HYPERPLASIA AND METAPLASIA IN SYNOVIAL MEMBRANE

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by

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(1) Introduction

I HAVE ENTITLED this lecture " Hyperplasia and Metaplasia in Synovial Membrane " because I wish to consider not only the changes which may occur in joints themselves but the formation of synovial membrane from other tissues. I shall refer especially to that uncommon but striking condition : Pigmented Villonodular Synovitis. I do not propose to deal with the changes which occur in arthritis nor with frankly neoplastic conditions.

(2) Anatomy and Physiology of Joints

In order to have a clear understanding of synovial membrane it is necessary to refer to the development of diarthrodial joints. At about the eighth week of embryonic life the definitive joint develops from the interzonal mesoderm, which is that tissue lying between the axial bony elements of the future skeleton. A number of lacunae appear which coalesce to form a cavity. The innermost lining of this cavity forms the synovial membrane and articular cartilage, while the outer layer forms the joint capsule and periosteum.

We should therefore regard synovial membrane as a mesodermal sleeve which, enclosing a large tissue space, the joint, envelops intra-articular structures but does not cover the articular cartilage. In man, the joint lining is usually complete although most joints communicate with neighbouring bursae. In animals the joints may communicate quite freely with tissue spaces in the limbs.

The membrane is highly vascular, expecially at its bony attachments, its surface is thrown into folds, and villi are normally present in certain areas. These villi may contain a central capillary loop, but the lymph plexus which runs superficially throughout the membrane in a rectangular pattern does not enter the villi. The synovial surface is covered by one or more layers of deeply staining cells which lie directly upon a corium of loose connective tissue, supported by elastic laminae. The deeper layers of the membrane contain minute lymph follicles, mast cells clustered around the blood vessels (Davies, 1943) and tissue histiocytes normally present in considerable numbers.

Three types of membrane have been described (Key, 1926) : (*a*) *areolar,* which represents the majority of the membrane, (*b*) *fibrous,* where the surface cells lie directly upon a fibrous sheath and surround tendons and ligaments or line bursae and (*c*) *adipose,* where movement of the membrane is required.

The synovial surface cell merits a more detailed examination. It is clearly of mesenchymal origin and Vaubel (1933) in his studies of tissue cultures of joints came to the conclusion that the synovial cell was more closely related to the cartilage and bone cell than to the undifferentiated fibrocyte. It cannot be considered to form an endothelium since it does not rest upon a basement membrane. It is highly pleomorphic and will assume pavement, polygonal or even a high columnar shape on appropriate stimulation. It has considerable powers of phagocytosis. It is capable of amitotic division into syncytial or multinuclear forms : it produces a fibrinolytic ferment. It possesses the property of excluding from the joint it lines any foreign material present in that joint by simply adhering to it and growing over its surface. In this way blood clot is removed from the joint cavity, loose bodies become sessile (Timbrell Fisher, 1921) and adhesions formed on the surface of the membrane first become covered by synovial cells and then bound to its surface. Under certain conditions the synovial surface is able to adhere to itself and coalesce to form complicated lace-work patterns and apparent cleft formation.

There is yet a further property of the synovial membrane which has interesting pathological implications. It is the ability to pass into the joint, or to have deposited within it, abnormal constituents of the blood stream while the deeper layers remain relatively unaffected. This is in direct contrast to the behaviour of the membrane after abnormal substances have been removed from within the joint, when the surface cells may remain clear while the deeper layers are loaded with the absorbed material. I refer to three rare conditions :

1. *Haemochromatosis.* Collins (1951) has recently shown that the haemosiderin is deposited at the surface of the membrane.

2. *Calcification of synovial membrane.* Here the joint or bursa is accurately outlined on a radiograph by the deposit.

(This was seen in a case of a child of 12 years dying from renal osteodystrophy in which a combination of renal failure and parathyroid hyperplasia caused the resorption of calcium salts from the bones and their deposit in the soft tissues, mainly the joints, bursae and blood vessels.)

3. *Synovial amyloidosis.* There is a similar outlining and distension of joints and bursae ; for example the shoulder joint in a case of multiple myelomatosis.

The synovial fluid is a product of the synovial cell but it cannot be regarded as a true secretion. Human joints normally contain only a small quantity of synovial fluid with 500-2,000 white cells per cu. mm. and these are mainly monocytes. The fluid acts as a lubricant of the joint surfaces and nourishes the central avascular areas of the articular cartilage. It consists of two moieties : a blood dialysate containing a little albumen ; and joint mucin, a complex protein, one constituent of which is hyaluronic acid and polysaccharide.

