5-1-1942

# Subacromial bursitis

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Senior Thesis Presented

to the College of Medicine

UNIVERSITY OF NEBRASKA

Omaha, 1942

TABLE OF CONTENTS

# INTRODUCTION

Duplay, working in Paris, is credited with the first description of bursitis in 1872, but it was not until 1896 that his paper received notice. Putman, 1882, and Monks, 1890, wrote on treatment. With the discovery of x-ray in 1895, the chief advances in our knowledge of the subject were forthcoming. Kuster, in 1902, published a classical description of subacromial bursitis. Codman, who was in Europe at this time, first became interested in the subject, on reading a monograph of Kuster's. In 1906, he gave the first complete thorough description of the bursae of the shoulder. His book, The Painful Shoulder, published in 1934, is considered authoritative.

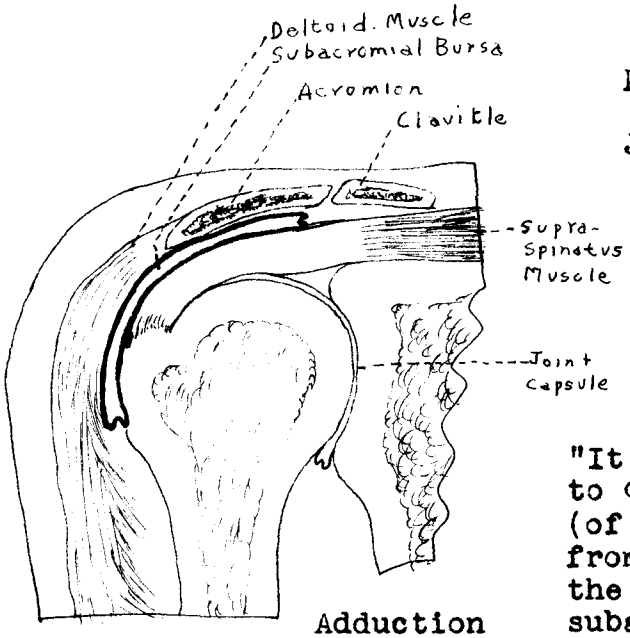
Bursae may be classified as deep or superficial. The deep bursae are those which lie between muscle and moving bony projections. Those most commonly found to give symptoms are subacromial; subgluteal, between the femoral greater trochanter and the gluteus maximus muscle; iliopsoas, between the capsule of the hip joint and iliopsoas muscle; supratrochanteric, in the muscle planes above the greater trochanter; semimembranosus, between the tendon of the semimembranosus muscle and the inner head of the gastrocnemius; subcutaneous calcaneal bursa, metatarsophalangeal bursa; pretibial, between

the quadriceps tendon and the tubercle of the tibia; and radiohumeral, between the extensor tendons of the biceps and supinator brevis.

The relationship of bursitis to occupations reflects itself in the layman's terms for these conditions: housemaid's knee, weaver's, tailor's or coachman's bottom, dustman's bursa and tennis elbow; however, subacromial bursitis is universal. Weeks, 1940 (1), believes that 90 percent of the pains of any moment about the shoulder are due to irritation within the subacromial bursa. Patterson and Patterson, 1940 (2), reported diagnoses of 104 cases of painful shoulder; 70 of this number were due to bursitis, 55 of which had calcification in the tendon, 15 did not. They reported three cases of rupture of the supraspinatus tendon.

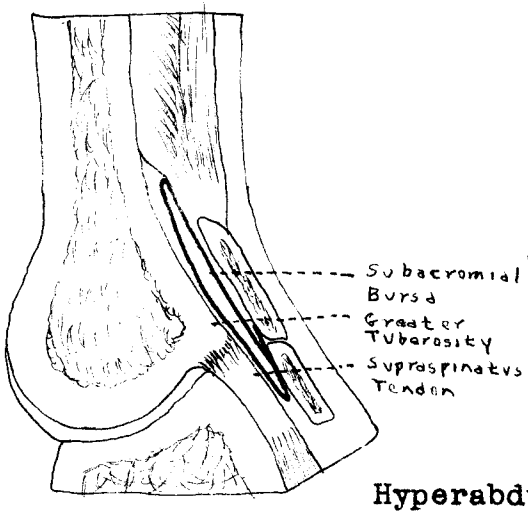
Codman (3) believes that subacromial bursitis is by far the most common cause of painful shoulder. Most writers consider subacromial calcifications as due to isolated deposits in tendons. However, Copeland and Michel, 1939 (4), consider calcifications in the subacromial bursa and supraspinatus tendon as local manifestations of the constitutional condition with calcification of ligaments, tendons, bursae, articular capsules and surrounding tissues known as peritendinitis calcarea. Discussion in this paper will be limited to the non-specific types of subacromial bursitis.

# DIAGRAMS OF THE SHOULDER JOINT AND THE SUBACROMIAL BURSA



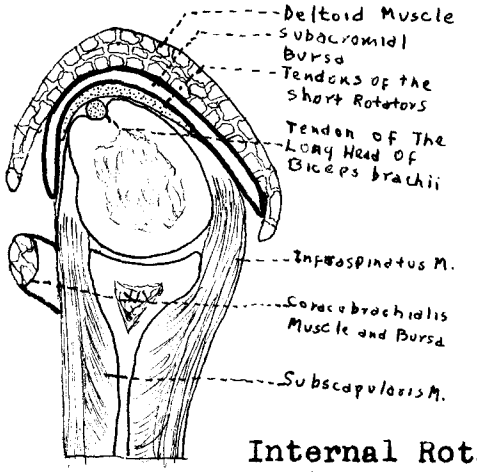

Adduction
Coronal Sect.

"It is impossible either to dissect these tendons (of the short rotators) from the capsule or from the synovial base of the subacromial bursa."
- Codman, The Shoulder, Page 16




Hyperabduction
Coronal Sect.

With the arm in hyper-abduction the scapula rotates superio-medially and the greater tuberosity passes under the acromion process.




Internal Rotation
Sagittal Sect.

The tendons of the short rotators form the base of the subacromial bursa, and roof of the joint capsule.

## ANATOMY OF SUBACROMIAL BURSA

The glenohumeral articulation is enarthrodial in type and as such is capable of a wide range of motion. The joint movement entails not only the passage of the cartilaginous surfaces of the humerus over the glenoid but also of the muscles running from the shoulder girdle to the humerus over their adjacent bone surfaces. The muscles involved would soon destroy themselves through friction were it not for the presence of bursae between the parts.

Bursae are small sacks with potential spaces, lined with a cobweb-thin synovial membrane which secretes a lubricating fluid. With its roof attached to a mobile surface and its base to a stable surface, all movement occurs within the bursa with a minimum of friction.

These sacks have an outer layer of fibrous connective tissue. Internal to this is a definite surface layer consisting of collagenous fibers interspersed with fibroblasts whose processes may extend for long distances. Formerly believed to be a "mesenchymal epithelium" forming a continuous lining of flattened epithelium-like cells, Kling, 1938 (5), has proven that the cellular surface is formed by the irregular massing of fibroblasts with a few macrophages, leukocytes and lymphoid wandering cells interspersed.

The synovial lining is supplied by a well-developed capillary network, accounting for the rapidity of accumulation of joint fluid.

This is the character of the bursa in general. Before describing the bursae of the shoulder in particular, a discussion of the local anatomy is proper.

Movement of the shoulder involves motion of the humerus, clavicle and scapula around the glenohumeral, sternoclavicular and acromioclavicular joints. It also involves sliding of the concave surface of the scapula over the dorsal chest wall. By the term "shoulder joint" is meant collectively, all those joints involved in the movements of the arm on the thorax.

A joint capsule attaches to the circumference of the glenoid cavity behind the glenoidal labrum on the scapula, and, in combination with the insertions of the short rotators, attaches to the sulcus composing the anatomical neck of the humerus. By short rotators is meant the supraspinatus, infraspinatus, teres minor and subscapularis, the first three inserting on the greater tubercle, the latter on the lesser tubercle.

The long head of the triceps brachii reinforces the inferior portion of the joint capsule, where it inserts on the infraglenoid tuberosity. The long head of the biceps brachii arises from the supraglenoid tuberosity and entering into the joint, passes out through

the bicipital groove, where it acquires a fibrous sheath which is an extension of the joint synovial membrane. In addition to atmospheric pressure, the glenohumeral articulation is kept in proximity through action of the biceps tendon. The coracoacromial ligament is a wide flat band attached by its apex to the summit of the acromion near its articulation with the clavicle, and by its base to the whole length of the lateral border of the coracoid process.

It forms practically a perfect hemisphere matching that of the humeral head when the humerus is adducted. The subacromial portion of the bursa completely fills the space between this ligament and the joint capsule. This ligament functions as a buffer between the humeral head and the acromioclavicular joint and acromion.

According to Gray's Anatomy, motion through the first 90 degrees of abduction of the arm occurs through the glenohumeral joint, the remaining 90 degrees occurring through the sternoclavicular joint. Codman, Cathcart (6) and Lockhart deny this, saying that motion occurs through both joints simultaneously. Codman uses alteration in this rhythm as a sign diagnostic of subacromial bursitis. He also maintains that ankylosis of the sternoclavicular joint will check abduction at 35 degrees.

The insertions of the short rotators have been described. The pectoralis major, latissimus dorsi and teres major insert in and along the bicipital groove. Instrumental in abduction, flexion and extension of the humerus is the tremendously developed deltoid, originating on the acromion and acromial end of the clavicle and inserting on the deltoid tubercle on the upper lateral third of the humerus.

The scapular and clavicular muscles figure into the joint movement also. The serratus anterior and levator scapulae rotate the scapula superio-medially while the sternocleidomastoid and clavicular part of the trapezius elevate the clavicle. The scapula is rotated inferio-laterally by the rhomboidei; the clavicle, by the clavicular portion of the pectoralis major, the motion in both cases being abetted by gravity. The bursae lie in relation to these bones, ligaments and muscles. The complete list of bursae around the shoulder was first compiled by Codman in 1906. They are:

Subacromial bursa

Subdeltoid bursa

Subcoracoid bursa

Bursa of the infraspinatus muscle

Subscapular bursa

Bursa of latissimus dorsi

Bursa of teres major

A distinction must be drawn between isolated bursae and bursae which are extensions of the joint capsule. Codman points out that the infraspinatus and subscapular bursa communicate with the joint capsule through small openings of variable size. They appear like diverticuli of the glenohumeral joint and on internal or external rotation of the humerus, the infraspinatus or subscapular bursae respectively become part of the joint capsule.

The subdeltoid, subacromial and subcoracoid bursae are all portions of one and the same organ, named for the structures which they underlie.

This separation is not justified even on topographical grounds. As the bursa moves with joint motion, each of the arbitrarily named sections flows into each other. The subacromial bursa in anatomic position becomes largely subdeltoid, and vice versa when the humerus is abducted. When the arm is behind the back, the subdeltoid bursa becomes largely subcoracoid as the humerus is rotated internally. In external rotations, the subcoracoid portion is nearly effaced as the movable periphery is stretched outward from beneath the coracoid process.

Codman says, "Even in cases in which there may be a synovial fold between the subcoracoid and subdeltoid portions of the bursa, the two portions are essentially

one . . . as they are composed of parts of the movable periphery . . . which fold on themselves when not stretched by extreme motion."

After injury or operation, these folds may become adherent and form cells or permanent divisions of parts of the bursa.

The subacromial portion extends beneath the acromion process and acromial portion of the clavicle. The subdeltoid portion passes laterally to the deltoid tubercle. The size of the bursa approximates that of a man's hand. The coracoclavicular ligament attaches to the roof of the bursa while the tendons of the short rotators and their firmly knit expansions are incorporated into the base. A tear of these tendons results in a tear of the bursa.

Dissection shows that the sheath of the biceps tendon (sometimes called the bicipital bursa) communicates with the subacromial and subdeltoid bursa. Consequently, the subacromial bursa, subcoracoid or coracobrachialis bursa, biceps sheath and the subdeltoid bursa will be considered as one bursa and called the subacromial bursa in this paper.

