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Bursitis and its treatment

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BURSITIS AND ITS TREATMENT

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INTRODUCTION

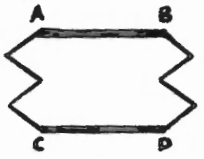
The purpose of this paper is to review the literature concerning the treatment of the more common forms of bursitis. To facilitate organized arrangement of this broad subject, bursitis will be discussed by regions, including the shoulder, elbow, hip and knee joints. This is not a complete list, for there are other bursae in in other joint regions of the body, but to deal with all the bursae of the body in a comprehensive manner is beyond the scope of a paper such as this.

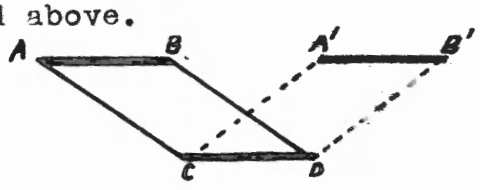
By way of preparation for a discussion of the very contraversial treatment of the commoner forms of bursitis, there will be set forth first the anatomy, the pathology, the etiology, the symptomatology and the pertinent points in diagnosis and differential diagnosis involved in disease of the bursae. For the sake of brevity, that more emphasis may be given to the treatment of bursitis, there will be reported what seems to be the most authoritative work done on the subject, rather than an involved discussion. Each of these "lesser" phases of the subject will be introduced by general considerations, followed by the special points involved in the particular bursae under discussion.

ANATOMY

To appreciate the general anatomy of the bursae, one must understand its function. In a book entitled THE SHOULDER, Codman tells us that "bursae are supplied where a considerable degree of motion between parts of anatomy is necessary, yet no cartilaginous joint is required". They are placed where muscles cross in different directions, or where bone, muscle and tendon must pass one another without actual articular contact. Some, largely the adventitious bursae, lie between the skin and some portion of the skeleton.

It is the motility of the periphery of the bursae which permits motion, rather than the attached roof and base. The following diagrams and explanation are taken from Codman's work mentioned above.





A-B the roof and C-D the base are fixed to attachments. A-C and B-D are the movable periphery. The periphery is very thin and flexible so that it may fold upon itself as the base and roof slide by one another. Normally, the bursae are spaceless sacs not filled with fluid, but supplied by a self oiling mechanism, that their walls may glide on one another with their surfaces

no farther apart than the thickness of the thinnest sheet of paper.

Before Codman's extensive work on the shoulder, published in 1934, three bursae were commonly described about the shoulder joint. These were the subacromial, subdeltoid, and subcoracoid. A fourth, unimportant from the clinical aspect, but usually mentioned, was a small bursa in connection with the tendon of the long head of the biceps. Indeed, the anatomy texts of Gray and Cunningham and the HAND ATLAS OF HUMAN ANATOMY by Spalteholz still describe the three common bursae of the shoulder joint mentioned above. Codman, on the other hand, believed that the subacromial, subdeltoid, and subcoracoid bursae are one and the same, though at times films of tissue may separate them. When the arm is abducted, the subdeltoid portion becomes subacromial. When the arm rotates inward the subdeltoid portion becomes subcoracoid. Among the many accepting Codman's theory on anatomy of the shoulder bursa are: Rogers, Stimson, Pelner, Wilson, Borak, and Haggart and Carr.

Codman states that the subacromial (this is the terminology now commonly accepted) bursa is the largest in the body and also the most complicated in structure and component parts. It could be called a secondary scapulo-humeral joint, though no part of its surface

is cartilage. It is a bursa between bone, tendon, muscle and bone. At its base, it is attached to the upper and outer 3/4 inch of the greater tuberosity, and to about 3/4 inch of the tendons of the four short rotators where they are attached to the tuberosities. Thus, part of the base covers the bicipital groove. Its roof is attached to the under side of the acromion, to the underside of the coraco-acromial ligament, and to the fibers of origin of the deltoid from the edge of the acromion. Its periphery extends loosely downward beneath the deltoid, backward and outward under the acromion, and inward under the coracoid, between it and the subscapularis, and under the common origin of the short head of the biceps and the coraco-brachialis. The roof and base are in close proximity and are lined by an exceedingly thin synovial membrane, which retains enough secreting properties to render the surfaces practically frictionless. Beneath this thin secreting membrane is a fine network of blood vessels, so that secretion can be increased or diminished on demand.

Codman describes very thin nictating and usually partial membranes which are often found in the subacromial bursa and which may thicken and become adherent due to traumatic and infection processes, giving rise to a number of smaller sacs. It is also pointed out that

bursae vary among individuals. He mentions two so-called "false bursae" (actually extensions of the joint cavity): the subscapularis, which may communicate with the subcoracoid portion of the subacromial bursa on the superior surface of the subscapularis muscle, thus forming a horseshoe shaped arch beneath the coracoid process and the tendons arising from it and the infraspinatus bursa. In addition to these and of no clinical importance, he describes some small bursae beneath some of the tendons at their attachments to the humerus on the ridges on each side of the bicipital groove. The teres major, latissimus dorsi, and pectoralis major muscles have such attachments.

In the elbow region there are two important bursae which frequently become involved in pathological processes. The first of these and by far the most common beset by disease is the olecronon bursa. Though there is very little in the literature on this subject, a fine paper was published in 1928 by Lasher and Mathewson. They describe it as a large subcutaneous bursa lying directly beneath the skin and which, when distended fully, measures 2 ½ inches in length by 3/4 inch in width. It varies in thickness. The upper half lies above the tip of the ulna while the lower half, according to Stimson (1940), is firmly attached to the periosteum by means of vertical

fibrous strands. This bursa lies in close proximity to the joint cavity, being separated from it only by the posterior portion of the orbicular ligament. It may in rare instances communicate with the joint cavity. Despite the presence of the bursa between the olecronon process and the skin, the bone here is very superficial. In addition, the cortex is thin and the cells cancellous in nature, all of which leads these men to believe that the bone is very often injured at the same time as the bursa.

The other clinically important bursa in the elbow region was perhaps best described by Osgood in 1922 and reiterated by Carp in 1932. It is called the radiohumeral bursa because it lies beneath the conjoined tendon, just distal to the epicondyle and over the radiohumeral joint from which arise a few fibers of the supinator radii brevis. It is normally about 1 by 0.5 cm. in diameter and its walls are very thin and friable. It may appear only as a slight depression or elevation, and when incised, is usually found to contain a little clear fluid. Carp expresses the theory that it is probably adventitious in nature resulting from a need for free play of the conjoined tendon over the supinator radii brevis and the radiohumeral joint. This bursa has also been mentioned by Vogt, Broesike, Bardenheuer, and

Heineke. In Black's study of the development of the human synovial bursae published in 1934, he states that none of the subcutaneous bursae develop prior to birth, thus lending support to Carp's adventitious theory mentioned above.

Stimson (1940) quotes Homans' text of Surgery (1931) in a description of two other bursae of the elbow region which are occasionally found but rare. These are the so-called interosseous and bicipitoradial bursae.

The most important bursa in the hip region is the iliopectineal bursa. According to Osgood, it is the largest in the body. In 1933 O'Connor published a good description of its anatomy. It varies in size within wide limits and according to Joessel, it lies between the partly tendinous portion of the iliac muscle and the front of the iliopectineal eminence. Anteriorly it is firmly attached to the iliopsoas muscle, posteriorly to the pectineal eminence and to the thin portion of the capsule of the hip joint. Its outer boundary is the cotyloid ligament. Occasionally the fibrous capsule at the thin point is lacking, and again the synovial membrane lacking, so that there is a direct communication with the joint cavity.

Chandler (1934) made extensive studies of the

anatomy of this bursa on some 206 cadavers at Loyola University. He reported that the iliopsoas (synonym for iliopectineal) bursa was present bilaterally in all the cadavers except 3. The sizes varied greatly, the average being 5-7 cm. long by 2-4 cm. in width. He also agreed with Lund (1902) that the proximal end of the bursa extends in some cases, over the brim of the pelvis. Chandler was also interested in the finding that 14.25% of the bursae communicated directly with the joint cavity. Of those lacking foramina, a worn area in the floor over the ramus of the pubis or on the capsule was present. He also demonstrated this bursa in the 5 month fetus and noted that in no fetus was there evidence of wear or apparent difference in thickness of the capsule, nor any communication between the bursa and the joint cavity. Chandler and Finder (1938) both mention that in the adult, that portion of the capsule of the hip joint between the pubocapsular ligament and the Y ligament of Bigelow is usually very thin and in some cases was even fenestrated. Kessel's work quoted by Finder, substantiates Chandler's findings also.

Stimson describes two more bursae in the hip region. First and quite important, is the trochanteric (gluteal) bursa which lies between the greater trochanter and the origin of the gluteus maximus and vastus externus muscles.

It is large, constant, and subject to trauma from blows or falls and when inflamed, causes pain at this site on flexion and internal rotation of the hip. It is rarely diseased, but may complicate a hip fracture. The other she describes as the ishial bursa. This lies between the ischial tuberosity and the origin of the hamstrings, and is in contact with the deep surface of the gluteus maximus muscle. When inflamed, it is called "Weavers" or "Tailors" bottom. Three other bursae of no clinical interest whatever are described in Spalteholz's HAND ATLAS OF HUMAN ANATOMY.

The bursa most often diseased about the knee joint is the prepatellar. Stimson describes it as lying between the skin and deep fascia overlying the patella and as usually adherent by tough fibrous strands to the bone or quadriceps aponeurosis. Slightly distal to this bursa is the infrapatellar bursa. This lies between the patellar tendon and the anterior surface of the tibia above the tibial tuberosity, and is separated from the anterior chamber of the knee joint by a thick pad of fat. According to Ghormley (1939), it is constant and does not communicate with the knee joint. Ghormley also describes a suprapatellar or quadriceps bursa. This lies between the quadriceps tendon and the lower end of the femur. According to Spalteholz it

communicates with the knee joint in 85% of the cases. A superficial pretibial bursa is described by Ghormley as being sometimes present under the skin, superficial to the tibial tendon at its attachment to the tuberosity.

