SHOULDER-ARM PAIN*

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The question of shoulder-arm pain is, perhaps, the most confusing of subjects, and one of those still largely unsolved in medicine and surgery. In an attempt to illustrate the problem, this paper considers the possibility of positions and movements causing compression of the neurovascular tree because of anatomical factors. Included are skeleton studies, a study of personal symptoms, and the application of conclusions to an assessment of clinical cases. A review of the historical development of the views on this problem is given.

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SKELETON STUDIES

The neurovascular tree was reconstructed and the scalenus anterior and pectoralis minor placed in relation to the tree (Fig. 1). It is, of course, impossible to claim that this truly represents what occurs in life. Movements caused dramatic alterations in the relations of the reconstructed

Fig. 1. Skeleton with reconstructed neurovascular tree in right arm.

tree to the anatomical structures, and it is at least possible that this may happen to some extent in the living person.

Scalenus Anterior and Medius

Information from these studies showed that, on elevating the shoulder towards the head, the neurovascular bundle is pulled against the edge of the scalenus anterior (Fig. 2). How much movement is permitted in life is difficult to say, but when the movement is allowed in the skeleton this is most noticeable. There is certainly a jamming upwards between the scalenus anterior and medius.

Pectoralis Minor

Further, on forward elevation of the arm, while keeping the shoulder absolutely level, the neurovascular bundle did definitely kink at the level of the pectoralis minor muscle. On abduction of the arm without elevation of the shoulder, there is a tautening of the neurovascular bundle with some kinking at the pectoralis minor.

A combination of elevation of the shoulder and abduction of the arm affords a double mechanism of tautness and kinking, with pressure on the scalenus anterior or in the wedge between the scalenus anterior and scalenus medius (Fig. 3). The pectoralis minor affords a second point of pressure from angulation and tautness beneath it.

Traction on the Neurovascular Tree

Studies were also made, on the skeleton, of tensions and tractions on the neurovascular tree. It is well known that traction injuries occur, e.g. as when the shoulders and the

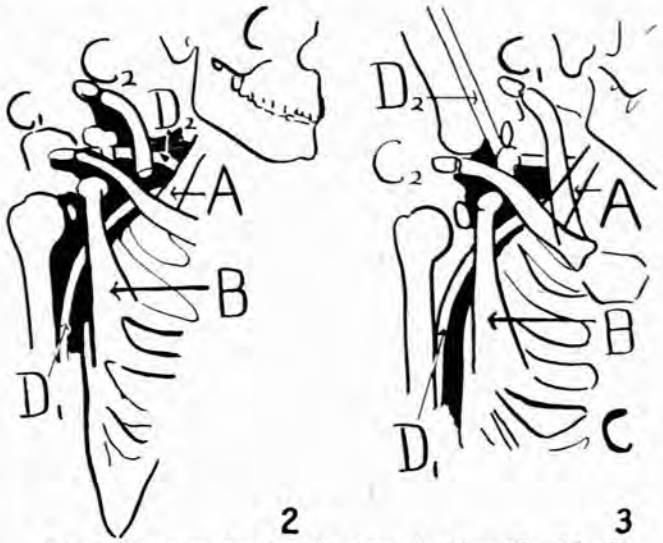

Fig. 2. Diagram showing shoulder elevation (C_1 - C_2) with elevation of neurovascular tree (D_1 - D_2). (A = scalenus anterior, B = pectoralis minor.)

Fig. 3. Diagram showing elevation and abduction of shoulder (C_2 - C_1) with elevation and kinking of taut neurovascular tree (D_1 - D_2). (A = scalenus anterior, B = pectoralis minor.)

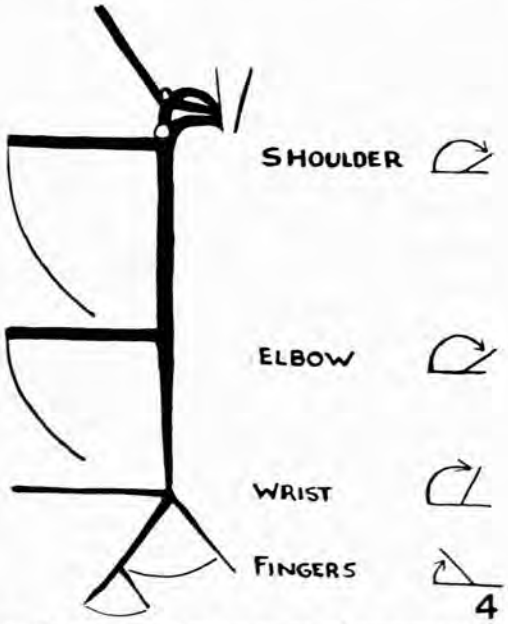

Fig. 4. Diagram showing tautening of neurovascular tree. Movements from flexion to extension at elbow and wrist. Abduction of shoulder.

head are separated in a fall on the shoulder, resulting in a complete or partial tear of the brachial plexus, often at Erb's point.