Piersol and older writers have stated that the subacromial bursa may communicate with the joint, but Codman, backed by Hitzrot, 1933 ( 7 ), emphasizes that such a condition is due to ruptures of short rotators,

causing development of pathological communications and that normally, this communication is never present, a view with which Brickner (8), and Carnett and Case (9), agree.

The bursa of the teres major, pectoralis major, latissimus dorsi at their insertions, and the subcutaneous acromial bursa are constant discrete structures. Other bursae of the shoulder which have been described but are not constantly found are the infraserratus bursa situated between the inferior angle of the scapula and the chest wall, the subtrapezoid bursa which lies over the triangular surface at the base of the spine of the scapula under the trapezius, or the subscapular bursa lying at the superior angle of the vertebral border of the scapula (not to be confused with the subscapular bursa which communicates with the joint).

Study of the bursae has been neglected by embryologists as well as anatomists until of late. Only one investigation of the development of the bursae has been reported. Black, 1934 (10), after studying a large number of human fetuses, showed that the subacromial bursa alone was present at birth, and this he was able to identify as such in 72.5 percent of the specimens. Connective tissue undergoes certain changes to form a fairly definite anlage composed of large closely packed cells. Slits develop between cells, coalescing to form the bursal cavity.

## THE ETIOLOGICAL FACTORS IN SUBACROMIAL BURSITIS

Stimson, 1940 (11), tabulates the causes of bursitis in general as follows:

1. Traumatic
    a) Actual wound of the bursa
    b) Direct contusion over the bursa
    c) Sudden violent motion or twist
    d) Repeated minor traumas
    e) Overuse sufficient to cause irritation
    f) Fracture or dislocation of the bones near the bursa
2. Infectious
    a) Common pyogenic organisms
    b) Gonococcus
    c) Tubercle bacillis
3. Toxic
    a) Obscure biochemical disturbances
    b) Distant foci of infection
    c) Absorbtion of abnormal substances by the intestinal tract

Rubert, 1938 (12), lists local trauma or inflammation and general constitutional changes as etiologic factors. Under the latter he includes arthritis, advancing age and metabolic and nutritional

disturbances. Bursitis, associated with arthritis, results from irritation by local arthritic osteophytes, he believes.

Granger, 1926 (13), was one of the first investigators to consider infection as playing a major role in bursal changes, especially in cases which are bilateral.

Dickson, 1931 (14), believes foci of infection are contributory causes, since he relieved several cases by removing infected teeth and tonsils.

Acute infection in a bursa may occur as a result of a wound, or, rarely, as a metastasis in pulmonary, otitic, upper respiratory, dental or dermal suppuration, or as a sequel to acute infectious disease, according to Cooperman, 1938 (15). He reports several cases of purulent subacromial bursitis following gonococcal bacteremia, one following mastoiditis, one following a carbuncle on the neck, and one following tonsillitis. Suppurative bursitis simulates septic arthritis or osteomyelitis.

Barnes, 1926 (16), reports a case of suppuration of the subacromial bursa in streptococcus septicemia following an induced abortion.

Hitzrot, 1928 ( 7 ), reported three cases of exudates of calcareous variety in the supraspinatus tendon in which a hemolytic streptococcus originating

from a tooth in one case, a paranasal sinus in another, and a hemolytic aureus originating from an ulceration of the cervix caused an adhesive bursitis of the subacromial bursa.

Steward, 1933 (17), reports a case of tuberculosis of the patellar bursa without involvement of the adjacent joint. James and Graham, 1930 (18), report several cases of pyemic bursitis in hogs, associated with Brucella suis infection.

Codman discounts the importance of local infection as an etiology factor, saying that in all his experience, he has found only one non-tuberculous suppurative subacromial bursitis. He believes that trauma is the cause of subacromial bursitis, either through partial or complete rupture or deposit of calcifications in the inserting tendons of the short rotators, particularly the supraspinatus. The trauma may be in the form of a blow directly on the bursa when the arm is in dorsal flexion, or it may be indirect, transmitted through the arm to the tendons. The indirect trauma may be a sudden sharp twist of the shoulder or it may be due to strain produced on the tendons by prolonged maintenance of the arms in semiabductions.

Consequently, trauma introduces the factors of sex, occupation, social status, age and physiologic state.

In those occupations involving heavy labor, supraspinatus tendon ruptures occur most frequently. Manual laborers, painters, dock workers, railroad switchmen, farmers, lumber yard workers, pipe layers and butchers all figure among Codman's list of cases. The mechanism here is usually a severe wrenching of the arm.

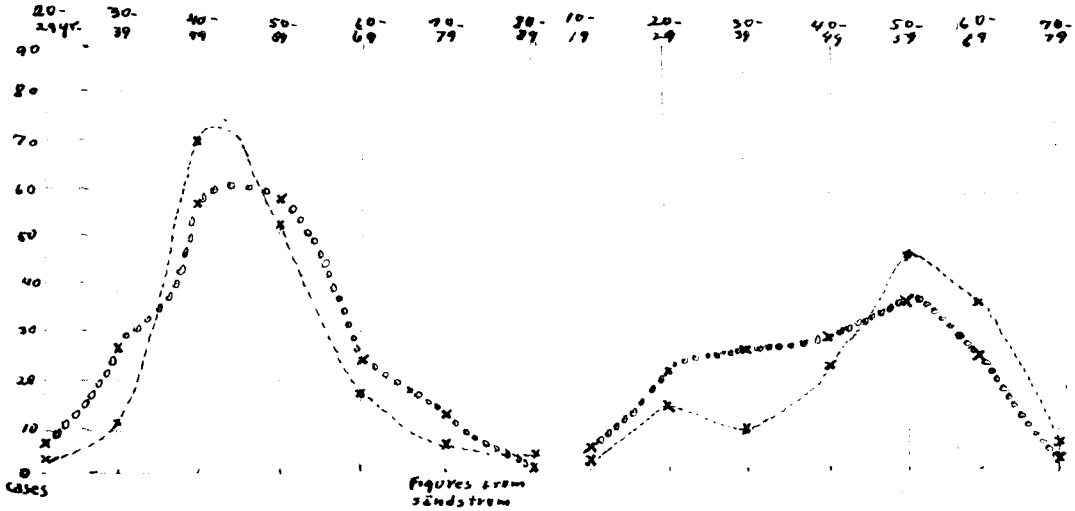
Those occupations requiring maintenance of semiabduction have the greatest frequency of tendinitis and calcified deposits. Codman lists stenographers, automobile drivers, office workers, store clerks, machinists, waitresses, bookkeepers, plasterers, tailors and housewives.

Sex figures into etiology through its relation to occupation. According to Codman, cases of rupture of the supraspinatus tendon are rarely found in women, except after severe exertion. However, 34 percent of the cases with calcified deposits occur in women. Sandstrom, 1938 (19), presents figures showing greater frequency of tendon deposits in females.

Social status is involved. The proletariat required to do manual labor, gets the partial and complete tendon ruptures, while the bourgeoisie and aristocrat get calcified deposits.

Codman states that the usual age for partial and complete tendon rupture is 49 and 55 years

respectively, for tendinitis 52 years and calcified deposits, 45 years. He would not suspect subdeltoid bursitis in an individual below 40. Sandstrom's figures conform with Codman's, although he does not classify his cases by pathology.




Sex distribution by age

Male ....
Female ----

Type of onset by age

Non-traumatic ....
Traumatic ----

The first peak occurs at an age of great activity and vigor, when direct trauma is a factor. The second peak occurs at an age when vascular and arthritic changes cause decrease in the integrity of the tendons, and the individual is yet active enough to be exposed to trauma. When degenerative changes have developed in the tendons, the amount of trauma needed to cause a partial or complete rupture may be so slight that the rupture is considered spontaneous.

Concerning the physiologic state, Codman believes that the menopause, being instrumental in loosening teeth, and causing atrophy of subcutaneous tissue, can also affect the tendons. He bases this belief on age-sex incidence.

Dickson cites two cases of hyperthyroidism with periarthritis which cleared up following thyroidectomy. Gunther, 1931 (20), declares that bursitis is not uncommon in acute rheumatic conditions. Painter, Haenisch, Petrignani, Dickson and Crosby believe that calcifications are the sequelae of periarthritis. Codman believes conversely that periarthritis is the result of chronic bursitis. He states that arthritis rarely occurs in the shoulder and so could not cause subacromial bursitis.

Kaplan and Ferguson, 1937 (21), feel that disability in older people following correction of fractures or dislocations at the shoulder may be due to the inflammatory process set up in the bursa.

Codman goes so far as to say that much of the pain and disability of fractures and dislocations of the upper end of the humerus are due to the resultant subacromial bursitis. Dislocation may be complicated by fracture of tuberosities, avulsion of facets or rupture of tendons, in addition to fracture of the glenoid rim and injury to the brachial plexus and axillary artery.

Stevens, (22), believes that an anterior dislocation is impossible without wholly or partially rupturing the tendons of supraspinatus, infraspinatus and teres minor. With the humeral head in subcoracoid dislocation, the distance from the origin to insertion of the supraspinatus is greatly increased, and in addition, the tendon is angled over the rim of the empty glenoid. Similarly, the posterior rotators are pulled over the posterior rim of the glenoid and are almost always injured. Placing the arm in the sling position and early mobilization will correct this condition.

The subacromial bursa survives fractures of the upper end of the humerus very well; as the subacromial and subcoracoid portions are protected by a heavy bone shelf, only the subdeltoid portion is usually involved. However, comminuted fractures of the head of the humerus may disrupt the subacromial portion, producing extensive lacerations of the bursa in addition to other complications. After replacement of fragments, the bursa need receive no further attention, as it heals graciously. The facet for the supraspinatus may avulse and form a bursa-joint communication, in which case Codman believes immediate repair should be undertaken to avoid a permanent bursitis. Avulsion of the facets of any of the short rotators may be corrected if the

raw bone surfaces can be approximated. However, if the facets get wedged between the acromion process and the humeral head and cannot be apposed, open operation will be necessary to prevent chronic bursitis and permanent partial disability.

## PATHOLOGY

When man changed from quadruped to biped posture, he not only made himself vulnerable for lumbo-sacral strain and visceroptosis, but also to injury to his supraspinatus tendon. In allquadruped vertebrates, the humerus hangs in line with the spine of the scapula, and the supraspinatus functions as an accelerator to pendulum action of the humerus. In man, the humerus hangs more or less at right angles to the scapular spine and the supraspinatus acts as an elevator on a short lever, at great mechanical disadvantage.

The supraspinatus initiates abduction and carries it through a 35° angle. Codman believes that the supraspinatus is constantly on the stretch in maintaining the humerus in the glenoid. In addition, it places the head of the humerus in the glenoid fossa and maintains the latter as a fulcrum. Without the intervention of the supraspinatus, the deltoid would elevate the humerus in its long axis, impinging the head against the acromioclavicular joint and the greater tuberosity against the edge of the acromion process, with compression of the intervening bursa. In this manner impairment of the supraspinatus function can cause damage to the articular surface and bursa.

Because of the situation of the inserting tendons in forming the roof of the articular capsule and the floor of the subacromial bursa, ruptures of the tendons or perforations through an area of softening in a tendon form a free communication between capsule and bursa. Due to its inherent functional disadvantages, the supraspinatus tendon would be expected to be most frequently involved, and Codman has found this to be true.

An area in the supraspinatus tendon just above its point of insertion into the greater tuberosity, called by Codman "the critical area" is the point where most tendon ruptures and pathologic changes occur. Apparently the point is the locus minora resistentiae of the bursa, as it is always affected in bursal disease and is usually the center of the disease process. This is the point of greatest mechanical stress in overcoming the inertia of the arms in initiating abduction. It may be jammed against the acromion or acromioclavicular ligament by sudden contraction of the deltoid without coordinating action of the supraspinatus. This is the most prominent point on the head of the humerus to pass beneath the acromion. Consequently, it is subject to constant friction and pressure.

Microscopic study of the supraspinatus tendon has demonstrated changes progressing from liquefaction necrosis to formation of rice bodies and calcareous degeneration, culminating in spontaneous or traumatic tendon rupture.

