Current Practice

JOINTS AND THEIR DISEASES

Disorders of the Shoulder Joint

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Though the shoulder joint is often considered to be synonymous with the glenohumeral joint, movement also takes place at the acromioclavicular and sternoclavicular joints, and a gliding movement of the scapula over the thoracic cage may contribute to shoulder movement.

The head of the humerus is held in the glenoid fossa by the musculotendinous rotator cuff, which is composed of the insertions of the supraspinatus, infraspinatus, subscapularis, and teres minor muscles, all of which blend with the joint capsule. The tendon of the long head of the biceps brachii muscle arises from the supraglenoid tubercle, and encased in its synovial sheath passes through the glenohumeral joint deep to the capsule and the rotator cuff and so to the bicipital groove on the anterior surface of the humerus. The subdeltoid (or subacromial) bursa lies superficial to the rotator cuff tendons, separating them from the arch formed by the acromion, coracoid process, the coraco-acromial ligament, and the deltoid muscle.

Clinical Examination

Inspection.—This may reveal muscle wasting (Fig. 1), effusion within the joint (usually presenting anteriorly), or redness of the overlying skin.

FIG. 1.—Wasting of muscles of left shoulder. Reproduced, by permission, from *Reports on Rheumatic Diseases*, 1968, No. 34.

Palpation.—This may indicate increased warmth over the joint, suggesting inflammation. Tender points should be located—for example in supraspinatus tendinitis or subdeltoid bursitis tenderness is maximal at the point of the shoulder; in bicipital tendinitis tenderness is maximal in the bicipital groove.

Passive Movements.—The most valuable are glenohumeral abduction, elevation, internal rotation, and external rotation. To test these movements, it is best to stand behind the patient.

(1) To test abduction, the scapula should be grasped with one hand to eliminate scapulothoracic movement and the arm abducted with the other hand. The point at which the patient's scapula begins to move marks the limit of abduction. This should be about 90°.

(2) Allowing full scapulothoracic movement on top of scapulohumeral abduction completes full elevation of the shoulder. This should approach 180°. During the full abduction-elevation movement the patient may experience pain at 60° which disappears when the arm is elevated beyond 100°. This sign is known as "the painful arc," and is positive in both supraspinatus tendinitis and subdeltoid bursitis.

(3) Internal rotation in the neutral position is tested by asking the patient to put his hand behind his back and touch his opposite shoulder blade. It should be about 90°.

(4) External rotation in the neutral position is tested by flexing the elbow to 90° and then holding the arm adducted to the side and, using the elbow as the pivot, rotating the wrist outwards. The range should again be 80-90°.

Differential Diagnosis of Shoulder Pain

SOFT TISSUE LESIONS

Supraspinatus tendinitis
Infraspinatus tendinitis
Subscapularis tendinitis
Subdeltoid bursitis
Bicipital tenosynovitis
Adhesive capsulitis (frozen shoulder)
Rotator cuff tears
Shoulder-hand syndrome

ARTHRITIC CONDITIONS

Inflammatory polyarthritis (e.g., rheumatoid arthritis, ankylosing spondylitis, etc.)
Osteoarthritis (often secondary to previous trauma)
Gout
Connective tissue disorders (e.g., systemic lupus erythematosus)
Septic and tuberculous arthritis
Neuroarthropathy
Haemophilic arthropathy

BONY LESIONS

Fractures and fracture-dislocations
Osteomyelitis
Primary tumours, e.g., sarcoma, osteoid osteoma, myeloma
Secondary deposits
Metabolic bone disease (e.g., hyperparathyroidism, Paget's disease)

NEUROLOGICAL CONDITIONS

Neuralgic amyotrophy
Herpes zoster
Poliomyelitis and Guillain-Barré syndrome
Syringomyelia

REFERRED PAIN

Cervical spondylosis
Myocardial infarction
Oesophageal pain
Pleural pain (e.g., apical bronchial carcinoma, diaphragmatic pleurisy)
Subphrenic lesions and bleeding into the peritoneum

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(5) Adduction may be tested by drawing the arm forward across the chest.

Resisted Movements.—These should be tested to see if there is muscle weakness or if isometric muscle contraction gives rise to pain.

The muscles that should be routinely tested are *supraspinatus*—resisted abduction with arm at the side, *infraspinatus*—resisted external rotation, *subscapularis*—resisted internal rotation, and *biceps*—resisted flexion of elbow.

General Examination.—Finally, it cannot be too strongly emphasized that the neck, chest, and abdomen should be examined routinely, since shoulder pain may be referred from these structures.