In the popliteal region of the knee there are numerous smaller bursae. According to Ghormley, a constant one is the anserina which is found over the tibial collateral ligament and beneath the tendons of the gracilis, semitendinosis and sartorius. Another is found beneath the tendon of the semimembranosis and the collateral ligament of the tibia. Still another between the medial head of the gastrocnemius and the capsule over the inner condyle of the femur. Voshell and Brantigan (1943) describe five variable locations for bursae deep to the tibial collateral ligament. These are: (1) between the ligament and the capsule, above the medial meniscus and often extending up to the medial epicondyle of the femur, (2) between the tibial collateral ligament and the medial meniscus, of separating the meniscus from the parallel portion of the tibial collateral ligament, with its main extent being mainly above the meniscus, (3) directly over the meniscus, extending equidistant above and below, (4) its extent principally inferior to the meniscus and (5) between the tibial collateral ligament and the tibia, but in

no way associated with the meniscus. Wilson, Eyre-Brook and Francis, (1938), describe a semimembranosus bursa beneath the deep fascia of the popliteal space in the interval between the semimembranosus muscle and the medial head of the gastrocnemius muscle and is intimatly attached to the posterior capsule of the knee joint and its bordering muscles. And finally, Stimsom completes the picture with a description of the so-called popliteal cyst, which is actually a long saccular extension of the joint cavity posteriorly, under the popliteal muscle.

PATHOLOGY

According to Ghormley, the pathologic picture depends upon the etiology and the stage of the process. The bursae react to acute pyogenic infections in a similar manner to the other serous cavities of the body. There is first extravasation of fluid and white cells into the layers of the bursal wall followed by an outpouring of fluid into the bursal sac. This is at first serous, but later may become seropurulent or purulent. As the process subsides, the walls thicken with scar tissue and often strands of fibrous tissue extend into the sac as pedunculated tags or even from a network and connect various parts of the bursa.

When the etiology is traumatic in nature, there is an extravasation of serosanguinous fluid into the cavity which is later replaced by fibrous tissue. In cases due to chronic trauma, there is gradual thickening of the wall until the whole bursa enlarges greatly, becoming a fibrous mass, with almost complete obliteration of the cavity.

One of the commonom complications of a chronically inflammed bursa is calcification. This developes in a bursa when sufficient fibrosis exists; when the hydrogen ion concentration is ideal and when the blood supply is

Inadequate. It may disappear when the processes are reversed.

Mumford in 1927, published a very enlightening paper on the other common pathological complication of bursitis, the formation of rice bodies. These may be found in bursal sacs when the inflammation is chronic, attended by excessive fluid in the sac, caused by a low grade pyogenic infection, trauma, or more frequently tuberculosis, and in all instances their formation is dependent upon the presence of some small nucleus, about which fibrin, derived from the bursal fluid, may be deposited. The physical characteristics of the rice bodies depends upon the origin of the nucleus and the character of the fluid. The nucleus may come from the fluid or from the bursal wall. Those from the fluid begin as small masses of fibrin which gradually collect more. They are usually found in fluid containing blood and when seen early, are soft flat masses, reddish in color and of a dull lustre. When seen later, they are firmer and round or oval in shape. If from the wall, the nucleus is a small tag of fibrin which may have acquired cellular elements from the underlying wall tissue, which later will lead to organization of the fibrin. In this way newly depositied fibrin may become organized, thus making this rice body much firmer in consistancy.

Mumford also believes that the nuclei for tuberculous rice bodies may result from giant cells which are pushed from the lower strata of the cell wall, losing nuclei as they approach the free surface and are finally liberated into the cavity. The life history is then the same as for the other types of rice bodies.

The pathology of the subacromial bursa is most completely set forth by Codman in his book on THE Shoulder. This study represents the conclusions from more than 200 explorations of living patients. Syphilis, tuberculosis and new growths are relatively rare and therefore will not be enlarged upon.

The roof of this bursa is seldom inflamed, but in cases of long standing bursitis with complete or incomplete rupture of the supraspinatus tendon, there are hypertrophic changes at the acromial edge. One of the most important points in this discussion, is that calcified deposites do not form in the bursa, but on the tendons, in this case the supraspinatus tendon. They may remain in the tendon for many years, but the usual sequence is that they gradually work through the base of the bursa producing a mound-like swelling with a turgid zone and a white center. Then some little accident or unusual effort causes a few superficial fibers to break, so that the little crater discharges

into the bursa. Acute bursitis is produced, fluid is secreted with which the calcified particles mingle, and fibrin appears and the calcium is entangled in it. The particles are then eliminated, probably by leukocytic action (this is not true while they remain in the tendon). This calcium absorption may take place within 3 weeks after the perforation and the patient is then free of symtoms. Codman believes that this is nature's method and that the average case would follow this course if allowed to do so. This is not always the outcome however, for if the inflammatory reaction is too intense, adhesions result in a "frozen shoulder".

The most common evidence of pathology is in the bursa itself. The whole floor is apt to be velvety, with lesser villi and thickened nictating folds. The center of this process is usually over the attachment of the supraspinatus muscle. Their presence indicates previous inflammation and adhesion of bursal surfaces. There may be present cord-like fibrous thick bands. Adhesions are commonest in the subdeltoid portion and in the fold separating this from the subcoracoid portion. These too are absorbed or rendered pliable in time by nature's own processes. A slight amount of fluid may occur; if there is a great deal, the supraspinatus

has ruptured completely and there is communication with the joint cavity. Codman also describes "straps", or damaged tendon fibers just over the supraspinatus insertion. There is a circular area about 1 inch in diameter which seems worn as from friction. Close inspection reveals thin straps of tendon fibers running with the tendon, attached above and below but separated by a space. They buckle up on abduction.

Wilson (1939) collaborated Codman's findings and in addition described the calcaneuous deposits. Grossly they may be uncoalesced collections of dry gritty sand-like particles without a surrounding hyperemia, or they may be confluent cavities filled with a soft greyish material having somewhat the consistancy of toothpaste and surrounded by intense inflammatory reaction. In chemical analysis, he agrees with Codman and Moschcowitz (1915) that is is chiefly calcium phospate and calcium carbonate in non-crystalline form.

In a treatise on chronic adhesive bursitis, Haggart and Carr (1943) pointed out the presence of atrophy of the shoulder muscles together with degenerative changes and scar tissue formation in the capsule and tendons of the shoulder joint. Kaplan and Hawkins in 1945 wrote a fine paper in which they reiterated Codman's original findings.

In dealing with the pathology of bursae at the elbow, Rosen in 1940 quotes from a description of the pathology in "Miners Beat Elbow" taken from the Lancet, Saturday, May 21, 1842; a letter to the editor from Edwin Gurney of Camborne, Cornwall. I will quote some from this description; ....."fluctuating tumour at the posterior part of the elbow joint is of frequent occurrence, in consequence of the narrow passages the miners must pass through going up or down or work in. The posterior part or (cap) of the elbow-joint is bruised, in consequence of which the bursal sac becomes inflamed, and effusion of serum follows....".

Lasher and Mathewson (1928) and Ghormley (1939) describe the typical inflammatory reaction of the wall and an outpouring of serous, seropurulent or even purulent fluid into the sac in the acutely inflamed bursa. The patient notices fluctuant swelling and pain of varying degree. Reddening of the skin and increased surface temperature is found if infection is present.

In a chronic olecronon bursitis, the typical inflammatory reaction is present with a variable thickening of the wall and the fibrous network within the sac. Calcification is seldom present. Osteomyelitis of the olecronon process is not a rare complication of infection in this bursa.

In 1922, Osgood gave us a small bit on the pathology involved in radiohumeral bursitis. He states that, "the essential pathology is an inflammatory reaction in a commonly existing bursa", previously described. Somewhat similar symptoms referred to the mesial condylar region may be explained by similar arealar tissue reactions beneath the common tendon of the flexor muscles. There is no constant bursa in this region, but adventitious ones can occur.

In 1929, Dittrich quotes a pathological description by Goeldel, who after removing the epicondyle in two cases, reported, "the bone is surrounded by scar tissue with metaplastic bone formation" in one case, while in the other, "partly calcified cartilage and periosteal new bone formation". Carp, writing in 1932, substantiates Osgood's anatomical and pathological findings and in addition, tells us that the position of the bursa can often be demonstrated by shadows in X-ray due to calcification of its walls. By this means, he has shown the bursa to sometimes encroach upon the epicondyle, or extend over the head of the radius.

Apparently, the most important bursa in the hip from the standpoint of pathology, is the iliopsoas bursa. Swindt (1923) tells us that infection or trauma in this bursa is followed by a great increase of fluid

within the sac, and a corresponding increase in the intravesicular tension. The walls of the sac are pushed out into the various muscle and fascial planes, a so-called burrowing capacity. Rupture may occur at any thin point in the wall. Fusing with neighboring bursa may result in a single cavity with a continuous flat cell lining. If during this process, the bursa establishes communication with the joint cavity, incomplete excision may be followed by regeneration from the synovial membrane. Infected bursa untreated, may rupture through the skin and establish a fistula which is unusually prone to remain open. O'Connor (1933) states that the first reaction of this bursa to mechanical or infectious irritation, is effussion which will subside if the part is put to rest for sufficient time. If proper treatment is not carried out, he believes that the bursal wall becomes many times thickened and degenerative changes may occur in the membrane, such as the formation of cartilaginous plaques. Chronic abscess cavities lined with granulation tissue may also result.