Codman states, "The starting point of most lesions of the shoulder centers in the tendon of the supraspinatus. Thence it involves the bursa and the adjoining tendons of other short rotators, but the inflammation of the bursa gives the most pronounced and often the only symptoms."

This statement should be modified in the following manner. An acute transitory bursitis will result from a direct blow on the anterior part of the shoulder when the arm is in dorsal flexion. There is acute local tenderness over the bursa and scapulohumeral spasm is present. As these cases promptly recover, these patients rarely present themselves to a doctor and hence are rarely seen. With the exception of this condition, Codman believes the acute form of subacromial bursitis is due to changes in the tendon of the supraspinatus, and the subacute and chronic forms are secondary, as shown by the following table:

A. Acute types

1. Local ischemia due to prolonged hyperabduction of over 1 to 2 hours, as from a surgical procedure like a radical mastectomy. This is a rare type.
2. Inflammation extending from about a calcified deposit which has either burst into the bursa or has approached the bursal surface closely enough to inflame its lining. This is by far the most common cause.
3. Rupture of the supraspinatus tendon by trauma of sufficient degree to make a direct opening between the joint and the bursa through a gap in the tendon. This produces a chronic permanent bursitis.
4. Partial ruptures of the supraspinatus tendon. They may be acute, subacute or chronic.
5. Suppurative infections. Codman states that these are rare.

B. Subacute and chronic types

Nonadherent - no restriction of motion but have a painful point on elevation

1. Inflamed villi, folds or bands. These do not have an acute phase.
2. Irregularities of the base due to calcified deposits in the tendon about which there is

only a little chronic inflammation. These cases have passed through an acute phase.

Adherent - limitation of motion from bursal adhesions or muscle contractures, in addition to muscle spasm.

1. Sequelae to inflammations from calcified deposits in the bursa or about to break into it.
2. Sequelae to partial ruptures of supraspinatus tendon and associated tendons. Adhesions rarely occur in complete rupture of the supraspinatus.
3. Inflammation of the floor of the bursa in association with early degenerative changes in the tendons as tendinitis. This condition has a brief acute stage and a prolonged chronic stage.

There has been much discussion over the exact location of the calcified deposits, Painter, Bergmann, Stieda and Haenisch saying that they were located in the bursa, Wrede, Brickner, Codman, Carnett and Case limiting them to the supraspinatus tendon, while Cooperman maintains that they are situated both in tendon and bursa. Sandstrom found them in the tendon and peritendinous soft tissue.

Codman suggests the following sequence in the formation of calcified deposits and Carnett and Case confirm it. They believe that the changes in the tendon are incident to the creation of an ischemic state of short frequent intervals or of permanent duration whether due to maintaining semiabduction or acquiring a sclerotic vasculature.

The net result is that a liquefaction necrosis develops. The initial change is a degeneration of the collagen of the tendinous tissue. This change is first demonstrated by staining reactions to hematoxylin and eosin, the degenerating tissue showing up by taking a darker red stain.

The next step in the process is a hyaline degeneration, without any leucocytic infiltration, so that no inflammatory signs obtain. At this time, the staining reaction varies so that certain portions of the section may stain blue while the remainder stains red. With the passage of time, calcium salts deposit in these areas. If the central portion of the tendon has degenerated, then a central necrosis and calcareous deposit develop. With the growth of the deposit and its final rupture out of the tendon into the subacromial bursa, as a milky mass of calcified granules and gelatinous tissue, the evolution of the process is completed. Codman does not believe that the process goes on to ossification.

Moschcowitz in 1915 (23) attributed the tissue degeneration to a coagulation necrosis, and believed that calcification takes place when the affected areas became anemic.

At first the deposit is merely a milky liquid, but as calcification proceeds the material becomes more solid, passing into a state in which calcium particles are mixed in a base of caseous material. The final result is the formation of calcareous material which may lie discretely in the tissue, leaving a sharp cavity on removal; may have a layer of necrotic tissue around it; or may be spread like granules of sand through the tendon.

The calcified deposit never moves toward the joint capsule but always toward the bursa. As it approaches the bursal floor, an acute inflammation appears around the deposit and surrounding tissue. Leukocytes migrate into the deposit and reduce to a buttery material.

The lesion, with a red indurated base and a whitish yellow elevated center, now resembles a furuncle. When this lesion is nicked by a scalpel, the contents squeeze out like zinc oxide from a tube.

In the meantime, the inflammation of the bursal floor has induced the formation of synovial

fluid which is rich in fibrin. With rupture of a deposit into a bursa, the calcified particles are received by the fibrin and are held there, as leukocytes, pass into the fluid and carry the particles. Within three weeks, the calcifications are removed. Codman considers the discharge of the calcifications into the bursa as the physiologic cure for the condition. It is noteworthy that there may be no phagocytic reaction to calcareous deposits in the tendons. However, Carnett 1931 (24) draws attention to his finding that when acute aseptic inflammation develops around a calcification in the tendon it is rapidly absorbed.

The deposits are rarely larger than lima beans. They may be single or multiple. The supraspinatus tendon is the most frequent siteof occurrence, followed by the tendon of insertion of subscapularis, infraspinatus, and teres minor, in that order.

Stoddard (3) has analyzed the calcareous material and has found it to be 55.8 percent calcium phosphate and 44.2 percent calcium oxalate and organic matter, mostly fibrin. Magnesium and iron carbonates and sulfates are present in traces. Case and Carnett have found the material to be sterile. On x-ray examination the calcareous masses show up as rounded coherent densities. After they have ruptured into the bursa, they appear like numberous small flecks in a cloudy shadow.

Sterns, 1925 (25), noting that rate of formation and disappearance of deposits in the supraspinatus tendon and subacromial bursa is not typical of that for calcified deposits in myositis ossificans and periostitis, states that on three occasions he removed the radiopaque substance and found it to be "a granular, noncrystalline, nonoily, nonfluid, brownish gray, not hard or bony" substance which gave positive tests for fat but was negative for calcium. Carnett and Case mention that thecellular reaction adjacent to a deposit may closely resemble that of a giant cell tumor.

The yellow-brown tumor-like lesion known as xanthoma, xanthogranuloma, giant cell tumor, xanthomatous giant cell tumor, has been long recognized as occurring fairly frequently on tendon sheaths. These tumors are composed of compact polyhedral cells, giant cells and lipoid and hemosideric cells. Jaffe, Lichtenstein and Sutro, 1941 (26), report their occurrence in synovial membrances of joints and bursa, forming in the latter case villonodular bursitis. The mistaken identification of these tumors as the typical tendon pathology in subacromial bursitis no doubt explains the confusion.

Tendon tears are second on the list of primary lesions in the development of subacromial bursitis. Rarely, one tendon may be completely ruptures without damage

to the others, but usually other tendons are completely or partially ruptured, or all may be partially ruptured.

Although the tendons of insertion of the short rotators are thought of as discrete tissues, actually they are conjoined andwoven together so that an expansion between the subscapularis and the muscles of the greater tuberosity bridges the bicipital groove. This fibrous bridge is a thin spot and tendon tears start there, usually proceeding laterally to rupture the supraspinatus. Because of its close relationship as it inserts laterally, posteriorly and slightly anteriorly to the supraspinatus tendon, the infraspinatus is frequently ruptured with it. Consequently, later muscular atrophy affects both suprascapular muscles.

The rupture may proceed medially to affect the subscapularis tendon. With the tendon expansion torn, the long head of the biceps brachii is exposed. Exposure causes an inflammation, as the tendon is found to be red and swollen. Sometimes, the trauma may be great enough to cause a biceps tendon rupture or a chip fracture of the supraglenoid origin. In the uncovered non-displaced tendon, a fraying of the edges which are adherent to the groove and have rice bodies attached to them can be seen.

Meyer, 1921 (27), reports finding numerous cases of attrition of thebiceps tendon at its origin, at the site of union of the muscle and the tendon, or in the intertubercular sulcus with resultant degenerative changes in the tendinous tissue as a complement of arthritis. Codman finds biceps tendon attrition following rupture of the biceps sheath and exposure to the bursal synovia.

The tendon ruptures may be of four types. First is that in which part of the tuberosity may be evulsed. The bursa is retracted by the withdrawing tendon sheath and facet, and the resulting space is filled with callus from the torn periosteum. Since the line of cleavage of the tuberosity is external to the bursal attachment, evulsion of the tuberosity can occur without rupture of the bursa.

The second site where tendon rupture occurs is at the point of insertion, so that the superficial part of the facet may be carried inward by the retracted tendon. This shell may be absorbed. This type of evulsion may be whole or partial.

True rupture of the tendon, leaving a stub on the tuberosity usually occurs at the "critical point," by reason of its narrowness, poor blood supply and vulnerability to trauma. In this case and in the case of evulsion of a facet the bursa is torn.

Partial rupture of the inserting tendinous cuff forms the fourth type of tendon rupture. Codman reports this as occurring with greatest frequency in the older age groups. The tendon istorn to a degree insufficient to tear the base of the bursa itself, leaving a film of tissue between the joint and the bursa. Repair takes place by a thickening of the film at the bursal base, and takes place in a few weeks or months. Frequently, however, the bone and the tendinous cuff do not unite, leaving a bare bone area at the anatomic neck.

The lesions which have been described are all primary and are found in acute bursitis. The pathology found in subacute and chronic bursitis is the direct consequence of the primary lesion. Secondary pathology may be divided into:

1. That located in the tendinous cuff
2. That in the joint, on the articular surface and in the subarticular bone
3. That within the bursa.

Sometimes the inserting tendon is laminated to form "straps" as Codman calls them. As different portions of the supraspinatus contract at different times, their perimysial extensions come under tension individually. Tendon fibers lie in loose laminae parallel to the joint capsule. Consequently, they

may develop into parallel layers of tendon tissue which are attached at both ends and loose in the center. On contraction of the entire muscle, the "strap" rises up like the handle on a valise. Whether this is the result of inflammation, discharge of calcareous material or of partial ruptures is not known. Grossly, the tendon seems to be ragged and frayed. Sometimes rice bodies are attached.

The short rotator tendons may be inflamed, frayed, partially or wholly torn, evulsed with facet, and may contain rice bodies or areas of liquefaction necrosis. When areas of rice bodies and liquefaction necrosis are visualized in the tendons through the floor of the bursa, they have the same appearance as the calcareous bodies - small white papules surrounded by erythematous areas. On scraping the tendon surface, the rice bodies or fluid are ejected.

Formerly, rice bodies were believed to form from the deposit of fibrin upon detached synovial tags as a nucleus. Hitzrot found rice bodies in one of his cases with multiple villi formation. Hyalinization had progressed far at the ends of the villi, and he assumes that this process led to the formation of rice bodies. Mumford, 1927 (28), found histologic proof that at least in the case of joint tuberculosis, the rice bodies formed about a nucleus of giant cells which had migrated into

the joint cavity. Due to their constant rubbing, the rice bodies develop their cigar-shaped form. Codman believes that rice bodies form in areas of liquefaction necrosis from condensation of collagen and burst out of the tendon into the bursa and joint capsule.

Bone changes are prominent. The greater tuberosity is principally affected. These changes are readily detected by x-ray. In many cases of old complete rupture, the tendon stub shrinks while the tuberosity enlarges, leaving a protuberance which causes a definite jog when the tuberosity passes under the acromion process. Due to the attendant friction, bone cells are stimulated to for irregular excrescences. This newly formed bone is very spongy and probably later atrophies, because in very old cases, the tuberosity is found to be recessed. The friction, bone hypertrophy and excrescences all add to each other to make a vicious circle. Hypertrophy of the acromion process develops to increase the obstacles to normal joint function.

Briefly, Wolf's law states that bone grows along the axis of stress and strain. Removal of the stimulae to bone growth which results on tendon rupture leads to bone atrophy. Atrophy, rather than hypertrophy, develops in the absence of friction. Frequently, an absorptive change develops beneath a superficial

osteitis of the tuberosity. The bone is replaced by vascular masses. This may be an early stage of recession. Atrophy of the tuberosity may proceed until no remnant of the tuberosity or tendon stub is left. The surface may be rounded with the anatomical neck obliterated and entirely covered with cartilage.

