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RECURRENT OR HABITUAL DISLOCATION OF THE SHOULDER-JOINT.

BY

A. S. BLUNDELL BANKART, M.C.CANTAB., F.R.C.S.,
 ORTHOPAEDIC SURGEON, MIDDLESEX HOSPITAL; SURGEON, ROYAL
 NATIONAL ORTHOPAEDIC HOSPITAL AND HOSPITAL FOR
 EPILEPSY AND PARALYSIS, MAIDA VALE.

RECURRENT dislocation of the shoulder-joint is an uncommon condition, the real nature of which appears to be little understood. It may almost be said to be peculiar to athletes and epileptics—a rather curious association which, as I shall show, is not without etiological significance. Most of the former class are powerful, healthy, athletic young men to whom the frequently recurring dislocation from trivial causes is a great and serious disability. In epileptics the dislocation sometimes recurs with every fit. The dislocation is nearly always anterior. I have only seen one case of posterior recurrent dislocation. That case was in a woman, and she was not operated upon.

The condition has been attributed to abnormal laxity of the capsule of the joint, and to weakness of the surrounding muscles. The abnormal laxity of the capsule is supposed to be due to stretching or imperfect healing of that structure after the reduction of an ordinary traumatic dislocation, and it has been thought that too early and too vigorous use of the arm may be the cause of the defect.

Various Operations.

Two types of operation have been performed for the relief or cure of this disability: (1) Operations designed to diminish the size of the capsule. These comprise various folding, plicating, plication, or overlapping procedures, with or without incision or excision of a portion of the capsule. (2) Operations designed to give support to the capsule, particularly at its lowest part where the dislocation is believed to take place during movements of abduction. The most favoured of these operations is that of Clairmont and Ehrlich, in which a strip of the deltoid muscle is transplanted in the form of a sling beneath the joint, and is supposed to contract and hold up the head of the humerus when the arm is abducted.

Both these types of operation are based upon erroneous ideas of the pathology of recurrent dislocation of the shoulder. In the first place, the capsule in these cases is not unduly lax. The capsule of the shoulder-joint is normally a lax structure, but more than one surgeon has commented upon the difficulty of raising a satisfactory fold when doing a plication operation for recurrent dislocation. Secondly, the muscles are not weak. There may be a little wasting after this, as after any other injury to the shoulder-joint, but the powerful musculature that is usually exhibited by these patients is a sufficient commentary on this point. Thirdly, the joint does not need support below, for it is not here that the dislocation takes place.

It must be admitted that these operations have sometimes been successful in preventing the recurrence of the

dislocation, but this result has usually been due to limitation of the normal movements of the joint by the operative procedure. One surgeon, indeed, has suggested that the operation might be simplified if it were done deliberately with this purpose, and he proposed to graft a band of fascia between the chest wall and the humerus in order to limit the movement of abduction to 60 degrees.

Causes of Recurrent Dislocation.

It has been too readily assumed that recurrent dislocation is an unfortunate sequel of ordinary traumatic dislocation, and I have known medical men reproach themselves quite unnecessarily for their treatment of the original injury. Recurrent dislocation has nothing whatever to do with ordinary traumatic dislocation of the shoulder-joint. It is an entirely different injury, and it is produced in an entirely different manner.

Ordinary dislocation of the shoulder is the commonest of all dislocations of the joints. It is caused by a fall on the abducted arm. In extreme abduction the neck of the humerus impacts against the acromion process, and by leverage the head is forced through the lowest and weakest part of the capsule between the subscapularis and triceps muscles. When such a dislocation is reduced, the rent in the capsule heals rapidly and soundly, and the dislocation rarely, if ever, recurs.

But the dislocation which afterwards becomes recurrent is caused, not by a fall on the abducted arm, but by a fall either directly on the back of the shoulder or on the elbow which is directed backwards and only slightly, if at all, outwards. The head of the humerus is forced out of the joint, not by leverage, but by a direct drive from behind forwards. In its passage forwards the head shears off the fibrous capsule of the joint from its attachment to the fibro-cartilaginous glenoid ligament. The detachment occurs over practically the whole of the anterior half of the glenoid rim. The reason why the dislocation recurs after reduction is that, whereas a rent in the fibrous capsule heals rapidly and soundly, there is no tendency whatever for the detached capsule to unite spontaneously with the fibro-cartilage. The defect in the joint is therefore permanent, and the head of the humerus is free to move forwards over the anterior rim of the glenoid cavity on the slightest provocation.