The origin of joint mucin has long been disputed. Kling (1938) and others maintain that there are definite areas of the membrane for its elaboration, but this has not been confirmed by other workers (Davies,

1943). However, in my studies of hyperplastic joints I have come firmly to the conclusion that there are certain especially active regions of the membrane, namely at its bony attachments and, to a lesser extent, overlying the fat pads.

Vaubel (1933) states : " One may regard the synovial fluid as a ground substance of synovial tissue analagous to the inter-cellular substance of cartilage which has become solid by the imbibition of chondroitin-sulphuric acid, or to that of bone which has become impregnated with lime salts." If we are to accept this remarkable statement we must look upon a joint simply as a biochemical interruption in the skeleton.

(3) The Response to Injury

Synovial membrane demonstrates only moderate powers of repair by its response to injury, and deficiencies made in its surface may be the site of dense adhesions. After excision, as in synovectomy, if movement has been successfully preserved, regeneration by metaplasia of underlying connective tissue occurs to form a joint cavity with a smooth surface lining and containing synovial fluid. Key (1925) excised the knee joints of rabbits and studied the development of a joint lining from ingrowing fibroblasts. He showed conclusively that a very complete restoration was achieved. Although this does not occur to the same extent in human joints, sufficient modification does take place to make practicable the operation of arthroplasty. I have examined the lining membrane of hip joints in which vitallium cup arthroplasty had been performed some years previously. Sections show a condensation of fibrous tissue bundles running parallel to the surface with a concentration of modified fibroblasts at the free margin, the whole being finally smoothed off by a deposit of fibrin. Although nodules occur in places, villi do not appear to be reformed. No doubt in these cases the age of the patients influences the degree of tissue modification.

In attempting to form new joints surgically, too much attention may be paid to preparing the bone ends of the future articulation rather than to encouraging the formation of a lining capable of producing synovial fluid and preserving the new joint cavity. The conditions for the formation of a nearthrosis are :—

(*a*) Distraction of the bone ends.

(*b*) Prevention of new bone formation and subsequent ankylosis, possibly by interposing an inert material between the bone ends.

(*c*) Careful haemostasis so that a blood clot does not form and become organised.

(*d*) Constant movement at the site of the new joint.

(*e*) Freedom from pain and its accompanying muscle spasm.

This last condition is important, and in arthroplasty of the hip, efforts are made to excise all the capsule containing nerve fibres. However, an intact nerve supply is necessary for the maintenance of joint integrity. Its pathological implications are seen in the Charcot joint where there is an

abnormal range of movement and marked changes in the articular surfaces and synovial membrane. I have successfully constructed a new metacarpo-phalangeal joint in the presence of peripheral nerve injury only to see the joint surfaces become absorbed and distorted, as is often the case.

Similar changes may occur inadvertently in the establishment of a pseudarthrosis. These false joints are invariably unstable and are very subject to degenerative changes and " arthritis " occurs in them.

The nature and origin of the simple ganglion is still a subject of controversy. I am convinced that a ganglion arises, as the result of trauma, from a hydropic degeneration or mucoid metaplasia of connective tissue. King (1931) compared its development with the formation of joints in the embryo, which I have already outlined and adventitious bursae are formed over bony prominences in this way ; but the differentiation is not completed since the ganglion wall is composed of compressed laminae of connective tissue and there is no characteristic lining cell. It is quite unnecessary to postulate a herniation or new growth of synovial membrane from a joint or tendon sheath. The fact that such cysts arise most commonly in the neighbourhood of joints and tendons is merely coincidental and indicates that the fibrous tissue overlying these areas is that most subject to stress. Ganglia do in fact arise in other situations ; I have excised one growing in the periosteum of the tibia. The cyst which frequently arises in the lateral meniscus of the knee is a simple ganglion. Although a ganglion appears to be a single cyst it does in fact arise in a " ganglion bearing area." Microscopic examination will reveal many minute cysts in its walls. The frequency with which a ganglion " recurs " after excision demonstrates the error of painstaking dissection of the cyst. The correct surgery is the removal, if possible, of a block of tissue in the area of, and containing, the ganglion.

I mention the term " Baker's Cyst " only to condemn it. It nowadays implies a cystic swelling in the neighbourhood of the knee which has arisen from a synovial protrusion and has become " nipped off." I have recently reviewed (1949) Morant Baker's two original papers and in them he described a collection of swellings around joints of widely differing origin and pathology. Cystic swellings lined by synovial membrane and arising in the neighbourhood of joints have been shown conclusively to have formed from pre-existing bursae. (Wilson *et al.*, 1938.)

