COSTO-CLAVICULAR COMPRESSION AND THE UPPER THORACIC SYNDROME*

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Contrary to what occurs in the lower limb and consequent upon our upright posture, the neuro-vascular bundle as it leaves the upper thoracic opening to enter the dependant upper limb, is sharply angulated. If because of this angulation, friction or stretching of the lower part of the plexus takes place over the first rib or a cervical rib, a band, or the tendinous fibres of the scalenus medius muscle, symptoms are produced which constitute the *syndrome of the upper thoracic opening*. As the subclavian artery leaves the upper thoracic opening over either the first rib or a cervical rib, it has to pass between this rib behind and the clavicle lying in front of it and in some subjects and in some positions of the arm it may be compressed between the two bones (*Costo-clavicular compression*).

Anatomy

The Upper Thoracic Opening

The superior aperture of the thorax is a narrow opening bounded by the first dorsal vertebra, the first pair of costal arches and the manubrium sterni, and from it issue the great vessels and nerves to the upper limb. The opening may be asymmetrical if a cervical rib is present which may vary in length from a few millimetres to a massive and complete costal arch. Such a rib may be only in part bone and represented in the remaining part of its length by a fibrous band.

Syndrome of the Upper Thoracic Opening

The chief symptoms are pain, paraesthesia and wasting and weakness of the small muscles of the hand, manifest by flattening of the thenar eminence (Fig. 1). There is a tendency for the patient to drop things. Two varieties of the syndrome may be recognized, (1) that in which the symptoms are predominantly neural in type, and (2) that in which they are essentially vascular.

Costo-Clavicular Compression

In 1934 Lewis and Pickering suggested that the

vascular phenomena associated with a cervical rib are due to direct injury to the subclavian artery by the clavicle, and this was confirmed at operation by Wilfred Trotter. Eden in 1939 reviewed the question and recorded further cases in which compression of the subclavian artery between the rib and the clavicle was clearly demonstrated. Murray Falconer and Weddell (1943) and others have since given details of similar cases.

Cases in which the subclavian artery is intermittently occluded by being squeezed between the clavicle and the cervical or first rib (costo-clavicular compression) form a relatively small proportion of those comprising the upper thoracic syndrome.

In a personal series of 46 operations performed for the upper thoracic syndrome on 41 patients aged between 17 and 55, of whom 25 were women and 16 men, the symptoms in 39 were ascribed to the presence of a rib or band or the tendinous fibres of the scalenus medius over which the lowest trunk of the brachial plexus was stretched and chafed by certain movements of the limb; only seven were considered to be due to pressure of the clavicle.

The type of cervical rib which is likely to play a part in costo-clavicular compression is the long well-developed and laterally placed one with a bulbous end to it, not the short stumpy rib with a pointed end to which a band is often attached. The costo-clavicular space is encroached upon by the massive rib. There is an asymmetrical upper thoracic opening and if exploration is carried out under a local anaesthetic, the narrowing of the costo-clavicular interval and the squeezing of the artery within it when movements of the shoulder-girdle take place, may be verified. It is important to recognize the mechanical compression in these cases because scalenotomy alone will not relieve these patients and the artery will only be effectively freed by a removal of one of the bony struts between which it is compressed. Scalenotomy alone may, in fact, make the patient with costo-clavicular compression worse, as in a case recorded by Le Vay (1945). The movement which causes the compression is usually backward and downward

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FIG. 1.—The characteristic wasting of the thenar musculature (right hand) is shown. There is little change in the interossei. The patient was an estate agent manager, aged 32, with a typical upper thoracic syndrome.

retraction of the shoulder, but the relationship of the abnormal rib will determine this and we have observed a case in which the bulbous end of a rather massive cervical rib bore such a relationship to the clavicle that upward and slightly backward movement of the shoulder produced the compression of the artery which was freed after removal of the anterior part of the rib (Rogers and Aldis, 1947).

If there is intermittent compression of the artery between the bony struts of the rib and clavicle, the vessel in the course of time is injured at the site and a local lesion such as periarteritis, an aneurysmal dilatation or thrombosis is likely to take place. Detached pieces of blood clot from the site of such injury may result in embolic obstruction of peripheral vessels and gangrene of fingers as has been noted by Eden (1939) and others.

Signs and Symptoms

Pain and colour changes in the digits, paraesthesia in the forearm and necrosis of the tips of the fingers or thumb or even of whole digits may occur. The necrosis is the result of small emboli dislodged from a damaged area of the artery where it is subjected to intermittent compression and where in some cases an aneurysm is apparent.

Alterations of the Pulse with Movements of the Shoulder Girdle

It has long been known that in some normal persons who are free from symptoms, changes in the radial pulse varying from a diminution in volume to complete obliteration, may be induced by certain movements of the shoulder girdle, and this phenomenon has been noted in more than 60 per cent. of a number of young adults (Weddell and Falconer, 1943). In retraction of the shoulder the clavicle moves backwards and also rotates in a counter clockwise direction, thus further approximating itself to the rib behind it (Stammers, 1950). In some subjects which I have examined, throwing back one shoulder will reduce or obliterate the pulse at the opposite wrist. Elevation of the arm may also affect the pulse.