In quite a number of the cases the original injury is stated to have occurred at football, and it should be noted that in this game, at least, a player is seldom sent sprawling with his arms out, but, when he falls or is thrown to the ground, it is usually in some moment of strenuous action, when his muscles are tense, and his arms are more or less close to his sides. Usually he is too preoccupied to make much attempt to save himself, and in falling backwards he is likely to strike either the back of the shoulder or the point of the elbow, which is directed backwards. The epileptic, too, falls with his muscles tensely contracted, and he, of course, makes no effort to save himself. Frequently the arms are drawn backwards and little, if at all, abducted. So that, if he falls backwards, he is likely to strike either the back of the shoulder or the point of the elbow. Thus both athletes and epileptics are liable to sustain dislocation of the shoulder-joint by direct violence rather than by indirect force or leverage.

Some cases have been described in which fractures, either of the anterior margin of the glenoid cavity or of the greater tuberosity of the humerus, have been associated with recurrent dislocation. Such fractures have not been seen in any of my cases, but it is easy to see how they might be caused by the direct force which produces the initial dislocation. They must, however, be regarded as incidental, and not essential, to the recurrent dislocation. The essential feature is the detachment of the capsule from the fibro-cartilaginous glenoid ligament.

The Operation Recommended.

I have now exposed this typical lesion at operation in four consecutive cases. A satisfactory exposure cannot be obtained by the anterior incision ordinarily employed for arthrotomy and plication operations on the capsule, and

no doubt this is the reason why the condition has not been generally recognized. The operation which I have done has been planned to expose completely the anterior margin of the glenoid cavity.

A small pillow or sandbag is placed beneath the scapula, so as to keep it forwards. The arm lies on the table by the patient's side, and it is rotated inwards, so as to relax the pectoralis major muscle. The incision extends from the upper border of the clavicle above the coracoid process downwards and outwards for about five inches. The deltoid and pectoralis major muscles are separated, and the coracoid process and the three muscles attached to it (pectoralis minor, biceps, and coraco-brachialis) are defined. Perthes, who used a similar incision in operating upon fractures of the glenoid, divided the pectoralis major, but I have not found this step necessary. The coracoid process is next divided with an osteotome or bone forceps, and drawn downwards with the three muscles attached to it. Lastly, the tendon of the subscapularis is divided close to its insertion, and this muscle is retracted inwards.

The damaged area is now fully exposed. Internally one sees the neck of the scapula with the fibro-cartilaginous glenoid ligament lying upon it. This ligament has usually been torn up a little way from the glenoid margin, so that it appears as a free edge lying upon bare bone. External to this is the glenoid cavity with the head of the humerus in contact with it. Running from above downwards over the joint cleft is usually to be seen a band of fibrous tissue which represents the free edge of the capsule which has been torn from the glenoid ligament. It appears as a band because immediately external to it is the not inconsiderable and perhaps enlarged opening of the subscapularis bursa. Lastly, external to this is the fibrous capsule of the joint covering the greater part of the head of the humerus. The mechanism of the recurrent dislocation can now be easily demonstrated, for on taking hold of the arm and pushing its upper end forwards, the head of the humerus can be made to pass freely over the anterior edge of the glenoid cavity.

To anyone who has seen this typical lesion exposed at operation it must be obvious that the only rational treatment is to reattach the fibrous capsule to the glenoid ligament whence it has been torn. It is clear that a plication or similar operation performed on some distant part of the capsule can only have the effect of drawing the detached edge outwards and further away from the glenoid margin. No doubt, if the capsule is sufficiently diminished in size, it may prevent displacement of the head of the humerus notwithstanding the defect at the glenoid margin, but this is at the expense of free movement in the joint. It should be remembered that the capsule of the shoulder-joint is normally a lax, and not a tense, structure.

It is exceedingly difficult to believe that operations of the Clairmont-Ehrlich type can have any influence in preventing recurrent dislocation, except by limiting the normal movements of the joint. Even if one could think that the transplanted strip of muscle acted in the way it is supposed to do, it is not clear how this would prevent the dislocation, for the displacement does not take place downwards, but forwards.