(4) Haemarthrosis

After haemorrhage has occurred into a joint the blood remains liquid, unless the joint has been severely damaged, and it will not clot after aspiration. It may be due to the fibrinolytic ferments elaborated in the synovial membrane, but I have not been able to demonstrate this property in vitro. There is an immediate outpouring of synovial fluid to dilute the blood, and it is then disposed of in three ways. Some of it seeps into the meshes of the synovium, some sedimented masses of cells adhere to the

synovial surface and become overgrown and excluded from the joint, while others are engulfed in the joint itself by amoeboid macrophages which enter the fluid in large numbers. The disintegrated corpuscles are broken into their principal constituents: lipoid, haemosiderin and bilirubin. The bilirubin diffuses back into the joint fluid, which is slowly absorbed, while the loaded phagocytes pass into the deeper layers of the membrane and their contents may eventually reach the circulation. The brown staining of the synovium may be seen if the joint is opened at a later operation and the iron may be readily demonstrated by special stains in granules or solution. Key (1929) noted the effect of repeated injections of blood into the knee-joints of rabbits; he found that the synovial membrane became thickened, hyperaemic and that there was a tendency to form villi upon its surface. The products of blood destruction, if excessive, tend to accumulate in the membrane and to evoke a sustained hyperplasia. It is a curious fact that there is no provision in joints for the disposal of particulate foreign material, and Adkins & Davies (1940) injecting indian ink into joints showed that carbon particles over a certain critical size could not be removed but remained permanently in the deeper layers of the synovium, at first lying in macrophages and later set free in the tissue by the death of these cells. *The Haemophiliac Joint* demonstrates well the effect of repeated haemorrhages, by the deep injection of the membrane and its considerable thickening. There is, in addition, marked destruction of the joint surfaces with cyst formation in the peri-articular bone. This is no doubt due to the sustained high intra-articular pressure, which may equal that of the systemic blood (Prip Buus, 1935). Microscopically the tissues show a considerable hyperplasia, especially the surface cells, with widespread deposits of haemosiderin, but there are few tissue histiocytes or giant cells to be seen and there is relatively little fibrous reaction.

(5) Pigmented Villonodular Synovitis

Pigmented villonodular synovitis is an uncommon condition which arises in adults between the ages of 20 and 40. It affects one joint only and this is nearly always the knee—in De Santo and Wilson's collected series 36 times out of 41 cases.

The uncertainty regarding its exact nature is indicated by the wide variety of titles which it has received: xanthomatous tumour, synovial membrane tumour, xanthogranuloma, myeloplaxoma, and so on. The name "pigmented villonodular synovitis" was first used by Jaffe, Lichtenstein and Sutro; these authors consider the condition closely related to xanthoma of tendon sheaths, but my study is confined to the articular form and is based upon 16 cases.

There are two main varieties which appear to differ so widely that they may not be recognised at first as the same condition. There is the generalised type in which the whole synovial membrane is affected by villous or nodular outgrowths; and the localised form which arises from normal synovial membrane as one or more pedunculated mass.

TABLE I

Pigmented Villonodular Synovitis

(Case series)

Case	Sex	Age	History	Site	Variety	Follow-up
1	F	20	½ year ..	Knee ..	Single	5 years
2	F	22	5 years ..	Knee ..	Villous ..	9 years
3	M	36	7 years ..	Knee ..	Villonodular ..	¾ year
4	M	25	Several years	Knee ..	Villonodular ..	2¾ years
5	F	47	15 years ..	Knee ..	Villonodular ..	1½ years
6	F	23	Several years	Knee ..	Villonodular ..	2 years
7	F	59	17 years ..	Knee ..	Single	¾ year
8	F	32	Unknown ..	Ankle ..	Single	½ year
9	F	38	5 years ..	Knee ..	Single	1½ years
10	M	25	2 years ..	Knee ..	Nodular ..	2 years
11	M	24	3 years ..	Knee ..	Villous ..	1¼ years
12	F	30	¾ year ..	Knee ..	Single	1¾ years
13	M	32	Many years	Knee ..	Villous ..	1 year
14	No details available		..	Knee ..	Villonodular ..	—
15	M	20	½ year ..	Knee ..	Villonodular ..	1½ years
16	M	23	1½ years ..	Knee ..	Single	¾ year