The extent to which the bursa can enlarge is somewhat contraversial. Durville (cited by Gatch and Green, 1925) states that its upper limit never extends above Poupart's ligament. Finder (1938) is agreed, stating that it may enlarge sideways from the iliopectineal

eminence to the anterior superior iliac spine. The lower border may reach into the region of the lesser trochanter. On dissection, the ordinary bursa is usually large enough to admit the index finger. When distended, it assumes surprising dimensions, usually as large as a hen's egg or goose egg. Lund, on the contrary, says that it may extend into the iliac fossa. Cullen, in 1910, reported one in a male which filled nearly half the pelvis and contained six large cartilagenous masses. Gatch and Green also quoted Charleston's description of a bursa which extended from the pelvis above Poupart's ligament and to the region of the knee joint.

In 1931, Lecoq was the first American to describe calcification in the iliopsoas bursa area causing inflammation. Later, in 1936, Goldenberg and Leventhal mentioned two bursa constantly present about the greater trochanter, (1) between the gluteus maximus and the tendon of the gluteus medius and (2) between the latter tendon and the bone. They described supratrochanteric calcification as occurring (1) in the gluteus medius tendon, (2) in the bursa between this tendon and the greater trochanter and (3) away from the trochanter on the under surface of the gluteus medius muscle. Hitchcock (1937) described periarticular calcification with acute symptoms in the shoulder and the supratrochanteric

region. Sandstrom (1938) was the first to bring out the analogy between the shoulder and the hip in reference to peritendinitis calcarea with involvement of the bursa in a manner similar to that in the shoulder described in detail by Codman. Subsequently Schein and Lehmann, (1941), also called attention to this analogy while describing seven cases. In five of these the calcification was lateral to the upper part of the greater trochanter and in the other two, above the tip of the greater trochanter. Ghormley (1939) reiterated Goldenberg's and Levanthal's previous report and included a work on the deep trochanteric bursa, lying between the greater trochanter and the tendon of the gluteus maximus muscle. He reports that its inflammation is usually subacute or chronic and is not often associated with an accumulation of fluid, however, pyogenic infection of the bursa with considerable purulent exudate may be found. The pathological response of the ischial bursa that of typical inflammation and effusion previously described.

The pathology of the bursae about the knee seems to be essentially that of effusion. Ghormley (1939) points out that the prepatellar is also predisposed to acute infections through abrasions or lacerations of the skin. This results in a seropurulent or purulent exudate. In most cases, however, there is a gradual thickening of

the bursal wall may continue and concentric rings of fibrous tissue may be laid down, so that it is several centimeters thick and the lumen is all but obliterated. Stimson (1940) tells us of the same danger of puncture wounds to the infrapatellar bursa. There is not quite the tendency for huge distension here as in the bursa previously mentioned. She points out that it may also become inflamed in connection with Osgood-Schlatter's disease. Breck and Higinbotham (1946) report that this, the infrapatellar bursa, is more frequently involved than the prepatellar bursa in infantrymen. The bursa becomes large and distended, the walls thicken, and it may continue on to an inflammatory phase which may be asceptic or suppurative.

Burman (1944) describes the usual popliteal cyst as an enlarged semimembranosus bursa. There is usually quite an effusion and later, the bursa may become fibrotic and palpable.

Voshell and Brantigan (1943) suggest that certain cases of Pellegrini-Stieda's disease may be accounted for upon the basis of calcification of the bursa between the tibial collateral ligament and the capsule superior to the medial meniscus. The cause for this calcification has been ascribed to a chip fracture of the internal epicondyle of the femur, periosteal tear, and metaplasia

of the tibial collateral ligament.

ETIOLOGY

For a generalized discourse on etiology of bursitis, the following outline was made from an article by Ghormley appearing in 1939.

I. Inflammation
   (1) laceration or abrasion of the skin as a portal of entry.
   (2) contiguous abscesses.
   (3) rarely hematogenous.

II. Injury
   (1) contused or torn.
   (2) direct or indirect.

   May become chronic traumatic bursitis.
   (3) constant irritation may lead to chronic inflammation with no acute phase.

III. Tuberculous bursae
   (1) found only when bursa connects with, or is in very close proximity to the infected joint.
   (2) only rarely is it found as a separate entity.

IV. Syphilitic infection--same as tuberculous.

V. Metabolic disturbances: (gout, endocrinopathies, toxic disorders, foci of infection, and etc.)

(1) are responsible for a few cases of chronic bursitis. May be aggravating factors.

Though Ghormley makes light of hematogenous bursitis, Barnes (1926) reported a case of suppurative subacromial bursitis in a woman suffering from septicemia. The pus revealed hemolytic streptococcus.

In 1928, James Hitzrot reported three cases of infectious calcific subacromial bursitis. These also yielded pus with positive cultures for hemolytic streptococcus; in one from a tooth, another from an infected sinus, and a third from an ulcerated cervix.

Martin (1929) reported a number of cases of bursal supperation following varicella. These included olecronon and trochanteric bursa.

In 1938, Cooperman described acute hematogenous bursitis as a rare but important complication that may arise in the course of or as sequelae to, the acute infectious diseases, septicemias; or as secondary, metastatic leasions in pulmonary, otitic, upper respiratory, dental, or dermal suppuration. He described six cases of metastatic bursal abscess: subacromial in four, gluteal in one, and prepatellar in one. These occured as a complication of acute mastoid suppuration (2), pulmonary suppuration (2), stapholococcic septicemia

secondary to a carbuncle (1) and unknown in origin (1).

When discussing the etiology of bursitis of the shoulder bursa, Codman's work must be reviewed. He states that the subacromial bursa "is not a structure where disease starts, so much as it is a structure which limits disease in adjacent structures by temporary adhesions causing fixation temporarily". He points out that the supraspinatus tendon (because it is inert and avascular) is the most vulnerable part of the joint. The common precipitating cause are: (1) a direct bruise on top of the anterior part of the shoulder while the arm is in dorsal flexion, (2) prolonged hyperabduction, that is 1-2 hours or longer, (3) inflammation extending from about a calcified deposit which has either burst into the bursa or is close enough to inflame its lining, (4) rupture of the supraspinatus tendon by trauma of sufficient degree to make a direct opening between the joint and the bursa (this leads to bleeding into the bursa and more or less distension with joint fluid), and (5) minor ruptures occassionally produce acute bursitis, but may be almost symptomless. These are usually subacute and sometimes become chronic.

In a study of 100 cases of subacromial bursitis in 1934, Rogers noted that the disease was fairly common among diabetics, coming on insidiously and

accompanied by almost complete loss of abduction. He theorized that this may be because diabetes makes the tissues "older", therefore a degenerative process. Stimson (1940) describes subacromial bursitis of a toxic origin, coming on suddenly with obscure cause, acute pain, sometimes crepitation, inability to abduct and rotate the arm outward, tenderness, swelling or not, and early deltoid atrophy. She also stated that other shoulder bursae are rarely inflammed, but noted the possible adventitial formation of an "acromial" bursa, due to habitual friction or weight lifting on the shoulder such as may be seen in porters and hod carriers, which may become inflamed and tender. Pelner (1944) suggests that subacromial bursitis may be caused by exposure. An article of interest written by Dynes in 1943, points out the frequency of subacromial bursitis seen following hemiplegia and Parkinson's Disease. The common factor in these neurological disorders is mechanical, a tendency toward immobilization of the arm for a variable length of time. He states that "one can only speculate on whether this promotes adhesions in the bursa because of inability to use the arm, or impairs circulation, or whether there is a trophic element present". Young (1944) substantiates Codman's theory that the condition is due to destruction and degeneration

of the tendons from ordinary use and that the symptoms develop when the deposits of calcium rupture into the bursa.

In the elbow, the olecronon bursa is most often diseased. It is very generally agreed, even by those first describing this entity known as "Miner's Elbow", that it is most frequently caused by direct injury. This is usually by fall, contusion, or by irritation due to repeated knocking of the elbow against objects such as working with tools in close quarters, (miners and plumbers and shipyard workers). Reder (1925) observed that chronically enlarged bursae are especially susceptible to acute inflammatory disease, and stressed the importance of inflammation via the lymphatics.

Osgood (1922) believes the etiology of radiohumeral bursitis to be an irritation by repeated violent extension in the pronated position, as in using a tennis racquet, hence the name "Tennis Elbow". This exercise is usually repeated. In an article by Carp in 1932, the theories as to the etiology of "Tennis Elbow" were summarized. They ranged from: (1) osteitis of the epicondyle (Bergman, Bertoichi, Ferraro, Merlini), (2) myofascitis of the extensor origins (Cooke, Romer, Edgar, Bryce, Cluzet, Hansson and Horwich, (3) arthritis of the radiohumeral joint (Wachendorf, Oschsenius), (4) ad-

hesions (Mills), and (6) radiohumeral bursitis (Schueller, Carter and Dittrich). Carp sides with Osgood's theory.

Disease of the other elbow bursae is so rare that it is clinically quite unimportant. The interosseous and bicipitoradial bursitis types may be caused by irritation from repeated violent motions, as in pitching a baseball, according to Stimson (1940).

To discuss the etiology of bursitis in the hip joint, the subject must be divided into two groups, bursitis of the trochanteric bursae and bursitis of the iliopectineal or iliopsoas bursae. Swindt (1923) states that traumatism seems to be the chief factor in the etiology of trochanteric bursitis. He attributes acute bursitis to sudden violence and chronic bursitis to prolonged irritation. The violence may be external (blows or falls) or internal (convulsive contraction of the overriding muscle as seen in active physical sports). Infection is a second factor of etiology and according to him, is usually secondary to traumatism. This is seldom due to penetrating wounds, therefore the infection is often metastatic in origin.

Sandstrom (1938) and Schein and Lehmann (1941) voice a theory of etiology similar to Codman's in the shoulder, that is that the low vascularity of the contiguous tendons leads to their calcification with

resultant irritation to the trochanteric bursa.