Eburnation is found in cases with low grade chronic inflammation. A placque of thickened hardened bone of increased mineral content, showing on x-ray as an area of increased density, develops around the insertion of the supraspinatus tendon. The increased bone density may decrease the bone blood supply to the tendon, laying the groundwork for degenerative changes. Eburnation around tendon insertions and osteitis of the acromion process are found in heavy laborers without joint symptoms and apparently is a physiologic change in them, but when these changes are associated with symptoms of tendon rupture Codman interprets them as confirmatory of the diagnosis.

The osteitis caused by this irritation on the tuberosity may sometimes involve the outer side of the tuberosity as well as its tip. The base of the bursa may be the seat of chronic inflammation, sothat irregularities will be shown by x-ray on the appropriate portion of the tuberosity, external and below the insertion of the tendon, on the outer aspect of the bone.

In all long standing lesions of the bursa, whether or not they are due to rupture of the tendons, the x-ray shows that the trabeculae beneath the base of the bursa are more or less atrophied. These changes occur on the tip and external aspect of the tuberosity. The trabecular markings are indistinct and vague. This kind of atrophy is sometimes marked in acute cases of bursitis due to calcified deposits. In such cases, it slowly disappears after the lesions have healed.

Eburnation may occur in the anatomical neck. The articular edge may be hypertrophied to form an elevation. This may be a chronic arthritic change. No symptoms can be attributed to this.

Another area where subacromial pathology appears is in the bursa itself. Normally, the roof of the bursa resembles the peritoneum, but when acute infection is present, it thickens and becomes opaque and red. In chronic cases, it is whitish and firm. No other changes occur in the roof.

Defects will be found in the bursal floor in relation to the changes produced by the bursitis. These will vary with the lesions, dependent on extent of rupture, amount of joint cartilage exposed through the rent, exposure of the biceps tendon and recession or hypertrophy of the tuberosities and other variables.

Villi may be present in the synovial lining of the bursa. Whether they are pathologic or physiologic is questioned, but when present in large quantities, are probably evidence of an old inflammation. The villi may be attached at both ends to floor and ceiling or may lie with one end free. They are pink or red and appear around the greater tuberosity at the point mentioned in the section on etiology as the locus minora resistentia of the glenohumeral joint.

Thick cordlike bands offibrous consistency may ramify the bursa and are associated with chronic adhesive bursitis.

Bursae have folds of synovial membrane which are held in reserve for extremes of motion. These folds may become inflamed and give rise to a chronic bursitis with intermittent symptoms such as twinges in certain positions.

Adhesions develop in the bursa just as in the peritoneum. They are the result of the inflammation, and form separations between portions of the bursa, generally developing between the subcoracoid and subdeltoid portion of the subacromial bursa. Joint motion is limited until resolution of the adhesions occurs. Absorbtion of adhesions is the usual result.

A small amount of fluid is secreted with an inflammatory process in the bursa. However, the presence

of large amounts of fluid indicates that communication has been established between bursa and capsule.

The bursal extensions of the joint beneath the infraspinatus and subscapularis may be stretched and torn to form a habitual dislocation, or they may be adherent. Fluid production may be a response to inflammation, although this rarely occurs. If use of a joint with an inflammatory bursitis is continued in spite of the pain, fluid will be produced which will give transient relief. The fluid is a clear, straw colored liquid and rather thick.

When it does occur, it can be recognized by the obtuse angle at which the humerus is held in relation to the axis of the spine of the scapula, or by palpation of a rounded swelling posterior to the acromion or in the axilla. Joint fluid is demonstrated by x-ray by the distance between joint surfaces.

If joint bursa communication is established through a ruptured supraspinatus tendon or partial rupture of the tendinous cuff, then on abduction of the arm, fluid is squeezed from the dependent portion of the joint capsule into the bursa, distending it in the subdeltoid region beneath the upper fibers of the deltoid. The continual pumping of fluid back and forth results in a stretching of the bursa, which may go to a hydrops.

Other results of communications between joint and bursa through the inserting teninous cuff are the osteitis of the acromion process; erosion of the articular cartilage which has been exposed through the rent and subject to direct trauma from the roughened acromion; degeneration of the tendon stub several months after injury; andrecession ofthe tuberosity, as described previously.

Codman believes the mechanism for the production of the pathologic findings of chronic bursitis in those cases with history of minor trauma may be as follows. Degeneration of the tendon rsults from a general toxic condition, or from poor blood supply to the part due to maintenance of an abducted position, arteriosclerosis, or increased calcification of the osseous plate of the greater tuberosity. This may give rise to partial ruptures, small areas of liquefaction necrosis, rice bodies or calcified deposits. The latter three may remain in the tendon, asymptomatic indefinitely, or later may empty into the bursa, causing an acute bursitis.

Partial ruptures cause a local inflammation making the patient limit his motion in that arm, so that a disuse atrophy may be superimposed. Then additional slight trauma will cause further tendon rupture until bursal-joint capsule communication is

established. With synovitis, comes the production of fluid so that the bursa is pumped up to several times normal size with use of the arm. On continuation of the process, more fibers are torn, the exposed biceps tendon becomes inflamed and the greater tuberosity undergoes its initial reaction to inflammation - that of hypertrophy and excrescence. This causes friction against the acromion process which results in a similar proliferative process.

The bursa, compressed between the roughened greater tuberosity and acromion with each abduction of the arm, undergoes an inflammatory process and villi formation. With continued tearing of the tendons, the exposed area of articular cartilage increases and being subject to trauma from the acromion, the cartilage erodes. Later, the greater tuberosity and whatever stub of tendon that remained on it, atrophy so that a rounded surface covered with a thin cartilage-like layer of fibrous tissue is left in the old chronic case. The tendinous cuff recedes from the anatomical neck, with the development of partial tendon ruptures on both the bursal and joint side. This process may be bilateral.

In cases in which the onset can be laid to severe trauma, the process begins with complete rupture of the supraspinatus and possibly other adjacent tendons, with development of communication between joint and bursa

and a similar sequence of events as in the previous case.

Inflammation of the bursa presents the story of inflammation of any other serous cavity, i.e., congestion, friction, pain, protective spasm, fibrinous exudate, adhesions, cicatrizations and partial absorbtion. The microscopic changes are characteristic. Dilatation of the blood vessels, transudation of lymph, cellular material and fibrin from the bursal lining into the bursal fluid occur. The bursa becomes thick and edematous. The bursal fluid may contain a thin fibrin net which may resolve or go to form adhesions. Besides adhesions, the fibrinous exudate may form a heavy plastic exudate which results in heavy bands of connective tissue running through the bursa restricting the range of motion and giving rise to creaking leather sounds.

Irritation of the bursal surface may cause proliferation of the fibroblasts forming the bursal lining to give rise to villi which may move freely within the joint.

Microscopic study of a calcareous deposit by J. W. Stevenson (3) revealed that particles varied in size from that of bacteria to ten to fifteen times the size of a white blood cell. They were mostly round or oval. The large ones were concentrically striated, resembling gall stones. The central nucleus resembles

a white cell. On Gram stain or Ziehl Nielsen, no organisms were seen.

In all cases of deposits, there is no inflammatory infiltration around the deposits, yet degenerative tendon change can be seen. Moschcowitz believes that this lack of infiltration is due to the avascular condition of tendon and that necrosis is the reaction until vascular tissues become involved in the process.

## SYMPTOMS AND DIAGNOSIS

The cardinal symptoms of subacromial bursitis are pain in the shoulder present when the greater tuberosity passes under the acromion process; tenderness over the greater tuberosity; and inability to voluntarily abduct the humerus on the scapula.

The symptoms of acute bursitis vary with the primary pathology, but the syndrome is characteristic, whether it is caused by calcified deposits, partial or complete tendon rupture, or tendinitis. The slight differences in symptoms, signs and story, first recognized by Codman, make diagnosis of the nature of the primary lesion possible.

Codman also was the first to recognize tendon ruptures, both partial and complete, as primary pathology. He agrees with the other authorities that the most frequent cause of acute subacromial bursitis is calcified tendon deposits. However, he says that the most common cause of prolonged disability of the shoulder is produced by complete supraspinatus tendon ruptures and that the most common cause of minor shoulder disability is partial tendon rupture.

Patterson and Darrach, 1937 (30), found that many of their patients had a white blood count of 14,000 with a percentage of polymorphonuclear leukocytes

as high as 75. Some of them ran an oral temperature of 101 degrees. However, every culture of bursal material that they secured was sterile.

In cases of acute bursitis Sandstrom found lowered or elevated temperatures in 70 out of 80 cases. Sedimentation rate was elevated in 70 out of 75 cases. Fifty-eight showed values of from 20 to 40 while one ran 96 mm. per hour. These changes indicate the presence of a toxic process, which may result in the degenerative tendon changes and spontaneous tendon ruptures described by Codman.

In the cases in which complete rupture of the supraspinatus tendon is suspected, the following history is usually presented. The patient is over 40, usually a laborer who has a definite history of trauma to the shoulder. A sharp pain is felt in the shoulder at the time of accident. The patient may actually feel something snap. Due to the shelter offered by the shoulder cap, the damage to the tendons is not direct. Indirect strain is transmitted to them along the shaft of the humerus due to the inertia of the arm during sudden movements, especially when heavy objects are in the hands. For a few hours after the accident, the pain is not bad. The patient may work out the day, favoring his arm. In about three hours the pain becomes severe and later intolerable. The patient is

unable to sleep that night and unable to work the next day. The pain is constant, nagging, unrelenting. The patient's family may not give him any sympathy because physical signs are not apparent.

Codman explains the three hour interval between the initial and the severe shoulder pain as that time taken for hemorrhage from a slowly bleeding ruptured tendon to distend the joint and bursa enough to cause tension pain. The pain causes scapulohumeral spasm and the tension decreases bleeding. After a few nights, the hemorrhage absorbs and tension spasm and pain leave.

The patient soon discovers that he is unable to abduct his arm. With section of the supraspinatus, the head of the humerus is not held to its fulcrum in the glenoid fossa, and contraction of the deltoid merely draws the head of the humerus up to the acromion without producing abduction. Exceptionally, the remaining short rotators may be able to maintain the humeral head in the glenoid fossa so that abduction can take place, but in these cases normal scapulohumeral rhythm will be lacking. By normal scapulohumeral rhythm is meant coordinated simultaneous movement through the glenohumeral and sternoclavicular joint, as described in the section on anatomy.

Physical examination will show that there is spasm of the scapulehumeral muscles. When the patient

is asked to hyperabduct his arm he will make all the motion pass through his sternoclavicular joint while he keeps his glenohumeral joint immovable. On being asked to carry the movement further, the patient will be unable to do so or will abduct slightly further with great pain and difficulty. In testing for voluntary movement of the hand, one must be sure that there is true inability to elevate the humerus and not inhibition because of pain, which could be caused by partial tendon ruptures or direct blows on the bursa.

An important finding is that there is no restriction to passive motion or to motions that do not involve the supraspinatus. The humerus can be passively moved from adduction to hyperabduction without pain except at that point in the arc where the greater tuberosity passes under the acromion process. At this point a slight jog can be felt as the tender greater tuberosity is deflected by the acromion and the patient gives evidence of his pain. A soft gristly crepitus can be heard at this time. When the greater tuberosity is not passing the acromion, these symptoms are silent.

The disappearance of tenderness on palpation of the head of the humerus when the greater tuberosity is pulled beneath the acromion process was reported by Dawbarn in 1906 (31), and is known as Dawbarn's sign.

A test which aids in identification of the ruptured tendons is one in which the patient bends at the hip with his knees extended so that his arms are hanging in anatomical quadruped position, or the patient may stand erect and the examiner elevate his arm 140 degrees. From this position, the patient with a supraspinatus tendon rupture can elevate his arms into dorsal flexion or abduction by movement through this glenohumeral joint. Then, if his scapula and humerus are set by the examiner the patient will be able to stand erect and keep his arms in abduction. On lowering the arms, the first part of the movement will be through the sternoclavicular joint. After the limit of movement has been reached there, the power to keep the arms elevated will be lost and they will drop to the sides, while the patient winces as the greater tuberosity slides from under the acromion.