Generalised type (9 cases). This variety is characterised by a history extending over several years of an injury followed by recurrent effusions which are often very large and persistent (Fig. 2). There is little pain or limitation of movement except that caused by the effusion itself. On palpation the synovium is found to be remarkably thickened and sometimes lobulated (Fig. 3). Clearly this type needs to be distinguished from tubercle and other forms of arthritis in which large effusions occur: haemophilia; the neuroarthropathies and synovial sarcoma. On the other hand, many perplexing cases finally dismissed as "idiopathic intermittent hydrarthrosis" are probably suffering villonodular synovitis. The condition is not usually diagnosed until the joint is opened, but it may be suspected if the joint is aspirated and orange cloudy fluid is withdrawn, and especially if this is found to contain red blood cells and large quantities of cholesterol and bilirubin.

I should now like to refer to a case which I only saw after repeated aspirations and in which a biopsy had been performed a year previously. It was thus possible to make a tentative diagnosis and to record the progress of the case in all its stages.

A sailor, aged 36, was torpedoed in 1942, injuring his left knee. He took three months to recover. In 1944 excision of a semimembranosis bursa was performed. In 1945 he was in hospital again for three months with a swollen knee, which was aspirated three times. In 1946 aspiration of the knee yielded "a blood-stained fluid . . . which was sterile on culture" and guinea-pig inoculation. In 1948 " ten ounces of yellow-brown viscid fluid " were withdrawn. Biopsy revealed " synovial membrane chocolate brown and villous." On examination his general health was excellent. His left knee was enormously swollen with much quadriceps wasting. Movements were full and free. In February, 1951, sub-total synovectomy was carried out by extrasynovial dissection through two curved incisions. At the last examination his knee was symptomless and flexion had returned to 70 degrees. He was back at work, but unfortunately he has returned to sea and I have lost track of him.

In the villous form the synovial surface is thrown into an immense number of delicate fronds (Fig. 1).

A land-girl, aged 22, gave a history of five years' pain and swelling of the left knee. The onset was gradual. There had been no locking or clicking. On examination there was quadriceps wasting, a small effusion and marked synovial thickening. A radiograph was normal. In September, 1942, excision of the synovium from the inside of the joint was performed. She made a complete recovery and the knee has remained symptomless for nine years.

Localised type (7 cases). This takes the form of a pedunculated mass ranging in size and colour from a cherry to an orange and usually arising near the joint line. It is often called a joint xanthoma or benign synovioma. It gives rise to attacks of pain and locking suggesting a torn meniscus or loose body. The attacks are accompanied by effusions of clear fluid in which fresh blood is sometimes present.

In July, 1945, a female, aged 20, had a spontaneous locking of the right knee, while running upstairs. There was no clicking or swelling, but she had many subsequent attacks of locking. On examination a loose body was not visible on the radiograph but was palpable over the outer side of the knee. In December, 1945, removal of a soft pedunculated tumour, the size of a cherry and brick red in colour, was performed. The tumour arose from normal synovium to which it was attached by a thin pedicle. She made a full recovery and has remained symptomless since.

Histology.—There are four characteristic features: First, there is a marked proliferation of the surface cells which form solid masses or become heaped up into villi and nodules. These are highly vascular and show a remarkable facility for coalescing with one another, forming a lacework pattern.

Secondly, there is a marked proliferation of connective tissue throughout the membrane and this is largely responsible for its increase in thickness.

The third feature is the extraordinary accumulation of intra- and extracellular haemosiderin and lipoid. This iron-containing blood pigment can be seen as granules or contrasted by the prussian blue reaction. Doubly refractile material is well demonstrated on frozen section and the fat when differentially stained shows the characteristics of cholesterol and its esters. The single nodules contain mainly lipoid while in the generalised form the haemosiderin preponderates. Reticulo-endothelial cells are present in great numbers and variety; monocytes and giant cells containing haemosiderin or with foamy cytoplasm are seen. Other sections show sheets of plasma cells or collections of lymphocytes.

Each type of cell has been observed in turn and considered the essential feature of the condition. In addition they may be actively dividing and give the impression that the tissue is malignant.