According to O'Connor (1933) the ascribed causes for iliopsoas bursitis are tied up in sudden muscular action involving the psoas muscle while the thigh is flexed upon the body. His explanation is that the bursa is in front of the hip joint and attached posteriorly to the joint capsule. In front of the bursa is the tendon of the psoas to which the bursa is also attached. When the thigh is flexed upon the body (or the body on the thigh) the psoas tendon rides away from the front of the hip joint. In this position, the bursa is stretched between the capsule of the hip joint and the psoas tendon. Thus, sudden vioent action of the psoas caused sudden stretching of the bursa and it is this injury which initiates the pathological changes.

Finder (1939) believes the two important etiological factors to be trauma and arthritis, frequently combined. He drew this conclusion because of the results of Gatch and Green (1925) in which they reported without exception, investigation of cases of rheumatic conditions disclosed a communication between the hip joint and the bursa. Therefore the inflammatory condition of the synovia would logically be mirrored.

by the bursal lining. He then substantiates O'Connor's theory on muscular trauma.

In bursitis of those bursae about the knee, everyone is agreed that trauma is far and away the single most important factor. In prepatellar bursitis and infrapatellar bursitis, kneeling occupations are especially important. In these two, the hazard of puncture wounds is also very important. Stimson (1940) points out that the infrapatellar bursa may also be inflamed in connection with "Osgood-Schlatter" disease (tibial tuberosity epiphisitis of adolescence). She also reports that prepatellar bursitis may rarely result from a cellulitis in that region. Ghormley (1930) points out that due to the frequent communication between the suprapatellar bursa and the joint cavity, any involvement of the joint usually means involvement in the bursa.

Voshell and Brantigan (1944) report that imflammation of the bursa in the region of the tibial collateral ligament is most likely due to compression or friction of the bursa between the tibial collateral ligament and the edge of the tibial tuberosity, which may be irregular or roughened due to arthritis or old trauma, by direct contusion (with or without hemorrhage into the bursa), unaccustomed frequent knee action especially that involving full flexion under muscle

tension (squatting). Burman (1944) states that trauma or excessive knee action causes enlargement of the semimembranosus bursa. He is not in agreement with Meyerding and van Demark that enlargement of the bursa is often associated with rheumatoid arthritis or osteoarthritis, for this combination of conditions is rarely seen.

SYMPTOMATOLOGY

Pain, swelling and limitation of motion in varying proportions are the characteristic symptoms of bursitis. Swelling is most prominent in the superficial bursae, while pain and limitation of motion are more prominent in disease of the deeper bursae.

According to Codman (1934) the characteristic symptoms of subacromial bursitis are: (1) pain, on any motion of the arm causing movement of the supraspinatus tendon, particularly abduction and external rotation, (2) apprehension before movement and often a sort of "catch" in the abduction, (3) there may be exquisite point tenderness, at the point of the shoulder or slightly anterior thereto, and (4) swelling which may be quite extreme when the supraspinatus tendon has ruptured completely, allowing communication between the bursa and the joint cavity. Rogers (1934) believes that internal rotation of the shoulder is also restricted. Pelner (1944) states that the pain may be so severe that morphine has to be used for relief. The pain may also radiate, to the neck, arm and forearm and even the finger tips. Wilson (1939), in his work, found that in cases due to rupture of the contents of a calcium deposit into a bursa, the pain was acute and could

come on suddenly enough to wake the patient out of a sound sleep. In 1943, Haggart and Carr did a study of chronic adhesive subacromial bursitis from which they concluded that: (1) marked loss of shoulder function may be the only complaint, (2) atrophy of the shoulder musculature is dependent upon the duration of the disability, (3) some patients may have pronounced loss of finger joint function, the hand being swollen, similar to atrophic arthritis, and (4) that localized tenderness is much less acute than that seen in subacromial bursitis with calcification.

The symptomatology of bursitis at the elbow is divided into that of the olecronon bursa and that of the radiohumeral bursa. Apparently the symptomatology and diagnosis of the diseased olecronon bursa is obvious and not difficult, for it is made very light of in the literature. In the acutely inflamed, according to Ghormley (1939), the patient notices a fluctuent swelling and pain, the intensity of which depends upon the degree of inflammation. Reddening of the skin and increase in surface temperature may also be noticed if infection is present. In chronic cases, there may be little or no pain even though acute trauma has caused an extravasation of fluid. Some tenderness, but no increase in the surface temperature is found and if fluid is present there

is fluctuation.

The symptomatology of radiohumeral bursitis is thoroughly described by Osgood in a paper published in 1922. He stated that the onset is rarely sudden, thought the symptoms may be well established within a few hours after some physical exertion. There is frequently discomfort, sometimes acute pain, arising in the elbow joint following a game of tennis, some continuous labor such as painting a ceiling in an awkward position, hammering, and etc. There is tenderness directly over the external epicondyle of the humerus, its location varying with the position of the elbow: flexion, extension, pronation and supination. In right angle flexion and pronation the tenderness may shift slightly mesially and distally. Another typical annoying symptom is a sudden sensation of weakness and pain, often running down the forearm, when one attempts to lift an object such as a glass of water or a book with the elbow partially extended with the forearm muscles, especially the extensors, set. It is difficult for them to anticipate this disability. These patients may be able to lift a heavy weight with the arm extended sans discomfort, but carrying a weight for any length of time produces a feeling of discomfort, weakness and ache, which may be come acute.

In the hip region, the symptomatology depends upon which of the two major bursae are involved, the trochanteric or the iliopsoas. In 1923 Swindt described the symptoms relevant to disease of the trochanteric bursa. He states that early there is inflammation tenderness over the site of the bursa, usually posterior and lateral to the greater trochanter. There is also pain which is greatly augmented when the limb is held firmly in counter rotation by the examiner. The characteristic attitude of the limb is flexion, abduction and external rotation, the position which best relaxes the aponeurosis of the gluteus maximus and tensor fascia femoris. The patient may also complain of a swelling or tumor formation posterior to the trochanter, and underneath the aponeurosis of the gluteus maximus, which obliterates the hollow behind the trochanter. These may be large enough to obliterate the gluteal fold and the patient may complain of pain along the course of the sciatic nerve due to pressure upon it by the tumor. Schein and Lehman (1941), in a paper dealing with acute trochanteric bursitis with calcification, tell us that the onset is often quite acute, the patient being unable to walk within 48 hours from the first indication of disease. In an article on tuberculosis of the bursae in the region of the hip joint, Farr (1944) tells us that

the presenting symptom in patients with tuberculosis of the trochanteric bursa, is usually weakness in the affected hip on exertion. Limping after exercise is not unusual.

Swindt (1923) also describes the symptomatology related to disease of the iliopsoas bursa. He states that the patient complains of pain, increased by extension of the thigh and decreased on flexion of the thigh. The tumor may present in Scarpa's triangle, and may extend into the inguinal canal and thus produce pain in the region supplied by the femoral nerve. In 1933 O'Connor presented a very complete description of the symptomatology of disease of the iliopsoas bursa. According to him the commonest symptom is pain in the anterior aspect of the hip joint. This is indefinite in character, throbbing or aching, and frequently radiates to the front of the knee due to irritation of the anterior crural nerve in its course anterior to the bursa in front of the hip. The pain is aggravated by activity. There is often dragging of the leg, a limp, stumbling, or weakness of the leg due to atrophy of the psoas muscle which takes place soon after the onset. This is accentuated in general fatigue or in presence of toxicity. Though rare, edema of the extremity may be present when the enlargement is sufficient to cause

pressure on the great saphenous vein. Localized swelling on the anterior aspect of the hip occurs in late stages and indicates an old advanced bursitis or an active infectious bursitis. A so-called flexion deformity may be present early to a slight degree, but is most often seen late when the bursa is very large. Finder (1938) describes an insidius onset, following a half forgotten accident or unnoticed repeated minor occupational injury. The pain gradually developes, first noticed after the fatigue of a day's labor, and later efforts such as arising from a chair may evoke pain in the inguinal region. There may also be point tenderness just below Poupart's ligament, halfway between the symphsis pubis and the anterior superior iliac spine. There is pain on active motion and less on passive motion. He quoted Timmerman's observation that the pain may occasionaly radiate to the abdomen or back.

As described by Swindt (1923) bursitis in the ischial-gluteal bursa is accompanied by great discomfort in sitting and pain and functional disturbances in the area of the distribution of the inferior pudendal nerve, the perineum and external genitalia.

Writers have been especially negligent in their work dealing with the symptomatology of bursitis about the knee. According to Stimson (1940), prepatellar

bursitis is characterized by swelling in particular. Patients complain more of discomfort than pain except in cases where purulent infection is present. In 1939 Ghormley lightly described infrapatellar bursitis and noted that pain was complained of immediately below the knee which can be increased on resisted extension of the knee. Here also may be point tenderness. These observations are confirmed by Stimson in 1940. In 1936 Snodgrass stated that painless swelling of the knee is often the complaint in disease of the suprapatellar bursa. Ghormley describes a painful fluctuant swelling above the knee which is especially evident on either side of the quadriceps tendon. According to Ghormley, the evidence of inflammation in the pretibial bursa is in finding a tender fluctuant swelling superficial to the tibial tendon at its attachment to the tuberosity.

The symptomatology of disease of the bursa in the region of the tibial collateral ligament was described by Voshell and Brantigan in 1944. The patient usually complains of repeated attacks of pain in the knee following weight bearing flexion (squatting), or perhaps contusions to the area. There may be complaints of swelling and point tenderness in the region of the tibial collateral ligament.

Symptoms related to inflammation of the hamstring

bursa are described by Ghormley (1939). He noted that pain is an early symptom and may precede visible swelling. In many however, painless swelling is the only complaint, the swelling being most prominent when the knee is extended. Even though the sac does not connect with the joint cavity, there is often pain in and restriction of motion of the joint and signs suggesting internal derangement. In 1940, Stimson stated that patients with popliteal bursitis complain of tenderness on pressure and pain on knee extension which is relieved on knee flexion. They are apt to obtain relief from pain by walking or standing with the knee flexed.