The shoulder is now palpated. The examiner stands behind the patient and while holding the patient's forearm in flexion and humerus in adduction with his hand of the same side, the examiner places his other hand on the shoulder to be examined. For example, the examiner's left hand would be on the patient's right shoulder. The hand is placed so that the thumb is posterior and the index finger anterior to the acromion process and the four fingers are wrapped around the

clavicle. If theindex finger is now moved laterally toward the point of the acromion process, a tender sensitive spot will be pressed, which the patient will describe as thecenter of his pain.

If pressure is made here, the finger will press into a groove, between the acromion and the greater tuberosity. The gap is between the torn ends of the tendon and was not felt previously because the tense tendon bridged it.

The position of this point of maximal tenderness may vary slightly, depending on the short rotators which are also ruptured. As the supraspinatus tendon is always involved in tendon ruptures of the shoulder, the location will be lateral to the bicipital groove around the insertion of the supraspinatus as midpoint.

X-rays which are taken after 24 hours are reported negative, which rules out fracture, dislocation and calcified material in the tendons. Codman suggests that the injection of radiopaque fluids into the joint would aid in positive diagnosis of tendon rupture.

If a patient presents most of the picture described, Codman says that an exploratory incision should be made into the shoulder. The incision is placed over the greater tuberosity and proceeds

vertically for one-half inch. Dissection is made through the deltoid. If supraspinatus rupture has occurred, a tear of the bursal floor with triangular retraction of the edges can be seen through the transparent bursal roof. The greater tuberosity with its red stub, the biceps tendon and the joint cartilage will be seen. The humerus should be rotated so that the ligaments of the infraspinatus, teres minor and subscapularis passing before the incision can be examined. Codman believes that an error on the side of too frequent exploration is justified because of the numbers of early ruptures that can be discovered.

If operation is delayed for over three weeks, the joint will develop fluid, going perhaps to a hydrops as the bursa balloons out. On abduction, the fluid can be felt under the deltoid. The deltoid is hypertrophied as it is working harder inattempting to abduct the arm, although it is not as effective. The supraspinatus and infraspinatus both atrophy, the former because its tendon is sectioned and the latter because a goodly share of its fibers of insertion are torn due to the crossing of the supraspinatus tendon as they proceed to their insertion lateral and posterior to the supraspinatus. As the infraspinatus is the larger muscle of the two, its atrophy is more evident externally. With the passage of time, an exploratory incision will show the

following findings in addition to those already described. The greater tuberosity will have excrescenses and possibly osteoporosis. Granulation tissue will fill in the anatomical neck between the greater tuberosity and the articular surface of the humerus; the biceps tendon may be in varying degrees of inflammation, or may be frayed, adherent to the bicipital groove or contain rice bodies; and a concave web of fibrous tissue will start to grow from the ruptured tendon end. Little repair will have taken place in the triangular rent in the floor of the bursa. As fibroblastic proliferation must proceed from the wound edges and await development of a vascular supply, union of this part is slow and sometimes never accomplished. In old cases in which the rent is not closed, the exposed articular cartilage becomes eroded.

With continuation of the injury, the patient cannot sleep, worries about his arm and his inability to work. Without working, the patient may be unable to buy good food and consequently may become poorly nourished. Late in the case, the patient is depressed mentally and physically.

Incomplete ruptures of the supraspinatus are those in which the section does not run the whole width or the whole thickness of the tendon. Associated with it may be partial tears of other portions of the tendinous

cuff. The picture varies from that of complete rupture, in that considerable degree of power of abduction remains and all of the signs are not definite. The cardinal symptoms of complete rupture, viz., Dawbarn's sign, jog, crepitus, atrophy, defect lateral to the bicipital groove and local tenderness may be present, although they may be altered or questionable. Accurate differentiation is important here as partial ruptures recover without suturing the tendon, while complete ruptures never do.

Consequently, when doubt is present as to diagnosis, Codman believes the exploratory incision is a proper procedure.

The partial ruptures may be of four kinds:

1. A few of the lower fibers on the joint side, together with the synovial reflection, may be torn out.
2. Some of the central fibers may be parted without tearing either the joint side or the bursal side of the tendon.
3. The rupture may extend vertically through the whole tendon, making a communication between the joint and the bursa without involving the whole breadth of the tendon.
4. The fibers on the bursal side may be eroded without complete communication with the joint.

Codman believes that type 1 is the cause of a widening of the bare area of the anatomical neck, which ordinarily would be covered by the tendinous cuff. He thinks that type 2 gives rise to calcified deposits and that types 3 and 4 might give rise to tendinitis symptoms.

The symptoms of bursitis from calcified deposits in the supraspinatus tendon might be divided into a pre-acute phase, at which time the calcified deposit is building up in the tendon and irritating the bursal floor, and an acute stage when the deposit ruptures into the bursa.

The presence of calcifications in the tendons may give no immediate symptoms. In fact, radiographic evidence of calcifications has been found in asymptomatic shoulders. However, Codman believes that these cases eventually reach a symptomatic stage in the course of their development. Spontaneous absorption of small calcified deposits has been known to occur but Codman has yet to see a case in which a good sized calcification disappeared without pain or restriction.

In the course of its development, the calcification migrates or extends toward the bursal floor where it sets up an inflammation due to mechancial irritation. The bursal floor then has a turgid red edematous area, in the center of which is a yellowish white

peak containing the concretion. The lesion resembles a furuncle. The central portion lies over the calcification and hence its location will depend on the tendon involved. This is usually the supraspinatus tendon, although other tendons, alone or with it, may also contain calcifications.

Most cases run a subacute course, corresponding to the pre-acute stage, the rest being acute, chronic, or a combination of all three. The usual history is not of acute pain at the beginning. In the pre-acute condition symptom first noted is a slight painful "hitch" during certain movements of the arm, usually those requiring abduction. This pain is irregular at first but soon accompanies every movement. The patient soon learns to rotate his humerus outward before abducting so as to avoid compressing the inflamed point under the coracoacromial ligament. At this stage, the deposit still presents a surface which is flush with that of the tendon. With extension or migration of the deposits, the surface of the calcification protrudes, and motion is restricted in both axes.

Eventually, involuntary scapulohumeral spasm develops and the normal smooth division of motion between the movement of the scapula on the chest wall and the elevation of the humerus on the scapula is destroyed.

In the normal shoulder, abduction of the arm through 180 degrees is accomplished by even, simultaneous motion of the humeral head in the glenoid and the scapula on the chest wall. The scapula is checked against inferior rotation on initiating motion by a slight contraction of serratus anterior. In the shoulder with spasm, this sequence is changed. The short rotators lock the glenohumeral joint and the scapula is rotated on the chest wall to elevate the humerus through the first 90 degrees. If the muscle spasm is still young enough to be partially under voluntary control, the short rotators are relaxed enough to take the other 90 degrees through the glenohumeral. With continuation of spasm, the glenohumeral joint becomes locked and the arm can be abducted only through 90 degrees.

If the spasm is of long duration, firm adhesions form, producing a fibrous ankylosis, which restricts joint motion even after the inflammatory process has resulved and muscle spasm has ceased. However, these adhesions gradually stretch and motion returns slowly. With disuse of the arm and development of spasm, disuse atrophy occurs. Hence, the cardinal symptoms of calcified deposits in the tendon are pain, spasm, limitations of motion and muscle atrophy. The degree of spasm regulates the degree of motion. The pain is directly

related to the degree of motion, and can be controlled by immobilizing the joint. However, this results in a fibrous ankylosis with a long period of disability so this treatment is contraindicated.

For history of the pre-acute stage the patient will recite the story of onset of pain, spasm and restricted motion as previously described. Before spasm sets in, the patient will tell of finding that the most comfortable position for sleep is with the arm abducted and in external rotation, that is, with the hand behind the head, demonstrating Dawbarn's sign. In this position, the tender point lies beneath the acromion when it has plenty of room and the supraspinatus is relaxed. Pain is felt in the lower fibers of insertion of the deltoid. This may be reflex or directly due to spasm.

Physical exam will indicate the "hitch," limitation in motion, changes in humeroscapular rhythm, spasm with its associated muscle tenderness and atrophy of the spinatus muscles. Palpation of the shoulder will indicate an enlarged raised tender point on the greater tuberosity, which moves as the arm is rotated. The diagnosis is confirmed by x-ray.

Although the supraspinatus tendon is the most commonly affected, the tendons of any of the short rotators may be the site of the calcification. Accurate

localization should be made before surgical interference is started. This may be done by palpation for tender spots, study of restriction of motion, and well posed x-ray pictures.

X-ray views should be taken both in internal rotation and external rotation, according to Sandstrom, who cites numerous cases of the calcified deposit appearing in only one of the two aspects.

This condition is differentiated from complete rupture of the supraspinatus tendon by the history of gradual onset, the degree of voluntary motion possible when the condition is young, or the limitation of passive motion because of spasm perhaps with ankylosis, when the condition is old. Palpation of the shoulder will show maximum tenderness on an eminence of the greater tuberosity rather than in the deep gap between acromion and greater tuberosity. Finally, x-ray will show the calcification while it is negative in cases of rupture.

Partial rupture is harder to differentiate. However, the muscle spasm, the location of the tender spot, and x-ray evidence again will make positive diagnosis possible. In case of doubt, resort can be made to the exploratory incision.

The condition which has been discussed is one in which the calcification has formed in the tendon

and which has caused but slight or no irritation of the bursal floor, the so-called pre-acute stages. The acute bursitis is precipitated when after trauma of varying degree, the discharge empties into the bursa. Here, the calcified material is received by an excessive quantity of synovial fluid which the irritation has induced. Fibrin is present in large quantities.

The presence of the sand-like particles in the bursa is very irritating, as they act as foreign bodies, and the inflammation is increased. At the time of discharge and for about three days afterward, pain increases markedly and is controlled only by large doses of morphine.

Then absorption takes place and is complete after three weeks, the pain subsiding as the process goes on and absent on completion.

In addition to the symptoms which were present with the undischarged calcified deposits, a hyperalgesia and hyperesthesia overlie the inflamed bursa and can be easily marked out. This line can be visualized when the bursa is distended with fluid and fibrin. Even when the greater tuberosity is the maximum tender point, the lower deltoid region is the seat of maximum pain. Sometimes, tenderness over the greater tuberosity is absent although deltoid pain persists. The pain is exquisite preventing the patient from sleeping.

Atrophy of the spinati appears in cases which have had severe symptoms for over three weeks. The spine of the scapula is prominent on the affected side and may even suggest the atrophy of paralysis. The deltoid may atrophy here in contradistinction to the cases of tendon rupture, in which the deltoid hypertrophies. All of the muscles of the arm show the atrophy of disuse in long standing cases. Palpation of the shoulder muscles will show them to be thick and hard with spasm.

Codman has observed what he calls a "down it will go" symptom, in which the individual, on picking up an object will involuntarily release it from his grasp on elevating his humerus to a point at which a sensitive part of the tendon is brought below the acromion process and pain is produced. This occurs in cases of complete and incomplete rupture, and calcareous tendon deposits.

A fourth cause of bursitis is what is called tendinitis by Codman. The early degenerative tendon changes which were present in the development of tendon rupture and tendon calcification are found in this condition. Tissue sections show increased staining with eosin hematoxylin. There is no inflammatory response present. Absorption from foci of infection has been credited as the cause. Codman believes that the

changes may be initiated by a partial tear on the joint side of the tendon. The surface of the bursa which has been incontact with the tendon gets the bullseye appearance which is typical of that accompanying calcified deposits in the tendon. There is an angry red swollen surface with a cream colored central papilla. However, it differs from that of calcified deposits in that this lesion does not enter the tendon but is confined to the bursa, and also, it does not contain calcareous material.

Because of the bursal sensitivity, muscle spasm is produced which results in adhesions within the bursa. The patient tends to flex the forearm and hold the arm in adduction and internal rotation due to overpullings of the pectoralis major, teres major and latissimus dorsi. In this position it becomes fixed by muscle spasm and adhesions.