Treatment.—With the solitary nodules simple excision is all that is necessary. Unfortunately there appears to be no reliable histological criterion or unanimity of opinion among pathologists to distinguish them for certain from malignant new growths. I must therefore plead for the use of clinical judgment in these cases, bearing in mind the macroscopic appearances of the joint concerned. The surgeon should

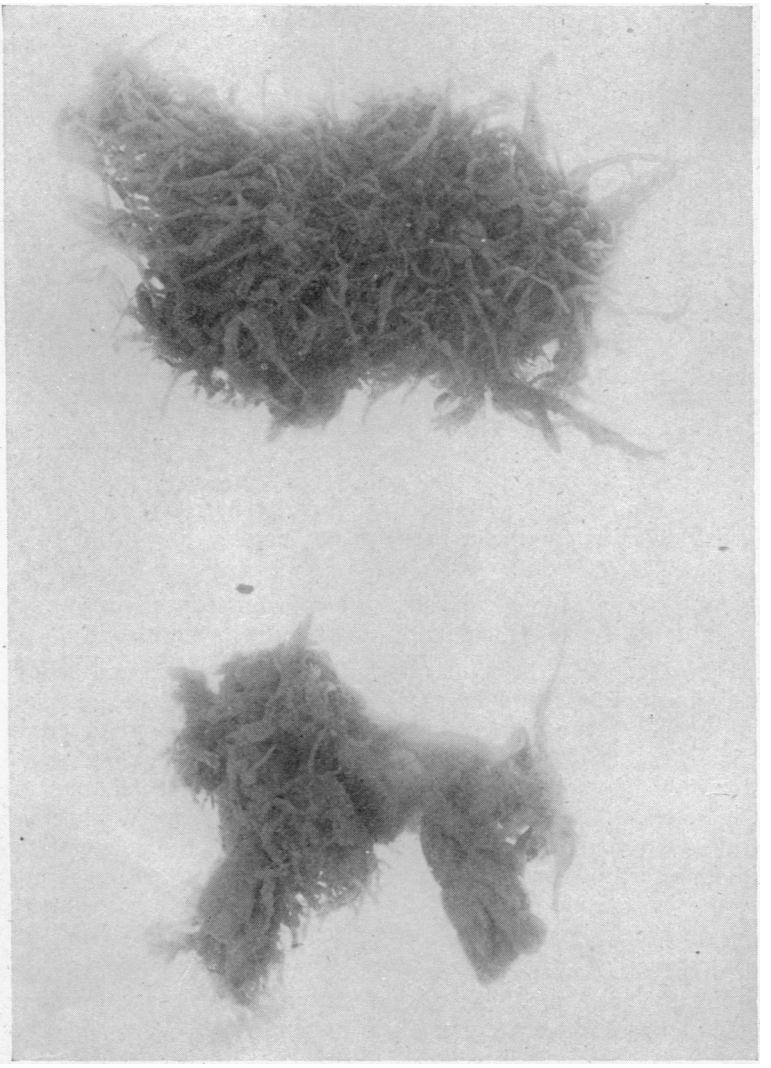

Fig. 1. Pigmented villonodular synovitis—Villous type.

certainly not be persuaded to carry out amputation on histological grounds alone. It is significant that in at least four of my cases malignancy was suspected and amputation seriously considered.

The diffused varieties can be cured for certain by synovectomy and this should be as complete as possible, either by extrasynovial dissection or from within the joint. The disadvantage of the latter method is that fragments may be left behind which continue to grow and give the impression that the condition has recurred.

Aetiology.—Why do such wide-spread changes occur in an otherwise normal knee-joint ?

Although there is a chronic inflammatory process at work, the condition cannot be due to infection. The synovial fluid is invariably sterile, the patient apyrexial and the blood count normal. There is no destruction of the joint surfaces, and the change may be more advanced in some areas of the membrane than in others.

Again it is unlikely to be due to a metabolic disorder. Although a raised blood cholesterol has been recorded by other workers, in my cases in which the blood cholesterol was estimated no significant rise was noted. In addition no other lipoid deposits are found ; joint changes are rare in the lipoidoses and, as far as I am aware, villonodular synovitis has never been recorded in conjunction with them.

What part does trauma play in the onset or maintenance of the disorder? Obviously, a single injury cannot be responsible, or the condition would be extremely common. However, many of my patients were able to recall a direct injury and in De Santo and Wilson's series this was recorded 23 times out of 30 cases.

Many authorities seem to imply that the condition is neoplastic. As far as the single nodule is concerned Matthew Stewart and others have long maintained that this is a benign new growth of synovial tissue. He uses (1948) the term " bionecrosis " to explain the accumulation of lipoid : the growth outstrips its blood supply, some tissues die, cell lipoid is released in an insoluble form which is taken up by local phagocytes. I believe, on the other hand, that the condition commences as an enlargement of a synovial fringe which becomes nipped between the joint surfaces. This is a very common occurrence in the region of the subpatellar fat pad, where these nodules are most frequently found. Haemorrhage occurs into the membrane with tissue necrosis. A nodule is formed and the products of tissue destruction accumulate. As the swelling arises at the joint line it becomes more liable to trauma so that is slowly increases in size.