DIAGNOSIS

The diagnosis of a bursitis can usually be definitely made. The superficial bursae in particular offer no great diagnostic problem while the deeper bursae present a more diverse picture. The use of inspection and palpation are invaluable in all cases. In addition, the use of the roentgen ray may show evidence of calcification in the region of certain bursae which makes positive the diagnosis. In some of the deep bursa, exploration alone can clinch the diagnosis.

The more important conditions to be differentiated from inflammation in the various bursae are: muscular and ligamentous sprains, acute and chronic tuberculous arthritis or synovitis, osteomyelitis, osteitis and periosteitis.

According to Codman (1934) there are five main points in the diagnosis of subacromial bursitis. These characteristic signs are: faulty abduction, pain, spasm, swelling, and hesitancy on abduction. The pain is most marked on abduction and external rotation. There is also localized tenderness over the greater tuberosity, just below the acromial process. In 1940 Stimson listed the important diseases of the shoulder one should carefully differentiate from bursitis. These are: traumatic

synovitis of the shoulder joint proper, arthritis, scapulohumeral fibrosis, rupture of the long head of the biceps, muscle sprain, brachial neuritis, myositis and myalgia. He also points out an important contrast between bursitis and true shoulder joint involvement, which is intracapsular. In the latter, the characteristic tender point is anteriorly over the region of the long head of the biceps brachii; while in subacromial bursitis, the tenderness is under the middle of the belly of the deltoid, laterally. Another clinical finding, described by Haggart and Carr in 1943, is the generalized bone atrophy seen in the shoulder girdle on X-ray diagnosis of the calcium deposits either in the supraspinatus tendon or in the bursa itself after rupture of the tendon.

Because of its subcutaneous location, the diagnosis of olecronon bursitis is not usually difficult, and is, according to Stimson (1940) characterized by fluctuant swelling over the proximal portion of the ulna. There may be reddening and increased skin temperature if there is bone involvement in connection with the bursitis; which is an important and none too frequent complication owing to the superficiality of the bursa and the cancellous nature of the bone underlying it.

In addition to the typical symptoms attributed to

radiohumeral bursitis described previously, Osgood (1922) states that careful inspection of both arms with each in the same position, there may be noticed a slight obscuration of the normal contours of the affected elbow, consisting of a slight fullness over the distal tip of epicondyle and extending distally and mesially, filling up the slight normal depression over the radiohumeral joint. On palpation, he describes a feeling of increased elasticity over the area of tenderness. When a fist is made and the wrist plantar is flexed, there is pain at the site of the lesion; but if the wrist is dorsally flexed, relaxing the extensors, this pain lessens or disappears. When the elbow is fixed in full or partial extension, especially if the hand is supinated, there is pain when lifting an object. In addition, there is often pain on sudden extension of the forearm. In 1929 Dittrich declared that there is nothing diagnostic on X-ray. This seems to be contested only by men who have other theories for the cause of "tennis elbow" than the bursitis theory advocated by Osgood and many others since his work. Dittrich also states that paresthesias and even anethesias have been found in the distribution of some of the nerves of the forearm and have thus lead to a diagnosis of a traumatic or toxic neuritis. Because the etiology and pathology of the so-called

tennis elbow is so contraversial, one must exclude several possibilities when making a diagnosis of radiohumeral bursitis, among which are: osteitis of the epicondyle, myofascitis of the extensor origins, arthritis of the radiohumeral joint, adhesions and an additional condition described by Prieser in which the contour of the epicondyle, normally on the same plane as the head of the radius, projects beyond the epicondyle.

Swindt (1923) based the diagnosis of trochanteric bursitis upon finding inflammation and tenderness over the site of the bursa, upon pain which is greatly augmented by firmly holding the limb in counter rotation and upon the characteristic attitude of the limb, flexion, abduction and external rotation. In advanced cases, a tumor often developes which can obliterate the gluteal fold. In differentiating it from conditions of the hip joint proper, he notes that in this type of bursitis there is no swelling in the hip joint and the head of the femur can be rotated in the acetabulum without pain. In 1941 Schein and Lehmann list exquisite tenderness over the hip joint proper as essential in diagnosis of this condition. In addition to these, they believe X-ray to be useful in picking up many of these conditions due to the presence of calcium deposits. To be ruled out are: (1) lesions of the hip joint proper,

(2) lesions of the trochanteric region. These may be: fractures, arthritis, osteomyelitis, traumatic periostitis and even neoplasms (Ewing's tumor). The relative freedom of flexion and extension as compared with abduction, adduction and rotation seen in bursitis is important in differentiating it from other joint disease. X-ray is also important in differentiating calcific lesions in the bursa from other hip diseases, but when there is a history of recent injury, it may be confused with an avulsion fracture of the greater trochanter.

In discussing the diagnostic signs related to iliopectineal bursitis, O'Connor (1933) states that early inspection is negative. Later there may be a flexion deformity or merely limitation of hyperextension. In later stages, a tumor mass presents itself in front of the upper end of the affected thigh. Before this, palpation may reveal a slight fullness in Scarpa's triangle, but here differentiation from lymph node enlargement may be difficult. He believes the most consistant finding leading to a diagnosis of this condition is a point tenderness, the area of which is never over two centimeters in diameter and is always located at a point over the front of the upper thigh or inguinal region. This point is just below Poupart's ligament, halfway between its attachment to the anterior superior

spine and the pubis. It is about two centimeters lateral to the femoral pulsation. In addition, active hyperextension of the body on the affected hip will cause pain in front of the hip. Active acute flexion of the hip from the sitting position with the knee extended gives pain in front of the hip and the maneuver is weak in its performance because of psoas weakness. Extreme passive flexion of the thigh on the body also causes pain at the bursal site. Finder (1938) included a history of a direct blow in the groin or a sudden hyperextension force at the hip in his list of diagnostic aids. He also reports that fluid may be aspirated to aid the diagnosis.

Finder also included a fine list of conditions about the hip to be differentiated from iliopectineal bursitis. Inguinal hernia can be ruled out by its reducibility, transmission cough wave, and frequent impediment of the motion in the joint. Tuberculosis of the spine or sacroiliac joint may be ruled out on X-ray. Acute psoas abscess usually tracts upward or downward into the sheath of the iliacus muscle and seldom goes deep to the inguinal ligament into the thigh. Malignant conditions develop insidiously and the patient declines rapidly; while bursitis patients are usually in good physical condition. Less commonly

confused are: dilation of the femoral vein, aneurism of the femoral artery and inguinal lymphadenopathy. In absence of swelling, osteoarthritis, (frequently concomitant with bursitis) cannot be excluded as a possibility. Here the greater age of the patient, frequent involvement of several joints, lateral localization of pain over the neck of the femur, and the X-ray picture furnish the differential. Primary psoaitis is sometimes seen in childhood and adolescence to be confused with bursitis, but the hip joint is free and X-ray may show an enlarged psoas shadow.

Because of its superficial location, the diagnosis of prepatellar bursitis offers no difficulty. The fluctuent swelling over the patellar region is characteristic. The diagnosis of inflammation in the infrapatellar bursa is likewise not much of a problem. Ghormley (1939) describes pain immediately below the knee which is increased on resisted extension. There is also characteristically a filling out of the usual depressions on either side of the ligament and tenderness on palpation. Stimson (1940) points out that with both infra and prepatellar bursitis, pain on kneeling is the common complaint. Breck and Higinbotham (1946) emphasize the fact that both of these bursae are often diseased at the same time and that they may imitate Osgood-

Schlatter disease. This differentiation can be made by the use of X-ray. In 1939 Ghormley described supra-patellar bursitis mentioning that the painful fluctuant swelling develops above the knee and is especially evident under and on either side of the quadriceps tendon. He noted that calcification can occur and if so, is easily demonstrated by means of X-ray. It was stated that the diagnosis of superficial pretibial bursitis is made by finding a tender fluctuant swelling at its location.

In 1940 Stimson described the diagnosis of bursitis of the hamstrings. These swellings are usually not large but can be brought out to inspection and palpation by having the patient hyperextend the knee. The patient usually complains of tenderness on pressure, pain on extension of the knee and notices relief from pain upon knee flexion. Burman (1944) stresses the importance of examining the popliteal space for diseased conditions of these bursae whenever one suspects tears in the internal meniscus, for these two conditions are sometimes found together.

In the diagnosis of bursitis in the region of the tibial collateral ligament, Brantigan and Voshell (1944) seem authoratative. The only absolute proof of diagnosis is excision of the bursal tissue followed by symptomatic relief. There may be visible and palpable enlargements

of varying sizes beneath the tibial ligament. These enlargements are always tender on pressure and when the ligament is tightened under hyperextension, abduction or external rotation of the lower leg they are semi-fluculent, as though there were fluid under pressure. They mention aspiration of fluid as an important positive sign. The area of tenderness is always pointlike, usually between the ligament and the tibial edge, though it may be over the base of the meniscus. Here it is of much more limited scope than a torn meniscus, which is apt to be confused with it. Fracture of the tibia and a torn ligament are much more severe and are usually associated with acute trauma, which makes confusion with these unlikely. X-ray is important in differentiating this type of bursitis from such formations or exostoses on the tibia and fibrositis of the ligament and subadjacent fascia. In the latter there is soft tissue calcification and swelling.

TREATMENT

The treatment of the various diseased bursae is exceedingly diverse and very contraversial. Two main factors influence the type of treatment to be instituted. The first is the type or stage of the disease; is it acute, subacute, or chronic? The second is the location of the bursa; is it deeply placed, or is it superficial?