A young doctor jumped over a tennis net and fell on his extended arm with the hand and fingers extended as well. He immediately suffered pain in the shoulder, and this was followed by pain in the areas of the anterior divisions of the nerves C 5 and 6. This lasted for several weeks, and was obviously from traction injury of the neurovascular tree.

In the case of the skeleton, fishing line was used. The plexus was fashioned to run under a reconstructed scalenus anterior muscle into the arm, under a reconstructed pectoralis minor muscle, and then under a reconstructed carpal tunnel to the tips of the fingers. Loose loops were used to maintain the reconstructed vascular tree in the fingers (to copy the action of the flexor tunnels) and were also used in the forearm and in the region of the humerus. A tensionometer was used, which was the facepiece and lever of an oscillometer, in which a cotton attachment to the needle allowed the dial to be adjusted to a standard position. Extension of the elbow from a semi-flexed position (a right-angle flexed position), to full extension, and abduction from the position of the arm by the side to 90° abduction, was studied. The results indicated that quite considerable traction and tension occur with these movements (Fig. 4).

Use of the Tensionometer

Extension of wrist and fingers. On fixing the indicator of the dial to 0 and adjusting the manometer to a standard position which was present at full flexion of the wrist, it was found that extending the wrist and the fingers fully caused the tensionometer to record in the region of 130 mm.Hg.

Extension of the elbow. The investigation was applied also to the elbow and shoulder and, from right-angle flexion to full extension of the elbow the needle was displaced to the full scale of the manometer and would probably have gone further if possible.

Abduction of the shoulder. This same experience was obtained on abducting the shoulder. It was thus possible to appreciate roughly the amount of traction that is exerted on a fixed point in the distal forearm by extension of the elbow and abduction of the shoulder.

Comment. Although these experiences are crude and there is difficulty in comparing these findings with what happens during life, there is no doubt from this simple experiment that a considerable traction effect on the neurovascular tree does occur during these movements. Kinking, and traction and pressure are possible from the taut kinking at the scalenus and pectoralis minor muscles in extension and abduction.

The wrist. Sunderland,[1] in 1945, carried out an interesting experiment. He injected the brachial artery in a fresh cadaver and was able to show the blood supply in the median nerve. On flexion of the wrist, an area corresponding to the carpal tunnel showed no blood supply in the nerve. Obviously, pressure sufficient to occlude nerve vessels occurs on wrist movements.

Discussion

It is apparent that tautness, kinking and pressure are common factors in the aetiology of shoulder-arm pain, apart from pathological causes of neurovascular abnormality. Normal movements will produce fleeting attacks of pain, whereas occupational or forced maintenance of abnormal positions and the prolonged effects of position when asleep may be the determining factors.

Abnormal anatomy, such as fallen shoulder-tip in costoclavicular compression, cervical ribs, a highly placed subclavian artery, etc., will play a part in postural decompensation.

It is known that the excision of the clavicle has relieved clinical costoclavicular symptoms when scalenectomy has failed. A case of this nature was presented recently by Prof. D. J. du Plessis in Johannesburg. In this patient relief had been afforded by leaning forwards. There was also night numbness and pain. On X-ray examination, osteophyte encroachment had been demonstrated, as well as notching on the under medial portion of the clavicle. Removal of the major pressure by excision of the clavicle gave dramatic relief even when psychiatric advice had been against the operation.

PERSONAL SYMPTOMS

Schultze[2] originally described the syndrome of numbness and paraesthesia of the hands, associated with certain positional alterations, as acroparaesthesia. Being a mild sufferer from these symptoms myself, I made observations on them, and attempted to correlate the signs and symptoms with the supposed pathology, and investigated symptoms developing in different positions. A proved encroachment of cervical intervertebral foramina was present, and its relationship to the development of symptoms will be described at a later stage.