Is this phenomenon of pulse change due to costo-clavicular compression or has it some other explanation? J. B. Murphy (1906), who noted that 'the change in the circulation is peculiar and striking and involves the arterial, never the venous current,' suggested it was due to compression by the scalenes and undoubtedly some cases appear to be so caused. The medial tendinous fibres of the scalenus medius are sometimes well developed and extend as a sharp crescentic band along the upper surface of the first rib. Between the scalenus anticus in front and these fibres of the

scalenus medius or a rib or band behind, the lowest trunk of the brachial plexus and the subclavian artery may be compressed as in a vice and the expression 'scalene vice' has been used in this connection by some writers.

Sometimes the subclavian artery is actually attached to the scalenus anticus, certain fibres of which are inserted into the sheath of the vessel and have to be cut away from it when clearing the artery. It is conceivable that the pull of these fibres may so disturb the artery as to alter the volume of the pulse at the wrist.

Wingate Todd (1913) and Telford and Stopford (1930) suggested that the vascular phenomenon could be accounted for by sympathetic nervous fibres being stretched over the abnormal rib or corresponding structure and later Telford and Mottershead (1947) considered that when the arm is pulled downwards, the two heads of the median nerve may between them grip the brachial artery so tightly that the radial pulse is affected.

Which of these mechanisms is responsible for the alteration in the pulse to be observed in so many normal subjects when the shoulder is depressed or thrown back or the arm elevated or pulled downwards and which of them may produce the vascular symptoms of the upper thoracic syndrome? The answer is almost certainly that no one mechanism accounts for all cases. That costo-clavicular compression is not always responsible is clearly shown in one case of my series, a patient who had a large right cervical rib against which the subclavian artery was compressed by the clavicle when the shoulder was elevated. The lower half of the rib was removed with complete relief of symptoms. Six years later the patient is working hard and is symptom free but retraction of the shoulder still stops the radial pulse. In those cases in which the artery is so damaged that an aneurysm has developed or the vessel has thrombosed, true costo-clavicular compression can be demonstrated. If vascular changes associated with symptoms are produced by movements of the shoulder girdle, exploration is called for so that the artery may be freed from its source of compression. Adequate exposure is obtained through a linear incision made across the root of the neck from a point over the middle of the sternum to the mastoid muscle, one finger's breadth above the inner end of the clavicle. If there is no gripping of the artery between clavicle and rib when the shoulder is moved, it is still not enough to divide the scalenus anticus alone. A source of deep pressure, i.e. the other limb of the vice, whether it be rib, band or fibres of the scalenus medius, must be sought and divided so as to leave the artery and the plexus free.

Conclusions

In many normal persons changes in the pulse, from diminution to complete obliteration, can be brought about by changes in the position of the shoulder girdle produced by movements of the arm, and in some, throwing back the shoulder on one side will obliterate the pulse on the opposite side. Probably no one mechanism is responsible in all these cases. Man's upright position, the shape of the upper thoracic opening as determined by the costal arrangement and the form and development of the shoulder girdle and its associated muscles are the chief factors involved.

The clinical features of the syndrome of the upper thoracic opening are either neural or vascular in type. The clavicle only rarely plays a part in their production and costo-clavicular compression may be associated with local damage to the subclavian artery.

Vascular changes with pain in cases of the upper thoracic syndrome call for an exploratory operation to free the artery and the lowest trunk of the brachial plexus from compression. Anterior scalenotomy alone will not suffice in most of these cases; the deep relationships of the artery and the brachial plexus must be examined and the other limb of the vice, if present in the form of a rib, a band or tendinous fibres of the scalenus medius must be divided as well.

Summary

An explanation is given of why the syndrome of the upper thoracic opening has no counterpart in the lower limb.

The effect of arm and shoulder girdle movements on the pulse is discussed. Costo-clavicular compression is shown to be comparatively rare and when it occurs may lead to vascular changes consequent upon damage to the subclavian artery. The principles of operation for the relief of these syndromes are stated.

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BASIC MECHANISMS OF ALLERGIC REACTIONS

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Introduction

The general integration into everyday medical thought of advances in fundamental knowledge in medical science is often delayed until it has become imperative in order to enhance precision of diagnosis and treatment. The role of allergic reactions has clearly reached this stage and their basic mechanisms will be discussed within the limitations of our present knowledge in which gaps bridged by new developments remain to be filled in later. The term 'allergy' has lost some of its precision and a survey of its origin will help to make its meaning clearer. Arising out of the fascinating and important study of immunity towards the end of the last century came v. Behring's

production of diphtheria anti-toxic serum derived from horses. Its extensive use in humans and guinea pigs soon resulted in numerous, unexpected and severe reactions which were not related to the disease and which were termed serum sickness (v. Pirquet and Schick, 1905). At this time Portier and Richet (1902), who were studying the production of immunity to actinia toxin in dogs, found that if they were suitably spaced the injection of doses of toxin far below the toxic level produced severe and often fatal reactions. This reaction seemed to be opposed to 'prophylaxis' or 'immunity,' and they termed it 'anaphylaxis' or 'removal of protection.' This dual capacity of the same agent required explanation and v. Pirquet