis follows a displaced capital epiphysis. This can be demonstrated readily at operation by moving the hip joint under direct vision after removal or incision of the capsule. Then the main limiting structure is found to be shortening of muscle, a shortening due to the contracture which may occur in any muscle that actively determines the direction of long-standing deformity in any joint (Steindler 1950). This contracture is, however, the secondary effect rather than the cause of the other deforming factors. Only if
the deformity is severe or long-standing will the adductor muscles prove to be tight on abduction under anaesthesia.

The effect of capsular shortening upon hip joint movement can be understood only when the functional anatomy of the capsule is studied by dissection of fresh cadavers. These dissections show that all parts of the capsule are tight in the position of full extension associated with some abduction and slight medial rotation. This fact was described by Walmsley (1928), who also pointed out that in this position there is greatest bony contact and stability between femur and pelvis. It is the position adopted when weight is carried on one leg, and it allows tilting of the pelvis and locking of the knee to occur readily. Walmsley also emphasised that flexion from this point is accompanied by progressive relaxation of the capsule. Medial rotation is brought to a halt by tension upon the posterior and inferior capsule, and abduction by the inferior capsule alone. Adduction and lateral rotation tighten the superior and anterior parts of the capsule respectively. It is also evident that for unhampered movement all parts of the capsule must move freely—this free mobility is particularly necessary posteriorly where the capsule is merely a thin membranous layer in contact with its reflection covering the femoral neck. We have confirmed these statements by dissections of fresh cadaveric hip joints.

Shortening of any part of the capsule will cause a loss of full extension because in this position all parts of the capsule should be tight. Shortening of any part will therefore check the full development of tension elsewhere and in consequence prevent extension. Should this shortening develop in the inferior part of the joint (which deals with the greatest amount of joint debris and which in consequence undergoes the earliest fibrotic changes) a loss of medial rotation and wide abduction will accompany or follow this loss of extension. Intra-articular adhesions in this area will have the same consequences. Flexion from the horizontal plane, during which the capsule is progressively relaxed, will remain free. Continuation of this restricting factor will clearly produce an actual deformity in the opposite direction—namely, flexion, adduction and lateral rotation. Limitation of adduction and lateral rotation will not occur until the capsular changes have spread to involve the superior and anterior aspects of the joint.

This early loss of extension may have an important influence upon the progress of the disease. As already mentioned, Walmsley emphasised that the surfaces of the femur and acetabulum were in greatest contact in this weight-bearing position. With loss of extension the area of contact will diminish progressively as the deformity increases. Consequently the body weight will be carried through a smaller area of articular cartilage, this will wear out more quickly and the accelerated detachment of debris will speed capsular fibrosis.

This description of the functional effects of capsular shortening has been verified at operation in several advanced cases. An exposure of the hip from behind, with vertical division of the grossly thickened posterior and inferior capsule, may be found to release the movements of medial rotation and abduction within the limits set by the contracted muscles. An anterior approach with transverse division of the lower part of the anterior ligament will often overcome the greater part of a flexion deformity. This approach combined with tenotomies also allows an assessment of the amount of the deformity that is caused by contracture of the flexor and adductor muscles. It should be emphasised, however, that a full range in any direction could seldom be obtained by selective capsular incision alone. In most, capsular changes had become so generalised when operation was performed that wide excision was required before a range of movement approaching the normal could be restored.

In this discussion the capsular abnormality has been ascribed to the progressive fibrosis secondary to sustained synovial hyperplasia and it has been regarded as a contributory cause of deformity. It is possible, however, that the capsular abnormality has an extra-articular cause, such as muscle spasm. Although operations performed for the correction of
fixed deformity caused by spasm or muscle imbalance may show the capsule to be shortened on the convex side, it is not as thickened and fibrotic as the capsule in osteoarthritis (Fig. 2). In addition there is neither a synovial abnormality in these joints nor a tendency for other areas of the capsule to become involved. Lastly, as Collins (1949) points out, intra-articular adhesions never form secondarily to deformities from muscle imbalance or paralytic disorders, whereas they are common in osteoarthritis. It may be concluded that, while periarticular factors may encourage shortening of the joint capsule in osteoarthritis, they do not bring about the generalised fibrotic thickening and synovial changes that occur and which precede contracture in the surrounding muscles.

The dominant deforming factor in most cases is muscle spasm. Spasm is a voluntary and involuntary response to movements which if continued would be painful, so that both pain and spasm may have a common origin. The intimate innervation of articular structures was fully discussed by Gardner (1950). He concluded that the joint capsule is richly supplied by somatic and autonomic fibres. Although nerve plexuses are seen in the synovial membrane their nature is obscure. Kellgren and Samuel (1950), studying pain sensibility in the human knee joint, commented upon the sensitivity of the capsule, which contrasted strikingly with the scattered areas of pain appreciation in the synovial membrane and the insensitivity of articular cartilage. During an operation upon a knee joint under local anaesthesia the writer found traction upon the capsule a particularly painful manoeuvre.