Symptoms Attributable to the Scalenus Anterior

The most typical symptoms which I attributed to scalene pressure were those occurring on sleeping on the affected side. Numbness in the arm, hand and fingers disturbed sleep. The symptoms were relieved by turning over to the other side. It was thought that the brachial plexus was elevated against the edge of the anterior scalene with pressure or traction effects. The following is a typical case history:

A woman, aged 58, had symptoms for 33 years following a fall from her bicycle, in which she injured her shoulder. This was diagnosed as 'brachial neuritis', presenting with numb 'dead' hands; waking in the night with 'dead', numb and painful hands; and inability to write or knit. Symptoms could be reproduced by lying on her side with shoulder-head approximation.

X-rays of the neck showed no cervical spondylosis or cervical ribs. She had seen numerous doctors for this condition. She had been treated with vitamin-B_{12} injections, X-ray therapy, injection of fibrositic nodules, neck traction, and ACTH, and had had treatment for tennis elbow.

Operative division of the scalenus anterior revealed a high subclavicular artery, and, after freeing this and the plexus, symptoms were completely relieved (follow-up 7 years).

Other similar results have been obtained from operative division of the scalenus anterior muscle and freeing the plexus in acroparaesthesia with night numbness. Degenerative changes have been noted in the brachial artery and the scalenus anterior muscle; these were mentioned by Oschner and de Bakey.[3] Other patients with similar symptoms in the presence of cervical ribs have obtained relief following resection of the ribs and scalenectomy. Careful assessment of symptoms in shoulder lesions has revealed further examples of plexus-scalene pressure.

Two patients suffering from fairly long-standing painful supraspinatus tendinitis later developed typical night numbness and tingling in the hand and arm, explainable only on a plexus-pressure basis. This was assumed to be the result of the characteristic protective position that is taken up in these cases. The shoulder is elevated towards the head, with or without the use of a sling. Relief of

the cause of pain and restoration of shoulder function followed treatment of the supraspinatus tendinitis.

Symptoms Attributable to Pectoralis-minor Pressure

On driving a motor-car with my arms extended straight in front and gripping the top of the driving wheel, symptoms of 'dead', numb fingers occurred.

Wright,[4] in describing the hyperabduction syndrome, an acroparaesthesia of occupational nature in riveters and ballet dancers, etc., regarded the pectoralis minor as an important pressure point associated with costoclavicular pressure.

It was thought that the symptoms following elevation of the arm holding the upper part of the wheel were almost certainly from pectoralis-minor pressure. However, the study was carried further. Symptoms in the left hand and arm invariably began when the arm was extended in front on the top of the steering wheel, with the clenched fingers (flexor digitorum sublimus contracted) and the thumb pressing firmly against the wheel (thenar-muscle contraction and carpal-tunnel tension). The numbness seemed, under these circumstances, to be in the distribution of the median nerve. Immediately the hand was rested on the wheel without gripping, the symptoms disappeared. I could only assume that what I had thought to be pectoralis-minor pressure was in fact carpal-tunnel pressure. The unreliability of explanations was thus obvious and portended great difficulties in the future study.

Another disconcerting problem arose when the same position on the driving wheel was taken up, i.e. forward elevation of arms and semi-extended elbows, fingers flexed and thumb pressing on the wheel, but with the forearm supinated instead of pronated. Then no symptoms could be reproduced. Immediately the pronated position was restored, symptoms recommenced, and the moment the forearms were supinated the symptoms disappeared again. This could only mean that the pronator teres had to be brought into action in these circumstances for symptoms to appear. As the median nerve passes between the 2 heads of the pronator teres, these muscles partake in the production of symptoms.

Symptoms Caused by Local Pressure

On rare occasions the wearing of a tight rubber glove caused numbness in the thumb. Also, leaning over a chair caused pressure on the medial side of the upper arm with resultant numbness in the hand. A strap over the shoulder occasionally did the same thing.

Symptoms Attributable to Head and Neck Movements

These were rather vague and localized to the posterior part of the neck and shoulders, prominent when high pillows were used, or during a visit to the planetarium.

Symptoms Caused by Costoclavicular Pressure

This pressure was thought to cause the occasional numbness that occurred with the arms by the side when carrying weights, etc. This did not constitute a major problem.