In an adhesive bursitis, the arm cannot be elevated over 90 degrees actively or passively. Passive glenohumeral movement is tested by fixing the scapula with one hand while elevating the humerus with the other.

The usual history states that pain had a gradual onset, that it originally was felt at the fibers of insertion of the deltoid. The patient is unable to sleep on the affected side. His arm assumes a sling position, from which abduction andelevation are limited

and painful. If the condition is of several weeks' duration, there will be atrophy of the spinati and the tuberosities of the humerus. Palpation will show no localized tenderness. X-ray findings will be negative except for bone atrophy.

Diagnosis is important no only as an index to treatment but also to prognosis. While complete tendon ruptures require surgery and calcified deposits may or may not, tendinitis is treated best by non-surgical means. The prognosis for tendon rupture is recovery after years; calcification, after weeks; and tendinitis, after months.

The symptoms which differentiate the bursitis of tendinitis from that caused by the three previously described agents are the gradual onset, the assuming of internal rotation rather than the external rotation of calcified deposits, the limitation of motion by adhesions, as can be shown when muscle spasm is removed by anesthesia, the absence of points of tenderness, the ability to perform abduction before spasm sets in, and the absence of x-ray findings of calcified particles. Diagnosis is confirmed when an exploratory incision discloses a lesion restricted to the bursa and lacking in calcified material. These cases are separated from partial rupture only by lack of a traumatic history.

Discovery of foci of infection suggests that the process is initiated by toxic absorbtion.

## DIFFERENTIAL DIAGNOSIS

Pain may be caused by local or generalized processes, or may be referred from a diseased portion of the viscera.

Patterson and Darrach, 1939 (30), suggest that bursal lesions are distinguished from intra-articular lesions by the finding that flexion and extension are not painful, but abduction is.

Local lesions causing pain in the shoulder, to be differentiated from subacromial bursitis are osteomyelitis, bone sarcoma, acute myositis, fibrositis and fasciitis, epiphysitis, fracture and dislocation of the shoulder, biceps tendon rupture or displacement, tumors of the superior pulmonary sulcus and ordinary contusions of the shoulder. Lesions with tumor but not pain are hygroma of the subacromial bursa and supracromial lipoma. With axillary or suprascapular nerve paralysis, glenohumeral motion is limited.

Brachial neuritis, neuralgia and rheumatism are not considered legitimate diagnoses for vague shoulder pain by Codman. He classes most of these types under the partial tendon rupture group, whose pathology he has demonstrated on autopsy.

Haggart and Allen, 1934 (32), believe that a diagnosis of brachial neuritis is warranted only in

cases of brachial plexus injury, cervical rib, or peripheral neuritis due to lead or diabetes.

As rheumatoid, hypertrophic and suppurative and specific arthritides are generalized diseases and as such are not limited to the shoulder, they should not be diagnosed when the shoulder is the only joint involved. This will be considered further under generalized disease affecting the shoulder.

Osteomyelitis in the acute stage is a disease with toxic manifestations. It is characterized by local pain and tenderness, high fever of sudden onset, nausea, vomiting, chill, rapid pulse, delirium, and other signs of toxicity. After two weeks, x-ray findings are typical. In chronic osteomyelitis, draining sinuses and bone deformity differentiate this from bursitis.

Bone sarcoma is indicated by local pain, and tenderness, presence of a tumor mass, weight loss and typical x-ray findings.

Acute myositis results from trauma, bruising, exposure to cold. If suppurative, it will have headache, fever, chills, sweating and localization of pain and swelling in affected muscles with later fluctuation. Non-suppurative myositis may be localized but is often generalized. Muscles atrophy and undergo a replacement fibrosis. Clinical features are weakness, loss of weight, stiffness of the muscles and clumsiness.

The condition is progressive and culminates in contractures.

Primary fibrositis is considered to be a type of insterstitial myositis with "rheumatic nodules." The pathology present consists of an inflammatory hyperplasia of white fibrous tissue anywhere in the body, that is, in fascia, aponeurosis, sheaths of muscles and nerves, tendon, ligaments, articular capsules, subcutaneous tissue and periosteum. A serofibrinous exudate in fibrous tissue produces trancient symptoms and then undergoes resolution. Slocumb (33) has separated inflammatory hyperplasia of fascial sheets into a distinct entity of fasciitis. The symptoms of primary fibrositis are local pain and subjective stiffness, exacerbated by cold or damp weather, exposure to drafts or fatigue, with relief produced by "limbering up," local heat or acetylsalicylic acid.

Epiphysitis occurs in pubescent and young adults, while bursitis rarely appears in an individual under 40 years. Pain is local, motion is not limited and the epiphysis exhibits changes to x-ray.

Fractures of the shoulder will have history of trauma, impaired mobility (unless impacted), deformity, ecchymosis, possibly bullae, crepitus and point tenderness. X-ray proves the diagnosis. Dislocation gives absolute immobility, change in anatomical relations, alteration in contour, and change in

length. Coracoclavicular dislocation can be shown by palpation and x-ray.

Codman believes that biceps tendon displacement occurs more commonly than is generally supposed. The condition causes symptoms only when the humerus is in external rotation, at which time snapping of the tendon is heard on abduction and pain is produced on flexion of the elbow or elevation of the arm, when a tear of the biceps sheath permits it to slip out of its groove. Biceps rupture is characterized by weakness of elbow flexion and presence of a mass at the belly of the biceps on muscular contraction.

Brown, 1919 (34), presents rupture of the tendons of latissimus dorsi and teres major as a cause of stiff and painful shoulder. A pathognomonic sign is point tenderness of the inner surface of the humerus high in the axilla. This sign has also been noted by Brickner. Deltoid atrophy of disuse follows. The mechanism of production is a sudden sharp adduction of the humerus in extension or flexion.

Nathanson et al., 1939 (35) (36), warn that with the persistance of shoulder pain after removal of a calcified deposit, the surrounding structures should be investigated, citing a case of theirs in which a patient underwent surgery for calcified tendon deposit, while his major lesion was a superior pulmonary sulcus

tumor of the same side, which brought about his death. Rubert reports one case simulating calcified supraspinatus tendon deposit in which Trichina spiralis was found although the muscle was intact.

Rubert mentions the occurrence of giant cell tumor of the humerus as a consideration in differential diagnosis, especially in patients under 20. Jaffe, Sutro and Lichtenstein describe a xanthoma of the tendon.

Ordinary contusions to the shoulder present a history of trauma, show ecchymoses, may hinder but do not restrict motion, are relieved spontaneously after a week or ten days.

Hygroma of the subacromial bursa is the diagnosis for a large, painless cystic tumor of the shoulder with the tumor placed subdeltoid and post acromial. Codman believes that hygroma develops from the traumatic development of a communication between the joint capsule and the subacromial bursa, with the resultant expression of joint fluid by joint motion into a gradually expanding bursa. Hygroma is rarely found under the acromion, usually occurring at the junction of the lateral border of the sternocleidomastoid and the clavicle. Diagnosis can be aided by aspiration, and injection of radiopaque fluid.

Supracromial lipomas are painless fluctuating masses, causing no symptoms and which lie above the acromion process.

Axillary nerve damage results in paralysis of the entire deltoid and teres minor. This is a complication of anterior dislocation of the shoulder. Often the biceps, coracobrachialis and brachialis anticus are also involved. The nerve supply to all five muscles is from cervicals 5 and 6. The chief sign of paralysis of the deltoid and teres minor is difficulty in abducting the arm. Abduction is weakly accomplished through action of the supraspinatus. One should determine that the weakness of action is due to paralysis rather than inhibition from pain. Reaction to galvanic and faradic current is typical. Atrophy of the deltoid is evident later. Teres minor atrophy is not evident because the muscle is covered by the trapezius. Eventually, all the muscles of the shoulder atrophy because of disuse. Codman points out that supraspinatus tendon rupture should be looked for in association with axillary nerve paralysis when the patient is unable to abduct his arm to any degree.

Suprascapular nerve paralysis has a history of a blow on the shoulder in the region of the suprascapular notch. Since the nerve exclusively supplies the supra and infraspinatus muscles, symptoms of a

traumatic neuritis appear. The patient is unable to abduct, and external rotation is weakened. Pain is felt over the scapula. Marked atrophy of the spinati appears more rapidly than in any previously described condition. Tests for nerve integrity as outlined in axillary nerve paralysis indicate the etiology of the condition.

The generalized diseases which may have metastases or foci in the shoulder make an imposing list. These are polyneuritis, lead palsy, diabetic neuritis, gout, pyemia with multiple abscesses, malignant metastases, bone tuberculosis, bone syphilis, acute rheumatic fever, periarthritis, and arthritis - hypertrophic, rheumatoid, suppurative, tuberculous or gonococcal. The manifestations of these diseases are widespread, so no difficulty should be anticipated in differentiating them from subacromial bursitis.

Formerly, the diagnosis of hypertrophic or rheumatoid arthritis or periarthritis was made for this condition. The pathology of those diseases closely resembles that found in chronic subacromial bursitis. To establish subacromial bursitis as a disease entity, Codman had to distinguish between it and these conditions.

Codman's fundamental premise is that arthritis and periarthritis are constitutional diseases

and as such will rarely be nonarticular. His next premise is that due to its architecture, the shoulder is the least affected and retains the least residual of arthritis and periarthritis, of any major joint in the body. Consequently, he concludes that the arthritis and periarthritis should be diagnosed with reservation as a cause of pain in the shoulder in the presence of a generalized arthritic or periarthritic condition and should never be diagnosed if other joints are not involved.

A basic difference in the pathology of arthritis and subacromial bursitis is that in the former, changes take place in the joint articular surface, while in the latter, changes appear first in the tendons and the bursa, with involvement of the joint capsule if the condition becomes complicated.

When, in the course of acute arthritis, pressure is maintained on a softened, swollen joint surface, permanent deformities of the joint surface develop. In the shoulder, permanent lesions do not develop or are held to a minimum because of the looseness of the shoulder ligaments, the status of the shoulder as a non-weight bearing joint, and theease with which the shoulder is immobilized.

Contrast this with the anatomical and physiological conditions under which the tibiofemoral,

acetabular, vertebral and phalangeal joints function. That arthritis should leave less distortion and calm down sooner in the shoulder joint than in other joints is understandable. Conclusions reached by deduction are borne out by clinical experience. Codman states, "Dealing with shoulders as much as I have for 30 years, I can recall but one case of a permanently damaged shoulder from a local non-traumatic or non-tuberculous arthritis."

In the series of painful shoulders upon which Codman operated, he was unable to find the pathologic changes characteristic of arthritis. Comparing the findings of generalized arthritis with those of so-called "arthritis of the shoulder" he states that fluid, while prominent in other affected parts of the body, rarely develops in the intact shoulder joint capsule because as soon as the pain of irritation is felt, the joint is put at rest. The villous conditions, so common in arthritic joints are unusual findings in the shoulder. There is no fibrosis of inflamed villae nor impingement of "lipoma arborescens" between articular surfaces. The ligaments are rarely thickened. Although absorption of cartilage does occur on the humeral head in bursitis, the erosion is superficial and is limited to that portion which is exposed through the gap in the capsule and is proportionate to the degree of rupture of the supraspinatus tendon.

Deformity of the articular surface does occur in the shoulder but only after very distinctive traumatic lesions or suppurative infections. In the worst cases Codman has seen due to these causes, the heads of the bones were rounded knobs, not much misshapen and with no hypertrophic excrescences. Lipping of the articular margins is the most characteristic change in cases of hypertrophic arthritis in all joints. Calcified spurs tend to develop in those tendons inserting on the articular edge. These changes are not found on the edge of the head of the humerus. The only change seen is a corona-like hypertrophy of the articular edge, which is symptomless. In the glenoid, there may be some ossification of the fibrocartilaginous labrum, but there is no hypertrophy. Variation may be seen in the shape of the glenoid surface and labrum, but the cases of distortion are limited to those with relaxed capsule due to paralysis of adjoining muscles, recurrent dislocation, or rupture of some of the short rotators. In these conditions the head of the humerus can ride over the edge of the glenoid, traumatizing it. Changes in the tuberosities cannot be considered arthritic as they are outside of the joint capsule.