It has already been suggested that shortened and thickened capsule becomes tense prematurely and so reduces the range of hip joint movement. It is now suggested that if movement continues beyond this limit imposed by the shrunken capsule, and if at the same time body weight is added to the movement, a considerable traction force is likely to fall upon this abnormal ligament, thereby producing pain and protective muscle spasm. Intra-articular adhesions will tend to reinforce the effect of capsular fibrosis in a similar way. Intra-articular and intracapsular novocain injections will often temporarily reduce both this pain and spasm.

Considering once again the lower part of the joint which is supplied mainly by the obturator nerve, but also by the nerve to quadratus femoris behind and to pectineus in front (Gardner 1948), I find in this hypothesis of traction pain a logical explanation for the occurrence of reflex spasticity in the muscles supplied by these nerves. The deformity produced by this spasm is furthermore in the same direction as that which I have shown to follow shortening of the capsule in the same area of the hip joint.

Support for this interpretation of the origin of reflex spasm is provided by the familiar clinical test for adductor spasm. An abduction or medial rotation strain is resisted by the posteroinferior capsule of the joint. If this part of the capsule is contracted the tension in this area produced by these movements may give rise to pain and spasm, whereas passive flexion loosens the capsule and may often be accomplished without spasm. Adduction and lateral rotation do not tend to stretch the inferior part of the capsule and are consequently less likely to induce pain and spasm.

A RADILOGICAL SIGN

Further evidence of capsular shortening is provided by a radiological sign. New bone formation visible below the femoral neck was seen in the radiographs of thirty-three out of seventy-three consecutive patients with osteoarthritis of unknown origin subjected to operation. This sign was fully described by Wiberg (1939) in congenital subluxation of the hip. He said that new bone formation occurred behind the lower margin of the femoral neck, an observation which my operative and radiological investigations confirm. He regarded it as a buttress formed in response to abnormal stress. Although in idiopathic osteoarthritis it occurs more commonly when subluxation is present, it may occur when subluxation is minimal.
Figure 12—Idiopathic osteoarthritis of the hip. A considerable amount of new bone is present below the neck of the femur in spite of only slight subluxation. There is calcification in the capsule above the neck.

Figure 13—Idiopathic osteoarthritis of the hip. There is new bone below the neck of the femur without subluxation of the hip joint.

Figure 14—Condition of hip five months after open reduction for traumatic dislocation. The new bone visible below the neck of the femur appears to be on the deep surface of raised periosteum. There had been no weight bearing.

Figure 15—Osteoarthritis of the hip. The new bone seen below the neck of the femur has appeared within five months of an exploratory operation and partial capsulectomy.
or absent (Figs. 12 and 13). I believe that this bone is subperiostal and is caused by elevation of the periosteum away from the neck of the femur by a traction force transmitted through the postero-inferior capsular reflection (retinaculum). In support of this view I submit, first, that capsular tension is most likely to develop here when subluxation occurs and, secondly, that similar changes may follow traumatic dislocation (Fig. 14) and exploratory operations upon the hip when the capsule has been stretched by bone levers (Fig. 15). Furthermore, of the thirty-three patients presenting this feature twenty-six had an adduction deformity, or had lost power to abduct beyond the neutral position—a sign which it is suggested indicates marked capsular shortening in the inferior part of the joint capsule.

**DISCUSSION**

An attempt has been made to correlate the symptoms and signs of osteoarthritis with the pathological changes that have been found in joint capsules removed from the hip joints of patients sufficiently disabled to require operative treatment. It is well recognised, however, that many patients in whom there is radiological evidence of advanced osteoarthritis, suffer surprisingly little pain. They present an important clinical problem and have been questioned and examined with particular care.

With advanced radiological changes movement is always restricted, but pain may be absent. Its source must therefore be in structures scarcely manifest in radiographs: I suspect stretching of the shortened capsule, as occurs when an attempt is made to force movement in such a joint. I suggest that the reason why a McMurray’s osteotomy may relieve pain is that the upper fragment takes up the position of deformity which the capsule dictates and so protects it from much of the strain to which it was previously exposed. Similarly replacement of the femoral head by a prosthesis may shorten the distance between the fixed points of the capsule and thus relieve pain, even if the capsule is not excised. Such a prosthesis will also remove the main source of joint debris.