Symptoms Attributable to Pressure at Wrist Level

Many tests were carried out to see whether wrist movements in any particular position caused the symptoms in the fingers. At no time was it possible to say definitely that wrist flexion or extension alone caused symptoms in the hand. Tests were carried out in patients as well, who were asked to sleep holding a newspaper to keep the hands from flexing or extending. Only one patient attributed symptoms to extension of the wrist from holding a telephone for long periods or pushing a pram up a hill, as well as to waking up at night with the wrist fully extended under the chin. This patient was a young housewife who also complained of night paraesthesia. According to Walshe this was a typical costoclavicular syndrome, and according to Nissen it was a typical carpal-tunnel syndrome. Activity of the hands was associated with symptoms in a few patients.

HISTORICAL SURVEY

Before 1947

This was the 'golden era' of scalenectomy. Scalenectomy was all that was necessary, even in cervical rib abnormalities.[5] The scalenus-anticus syndrome was recognized[5,6] and, in fact, histological signs of degeneration and inflammation were actually described.[3]

The Debunking Era

About 1947 there was a period which could be called the 'debunking era' of scalenectomy. Telford and Mottershead,[7] in 1947, showed that the Adson arterial-obstruction tests of head and neck movements, and deep breathing obstructing the pulse, occurred in normal subjects. They stressed that there was no agreement as to the cause of the compression of the brachial plexus. The scalenus medius was implicated as well, and direct traction on the nerve roots was a factor in certain circumstances. Cervical ribs and their remnants, according to these writers, still accounted for the largest proportion of cases.

At this time, too, Brain, Wright and Wilkinson[8] described the division of the carpal tunnel in 6 well-selected cases of median-nerve compression. The focus of interest was passing from the thoracic outlet to the wrist. There were, however, other workers interested in the thoracic outlet.

The Costoclavicular Syndrome

Falconer and Weddell,[9] in 1943, described the costoclavicular syndrome of compression between the clavicle and the ribs, referring mainly to arterial compression. Walshe *et al.*[10] strongly supported the view that acroparaesthesia in middle-aged women was caused by the pressure of the plexus against the first rib following muscle decompensation and loss of tone (Fig. 5). During the War middle-aged women found that they had more unaccustomed work to do. This syndrome was also described in pregnancy and in the nursing mother. It was also caused by unaccustomed muscular strain and increased activities in patients lacking in general muscular tone. The shoulder

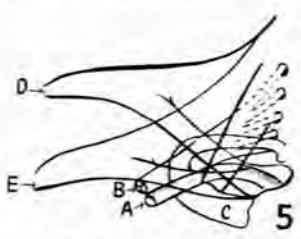

Fig 5. Diagram showing pressure of the brachial plexus and subclavian artery against the first rib with a fallen shoulder tip, the result of muscle decompensation and loss of tone. (A = subclavian artery, B = brachial plexus, C = first rib, D = normal shoulder tip, E = fallen shoulder tip.)

tip was consistently found to be lower than the sterno-clavicular joint.

In my series, costoclavicular compression was regarded as important in several cases. In 2 patients, fractured clavicles with bosses of callus causing pressure on the plexus brought on these symptoms on walking, carrying bags, and depressing the shoulders and retracting them backwards. These symptoms did not cause the typical night paraesthesia.

Two males got the symptoms lying in bed after abdominal operations. In each case the neck was flexed by too many pillows, the arms hung down at the sides, and the patients could not turn on their sides. The symptoms were relieved when the patients became ambulant and were able to sleep on their sides.

The Hyperabduction Syndrome

In 1945, Wright, a colonel in the American Army, investigated what he called the 'hyperabduction syndrome',[4] a syndrome of occupational nature in which riveters, ballet dancers, and other workers with arms elevated above the head had typical symptoms of acroparaesthesia. He studied normal subjects and came to the conclusion that the compression of the neurovascular tree arose from costoclavicular compression together with pectoralis-minor compression. Spontaneous axillary thrombosis has been linked with this positional state and with pectoralis-minor pressure, but radiologically it has also been shown to occur following pressure from the scalenus anterior muscle.

Cervical Spondylosis

During the period 1946 - 1956 the syndrome of cervical spondylosis became recognized. At first cervical discs, like lumbar discs, were blamed. Later, encroachment of the intervertebral foramina and osteophytes were considered the major cause. Patients with acroparaesthesia without wasting or neurological signs often respond to neck traction.

This has not been the experience of a neurological colleague, and I can only assume that his patients have obvious neurological signs and wasting, whereas my patients have presented only with night paraesthesia.