Bony ankylosis of the shoulder joint rarely occurs except in children following tuberculosis and osteomyelitis. Both of these conditions are very rare

in adults. Even a fibrous ankylosis is rare in the shoulder, as muscle spasm checks joint movement before fibrotic bands are developed. The shoulder is capable of rapid resolution of fibrous bands should they occur.

With this discussion of the pathological changes of arthritis, one can conclude that arthritic changes either do not exist, or remain at a minimum in the shoulder.

Slocumb defines periarthritis as a generalized inflammation of the capsule and bursa and of the tendons as they pass through the joint capsule. He believes that periarthritis is one of the causes of subacromial bursitis. This condition most closely resembles the tendinitis of Codman. However, Codman contends that when the condition is localized to one joint, it cannot be diagnosed as periarthritis. Any limitation of motion is due to inhibition, as tendon ruptures do not occur in this condition. Calcified deposits or bone changes do not occur, so x-ray will be negative for these findings.

Consequently, subacromial bursitis is distinguished as a clinical entity from the arthritides and periarthritides, and these two diagnoses are eliminated when the lesion is confined to the shoulder. When generalized arthritis is present, one must still consider subacromial bursitis when shoulder pain obtains.

Finding the condition in joints other than the shoulder, aspirating specific organisms from the joint, finding the concomitants of a disease in other parts of the body (such as pulmonary findings in tuberculosis or tophi in gout) or a history of previous septic disease (such as gonorrhea, pneumonia or osteomyelitis) all tend to eliminate subacromial bursitis as a diagnosis.

Paterson, 1928 (37) in South Africa reported a case of sanguinous scorbutic swelling simulating a prepatellar bursitis.

Pain referred to the shoulder from sites of visceral inflammation is transmitted by way of the phrenic nerve from roots of the cervical nerves three, four and five which also supply the skin over the trapezius ridge. Codman believes that neurologic lesions which locate pain in a site other than that of the lesion should also be considered as referred.

The involved neurologic lesions act by producing a traumatic neuritis of the nerve roots forming the brachial plexus. Pain is referred to the distribution of these roots.

The most common causes of pain referred to the shoulder are coronary sclerosis, aortitis, aortic aneurism, splenic rupture and splenic infarcts. These generally refer to the left shoulder.

The following types, listed by Cope ( 3 ), while commonly causing referred pain, rarely but occasionally may refer to the shoulder.

1. Pleural, Pulmonary and Mediastinal Lesions: Pulmonary infarcts, pneumonia, diaphragmatic pleurisy, intrathoracic or axillary new growths, pneumothorax or pericarditis.
2. Abdominal Visceral Lesions: Gastritis, gastric ulcer or carcinoma, gastric dilatation, perforated gastric or duodenal ulcer, cholecystitis with adjacent peritonitis perforation of the gall bladder, hepatic abscess, acute pancreatitis, ruptured liver, appendicitis and ruptured ectopic pregnancy.
3. Peritoneal Lesions: Subphrenic abscess, ascites, pneumo or hemo-peritoneum and any of the local peritonities caused by visceral perforation.
4. Neurological Lesions: Herpes zoster, spinal new growth, cervical Pott's disease, cervical arthritis and cervical ribs, or the scalenus anticus syndrome.

Codman points out that these lesions can cause pain but will not cause limitation of motion, localized tenderness or muscle spasm with their consequent atrophy and capsule relaxation. He maintains that with the ex-

ception of certain neurologic lesions organic change will not take place in the absence of local pathology. Hyperalgesia may be present but not deep local tenderness. Muscle atrophy and bone decalcification may develop from disuse.

It must be remembered that due to thefrequency of shoulder lesions, possibility of their presence should not be dismissed when visceral lesions are found.

In all these lesions with referred pain, the site of origin is made evident by local physical signs, with the possible exception of spinal new growths, cervical Pott's disease, cervical arthritis or cervical rib. Consequently, confusion in diagnosis exists only in the last named group.

The symptoms of cervical rib are a bony tumor in the supraclavicular region, disturbance of blood supply to the fingers, variation in blood pressure of arm in adduction, pain of segmental distribution, numbness and tingling. Diagnosis is confirmed by x-ray.

In all cases, there is no stiffness in the joint or atrophy of the shoulder muscles. Abduction can be accomplished. Onset is slow. None of the signs of bursitis such as crepitus, fluid, restriction of glenohumeral motion or muscle spasm are present.

Cervical hypertrophic arthritis is usually part of a generalized condition, with other lesions

occurring in the hips, knees and distal phalangeal joint, but may occur in their absence. Presumptive diagnosis is made by finding rigidity of neck muscles. Lateral motion of the head and neck will be restricted more on one side than another. Pain is of a transient nature. There is no limitation of glenohumeral motion, nor muscular atrophy. X-ray of the spine shows lipping of vertebral border and spur formation.

Tumors of the spinal cord and nerves may cause shoulder pain, but they are rare. Those located in the cervical region form 25 percent of the total cord tumors. The first symptom is usually root pain which is sharp, sudden and severe. It is aggravated by motion and the patient holds his neck stiff. Tumors in the region of cervicals four and five give pain on the top of the shoulder which is distributed along the roots. Uni- or bilateral numbness, paresthesia spasm and paralysis follow. Muscle atrophy is marked. Lumbar puncture, showing decreased lumbar pressure, no change on jugular compression, and xanthochromic reaction all indicate spinal block. Muscle weakness and atrophy set in gradually and without initial spasm, contrast to cases of bursitis.

Tumors of the peripheral nerves sometimes occur in the shoulder region and give rise to constant

pain. Pain has nerve distribution and may be associated with a palpable mass. The condition occurs very infrequently.

Cervical Pott's disease may cause referred pain to the shoulder. As it occurs in children, it is rarely found in the bursitis age distribution. The lesion is part of a general tuberculosis and presence of pulmonary lesions with their accompanying signs and symptoms will be found.

Traumatic neurosis, hysteria and malingering may be mistaken for shoulder injury, or conversely, organic lesions may receive those labels. A traumatic neurosis should be considered as a concomitant to any injury which makes an individual into an invalid temporarily. The patient's entire attention is focussed on himself, his pains, his limitation of motion and his fear of not recovering. This can be treated by placing the patient in a cheerful atmosphere with good supportive treatment and reassuring the patient of his ultimate recovery.

Hysteria is a psychoneurosis. It is diagnosed by complaints of motor and sensory derangements of bizarre type and distribution. It should not be diagnosed where muscle spasm and muscle and bone atrophy are present. Jog on rotating the head of the

humerus or changes in scapulo-humeral rhythm, also should rule out this diagnosis. It should not be suspected in individuals who have gone through most of their life without exaggerating their illnesses, nor where there is history of a bona fide injury and a persistence of the original symptoms for a long period of time.

Frequency of malingering has increased since the development of accident compensation insurance. It usually occurs in an individual who suffers a genuine trauma and cocking an eye to a disability award, claims the persistance of symptoms. These patients can be picked out by their lack of muscle spasm and muscle and bone atrophy. They may fake limitation of motion, in which case contraction of opposing muscle groups can be palpated. A portrayal of ruptured supraspinatus tendon may be exposed by the individual's frequent inability to abduct when he is in the stooping position. A spotty work record prior to injury suggests the type of individual who would malinger. Changes and exaggerations in the descriptions of the symptoms tend to do this also. One should always remember, however, that many truly suffering individuals have been termed hysterical types or malingerers because of our own ignorance.

In summary, although numerous diseases cause pain, point tenderness, inability to abduct over 90 degrees and limitation of motion - any one or combination of these symptoms - subacromial bursitis is still a separate entity capable of diagnosis on the basis of history, physical examination and radiological findings.

## TREATMENT

As subacromial bursitis develops from several types of pathology, the methods of treatment are varied. This also explains why any one treatment is not uniformly successful. As small calcifications and tendinitis are labile lesions and tend to resolve spontaneously, numerous types of therapy have been erroneously credited with effecting a cure. Consequently, treatment runs the gamut from diathermy and light rays to massage and surgery.

Therapy is aimed at the primary pathology. Most writers consider only calcified deposits and tendinitis when discussing treatment. Papers on the treatment of partial and complete tendon ruptures are scarce. Many writers make no consideration whatever of the primary pathology in their method of treatment.

Because of its depth, thesubacromial bursa cannot be treated by sclerosing solutions or excision, which are successful procedures for correcting inflammation of superficial bursae. Due to the nature of its attachment to the acromion, clavicle and tuberosities of the humerus, complete removal is an impossibility, according to Codman.

Numerous types of therapy are used in treating calcified deposits and tendinitis. Palmer, 1935 (38),

put his patients to bed. In the acute cases, he put the arm in a sling. He then applied heat by means of diathermy. Small rounded electrodes, about 2½ inches in diameter are carefully moulded to the contour of the shoulder and are bound down to the anterior and posterior surfaces of the shoulder by Ace bandages. Diathermy is given for thirty minutes at a current intensity of 400-500 milliamperes. Palmer emphasizes that the electrodes must be attached so that they cause active hyperemia in the shoulder.

In the subacute or chronic case, diathermy is followed by massage motion and exercise. In adhesive bursitis, conservative attempts should be made to gradually stretch the adhesions. Massage, consisting of light stroking or effleurage, is followed by gentle kneading. After the pain has subsided, active and passive motion are used. Palmer suggests wall creeping exercises. A mark is made on the wall, and the individual is encouraged to move his fingers up the wall to it. The patient is encouraged to beat his past record until full abduction is attained. Circumduction exercises are given.

Another physical agent on which Palmer and Heald of the Royal Free Hospital of London report satisfactory results is exposure of the shoulder to water-cooled ultra violet radiation. The skin is cleansed

with alcohol and the quartz lens is applied in direct contact with the skin in the neighborhood of the bursa. Increasing exposures of from thirty to sixty seconds are given to two or three areas, repeated every few days. The purpose of the treatment is to produce an erythema and counter-irritation of the overlying skin. This is supplemented by diathermy, massage, motion and exercise.

Granger, 1926 (13) believes that physiotherapy will give relief if not cause the resolution of a deposit. He endorses a course of heat, chlorine ionization, massage, stretching, static brush discharge and abduction exercises.

Mumford and Martin, 1931 (14), treated 14 cases of subacromial bursitis with calcification by diathermy giving complete relief of symptoms and complete function in all cases. They believe that surgery should be resorted to only after the failure of diathermy to give relief.

Cambell and Deering (14) state that conservative treatment should be tried before surgery. Cooperman (15), reporting 50 cases and Titus (39), reporting 150 cases, believe that the deposit can be dissipated under the influence of diathermy.

Echtmann, 1936 (40), believes that bursitis can be treated in all degrees with physical methods.

He uses heat, cold, galvanization, ionization, infra red ray and diathermy.

Lattman, 1936 (41), treated 20 cases of subacromial bursitis with roentgen therapy with relief of pain and restoration of function. He believed relief was obtained more rapidly by this method than by other non-surgical means of treatment. The treatment customarily given was 350 roentgens with the following factors: 200 kv., 0.25 mm. Cu filter, 50 cm distance, 15 by 15 cm field applied to the anterior and posterior shoulder.

During the first 24 hours following treatment, the symptoms are somewhat aggravated, but within the second 24 hours there is a marked diminution of pain and a greater ability to move the arm. As a rule only one treatment was necessary.

Weinberg, 1940 (42), reported excellent results with x-ray on subacromial bursitis with or without calcifications. The shorter the duration of symptoms, the sooner was the condition relieved. So sure is he of the efficacy of x-ray in cases of calcification that he assures these patients of certain cure.

As the requisite of treatment of calcification is removal of the deposit, a method which would cause expulsion of the deposit into the bursa and removal from the bursa should result in a cure.

