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Occupational cervicobrachial syndromes

A review

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Key words: cervical syndrome, humeral tendinitis, tension neck syndrome, thoracic outlet syndrome.

The cervicobrachial syndromes related to work form an important group of rheumatic diseases. Surveys of rheumatic complaints in different occupations have been made for half a century (2). In these studies the cervicobrachial pain syndromes are preceded only by back diseases as a cause of occupational rheumatism.

The nomenclature of rheumatic diseases is difficult to systematize, and the differences in diagnostic criteria are partly reflected in the results of field surveys. Moreover, official statistics based on the International Classification of Diseases (114) are only of limited value when the socioeconomic burden of these diseases is being estimated.

What is also confusing is the role of work in the etiology or symptomatology of rheumatic diseases. Obviously there is no universal cause, such as "wear and tear" or "usage," for the common rheumatic syndromes. The role of occupational medicine today is to detect and report the defined patterns of occupational overuse associated with defined clinical syndromes.

In the present review the cervicobrachial syndromes are divided into four entities, namely, the cervical syndrome, the tension neck syndrome, the humeral tendinitises, and the thoracic outlet syndrome. Each of these syndromes is discussed separately, and their incidence, etiology, pathogenesis and clinical signs are considered from an occupational point of view.

The frozen shoulder and acromioclavicular syndromes have not been included because of the scant relevant literature focusing on the occupational aspects of these entities.

CERVICAL SYNDROME

Definition

The fundamental cause of the cervical syndrome is degenerative changes of the cervical spine. Radiological findings of cervical spondyloarthrosis, such as disc space narrowing, osteophyte formation and sclerosis of the apophyseal joints (81), are

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common. The encroachment of the intervertebral foramen or the spinal canal can compress and irritate nerve roots, the spinal cord, or vertebral arteries and result in neck stiffness, vertigo, pain radiating to the head, neck or arms, or silent cord involvement with slowly progressive spastic weakness in the legs (26).

Incidence

The presence of vertebral spondylosis has been recognized by pathologists for over a century (49, 81). Most of these studies have, however, been focused on the lumbar or thoracic spine. Cervical spondylosis has been found in autopsies of 30-year-olds and shown to occur in 70 and 90 % of 70-year-old females and males, respectively (28). With more sensitive criteria Nathan (60) showed the occurrence of this condition in 100 % of the 40-year-olds examined. Utilizing radiographs, Lawrence (46) noted similar frequencies for the occurrence of cervical disc degeneration.

In Lawrence's study there was a male predominance, which was attributed to the more severe degenerative changes occurring in males in heavy industry. He also noted a relationship between cervical disc degeneration and cervicobrachial pain, but only for moderate to severe changes was it significant. The same marginal correlation between radiological spondylosis of the cervical spine and the related clinical findings has been stressed by, e.g., Kellgren and Lawrence (36), McRae (55) and Friedenberg and Miller (25), whereas Hult (33) found a distinct correlation between cervicobrachial symptoms and an increasing severity of degenerative changes.

Etiology

The etiology of cervical disc degeneration is clearly multiple. The role of hereditary factors is indicated in the interindividual variation of the occurrence of the degenerative changes (107). The differences in the frequency of the condition for the two sexes (37, 44, 60) or different races (60, 94, 95) may suggest a genetic susceptibility or reflect environmental differences.

The high incidence of spondylosis in areas of excessive mobility or maximal pressure of the spine has been confirmed by many radiographic or autopsy studies of lumbar and thoracic (81) and cervical (60, 74) areas. The appearance of osteophytes has been considered to be a protective reaction of the body secondary to disc degeneration (74, 81), vertebral osteoporosis (60), excess mechanical stress (60), or trauma (99).

The effect of excessive mechanical stress on spinal degeneration has been investigated in several cross-sectional studies. In many of these interoccupational studies there has been an excess of symptoms and signs, as well as radiological evidence of lumbar degeneration in heavy occupations (33, 36, 43, 44, 80), but in the cervical area only slight, if any, differences have been found. Results supporting the theory of wear and tear as an occupational cause of cervical spondylosis are few. However, excessive loading on the head and neck has been shown to provoke significantly more degenerative changes in the cervical spine in meat carriers (85). Also Bremner et al. (11) found an increase of cervical, but not lumbar, spondylosis in Jamaicans when they were compared to Caucasians; he contributed this increase to the habitual carriage of head loads.

The question of static posture as a cause of cervical degeneration has been debated, and the results are still contradictory. Schröter (84) found as much cervical degeneration in stenotypists as in controls, whereas male dental surgeons showed an increased prevalence of grave degenerative changes as compared to load carriers, miners and office workers (83). The problems of selection, exposure and age-matching were, however, not adequately reported in these studies.

Aside from being a possible cause, work can also provoke the symptoms of cervical degeneration. The elevated incidence of cervical degenerative symptoms and incapacity in strenuous jobs can often be contributed to this fact (44). Ergonomic analysis of the load factors involved has not been common, but static and kinetic stress, extreme postures, and minor injuries have been claimed to be the factors in question (10, 13).

Pathogenesis

Degeneration of the intervertebral discs starts after adolescence and progresses continuously with increasing age (44). A decrease in water content with simultaneous alterations in the collagen and glycoprotein matrix of the intervertebral disc leads to a decrease in disc height and loss of shock absorbing capacity (61, 90). Secondary, partly protective, hypertrophic spurs develop on the edges of vertebral bodies and apophyseal joints (81). The clear association of the degenerative process with ageing has generally led to its acceptance as more or less "physiological" (33, 81).