It was found that true nuclear herniations in the cervical region are rare[5] and the diagnosis of motor-neurone disease, syringomyelia, spinal disseminated sclerosis, and progressive lateral sclerosis are considered by some to be entities apart from this condition of cervical spondylosis, whereas others consider them to be caused by different selective sites of compression. Osteophyte formation is recognized as a cause of local pressure and the nerve roots may be affected by direct pressure or ischaemia. Canal capacity and root-sleeve fibrosis are important factors as well.

That acroparaesthesia occurs in a large number of patients with cervical spondylosis with encroachment is undoubted. I myself have the typical night numbness and tingling. Why should symptoms from this condition be apparently identical with those in patients suffering from cervical-rib or scalene syndrome, and even carpal-tunnel syndrome?

It is thought that segmental spasm of the scalene could be the cause of 'secondary' scalene symptoms, or it could be that the pathology in the neck makes the nerves more susceptible to pressure effects which would not be noticeable in a normal subject. Can it be that the state of the major nerve roots is one factor, and that pressure points and traction effects pertaining to several anatomical sites can be other separate factors?

Carpal-tunnel Syndrome

The carpal-tunnel syndrome was well known even in 1909, when Hunt[11] described division of the carpal tunnel and found a neuroma under it. Marie and Foix[12] described similar cases in 1912 and proved one by necropsy dissection.

Brouwer,[13] Harris,[14] Lhermitte and de Massany,[15] Dorndorf,[16] Moersch,[17] Wartenburg,[18] Woltman,[19] and Zachary[20] all contributed to knowledge on this subject, and Brain, Wright and Wilkinson,[8] with their 6 cases of operative division of the carpal tunnel, popularized this diagnosis.

We are all occasionally confronted with obvious cases of median-nerve involvement in the hand, with thickening and neuroma formation under the carpal ligament, associated with sensory abnormality and wasting of muscles supplied by the median nerve.

The extension of the operation dividing the carpal tunnel to the treatment of a large number of patients with acroparaesthesia is, however, a new concept. Nissen[21] wrote: 'Division of the transverse ligament of the carpal tunnel for carpal-tunnel compression of the medial nerve, thanks to the warm advocacy of the Middlesex School, has become by far the most frequent of all operations for the relief of pain in the upper arm'.

Nissen[22] also wrote: "Since 1947, at the Nuffield Orthopaedic Centre at Oxford, this operation has become a common one and the volume of patients requiring decompression justifies a procedure being performed in the out-patients' department, under local anaesthetic".

I have known of physicians who state that in their experience at least 80% of patients with these symptoms are relieved by division of the carpal tunnel, and they seek out trained surgeons to perform the operation. The indication is no longer the obvious median-nerve lesion with wasting and sensory changes in the distribution of median nerve, associated with a neuroma under the carpal ligament, but the operation is urged in patients with the syndrome of acroparaesthesia, even when the distribution of pain and numbness extends far above the arm, even into the shoulder.

As one who has seen the different eras and developments in this syndrome, I am not so easily converted to this line of thought. I can quite willingly accept that division of the carpal tunnel does relieve a large number of patients. It is difficult, however, to explain certain features. Why is night pain typical of this disorder when it is also typical of other lesions? Why also, when a sphygmomanometer cuff is blown up to 200 mm.Hg, causing tingling within 1 minute in the hands and the fingers in the median-nerve distribution, is this considered diagnostic of the carpal-tunnel condition, when tingling of this nature occurs in many lesions?

It is difficult to accept the statement of Nissen,[21] who said: 'Clear thinking on the subject is essential since there are two definite types — (*i*) the spontaneous primary compression which occurs in females, usually middle-aged

females; (*ii*) secondary compression from early rheumatoid tenosynovitis, long-standing carpal-bone lesions, acromegaly, and fluid retention such as occurs in pregnancy and myxoedema'.

It would seem that even clearer thinking is required to explain the empirical relief obtained by carpal-tunnel division and its use on a wide scale for lesions not necessarily in the distribution of the median nerve.

CASE HISTORIES AND DISCUSSIONS

The following specially selected cases are described to demonstrate problems arising in the shoulder-arm syndrome:

Case 1 — Mrs. H., Aged 58 Years

This patient had a scalenectomy for acroparaesthesia and night numbness caused by sleeping on her sides. Relief was obtained. This demonstrates that the operation helps in this condition, and also shows how a high subclavian artery, bunching the plexus under the scalenus anterior muscle, can be a factor in causing this syndrome.

Case 2 — Mrs. W., Aged 44 Years

This patient had Raynaud-like symptoms and night-numb-pain for 1 year, with blanching of fingers and sleepless nights from pain, as well as numbness on sleeping on her sides. X-rays were normal with no evidence of cervical ribs or cervical spondylosis.