There is little evidence to suggest that the generalised form is a neoplasm ; invasion of the surrounding tissues or distant metastasis has never been recorded. In my view the condition commences as a villous synovitis ; once this change has become sufficiently widespread it also becomes self-perpetuating. The delicate and hyperaemic villi readily bleed and this

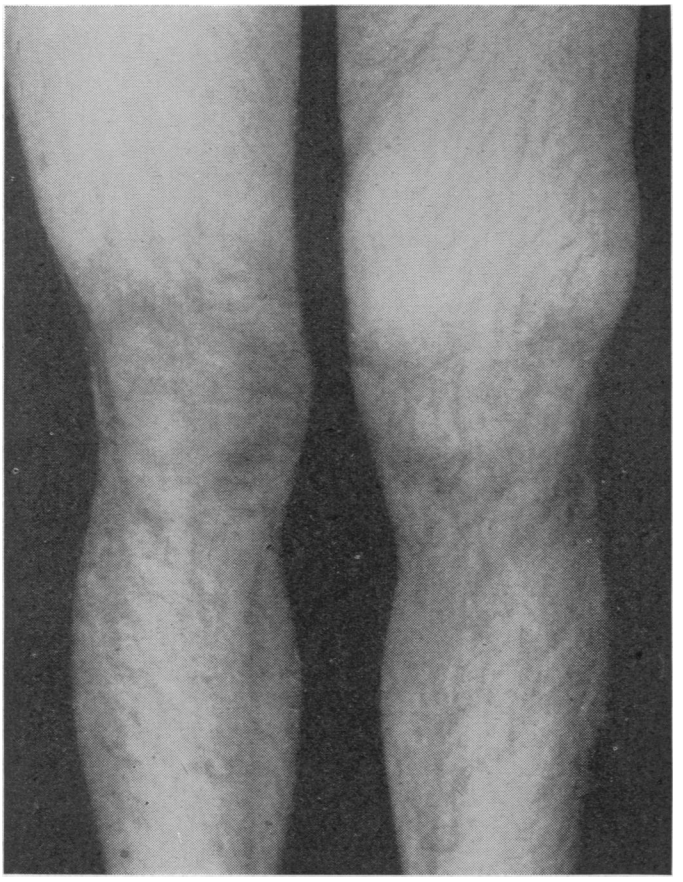

Fig. 2. Pigmented villonodular synovitis.
Enormous distension of quadriceps bursa.

gives rise to the characteristic episodes of haemorrhagic effusions. Absorption of the haemosiderin and lipoid takes place with further proliferation of the membrane. We have here therefore a combination of a primary villous hyperplasia followed by a metaplasia of the synovial cell into various phagocytic forms to deal with the products of blood destruction.

I am not qualified to speak about the corresponding swellings occurring in tendon sheaths, but their histological picture closely resembles that of the single joint nodule. These undoubtedly recur locally, but this may be due either to incomplete removal or to a continuation of the change in the remaining synovial sheath.

I am of the opinion that a precursor of villonodular synovitis is the condition which I shall now describe : Intermittent Hydrarthrosis.

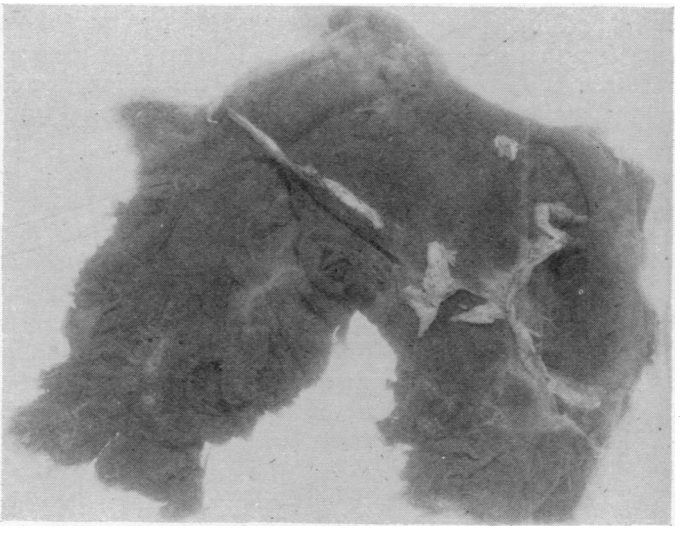

Fig. 3. Pigmented villonodular synovitis.
Membrane chocolate brown and in places an inch thick.