Patterson and Darrach, 1937 (30), reported a method of irrigating the bursa by means of introducing saline by needle at one point of the bursa while it is being withdrawn at another point. This was at the suggestion of Smith-Peterson. They report quick relief in most cases. Kaplan and Ferguson (21) and Patterson and Patterson ( 2 ), using a similar procedure, try first to place an aspirating needle into the deposit with the aid of fluoroscopy and if this fails, they insert a second needle and irrigate.

Patterson and Darrach irrigate bursa in the absence of x-ray findings of calcified material as their experience has shown them that the bursal washings nevertheless contain calcified material. Calcifications may not be visualized because of poor placement of the arm, bad x-ray technique, or the smallness of the calcified deposits. However, if they make this finding in all cases, without differentiating from partial or complete tendon rupture of tendinitis, their findings are contradictory to those of Codman, Rubert, Ferguson and Kaplan.

Fields, 1915 (43), is unique in advising the injection of five per cent codoform in glycerin into the bursal sac. This treatment has not received acceptance.

Richards, 1931 (44), empirically gave patients with acute subacromial, subscapular and infrapatellar bursitis iron arsenite and iron cacodylate intravenously and in 70 cases reports immediate relief of acute symptoms in all cases. He makes no claims for the end result, however.

In acute cases, Stewart, 1928 (45), put the arms in abduction and external rotation by traction to overcome the pull of the stronger abductors and internal rotators of the shoulder, thus relieving muscle spasm.

As downward traction on the arm has been found to relieve pain in bursitis of the shoulder, Ferguson, 1933 (46), improvised a heavy lead cast, placed on the forearm but attached by skin traction to the supracondylar region, to be used in conjunction with conservative treatment.

Steindler (47) believes that surgery should be reserved for cases of long standing or cases showing lime deposits not yielding to conservative treatment.

Jones and Lovett state that " a definite indication for surgery lies in the presence of a definite lime deposit shadow in the roentgenogram existing in connection with marked symptoms." Albee and Bartels (48) concur with them.

Brickner used conservative forms of treatment without success, stating that none of his cases with lime deposits had been relieved except those in which surgical removal had been performed. He advises operation first in chronic cases with exacerbations and secondly in all other types which did not respond to his methods of automatic abduction through the use of dumbell exercises, heat and passive manipulation.

Collins, 1938 (49), gives the acute fulminating cases morphine with hot or cold packs locally, together with complete rest. If this fails, operative measures are required. The usual procedure is exploration of the bursa and drainage of the calcified material. Collins curetts the area, and inserts his finger into the bursa to break the adhesions. He excises a portion of the roof of the bursa and closes without drainage. He starts mobilization after 48 hours, supplementing this with infra red ray, massage and hot packs. Collins suggests injecting the bursa with procaine hydrochloride, also injection with procaine followed by the introduction of 500 ccs of saline to rupture the bursa, after the method of Ober, 1938 (49).

In the case of calcifications, Codman believes that a deposit should be sought out and removed or at least have its surface nicked so that it can empty into the bursa.

On opening the bursa, a reddened area with a pale center which is flat or elevated can be seen on the bursal base overlying the tendons. If this area is not evident, the arm is rotated so that the tendon insertions are progressively swung past the incision. The bursa is examined for multiple lesions, pricking suspected areas that may not be inflammatory. X-ray aids in finding the deposits. When the calcifications have all been located, the overlying bursa is incised.

The calcified material will be pasty or stony, depending on whether process is acute or chronic. Sometimes the paste will squeeze out by itself on nicking its papilla. At other times the solid deposit will be dislodged, leaving the cavity in the tendon clean and sharp. An intermediate type will leave an accumulation of rubbery material which has to be curetted out.

Although small calcifications may be symptomless and spontaneously disappear, Codman believes that large deposits will cause severe symptoms eventually and cure results only from surgery. Non-operative treatment such as foreign protein, ultra violet ray and diathermy are worthless in his opinion. He attributes cases of reported cures by this method to spontaneous discharge or absorption of particles. Carnett (50) (51) agrees with Codman that excision of the deposit

is indicated in all cases as this is the simplest, speediest and most efficient means of affecting a cure.

Rogers and Kaplan and Ferguson (21) believe that partial ruptures of the supraspinatus tendon will heal if the arm is held in abduction, but complete rupture requires open operation. Codman agrees with them, ordering enforced rest of the arm for a few days, followed by early mobilization.

To repair a torn supraspinatus tendon, a channel is made through the anatomic neck of the humerus and the inner face of the greater tuberosity. Several drill holes are made through the greater tuberosity into the channel. The fibrous edge of the tendon is excised, and with the arm in abduction the tendon is approximated to the channel and sutured in place through the drill holes with silk or fascia lata.

The operation for suturing the torn tendon of the supraspinatus should be done before atrophy of the tendon stub and tuberosity and formation of granulation tissue has occurred. Unfortunately, this injury is seldom seen until it is three weeks old and changes have already developed. Incision is made by either of two methods which will be described, and the torn tendon ends are located and freshened. They are then apposed and sutured. Codman claims that the arm is ready for light work six weeks after surgery.

If the patient declines operation, treatment is aimed at improving the blood supply and approximating the torn tendon ends plus rounding off the irregular surface of the articular head. The first aim is best served by fixing the arm in elevation to relax the tendon, the second, by gentle exercises in the stooping position.

To be effective, fixation would have to be instituted soon after the injury and would have to be continued for three weeks. In old injuries this treatment is not practical as the tendons retract, leaving a gap too wide to be easily approximated. Also, the formation of adhesions are encouraged.

These patients are instructed to sleep with their arms abducted and behind their head to relax the short rotators. Palliative drugs are not often required. Diathermy is useless and manipulation contraindicated because of the nature of the pathology. Massage is beneficial in maintaining muscle tone, but does not affect the condition of the tendon.

Codman says that bursal adhesions develop as sequelae to tendinitis, calcified deposit and partial tendon rupture.

For chronic adhesive bursitis or tendinitis, Rogers (52) first applies heat in the form of diathermy baking or fomentations. Then gradual stretching under

guidance is started. Exercises are then given up to the point of reaction. If no improvement is seen after two weeks, the adhesions are broken up under anesthesia. Manipulation must be gentle or tendon rupture or fracture of the humerus might result.

Codman declares that this may be treated surgically, although he prefers to use non-surgical treatment. Surgery involves making small incisions into the tendon at probable sites of necrosis. These incisions are made along the axis of the tendon fibers just above their insertion into the tuberosities. Often, small amounts of liquid will be expressed through the incisions. Codman formerly broke the bursal adhesions, thus freeing the arm.

He no longer operates on these patients, having found that he gets better results from gentle traction on the arm in abduction. With the patient supine, in bed, he applies a splint to the back of the forearm and ties each end of the splint with a loop over the railing at the head of the bed. A light dose of morphine is given the first night to secure relaxation. In 12 to 24 hours, the spasm relaxes, adhesions yield and the tuberosity passes under the acromion. The arm abducts easily and becomes externally rotated. In a day or two, he removes the splint, maintaining abduction by a gauze loop around the wrist and bedstead. The

patient arises daily and does stooping exercises. These lengthen his shoulder tendons take out the stiffness and improve vascular supply without strain or friction on the tendon insertions at the tuberosities. The patient stays in bed until he can freely move his arm about in any direction above his head. This takes about one to two weeks. For a few weeks more, he sleeps with his arm above his head and continues his exercises gradually doing them when erect. Codman reports good results from this treatment.

Forcible rotation of the arm to break adhesions when the patient is under anesthetic more often than not will fracture atrophied bone or rupture shoulder tendons first. Immobilization in the sling position is contraindicated as the patients cannot mobilize their arm after this.

If the patient cannot spend two weeks in bed, Codman suggests diathermy, although he does not believe that it is very effective. Brickner uses dumbbell exercises in these cases.

Often, in the course of traumatic rupture of the tendons of the short rotators, the tendon of the long head of the biceps is ruptured also. Biceps function of flexion of forearm on the arm, arm on scapula and supination of the forearm are accomplished mainly by muscle developing from the short head of the biceps.

The long head of the biceps is chiefly a stabilizer of the glenohumeral joint and is of secondary importance so far as application of power is concerned. In cases where rupture of the long head of the biceps is the sole shoulder injury, function of the shoulder remains almost normal. Stability is maintained by the short rotators.

When biceps tendon rupture is associated with short rotator tendon injury, the tendon of the long head of the biceps must be repaired to maintain joint stability. This can be accomplished only by open operation. The biceps tendon may be found high in the groove or retracted below the groove. In either case, the tendon is secured, the end is freshened and attached to any of several points. It may be sutured back to the fibrocartilage of the supraglenoidal labrum. It may be sutured to the capsule. As the tendon lies stationary in the groove, it may be anchored to the groove without interfering with motor power of the arm. However, this insertion would not make for shoulder stability. When the supraspinatus tendon is torn and the gap between it and the greater tuberosity is too great to bridge, the biceps tendon may be sutured to the supraspinatus tendon and the two then anchored on the greater tuberosity and supraglenoid tubercle, after the method of Codman. A portion of the biceps tendon may be excised to furnish a tendon graft for a short ruptured supra-

spinatus tendon.

Thick cordlike fibrous bands may form in the subacromial bursa although they are more common in the prepatellar and olecranon bursae. When present, they are associated with a very painful chronic bursitis with a loud creaking crepitus. Removal of these bands by simple excision is an easy procedure giving immediate relief.

Red inflamed villi may be present over the site of a calcified deposit or tendon rupture. When in the course of an operation for correction of tendon pathology Codman finds greatly hypertrophied villi, he excises them. Otherwise he does not interfere. He does not consider them indications for surgery per se in the shoulder because the relationship of the humeroglenoidal articular surfaces preclude their interposing between joint surfaces as occurs in the condylar type of joints such as the knee.

Various types of surgical technique have been developed by the numerous operators. The saber cut incision is one used by Wilson (3) to approach the shoulder joint. An incision is made through the acromioclavicular joint and continued with a saw through the base of the acromion. The anterior end of the incision is continued into the deltoid for two inches and the acromion process is now retracted laterally to give adequate

exposure of the glenohumeral joint and subacromial bursa. A simpler shorter incision is used by Codman, who places his patients in a specially adapted position. The patient is supine, with his arm alongside, off the table, in hyper-extension. The shoulder is then elevated and internally rotated by tension of the pectoralis major. On opposing this pull by pectoralis major by rotating the humerus at the elbow, the humerus is always under control and the humeral head can be placed into position at will. Codman starts his incision directly anterior to the head of the humerus, with the upper end going to the acromioclavicular joint and the lower end at about the level of the top, of the bicipital groove for a distance of one and one-half to two inches.

In case the diagnosis is questioned, a one-half inch incision is made in the same location from the acromioclavicular ligament. The humerus is rotated to expose the tendons and the incision is lengthened to the top of the bicipital groove when diagnosis is confirmed. The deltoid is cut through to equal the length of the skin incision. Incision into the bursa is then made when the arm is placed so that the bicipital groove lies directly in line with skin and deltoid incision.

The manipulations indicated are then accomplished and the incision is ready to be closed. First,

it is sponged out. The roof of the bursa may be sutured, although Codman does not do this, believing that the auto-irrigation produced by the synovial fluid clears out debris and prevents adhesions. The muscle is closed with a few loose catgut stitches. Skin closure is obtained in the ordinary manner.

Codman formerly placed the arm in abduction for several days post operatively. He discontinued this because of the drainage of synovial fluid into the tissues due to the compression of the subdeltoid portion of the bursa which acts as a reservoir when the arm is in adduction.

Codman believes that early mobilization is the keynote of post operative treatment. At present, he has his patients carry their arm in a sling for a week to ten days. Then they gingerly try to use it for eating and dressing. The sling is then removed and swinging exercises in a stooping position are started. In this way, the short rotators are passively stretched by gravity and the scapula is abducted on the humerus. As these become free, that is complete motion is quite painless, standing exercises are prescribed. These exercises involve dorsal flexion and elevation of the arm to degrees at which pain begins to get severe. After three to six weeks, the patient is well except for twinges of pain that are probably

due to passage of the rough edge of the bursal incision under the acromion and acromioclavicular ligament.

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