Complete relief of the night-numb-pain and the vascular symptoms followed scalenectomy, freeing of the neurovascular bundle, and bilateral cervical sympathectomy. The subclavian artery was higher than normal at its origin.

Discussion

The co-existence of Raynaud's syndrome and brachial-plexus compression symptoms focused attention on the scalene muscle region. Relief of all symptoms followed scalenectomy.

Case 3 — Mrs. M., Aged 32 Years

This patient was relieved of 5 years of night-numb-pain by scalenectomy. There had been no evidence on X-ray of cervical ribs or encroachment of the intervertebral foramina. Bilateral high subclavian arteries were found. A physician treating the patient had asked for a carpal-tunnel release operation to be performed, but this was done only on the right side, whereas a bilateral decompression of the neurovascular tree and scalenectomy were performed.

After the operation symptoms were relieved on both sides; some recurrence occurred after 6 months on the right side. Knitting had been impossible until the carpal-tunnel release operation had been performed.

Re-exploration of the right supraclavicular region and freeing of the artery and the nerve plexus resulted in relief of the recurrent symptoms, and 1 year later the patient was still free of symptoms. At the second operation the artery on each side was adherent to scar tissue, and the plexus, especially on the right side, was being displaced posteriorly by a fibrous band.

Discussion

This patient was to some extent a test case. The physician diagnosed the carpal-tunnel syndrome. No swelling of the median nerve was visible on exposure beneath the right carpal tunnel. Further, symptoms were bilateral and relieved on both sides by scalenectomy and decompression, although carpal-tunnel release was only performed on the one side. Recurrent symptoms were relieved immediately by release of scar tissue in relation to the subclavian artery and the nerve plexus.

Case 4 — Miss G., Aged 34 Years

Following an attack of encephalitis, the patient was left with residual shoulder-arm pain and a typical night-numb-pain syndrome. On the right side the symptoms were so severe that sleep was lost. Relief was sought following continuance of the pain after deep-X-ray therapy to the spine, and 8 months' conservative treatment without improvement.

There was a history of numbness when the arms were extended on the steering wheel while driving a car, and a typical middle-of-the-night syndrome on lying on her side.

After the anterior scalene and the pectoralis minor were divided on the worst side, symptoms were dramatically improved. Only some residual numbness occurred on using the fingers and hands, but no further night-numb-pain.

Discussion

The occurrence of the typical syndrome of acroparaesthesia in a radiculitis following encephalitis was noted and the

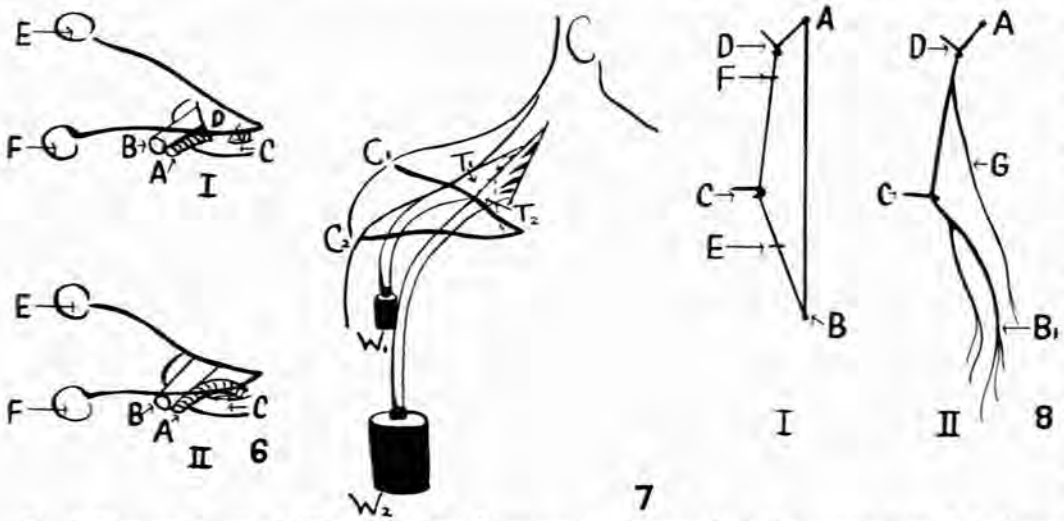

Fig. 6. Diagram showing buffer action of scalenus anterior in costoclavicular compression. I. Scalenus anterior present. II. Scalenus anterior removed. (A = subclavian artery, B = brachial plexus, C = first rib, D = scalenus anterior, E = normal shoulder tip, F = fallen shoulder tip.)