Diagnosis and Management of Common Soft-tissue Lesions

Supraspinatus Tendinitis.—This tendon is subjected to more stress than other components of the rotator cuff and is therefore most frequently affected.¹ Lesions tend to occur in middle age and may result from degenerative changes, overuse, or trauma. The painful-arc sign is positive, but passive movements are otherwise full and painless. Resisted elevation usually provokes extreme discomfort, and tenderness is maximal at the point of the shoulder or in the supraspinous fossa. X-ray of the shoulder is usually normal, though owing to small haemorrhages into the tendon calcium may be deposited in the tendon and is then visible on the x-ray film. Calcification in the rotator cuff is found in 8% of the population over the age of 30 years,² but does not necessarily give rise to symptoms.³

Most patients who develop this or other soft tissue lesions of the shoulder fear the onset of a crippling and progressive arthritis and need firm reassurance.

The patient should be encouraged to rest the shoulder, and all movements provoking pain should be avoided. The condition usually responds readily to one or more local injections of hydrocortisone. The patient sits with the arm hanging loosely to the side, and the gap between the humeral head and acromion is found by palpation. The needle is inserted through this gap perpendicular to the skin and passes through the subdeltoid bursa and into the supraspinatus tendon at a depth of 2–3 cm. The area is first infiltrated with 1% lignocaine; correct positioning of this injection should abolish pain in a few minutes. Then 50 mg. of hydrocortisone is injected into the same site (Fig. 2).⁴ If there is no response to

FIG. 2.—Site of injection into supraspinatus tendon.

three injections of hydrocortisone, friction massage to the tendon and local heat treatment may be instituted by the physiotherapist. Analgesics such as salicylates or phenyl-

butazone may be required to relieve the pain. Rarely, surgical removal of calcified material may be needed to relieve symptoms, especially in the chronic case.

Subdeltoid Bursitis.—Owing to the close anatomical relationship of the supraspinatus tendon and subdeltoid bursa, it is not always possible to differentiate between lesions of these two structures: indeed, they may often both be involved in the same inflammatory process. However, acute subdeltoid bursitis is usually a well-defined clinical entity. Pain is extremely severe, and the patient may fear even the smallest movement of the shoulder. Exquisite tenderness is present over the bursa, and the overlying skin may be reddened or warm. Acute deposition of calcium may occur within the bursa and is readily demonstrated on x-ray (Fig. 3).

FIG. 3.—Calcification in subdeltoid base.

The arm should be rested in a sling, and powerful analgesics may be needed to control pain. Local injection of hydrocortisone may be attempted, but is often difficult owing to the severe pain and tenderness. The condition usually responds rapidly to steroids given by mouth (e.g., prednisone 5 mg. t.d.s.), and since such treatment is not usually necessary for more than 10–14 days long-term side-effects need not be feared.

Acute subdeltoid bursitis also responds rapidly to a short course of deep x-ray therapy, and this may be used if steroids are contraindicated for any reason.

Finally, the condition may become chronic, particularly when there are deposits of calcium in the bursal sac. In such cases surgical evacuation of the bursal contents is usually indicated.

Bicipital Tenosynovitis.—This is an inflammatory disorder of the tendon sheath of the long head of the biceps muscle, usually due to excessive frictional wear or overuse. There may be some roughening of the bicipital groove (such as after a fracture). There will be tenderness over the tendon in the bicipital groove, a painful arc on shoulder abduction, and pain over the biceps tendon on resisted flexion or supination of the elbow.

This condition also usually responds rapidly to local hydrocortisone injection. The bicipital groove is most easily found if the shoulder is externally rotated, and lignocaine and then hydrocortisone 25 mg. are injected into the tendon sheath at the point of maximum tenderness. Two or three injections may be needed. Other treatment is rarely needed, but, again, the patient should be encouraged to rest the biceps muscle for a few weeks.

Rotator Cuff Lesions.—Tears of the rotator cuff occur most frequently in the supraspinatus tendon, which is the weakest point and subject to the most wear.¹ There may be a history of a sudden strain in abduction, such as a fall on the shoulder usually in middle-aged or elderly people. The pain comes on abruptly, and there may be skin discoloration overlying the lesion. On arthrography, contrast medium will spill into the subdeltoid bursa if the tear is full-thickness. In addition to a painful arc and pain on resisted abduction of the shoulder there will be weakness or complete inability to initiate shoulder abduction. The degree of weakness will depend on whether the tear is partial or complete. When the acute phase passes, the patient may have no disability—there may be no pain and abduction may be initiated by a trick movement, so that the patient notices no weakness even with a complete rupture of the supraspinatus. In the acute phase pain is relieved by immobilization, local injection of lignocaine and hydrocortisone, and analgesics by mouth. After a few days, mobilization of the shoulder will prevent the supervention of a capsulitis. If weakness is prominent, especially after complete rupture, surgical repair is indicated.