(6) Intermittent Hydrarthrosis

This condition is characterised by recurrent painful swelling of a joint, usually the knee, occuring at regular intervals. As this title only describes a symptom it is clear that there are numerous diseases which can produce recurrent effusions into joints. However, when mechanical, degenerative, infective and metabolic factors are excluded, there remains a group of cases in which no obvious cause can be found, and in which conservative treatment is of no avail. Many suggestions have been made as to its aetiology, among them heredity, allergy and hormonal imbalance.

Although such joints have been frequently explored, there are few recorded descriptions of the appearances of such a joint as seen at operation. I have now operated upon three such joints, all knees, and I have made the following observations. The synovial membrane is not thickened to any extent but it is deeply congested. There is little or no pigmentation present ; indeed when a fragment of excised tissue is washed free from blood it is almost colourless. The bony attachments of the synovial membrane are covered by reddish granular areas, but it is not until these areas are excised and floated in saline that the gross changes become obvious. These granular areas are in fact bunches of enlarged and elongated villi, too delicate to remain erect without the support of the fluid. Similar changes can be seen on the surface of the fat pads, many of the lobules carrying fringes of villi. The rest of the membrane is quite smooth as though the chronic distension of the joint had flattened its normal contours. In one case the synovial surface was covered with a

reticulation of fine fibrous strands, which I believe to be adhesions formed on the surface of the membrane and then overgrown by the synovial cells.

These observations strongly suggest that the villous areas are responsible for the recurrent effusions and I have therefore excised them as completely as possible. Although my series is, at the moment, too small from which to draw conclusions, I have in these cases succeeded in preventing the recurrence of these large effusions. In addition, one of these cases has been given a course of X-ray therapy with, I believe, further benefit. Histologically, the membrane is highly vascular and somewhat oedematous, the surface is a little increased in thickness and the whole tissue is infiltrated by plasma cells and collections of lymphocytes.

The present tendency is to regard intermittent hydrarthrosis as an atypical form of rheumatoid arthritis (Collins, 1949), but although one of my cases showed a raised blood sedimentation rate no other stigmata of this disease were present. Whatever the aetiology of the condition, the relief of the patient's main symptom by partial synovectomy is surely of value and justifies persistence.

I am able to report a case of a young man, aged 24, with a history of three years' recurrent effusions in the right knee following an injury at football. Aspiration of his knee on three occasions over a period of six months yielded a clear fluid once but a cloudy orange effusion typical of villonodular synovitis at the other times. Examination of the joint and histological section were typical of intermittent hydrarthrosis, but in addition iron was beginning to accumulate in the villi. I therefore regard this case as a link between these two conditions, and it is not without interest that villonodular synovitis has on occasions been called " plasma cell synovitis."

(7) Synovial Osteochondromatosis

The attachment of the synovial membrane to the bone at the edge of the articular cartilage is, like all tissue junctions, a histologically unstable area. The formation of cartilaginous nodules at this site is very common and this metaplasia is in part responsible for the lipping which is a feature of osteo-arthritis. Occasionally single " cell rests " of cartilage are found in normal synovial membrane, but the most striking changes are found in synovial osteochondromatosis.

This is a rare condition which may arise in the synovial membrane of both joints and bursae. Great numbers of cartilaginous bodies are formed within the membrane and then shed into the joint. These bodies give rise to attacks of effusion and locking ; they may erode the joint surface by pressure but the articular cartilage itself is unaffected. Macroscopically, the membrane appears congested and hyperplastic, and upon its surface can be seen numerous pearly nodules lying within villous projections.

Section of the membrane shows that these nodules commence as intercellular accretions of cartilage, and that as they grow they show increasing organisation. Finally, the nodule becomes surrounded by a

lamina of compressed fibrous tissue into which the cartilage continues to grow, and the nodule now contains a central matrix of delicate cellular bone. When the surrounding lamina becomes thinned, the surface cells disappear and the body ulcerates through into the joint. Here a layer of fibrin is deposited on its surface and the body slowly enlarges, deriving its nourishment from the synovial fluid.