Fig. 7. Diagram showing traction on brachial plexus as a result of a fallen shoulder tip. (C_1 and C_2 = positions of shoulder tip, T_1 and T_2 = brachial plexus, W_1 and W_2 = representation of degrees of traction.)

Fig. 8. Diagram showing the effect of carpal-tunnel release on the neurovascular tree. I. Carpal tunnel intact. II. Following carpal-tunnel release operation. (A = fixed point at the root of the neck, B = fixed point in certain positions at carpal tunnel, B_1 = effect of carpal-tunnel release, C = effect of flexion of elbow, D = effect of abduction at shoulder, E = pronator teres point, F = pectoralis minor point, G = radial nerve, which skips the carpal tunnel.)

secondary part played by the scalenus anterior and pectoralis minor was established by these observations.

Case 5 — Mrs. D., Aged 25 Years

This patient was a nursing mother with a 3-month-old baby. She had typical acroparaesthesia and night-numb-pain in her hands, arms and shoulders, which woke her at night. X-ray of the neck had shown no cervical ribs or spondylosis. Neck traction had not relieved the symptoms. The cuff test showed no typical findings, i.e. the numbness would come on late and in the ulnar-nerve distribution as well as medially. Pushing a pram and holding heavy objects would bring on symptoms.

A bilateral scalenectomy relieved the symptoms immediately, but within 2 months she had a recurrence of night-numb-pain in the hands. Scalenectomy had removed all arm and shoulder pain, but not the hand symptoms. Bilateral carpal-tunnel release then relieved all symptoms including the night-numb-pain. No evidence of nerve thickening or neuroma was seen under the carpal tunnel.

Discussion

The features of this case fit into the syndrome of costoclavicular compression of Walshe, but they are also typical of the carpal-tunnel syndrome, according to Nissen's standards.

The recurrence of night-numb-pain after scalenectomy had not yet been observed to any extent except for case 3, where re-exploration and freeing of the subclavian artery and plexus from adhesions was followed by relief. Here, however, recurrence occurred, but carpal-tunnel release cured the symptoms. The reason for this could be one of the following:

1. The irritable neurovascular tree was still subject to costoclavicular compression following scalenectomy, in view of the absence of a buffering scalenus anterior (Fig. 6). More traction is exerted on the plexus with a fallen shoulder tip (Fig. 7); in this case the protecting action of the scalenus anterior would be even more important.

2. Carpal-tunnel release so relaxed the neurovascular tree that the compression at the scalene level no longer mattered (Fig. 8).

3. The original cause of the nerve irritability (? oedema, ? calcium deficiency) and increased muscle tone had disappeared at this stage of recovery.

Case 6 — Mrs. S., Aged 44 Years

This patient is a diabetic and suffers from a diabetic neuritis and tenderness in the legs as well as severe acroparaesthesia in the arms. For 5 years she had night-numb-pain symptoms with lack of sleep, and symptoms on driving a car.

A small cervical rib was present on the right side. Bilateral scalenectomy decompression and removal of the rib remnant relieved her symptoms for 6 months, but at the end of 2 years, because of recurrent night-numb-pain symptoms, a bilateral carpal-tunnel release operation was performed as recommended by her attending physicians. There had been only partial relief from neck traction.

Following operation she complained of burning in the median-nerve distribution, and also on the finger tips of the ring and little fingers. Symptoms in the right arm and hand were aggravated, whereas the left side was almost symptomless.

Discussion

The probability of diabetic neuritis as one of the factors in the abnormality of the neurovascular tree had to be considered in this case. Recurrence of night-numb-pain symptoms after relief for 6 months suggested either: (1) adhesions; (2) recurrence of cervical rib growth; or (3) a costoclavicular syndrome secondary to scalenectomy, whereby the rib and clavicle would impinge directly on the neurovascular tree without the intervention of the scalenus anterior 'buffer'.

A burning median-nerve neuritis with mild depletion of sensation occurred in case 7 as well. These 2 patients both complained tremendously regarding this. Where immediate and dramatic relief is considered a *sine qua non* of carpal-tunnel release, this complication was most disconcerting.

After about 2 months the symptoms gradually improved and the final result has been relatively satisfactory.