Frozen Shoulder

This condition may be secondary to any of the disorders already described, or it may come on de novo following a trivial injury or after a period of immobilization of the shoulder.⁵ Gradually increasing pain disturbs sleep. There is increasing limitation of movement, external rotation often being most affected. Pain is maximal at the extremes of movement, but isometric resisted muscle contraction is painless. In most patients the condition is self-limiting and clears up spontaneously in six months to two years, relief of pain being followed after a few weeks by increasing range of movement. In the early mild case, where movement is not much restricted, recovery may be accelerated by injecting 50 mg. hydrocortisone into the joint on repeated occasions at weekly intervals. The same approach is used as for the supraspinatus tendon, but the needle is tilted slightly upwards and is advanced to about 5 cm. depth. Physiotherapy is usually advocated—combining gentle active mobilization exercises with local heat (radiant heat or short-wave diathermy), local cold (ice packs), or ultrasonic rays. Hydrotherapy may give comfort and increase the range of movement. Analgesics are almost always needed in this condition, and salicylates, indomethacin, phenylbutazone (or

oxyphenbutazone), or the fenamates may all be tried. If the condition does not respond to the above regimen, local x-ray therapy or systemic corticosteroids⁶ may be effective in 40–60% of cases. Manipulation of the shoulder under anaesthesia has been advocated,⁷ but may be harmful.⁸

Shoulder-Hand Syndrome

Steinbrocker⁹ coined the phrase “shoulder-hand syndrome” to describe stiffness of the shoulder shortly followed by swelling, hyperaesthesia, and vasomotor changes in the hand. The redness and swelling of the hand may be followed by atrophy and contractures. The underlying cause is thought to be a disorder of neurovascular reflex mechanisms in such conditions as myocardial infarction, hemiplegia and other neurological disorders, cervical disc prolapse, trauma to the shoulder, and drugs such as phenobarbitone.¹⁰ Early mobilization of the shoulder may prevent this disorder, but once started the process is difficult to reverse. If phenobarbitone is the cause, stopping the drug results in a rapid amelioration of symptoms.¹¹ Adequate analgesia, heat, and exercises to the shoulder and hand in the early stages may be effective. Where there is a personality disorder, antidepressive drugs may be helpful. Stellate ganglionectomy and oral corticosteroid therapy are advocated^{12 13} but are not always effective.

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TODAY'S DRUGS

With the help of expert contributors we print in this section notes on drugs in current use.

Oestrogens

Oestrogens have been defined as compounds which cause uterine enlargement and oestrus in spayed animals. This biological definition has little application to clinical practice. Oestrogens are seldom prescribed for their uterotrophic activity in the human female, and they do not influence sexual receptivity. The main sources of oestrogens in the human are the ovaries and the foeto-placental unit, though small amounts are also produced in the adrenals and testes, and in some animals, e.g., the stallions, the testes may be a very rich source of oestrogens.

Some 20 different oestrogens have been isolated from the urine of pregnant women. For the most part they are only metabolites of a few primary hormones and are of interest in reproductive physiology, but have no therapeutic application. By a happy chance the three primary hormones—oestrone,

oestradiol-17 β , and oestriol—were the first to be identified, and these “classical” oestrogens are still the point of departure for many synthetic preparations. Oestrone and oestradiol are the primary oestrogens produced in the ovary. They are interconvertible, and the enzymes which do the conversion exist in many tissues, so that the active circulating physiological oestrogen in non-pregnant women is probably an equilibrium mixture of oestrone and oestradiol. In such women oestriol is an irreversible metabolite of oestradiol. On a weight-for-weight basis oestriol is much less potent than oestrone or oestradiol in terms of its effect on the endometrium. It does, however, exert marked effects on the cervix and vagina, and there are grounds for believing that these organs are the target for oestriol just as the endometrium is the physiological target for oestradiol.¹ In pregnancy the situation is far different. The foeto-placental unit produces large quantities of oestriol, mainly by routes which do not involve oestrone or oestradiol.² This may mean that there is some function in pregnancy peculiar to this particular oestrogen. The therapeutic application of