In 1931, Foster advocated surgical removal whereever possible. Where complete excision was not possible, he condoned amputation of the anterior wall and as much of the edges as possible, for on occasion the bursal floor is thickened and firmly tied down. This procedure was followed by curettement of the lime salts from the part of the wall left in situ. His postoperative care was complete rest for the joint for a period of ten days plus infra red ray treatments twice daily for 30 minutes, continued one week after motion is permitted. The average disability time for deep bursae treated in this manner was 4 weeks and that for superficial bursae, two weeks.

In 1939 Coulter published an article on the efficiency of short wave diathermy in producing deep heat for the treatment of bursitis. His experimental work showed that the temperature rise in the quadriceps

muscle 2 inches below the skin was 0.25 degrees Fahrenheit for a hot water bottle and 2.25 degrees Fahrenheit with long wave diathermy as compared with 7 degrees Fahrenheit for short wave diathermy. The dosage is regulated by the patient's skin tolerance to temperature. This is tested with hot water in a test tube for skin tolerance to temperature beneath electrodes is subnormal. He listed contraindications to this form of therapy as: (1) acute inflammatory precesses, (2) any condition in which there is a possibility of hemorrhage, (3) the presence of peripheral nerve injuries where sensation to heat on the area is diminished, (4) through the abdomen or pelvis in pregnancy, and (5) over areas where there is a suspected malignancy. Application by electromagnetic induction may be accomplished in two ways: (1) by coiling the cable about the extremity over a half inch of terry cloth and (2) by means of the "pancake" or flat coil technique on a terry cloth pad.

Stimson (1940) condemns aspiration of fluid except for diagnosis because (1) it frequently leads to complications such as infected bursae and (2) it is replaced in a short time by more fluid. Also condemned by her are stab wounds and excision used alone. Her conservative treatment for simple or uninfected superficial bursitis is the use of a rubber sponge dressing, cut

to a shape larger than the bursa, and bandaged in place to compress the bursa firmly without embarrassing the circulation. She claims cures within 2 to 3 weeks. A saturated solution of potassium iodide, 10-15 drops three times daily, is said to hasten resolution of the effusion and bursal swelling. If the bursa is acutely inflamed with cellulitis or lymphangitis, she advocates continuous hot boric acid fomentations and elevation and rest of the member until it subsides or localizes, followed usually by bursectomy for a cure. Her surgery is accomplished by a curved semilunar incision, approximately in line with the bursal edge, extending halfway around its border. The flap is then reflected and the bursa removed in mass. A rubber drain is used to keep the scar from spreading.

In 1942 Burrows described some general principles of treatment. For acute suppurative bursitis: (1) incision and insertion of a rubber drain (preferably at the lowest part), (2) if an abscess is pointing, conservative measures in the form of heat, (3) local rest and general treatment as with any acute infection and (4) do not excise the bursa while infection is present. For acute serous bursitis, removal of the source of irritation is often followed by disappearance of swelling and other inflammatory changes. Improvement

may also be hastened by aspiration and pressure dressing. In dealing with chronic bursitis, excision is desirable.

From the standpoint of treatment, subacromial bursitis has attracted by far the most attention in the literature. In 1934 Codman described the surgical removal of calcium deposits from the bursa itself, and the tendon adjacent to it. In most instances, the calcareous deposit is still within the supraspinatus tendon, and when incised through the bursal floor, it exudes with somewhat the consistancy of toothpaste. Codman makes no attempt to curette out all of the material for he feels that the less damage done the tendon the better. The cavity is merely wiped out and no attempt is made to close either the incision in the tendon or in the bursa, because he believes that it is better to allow the fluid formed by synovial secretion to seep into the areolar tissue, washing out any particles of calcium or blood remaining in the bursa, thereby decreasing the chances for the development of adhesions or prolonging inflammation. Postoperative pain is also less frequent because there is no distension of the bursa. The muscle is closed with a few loose cat gut sutures to prevent a depression between the fibers of the deltoid. Postoperatively, he is inclined to allow any free use of

the arm which does not cause pain. Let it be reiterated here that Codman believes that in most cases, the particles of calcium would be eliminated through leukocytic action within a few weeks, even without treatment. Treatment of the above description is, however, indicated in those cases where the inflammatory reaction is so intense as to cause the formation of adhesions, resulting in "frozen" shoulder.

Following certain ideas and suggestions of M.N. Smith-Peterson of Boston, Patterson and Darrach (1937) treated 63 cases of subdeltoid (subacromial) bursitis by double needle irrigation. The affected shoulder is prepared with alcohol and iodine, then a small wheal is made with novocain over the area of maximum tenderness (usually a spot one inch lateral to, and on the same horizontal line, with the coracoid process of the scapula). The skin is then nicked with the point of a scalpel and in a like manner, a second area is infiltrated, about ¼ inch posterior to the greater tuberosity of the humerus on a level with the superior facet. Now nick the skin here. Introduce one large needle through the anterior nick directing the point posteriorly and upward toward the undersurface of the acromial process. When inserted 1/2 to 3/4 inch, resistance is felt, one quick stab places the needle within the bursa, and when

the bursa is greatly distended, a cloudy fluid may push the plunger back. Now introduce a second needle into the posterior nick, pushing it down gently to the superior facet of the greater tuberosity until actual bone is felt; now withdraw 1/8 inch and direct the tip toward the assumed position of the tip of the anterior needle. When this needle has been inserted ½ inch, the bursa has again been entered. Novocain is used in both needles on the way in. Using a 20 cc. syringe, saline can be flushed in one needle and out the other. A show of calcium indicates that the needles are in correct position and that the bursae are being washed clean. Use 30-60 cc. to wash, then withdraw and apply sterile dressings. Now the patient can usually move the arms in all directions without pain. Early motion is requested, for usually the acute pain is gone. The period of disability is one week or less and complete relief was obtained in 57 of the 63 cases. This method was most successful in: (1) very acute cases with no history of previous attacks, (2) cases in which the calcium on X-ray was very fuzzy, and (3) cases in which the pain was well localized.

Wilson, in 1939 and Milgram in the same year, reported excellent results from needling the bursa only. They theorized that the needling results in opening

channels for capillaries to remove the deposits, thereby alleviating the symptoms.

Kaplan and Hawkins (1915) reported on 18 patients treated by needle aspiration of the calcium deposit (usually not successful), followed by infiltration of the deposit and surrounding area with 20-30 cc. of normal saline, at the same time making numerous punctures into the bursa. There is usually immediate relief to be followed by an increase in symptoms for the first 24 hours. The patient should be encouraged to limber up the arm under a hot shower the first few days. Of the 18 treated, 77% responded favorably, the average time for symptom disappearance being six days and the average time for disappearance of the deposit, 7 weeks. More than one such treatment was required in the other 23%.

Coulter (1939) claims that an acute attack of subacromial bursitis can be relieved in 2 weeks when the following treatment is prescribed to: infra red radiation for 30 minutes daily, short wave diathermy for 20 minutes daily, and careful massage and passive motion as pain diminishes followed by exercise later. In a few cases, diathermy aggravates the pain; these he puts to bed in adhesive traction and continuous moist heat. He recommends surgical removal if these conserv-

active measures are not rewarded.

In 1943 Echtman published the conclusion that calcified bursitis can be cured 100% in 95% of the cases if physiotherapy is properly given. He classifies his problem into 4 conditions to deal with: (1) acute pain, (2) calcium deposits, (3) adhesions and (4) muscle atrophy. There are two points for the operator to bear in mind: (1) heat in any form aggravates symptoms and prolongs the stage when given to acute cases and (2) the only heat giving modality which is specific for exerting absorptive effects on calcium deposits is long wave (conventional) diathermy. Heat is the primary and basic form of treatment for calcium deposits, provided one uses the proper thermal measure. For the management of acute pain, he uses ionization with magnesium sulfate solution. The technique is too involved for explanation in this paper, but the rationale of this so-called ion transfer is very interesting. First Echtman explains that in any inflammation, three factors are responsible for pain: (1) disturbance in pH, (2) hyperemia and (3) edema. In acute inflammation, local or systemic, due to trauma or disease, the hydroxyl ions increase in the involved tissues, thus alkalinity rises. Alkalinity is an irritant and causes pain. Ionization with the magnesium ion causes the

the tissue to lose alkalinity, thus resulting in pain relief. Furthermore, the positive pole liberates oxygen from which acid is formed, which in turn helps neutralize the alkalinity. The positive pole also acts as a vasoconstrictor, thus reducing hyperemia, and the magnesium ion has a favorable effect in reducing edema. Finally, the magnesium sulfate has an anesthetic effect, which is enhanced by the ionization. If the pain is too severe, he resorts to cold applications until the acute process is ended. He reports these results to be good, but much slower than iontophoresis.

In the subacute stage, Echtman uses some form of heat modality with or without massage. This has a double purpose, to learn whether or not the inflammation is under control (for heat will aggravate the pain if the acute stage has not passed) and to accustom the shoulder to the thermal measure to be employed in causing absorption of calcium deposits. He believes long wave diathermy best for removal of calcium deposits because, though both long and short wave provide thermal heat (the most important measure,) long wave is superior for thermal concentration. There seems to be proportionately more heat caused in bone and calcium deposits than in muscles and circulating blood due to the resistance of fixed by the former to this current.

With other forms of heat, the opposite is true.

For adhesions, Echtman prefers ionization with sodium chloride solution, followed by interrupted sine wave current producing about 22 rhythmic contractions and relaxations per minute. This interrupted sine wave current, he also recommends for treating muscle atrophy.

In 1936, Lattman was instrumental in popularizing the use of X-ray therapy in treating subacromial bursitis. Briefly, his technique consisted of 200 peak kilovolts though a 0.25 mm. copper and 1 mm. aluminum screen. The field was 20 by 20 cm. at a distance of 50 cm. 350 Roentgen units were given at each treatment. During the first 24 hours, patients complained of aggravated symptoms, but were much improved within 48 hours (15 of 20 patients responded well).