Case 7 — Mrs. R., Aged 58 Years

This patient had indisputable cervical encroachment with symptoms of acroparaesthesia, night-numb-pain, and disturbed sleep. There had been no relief from neck traction and a physician and surgeon had both previously agreed that a carpal-tunnel release operation was indicated; they referred the patient to me.

The cuff test on the right side was positive, but on the left the tingling was somewhat delayed and was also disturbed in the ulnar nerve region. A ganglion was visible at the wrist on the right side, extending under the carpal ligament, and there was some osteoarthritis of the wrist joint.

A carpal-tunnel release operation was followed by the appearance of a mild, hot swelling which was attributed to the reaction, especially in the degenerated tissues in relation to the ganglion wall. It remained for a long time. A mild hypoaesthesia and a persistent intolerable burning were present in the distribution of the median nerve. Symptoms were worse on the right side and mild early Dupuytren's contraction became visible in the palms.

Treatment with antibiotics, physiotherapy, deep X-ray therapy and hydrocortisone all failed to help the patient. It was even considered that a possible psychoneurosis was the cause. The swelling slowly subsided, but the patient still complains of pain and burning, especially when she uses her hands and carries anything.

Discussion

This patient had osteoarthritis of the spine with encroachment of C 5, 6, and 7, but, according to clinical tests, she was diagnosed as having a carpal-tunnel syndrome with an associated ganglion. Wearing gloves caused a numbness of the dorsum of the thumb. After carpal-tunnel release there was a burning neuritis of the median nerve, and no relief of symptoms.

This case illustrates the fact that carpal-tunnel release is not a universal panacea for acroparaesthesia on the grounds suggested. Diabetics and patients with cervical osteoarthritis present a special problem.

Case 8 — Mrs. W., Aged 41 Years

On 14 July 1960 this patient presented with a history of 3 weeks' numbness and pain, with night-numb-pain in the hands and arms causing loss of sleep. The radiation was in the median-nerve distribution, and the tourniquet test was positive. X-ray showed no encroachment on, or narrowing of, the intervertebral spaces. However, a small, left-sided cervical rib was present, as well as a long transverse process of C 7 on the right side. Operation was advised.

On 9 August she fainted and cricked her neck. Since then there has been no further trouble.

Discussion

This case demonstrates how acroparaesthesia with typical night-numb-pain can be relieved by a crick of the neck. It would be almost impossible to call this lesion a carpal-tunnel syndrome, in spite of the diagnostic similarity of signs and symptoms, based on the 'modern' concept.

In a class of 18 students, 6 admitted to night-numb-pain symptoms on lying on one or other side. Such a common set of symptoms associated with shoulder-head

approximation is unlikely to be caused by carpal-tunnel compression, in spite of the claims that this syndrome is typical of that lesion.

CONCLUSION

From this study it would appear that:

1. Symptoms of acroparaesthesia can result from several different factors.

2. Tautness, kinking and pressure of a prolonged nature, either from occupation or from position in sleep can cause symptoms by involvement of several anatomical points.

3. Altered anatomy, e.g. cervical ribs and bands; a high subclavian artery; relative postural decompensation following changes caused by operations; etc., will aggravate these factors.

4. Debunking of scalenectomy is unnecessary. There is some justification for scalenectomy or for palmar release in selected cases, and a unified concept embraces the historical complexity of the problem.

5. Pathological irritability of the neurovascular tree may be from medical causes, and radiculitis, diabetes, arteriosclerosis and oedema, for example, must not be overlooked.

6. Night-numbness and pain from lying on the affected side, and waking in the early hours of the morning from these symptoms, seems most frequently to be caused by scalenus-anterior pressure on the neurovascular tree, but relief is sometimes afforded by attack on apparently unrelated structures, e.g. excision of the clavicle and carpal-tunnel release.

7. The scalenus-anterior buffer is described, and the possibility of aggravation of costoclavicular symptoms after division of the scalenus should be borne in mind.

8. The shoulder-elevation factor is suggested as the explanation for secondary acroparaesthesia, night numbness and pain arising in patients who have had a painful shoulder for a long time and who protect that shoulder by elevation.

SUMMARY

1. Skeleton studies were done to show the effect on the reconstructed neurovascular tree of movements which made the tree taut and which kinked the tree in relation to muscles.

2. Personal symptoms were analysed in relation to the knowledge gained from anatomical and skeleton studies.

3. Historical developments in the diagnosis of cases of acroparaesthesia are presented.

4. Case summaries and discussions illustrate the problems involved.

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