Rest is known to relieve the pain of most osteoarthritic joints. It seemed desirable therefore to enquire into the amount that patients, with radiological evidence of marked osteoarthritis but with little pain, demanded of their hip joints. By this means I have found an explanation for the absence of pain in many patients with osteoarthritis. On careful questioning, most mentioned a reduction of their activities to conform to the standards set by their hip joints. In some this was a conscious restriction; in others it occurred from advancing age or was imposed by some coincidental disease such as cardio-vascular failure. Patients suffering from osteoarthritis of both hips may have no pain in one side because the disability caused by its fellow has restricted their activity greatly. A pain-relieving operation on the painful hip often leads to pain in the previously symptomless hip when activity is increased.

Some patients complain of stiffness only. Most of these have had pain in the past which has been forgotten or is now regarded as unimportant. Among them some have noticed their disability after a period in bed for some other condition. Their deformity is often severe, and movement reduced to a few degrees of flexion only. Lastly there are those whose pain is referred to the knee or low back (Law 1952), and those who make light of their troubles.

In considering the symptoms of osteoarthritis it is also important to appreciate that the degenerative changes that occur in the articular cartilage and subchondral bone with ageing are identical with those in osteoarthritis. Bennett et al. (1942) have studied the changes in previously symptomless knee joints obtained at necropsy. In subjects of between eighty and ninety years old they found marked degenerative lesions which were very like those in younger individuals operated upon for osteoarthritis.

It has already been suggested that restriction of activity or stiffness tolerable at eighty may be less readily accepted at the age of fifty, but it is possible that the speed with which
debris is cast into the joint cavity influences the symptoms arising in the joint. Bennett et al., although aware of the work of Hultén and Gellerstedt, did not describe debris within the synovial membrane—unlike Horwitz, who found these fragments twice in clinically osteoarthritic knees during his investigation of Charcot’s joints. These observations, in addition to those presented in this communication, suggest that joint debris may become apparent only when it becomes dislodged at an abnormal rate or in abnormal quantity. The scantier and slower physiological loss of cartilage associated with ageing may be digested by the synovial membrane, as it occurs, within the recesses of capsular reflection. Consequently it may escape recognition; and it will induce capsular fibrosis at a much lower rate. Under these circumstances a balance may be set between increasing capsular changes on the one hand and decreasing activity on the other so that a joint, though degenerate by radiological and pathological standards, may continue to serve well. Anything (such as dysplasia of the hip joint) which accelerates the formation of joint debris will tend to hasten the onset of capsular shortening by the mechanism that has been described. In this way the patient may present at an early age the clinical features of severe osteoarthritis, but a radiograph showing only localised diminution of joint space and subchondral sclerosis.

In conclusion, it is submitted that this interpretation of the etiology and effect of capsular fibrosis may have an application in osteoarthritis of other joints. In hallux rigidus joint debris and synovial hyperplasia have been found within the reflection beneath the head of the metatarsal bone. Any shortening or adhesion of the capsule in this position would cause a loss of dorsiflexion, though plantar flexion would remain unaffected.

**SUMMARY AND CONCLUSIONS**

1. The synovial membrane and capsule in osteoarthritis of the hip have been studied in twenty-five cases. Dissections have been made on fresh cadavers to establish the normal structure and function of these tissues at different ages.
2. Fragments of bone and cartilage were found beneath the synovial surface in twenty-three cases of the twenty-five cases of osteoarthritis.
3. The source of these fragments is the degenerate articular surfaces.
4. The fibrosis of the synovial membrane and capsule follows the synovial hyperplasia which accompanies the phagocytosis of these fragments.
5. A similar histological picture has been produced by injecting fragmented cartilage into the knee joints of rabbits. The injected fragments are found beneath the surface, and synovial hyperplasia is followed by subsynovial fibrosis.
6. The greatest amount of this joint debris is found in the lowest part of the joint cavity.
7. The joint capsule is particularly sensitive to traction.
8. All parts of the capsule are tight in extension, which is the weight-bearing position.
9. Fibrotic shortening of the capsule in the lowest part of the joint cavity explains many of the symptoms and signs of the disease: pain is caused by an attempt to stretch the capsule; muscle spasm occurs in the muscles supplied by the sensory nerves of this part of the capsule; extension, medial rotation and abduction, which tighten this area, are lost first; progressive shortening causes deformity in the opposite direction, namely flexion, lateral rotation and adduction; the loss of extension causes a more rapid wearing of articular cartilage on weight bearing; subperiosteal new bone is formed on the under-surface of the neck of the femur.
10. The symptomatology is discussed.

I wish to thank Dr H. A. Sissons for his collaboration and valuable assistance in the experimental and pathological aspects of this study. Mr H. J. Seddon for his helpful criticism and assistance throughout, and Mr A. T. Fripp and Mr K. I. Nissen for their interest and the provision of pathological material. Mr B. H. Burns and Mr R. H. Young have very kindly allowed me to examine and operate on some of their patients and have given me considerable help and encouragement.
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