Jones (1941) reemphasized Lattman's work and quoted a series of cases in which he used Lattman's technique to obtain the following satisfactory results: 14 of 20 needed but one treatment and 2 of the 20 needed 3 treatments to obtain relief. Within 2 months, 3 of the patients had no calcium present (he was quick to acknowledge that this could occur without treatment).

In 1943, Harris published a paper on a series of 40 cases treated similarly by X-ray. His field was

10 by 10 cm. and centered on the point of maximum tenderness, being careful to direct the beam away from the lung field. He described the response as marked relief from pain in 24 to 36 hours with complete disappearance of pain by the third day. The remaining stiffness gradually subsides, usually within a week or 10 days. The calcium absorbs slowly, taking several weeks.

Brewer and Zinc (1943) also claimed good results in 11 of 14 cases using similar X-ray therapy. In acute cases, they considered those lacking improvement in 48 hours to be failures, and stated that operative proceedures should be considered. Neither did they claim "quick" responses for chronic cases. 30% were improved in varying degrees, but cures were rare. Lack of improvement in chronic cases treated for 10 days was considered a failure.

Borak (1945) credits Rench (1926) and Sandstrom (1929) with the early work in X-ray therapy of subacromial bursitis and providing the stimulus for later work. Borak's contribution is important because he outlined the theory behind the improvement after X-ray treatment. First, it is essential to realize that in the average case of subacromial bursitis, we are dealing with loose attachments caused by fibrin, <u>not</u> fixed adhesions caused by fibrous tissue. Experimental work

has shown that wounds he ling under X-ray have less fibrin between the edges of the wound than those not radiated. As a consequence, the resulting scar is thinner and finer in those irradiated. X-rays do not dissolve the fibrin directly, they primarily dilate the capillaries and increase the permiability of the capillary walls. This is followed by an increased flow of plasma from the blood, carrying along lymphocytes, monocytes and leukocytes which have the power to phagocytize and carry away the fibrin and necrotic tissue debris. This shows that X-ray acts not upon the lime deposit, but on the underlying inflammatory process, and the calcium salts disappear in the course of events. It is the postradiation edema carrying phagocytic cells which causes dissolution of the calcium deposits. In acute cases where hyperemia is present, a small X-ray dose is sufficient to increase their permiability and subsequently the precapillary edema. In chronic cases more irradiation is necessary because hyperemia does not already exist.

Borak divides his patients into three arbitrary groups depending upon the degree of motion being present. In those nearly immobilized, he obtains good results with small doses (200 Roentgens on 2 successive days). In those with only 40 to 45 degree abduction he gives

200 Roentgens at 6 to 8 intervals of 2 to 4 days. And for the third group (no loss of motility, but pain at the extremes of movement) he uses 250 Roentgens at 8 intervals of 2 days. He uses 2 portals of entrance, one anterior and one posterior.

In 1946, Gebler published a paper again stressing the value of X-ray in treatment of subacromial bursitis. He irradiates through several portals of entry at each sitting. His chronic cases recieve a treatment or two each week for 6 to 8 weeks.

In 1943, Haggart and Carr published a paper on the treatment of chronic adhesive subacromial bursitis, also known as "frozen" shoulder. They stress first that the patient must have a thorough understanding of his affliction, for cooperation of the patient is necessary for the success of this method. The patient is hospitalized and the arm is suspended in a Balkan frame apparatus, the arm in the maximum degree of abduction that will not cause increased discomfort, and adhesive traction is applied (6 to 8 pounds). Then they employ intense local heat and massage and urge the patient to move the arm as much as possible with comfort. Then intensive active exercises are instituted; sometimes patients increase in passive abduction as much as 30 to 40 degrees in 10 days. When the maximum of improvement has been

accomplished, shoulder joint manipulation is carried out under sodium pentothal anesthesia. Normal range is not attempted the first time, and the arm is grasped at the upper end of the humerus so as to decrease the chances of fracturing it. With the manipulation completed, 1% novocain is injected into the bursa, and periarticular structures to reduce postmanipulative discomfort. The patient is allowed to rest the arm 24 hours, then the arm exercises are instituted again. The manipulation is performed again when the patient has obtained a new maximum of improvement. If the chronic adhesive bursitis is complicated by calcification, they use the needling technique in the manner of Patterson. They describe two indications for open operation: (1) those so resistive to manipulation that it seems dangerous to try to rupture them (in this, they explore the bursa with a knife and thereafter employ the usual manipulative proceedures) and (2) patients who have a calcium mass which does not respond to needling. Of 100 cases, 47 obtained complete relief, 34 were markedly improved, 5 were improved but lacked an adequate follow-up, 5 were unimproved and 9 lacked any follow-up at all.

In 1931 Richards and in 1944 Pelner wrote upon a unique treatment for subacromial bursitis, the intravenous injection of one grain (5 cc.) of iron cacodylate

into the anticubital vein of the forearm. They claim effective results over many years. In fact, they believe that if no relief results, two conditions may exist: (1) wrong diagnosis and (2) calcification of the supraspinatus tendon present as a "complication". In typical acute cases, they describe immediate increase in range of motion with relief of pain. Pelner cited 75 cases over a 5 year period in which the results were invariably favorable. In the failures, morphine gave less relief than the iron cacodylate. The rationale is not known. Its toxicity is considered negligible, but there is one important unrelated side response. Iron cacodylate delays initiation of clot retraction, or even entirely inhibits it for 15 to 20 hours.

The treatment of olecronon bursitis has received its share of attention over the years. Of historical interest only is Rosen's (1940) description of the treatment for "Miner's Elbow" taken from the LANCET, Saturday, May 21, 1842. The article is a letter written to the editor from Edwin Gurney of Camborne, Cornwall, in which he writes: "in the early stage tepid bathing with water and a warm bread and water poulice, three times daily; and after this has been applied for a few days, and the inflammatory reaction subdued, the dispersion of the fluid should be accomplished, either

by application of about ½ drachm of the hydriodate of potash and iodine ointment combines, or of a lotion of 2 drachms of tincture of iodine to an ounce of rectified spirtis of wine; to be used in drachm applications over the tumour three times daily".

Lasher and Mathewson (1928) advocated the earliest possible removal of the bursa. They suggested an elliptical incision so the flap is toward the radial side, thereby lessening the danger of injury to the ulnar nerve. Due to the difficutly in separating the attached portion of the bursa without opening the periosteum, this small area was curetted thorougly and phenolized to destroy any secreting cells and to remove completely, any infectius tissue. The wound is closed without drainage and a tight pressure dressing applied to prevent reaccumulation of fluid. They preferred no splinting, rather motion immediately and continued. Van Alstine (1922) devised a pressure dressing using an old auto tube to maintain pressure, yet allowing for any motion. The tube was cut thus:

In 1939, Coulter recommended short wave diathermy for olecronon bursitis in the acute phase. The elbow immobilized in extension and infra red or baking heat

used 30 minutes with short wave diathermy for 20 minutes once each day. He supported operative removal in chronic cases. Burrows (1942) also advocated surgical removal, often under local anesthesia.

Burgess (1943) is an advocate of internal paracentesis or bursotomy in treating olecronon bursitis, of the chronic, often occupational type. The area is prepared surgically and the skin anesthetized 1/3 the distance from the distal bursal margins and directly over the center with novocain. 5 to 10 cc. of procain is then placed within the bursal sac. A cataract knife is thrust directly through the anesthetized area into the bursal cavity, then tilted distally and with a semicircular motion, the entire distal bursal sac is incised into the adjacent subcutaneous tissues of the forearm. A sterile dressing is applied and the patient is instructed to use the arm freely. Motion tends to keep the communication open and the bursa empty. Surgical cleanliness in redressing is axiomatic. Contraindications for this method of treatment are: (1) rice body formation, (2) presence of acute hemorrhage (use aspiration here), (3) when infectious processes or a neoplasm are suspected. The time to perform is after the acute reaction has subsided and fluid persists.

In 1946 Breck and Higinbotham published a paper

on a new surgical technique in treating olecronon bursitis. Their belief is that any recurrent bursitis is an indication for surgery. Their incision is new because they prefer a midline incision directly over the bursa. This is because large flaps are considered to have a poor blood supply. The bursa is dissected out completely, piecemeal if necessary. They prefer not to use buried ties or even electro-coagulation in destroying secreting cells (because of the charred material this leaves). The important step in their technique is the closure. They firmly mattress the overlying skin to the deeper structures with sutures placed over buttons. The incision is closed without drainage and covered with a moderately firm large pressure dressing. The patient is cautioned not to bend the elbow until the sutures are removed on the fourteenth day. This technique is claimed to be effective in preventing postoperative accumulation of blood at the site formerly occupied by the bursa, which minimizes the chance for recurrence and reduces the possibility of post operative infection. They boast complete cure in all but one in 44 cases with follow-ups of 3 to 10 months. These were performed on infantrymen.

Also in 1946, Cotrell describes a new drainage

technique which resulted in 14 cures in a series of 15 cases of olecronon bursitis characterized by chronic effusion. He first employed sterile aspiration followed by compression bandages, but when these failed, he adopted the following new technique. The skin is surgically prepared and infiltrated over the bursa with 2% procain hydrochloride. A 0.5 cm. transverse incision is made into the sac, into which is placed a specially devised hollow tube, spool shaped soft rubber drain. The contents are allowed to run out; when empty, the sac is refilled through the drain with 2 to 5 cc. of some sclerosing agent. A sponge rubber pad is then cut out about the drain, covered with a fluff dressing and held in place with an elastic adhesive bandage which does not encircle the limb. The drain is left in situ 48 to 96 hours, then removed, continuing with the pressure dressing. Reexamination in 5 to 7 days usually shows obliteration of the sac. Reeffusion is rarely needed and in no instances did infection develop, possibly due to the barrier set up by the reactive inflammation toward the sclerotic agent. The sclerotic agents used were: sodium morrhuate, 5 and 10%, proliferal solution and sodium psylliate, 5%. He attributes the success of the treatment to the use of the spool-like drain.