In my cases the joint defect has been repaired by interrupted sutures of silkworm gut passed between the free edge of the capsule and the glenoid ligament. It is well to freshen the bone on the neck of the scapula, so that the glenoid ligament may adhere to it. Having repaired the joint defect, the divided subscapularis tendon is reunited, the detached portion of the coracoid process is sutured in place, and the wound is closed. After the operation the arm is kept at rest for four weeks, and then active and passive movements are begun and persisted in until the movements of the joint are normal.

Two of my cases were epileptic, and both the others sustained the original injury at football. One case—a powerful epileptic—had some limitation of abduction two months after operation, and he has not been seen since. This relatively poor result was due to his neglect of after-treatment. The others recovered full movement at the shoulder-joint, and in none of them has the dislocation recurred.

TREATMENT OF MIGRAINE BY CALCIUM LACTATE.*

BY

A. DOUGLAS BIGLAND, M.A. CAMB., M.D. LIVERPOOL,
M.R.C.P. LOND.,

HONORARY PHYSICIAN, DAVID LEWIS NORTHERN HOSPITAL, LIVERPOOL;
LECTURER IN CLINICAL MEDICINE, LIVERPOOL UNIVERSITY.

MIGRAINE, passing under many names and treated by a host of different remedies, is a condition of surpassing interest on scientific grounds alone. To practising physicians the condition is of similar importance for three reasons: (1) Migraine is a very common ill; although referred to by those who have never suffered from it as a minor malady, yet in reality it accounts for an enormous amount of wretchedness and impaired efficiency. (2) Migraine appears to attack with great frequency the professional classes in general and medical men and women in particular. In the literature splendid accounts of the condition have been supplied by doctors who were themselves sufferers, and in my series of twenty cases eight were members of the medical profession or medical students. (3) The old treatment of migraine, as evidenced by the long list of so-called remedies, is not satisfactory. More recently fresh conceptions and new drugs have been tried with some success, and the results obtained with one of these remedies is the justification for this communication.

It appears probable that the fundamental cause of migraine is an anaphylactic shock. How this shock is manifested, and indeed in what particular part of the brain the manifestation occurs, is not known. The accurate, subjective localization of the headache and the external phenomena associated with the superficial temporal artery suggest the area of the brain underlying the squamous portion of the temporal bone as the probable site of the lesion; the occasional aphasia and even rarer hemiplegia met with in migraine point to the motor cortex. On the other hand, the visual phenomena, which are so common, can only be explained by referring the disturbance to the occipital cortex, and the vomiting is possibly associated with the medulla. It has been suggested that the pituitary gland may undergo temporary enlargement and thus cause symptoms, but the type of hemianopsia met with in migraine is opposed to this view. It would appear, therefore, that no single cerebral area can be held responsible for the various phenomena of the disease.

The undoubted relation between eye-strain and migraine is difficult to explain upon an anaphylactic basis unless it be that a brain already sensitized will react unduly to any stimuli and among them impulses connected with the ocular apparatus. On the other hand, the equally well known connexion between migraine and other nervous disorders, such as epilepsy, can best be explained on an anaphylactic basis; these conditions also are considered by many to have a similar origin. On this assumption it is not exactly correct to say that migraine is hereditary, but rather that a liability of the nervous system to become sensitized and react to certain stimuli in themselves innocuous may be transmitted. Migraine in the parents may be represented by asthma or angio-neurotic oedema in the children.

It is remarkable that organic disease, especially perhaps that of the cerebral vessels, is not more often associated with migraine, for temporary aphasias and hemiplegia are comparatively common in the former (Case v).

Of the symptoms occurring in the attack those connected with vision come first in point of time and in importance, since they may be the sole manifestation. A sudden general dimming of vision is a very common prelude, followed by scotomata, coloured dazzling spectra, and hemianopsia. These constitute the aura of an attack and, as might be expected, they are often associated with vertigo. In a short time follows the headache, which is usually localized very accurately to one temple and practically always to one side of the head. The least movement or any temporary increase in intracranial tension, as by coughing, is accompanied by

* Based upon a note read before the Liverpool Medical Institution on January 4th, 1923.