The cause of synovial osteochondromatosis is unknown but it seems to be related to injury. The condition need not be regarded as neoplastic, but it can be easily explained as a metaplasia. Bearing in mind Vaubel's statement concerning the intimate relationship between the tissues of the skeleton, quoted above, it can be readily understood how a slight modification in the metabolism of the synovial cell will cause the formation of cartilage and bone, rather than joint mucin. Indeed, every stage between a synovial cell and a fully developed cartilage cell can be observed in serial histological sections.

(8) Synovial Lipomatosis

Fat in the synovial membrane shares in the changes which affect the body tissues generally; for instance, it becomes increased in obesity and at the menopause, but little is known about its metabolism. Some investigations into the biochemistry of synovial fat kindly carried out for me by Dr. Kendall Dixon have failed to demonstrate any differences in its composition from subcutaneous adipose tissue.

Occasionally in osteo-arthritis of the knee the synovial surface is covered by grape-like protrusions of fatty tissue and it is then termed *lipoma arborescens*. This is probably not a neoplastic change, but potentially malignant lipomata, which break out of the confines of the joint or tendon sheath and invade surrounding structures, have been reported (White 1924). *Lipoma simplex symmetricum* (Strauss 1922) is an interesting condition unassociated with general lipoid disturbance, in which the same tendon sheaths on the two sides of the body undergo a smooth and regular distension by overgrowth of synovial fat. Enlargement is confined within the sheaths, and there is no interference in the function of the tendons. I have seen such a case in an adolescent girl in which the extensor sheaths of both hands were accurately outlined. Treatment is generally sought because of the unsightly appearance.

(9) Relationship to Malignant Neoplasms

I should finally like to discuss the relationship of these conditions to malignant new growths arising near joints. In the first place it is very odd that joints hardly ever, or should I say never, become the site of secondary deposits from any type of tumour. This is all the more surprising in view of their rich blood supply and the ease with which micro-organisms can establish themselves and flourish within joints.

It is, I believe, unfortunate that the term " synovioma " has been used to cover a wide range of greatly differing conditions which arise within joints. Although much evidence has accumulated (Key 1952) that

there is a group of highly malignant tumours which possess characteristic histological features resembling normal synovial membrane, I have tried in this lecture to show that all connective tissue under appropriate conditions may produce joint-like cavities lined by a membrane and containing synovial fluid. It is therefore more logical to consider such tumours as a special form of connective tissue sarcoma rather than to search for their origin from pre-existing synovial membrane in their neighbourhood. Of the many recorded cases of so-called malignant synovioma, there is little or no evidence that these tumours did in fact arise from a synovial surface and then invade surrounding tissues. Ross (1951) in his recent Arnott Demonstrations before this College, cast considerable doubt upon the pathological classification of these tumours, and has pleaded for the abandonment of the term " synovioma." The use of the term for the villonodular type of synovial change is inappropriate, misleading and a danger to the patient. As for the malignant new growths, if the reference to synovial membrane is to be retained, I would suggest the term " parasynovial sarcoma, " implying not only that the tumour may arise in the neighbourhood of joints and bursae, but also that its microscopic appearance is reminiscent of that seen in normal synovial membrane.

It only remains for me to thank the President and Council of this College for the privilege of delivering this lecture before you to-night. I must also thank my many surgical friends and colleagues for allowing me to use their material, Dr. Barrett of Cambridge University and the members of the Pathology Department of this College for their help with the histological preparations.

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PROCEEDINGS OF THE JULY COUNCIL

At a meeting of the Council held on the 31st July, with Sir Cecil Wakeley, President, in the Chair, Dr. O. C. Carter and Dr. Bernard Johnson were admitted to the Council, being co-opted members representing General Practice and Anaesthetics respectively.

A resolution of condolence was passed on the death of Sir Hugh Cairns, Past Member of the Council.

Mr. R. C. Davenport (Moorfields) and Mr. F. C. W. Capps (St. Bartholomew's) were elected Members of the Court of Examiners to examine respectively in Ophthalmology and Otolaryngology for the Fellowship.

Professor F. Wood Jones was appointed to the post of Honorary Curator of the Hunterian Collection of Human and Comparative Anatomy on his retirement from the Sir William Collins Professorship of Anatomy.

Mr. Hedley Whyte was re-appointed as representative of the College on the Council of King's College, Newcastle-on-Tyne.

Mr. L. E. C. Norbury was re-appointed as representative of the College on the Examination and Tuition Advisory Board of the Association of Medical Records Officers.

Dr. B. E. Heard of St. Mary's Hospital was appointed Lecturer in Pathology.