The treatment of radiohumeral bursitis was first

described by Osgood in 1922. His treatment was surgical and consisted of splitting the conjoined tendon over the bursal site; opening the bursa; evacuating its contents and curetting the walls. In cases of "Tennis Elbow" due to inflammation of this bursa, this procedure is always successful in relieving the symptoms.

Dittrick (1929) reaffirmed Osgood's work concerning the diagnosis and treatment of radiohumeral bursitis. In addition, he pointed out the advantages of using a local anesthia in performing the operation. By infiltrating each layer separately before proceeding through it, the surgeon can be guided directly to the area of inflammation by cooperation of the patient. He also pointed out that the pathological changes are not commensurate with the severity of the clinical symptoms. Carter (1925) is another supporter for surgical excision in treating radiohumeral bursitis.

In 1932 Carp described a conservative measure in treating radiohumeral bursitis. In four of eight cases, he simply used firm digital pressure over the bursa in order to rupture it. Immediate alleviation of symptoms and complete disappearance of signs were dramatic. If this simple manipulation fails, he is quick to advocate surgical removal.

Hanson and Horwich (1930) described a simple

treatment for radiohumeral bursitis in merely using rest, with the arm in a cock-up splint to relax the extensor muscles of the forearm. Coulter (1939) prefers this method also, but in addition uses short wave diathermy for 20 minutes each day followed by radiation.

The treatment of iliopectineal or iliopsoas bursitis has not received attention from a great many men, though it has been recognized as a disease entity since 1934. Gatch and Green (1925) reviewed the literature on the subject and found but 32 cases. They added one of their own and noted that in most cases the etiology was trauma. Therefore, they named rest as the essential principle in treating the acute stage; surgical exterpation in the chronic stage. Eight years later O'Connor (1933) added his support to this principle and noted that heat may aid the restoration of the membrane to normal thickness, and the absorption of fluid. The length of time necessary for complete restoration varies with the efficiency of the treatment, but at best consumes many weeks in chronic cases and is slow even in acute cases. He also recommends repeated aspiration in addition to rest, but condemns incision and drainage in non-infectious bursitis, believing that the only surgical procedure contemplated should be whole excision. In the presence of virulent organisms, he condones sur-

gical drainage--not to be delayed.

In 1934 Ramage and Morton reported on two cases of iliopectineal bursitis, one due to direct trauma, the other due to indirect trauma. They successfully treated them with surgical removal followed by closure with drainage. They suggest cauterization (with carbolic acid) of the posterior portion, should it communicate with the joint cavity.

Finder (1939) described the following treatment for iliopectineal bursitis: for the early stages, bed rest, relief by allowing flexion of the thigh or better still, Buck's extension; heat to hasten resolution, but cold may give more comfort; aspiration to temporarily relieve tension (fails as a cure). For the chronic stage, Finder believes total exterpation is technically too difficult because of the important structures adjacent to the area (femoral nerve, vein and artery), therefore he recommends obliteration of the sac, being careful to irradicate any communication with the joint cavity as suggested in the preceeding paragraph.

In 1923 Swindt wrote on bursitis of the hip region and briefly gave his opinion in regard to treating the trochanteric bursal inflammations. He believed in rest for the acute stages and firmly stated that absolute removal surgically, in the chronic stage, was the only

method resulting in cures.

On the subject of acute trochanteric bursitis with calcification, Schein and Lehmann (1941) supported the following methods of treatment: bed rest, cold or hot applications, or local infiltration of the the calcified area with novocain. In four cases treated in this manner, there were no recurrences with follow-ups from 9 months to 2 years. Disappearance of calcification was verified by X-ray in two of the cases.

Farr (1944) described a treatment for tuberculous bursitis in the trochanteric region, a rare lesion for only five cases were diagnosed at Massachusetts General Hospital from 1870 to 1920. Farr excises the affected bursa, does a partial closure and packs with gauze impregnated with "Bipp". The joint is then immobilized in a hip spica and the pack is changed once per week. Immobilization is continued until healing is complete.

One of the commonest types of bursitis encountered in any region, and by far the most common in the knee joint region, is prepatellar bursitis. Because of this, its treatment has received a relatively large degree of attention. Because of its similarity to the olecronon bursa, much of the treatment is common to both bursae.

In 1925 Reder suggest that surgical drainage is the proper treatment for chronic prepatellar bursitis

(hygroma). He prefers a lateral and medial incision with the use of a through and through rubber dam. This is left in place until all evidence of drainage has ceased (week to 10 days). After the walls have collapsed, the wound may still ooze for several days (drain is left in place until this ceases). After removing the drain, the wound is dressed aseptically. When the hygroma is infected (chronically enlarged bursa are especially susceptible to inflammatory disease) he irrigates the wound with 1:200 bichloride or 3% carbolic acid or 2% tincture of iodine. The limb is preferred splinted to secure adequate rest and drainage. Some are resistant to this form of treatment; he then advocates complete excision to cure.

Carp (1931) reports successful end results in 24 of 27 cases without operative therapy. He aspirates the fluid without anesthesia, then injects 2 to 5 cc. of half strength 3.5% tincture of iodine. The needle is withdrawn and the skin massaged thoroughly to disseminate the iodine in the bursal sac. The patient now complains of a burning pain. The knee is placed in complete extension and an attempt is made to approximate the roof and floor of the bursa by firm adhesive strapping. A pressure bandage is then placed over the knee so that the patient must walk with the knee in full

extension. In 50% of the cases, the dressings can be removed in 5 to 7 days without further treatment. In the other half there is re-effusion to the extent that aspiration must be repeated, and the pressure dressing continued. The average follow-up was 16.3 months.

Diamond (1933) reported on prepatellar bursitis treated with injection of tincture of metaphen 1:200. The technique was similar to that in the above paragraph. In a series of 3 patients it was 100% successful with follow-ups of 8 to 20 months.

Eising in 1934 had a series of 3 patients (1 olecronon, 2 prepatellar). He used aspiration, injection of sodium morrhuate (1 cc. of 5% solution) and a compression bandage. The results were obliteration of the sac in 5 to 14 days, no effusion and the development of a painless, flat fibrotic plaque at the bursal site. He cautions against this treatment in bursae where there may be connections with the joint cavity or tendon sheaths.

In 1939 Coulter treated prepatellar bursitis as he did olecronon bursitis with the same favorable results. Burrows (1942) recommends surgical excision for chronic prepatellar bursitis. He prefers general anesthesia and the application of a tourniquet, retained until the final compression bandage is in place.

He also likes to use a lateral curved incision best.

Burgess (1943) has used internal paracentesis as in the treatment of olecronon bursitis (previously described) with the same success. In the prepatellar bursa, the incision is made through the superior portion, taking care not to enter the joint capsule. If the first incision is inadequate, and premature closure results, the procedure can be repeated. He boasts of no work time lost in this method, other than that necessary for the operation. Breck and Higinbotham (1946) also recommend their surgical technique, previously described in the treatment of olecronon bursitis, for treating cases of "patellar bursitis". They use this inclusive term so as to include infrapatellar bursitis, which in their experience was more common than prepatellar bursitis. And finally, Cotrell (1946) recommends (as he did in the treatment of olecronon bursitis) aspiration, injection of an irritant, withdrawal of some of the irritant and the application of a pressure dressing.

The treatment of semimembranous bursitis has been sorely neglected in the literature, probably because of the rarity of the condition, and because the complaints relevant to it are not serious. Burrows (1942) suggests that bursectomy is justifiable, taking care to

ligate any connection with the joint cavity. Cotrell (1946) has used his drainage technique, with the spool-shaped rubber drain, effectively in two cases of semi-membranosis bursitis (Baker's cyst).

In 1936 Snodgrass treated a case of so-called popliteal bursitis (thought to be a posterior sacculation of the posterior wall of the joint capsule). He dissected the sac free in its entirety in a retrograde manner, until the neck passed around the medial head of the gastrocnemius muscle and joined the synovia of the knee joint. The neck was ligated close to the joint, touched with full strength iodine and closed without drainage. He treats suprapatellar bursitis in the same manner.

Bursitis in the region of the tibial collateral ligament, is a relatively new clinical entity first describe the following treatment of this condition, which is so far as known, the only contribution in the literature. When swelling is present, they aspirate if possible. This is followed by infiltration of the area with several cc. of novocain. If fluid has been obtained, novocain can also be injected into the bursal cavity. The knee is then protected by bandaging or a splint, in order to rest the joint. Diathermy is then used for deep heat. Return of the knee to active use

is done slowly and carefully, avoiding strain and repeated flexion of the joint. If recurrence occurs, repeat the treatment until convinced that conservative measures are of no avail. As a last resort, or if the enlargement causes constant mechanical hinderence of free action of the tibial collateral ligament, excision of the thickened or swollen tissue is recommended. They have treated 10 cases, 4 proven by excision. Novocain injection is often successful, particularly if there is no palpable enlargement.

CONCLUSION

In view of the diversity of methods of treatment for the different forms of bursitis, and the success claimed for each of these methods, one cannot but conclude that there is no one cure. Contrary to the rule that "many methods of treatment mean no successful one", bursitis apparently is quite amenable to more than one form of therapy.

The acute types of bursitis seem particularly responsive to the more conservative forms of therapy, such as rest, hot or cold applications, needle aspiration and injection of various irritating substances, incisional drainage, pressure bandaging, physiotherapy and Roentgen therapy. Indeed, many acute attacks are wholly disposed of by nature's efforts.

Chronic bursitis, on the other hand, is a very stubborn disease entity, and though many men report a few cures with conservative measures, nothing short of surgical extirpation succeeds in relieving symptoms with any degree of surety.

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