The process of disc degeneration per se seems to be affected only slightly by external factors (14, 61). The relative instability of the degenerated disc (53) seems to lead to enhanced hypertrophic changes of the vertebral end plates and apophyseal joints in connection with external stresses and strains (44, 81, 104).

Conclusions

Cervical syndrome due to cervical disc degeneration develops from a combination of hereditary, constitutional, and environmental causes. Extreme occupational stress factors, such as heavy-load carrying on the head and neck, can cause secondary changes of cervical disc degeneration, e.g., osteophytosis of the vertebral bodies. The correlation between radiological cervical disc degeneration and the clinical symptoms of the cervical syndrome is only slightly positive. Nevertheless, the static and kinetic stresses of work can, in the presence of cervical disc degeneration, initiate or aggravate the symptoms of cervical syndrome. These stress factors, e.g., static posture, extreme movements of the cervical spine and minor injuries, should be avoided in the work environment.

TENSION NECK SYNDROME

Definition

The various nonarticular pain syndromes of the cervicobrachial area have been given a host of names, e.g., fibrositis, fibromyositis, myofascial syndrome (6, 92, 105), muscular rheumatism (87, 88, 89) or tension myalgia (96). These vague and poorly defined syndromes account for a substantial number of musculoskeletal disorders and much work absenteeism (70).

During the last 20 years cervicobrachial myalgia has been noticed often among office and factory workers with mental stress and repetitive or static loading of the muscles (50). Thus the syndrome has been called occupational cramp or myalgia (23) or occupational cervicobrachial disorder (51, 65).

Tension myalgia consists of a variety of symptoms such as pain, tenderness and stiffness of muscles, signs of hardened bands or nodularities, and muscle spasm. The complex usually has a point of origin — trigger point — from which the pain and numbness is referred to the reference zone, and the common boundaries of nerve distribution are often ignored (87, 88, 91). Various reflex phenomena mediate via the sympathetic nervous system, such as involvement of the central nervous system (50). The psychogenic basis of the syndrome has been stressed by many authors, and it has led to terms like "psychogenic rheumatism" (12, 106) or occupational neurosis (23, 34).

Incidence

As there are no universally accepted criteria for diagnosing tension myalgia of the cervicobrachial area, reports of this syndrome are not comparable (105). In the cross-sectional studies by Partridge et al. (71) vague pains of the neck and shoulder girdle were noted in 6 to 14 % of the workers, and no correlation to occupation was noted. However, the method did not allow the exclusion of articular pains from the shoulder or cervical spine. In one of the large investigations by Lawrence (45) in the 1950s undetermined neck-shoulder pain was reported by 8—6 % of the miners studied and their controls. In a smaller sample, after those with cervical disc degeneration had been excluded, 24 % of the office workers, but only 4 % of the miners, complained of undetermined cervicobrachial pain (36). In recent Japanese literature,

"occupational cervicobrachial disorder" has been widely discussed, and its prevalence stated among, e.g., assembly-line workers (50, 68), cashiers (65), packers (66), and office workers (51). By using different gradings of the syndrome, Komoike et al. (39, 65) estimated its overall prevalence to range from 4 % in management staff to 21 % in assembly-line workers (51).

Similarly, Ferguson in Australia found occupational cramp in 22 % of the telegraphers examined (23), and mild symptoms without disability were reported by 42 % of the workers (24).

Etiology

Today the etiology of tension myalgia is still partly speculative. If secondary myalgies due to clear injuries, infections and inflammatory or degenerative musculoskeletal diseases (87, 88) are ruled out, there remains an inconstant, functional entity which can be called "primary tension myalgia" (96). In his extensive review Simons (88) summarized the factors of importance, namely, local chilling, acute strain, overexertion and excess fatigue of the muscles, or immobility and inadequate exercise. Also chronic straining, habitually poor posture and psychogenic factors such as depression or emotional stress and tension (12, 106) have been considered important.

The occupational etiology of cervicobrachial myalgia was investigated in the 1970s, mostly in Japan, where the Committee on Cervicobrachial Disorder, organized in 1971, formulated standards for diagnosis of the disease. The Committee stated that the disease is a functional and organic disorder due to muscular and mental fatigue in static and/or repetitive arm and hand work (51).

Typical occupations with cervicobrachial disorder were cash register operators (65), typists, packers and assembly-line workers (51), but also in 35 % of the office workers shoulder muscle tenderness and stiffness were noticed (68).

The causative dynamic muscle loads were repetitive, high speed finger, wrist and arm motions hundreds or thousands of times per workday (51, 66). Unnatural and static positions of the arms with raised elbows and shoulders (21, 50, 51, 103), as well as the need for excessive force (51), increased the static muscular load. Mental strain such as monotonous work conditions, need for concentration and much responsibility, together with poor illumination and noise, were characteristic of most of the occupations (50, 51, 66). In the work tasks, e.g., 1,000—2,000 articles per day were handled on the assembly line (51, 68), or 15,000—36,000 information inputs per day were processed in data machine operation (51, 68).

The effect of static shoulder muscle load has been examined in Swedish welders and construction workers by Petersen et al. (75), Kadefors et al. (35) and Ekholm et al. (22). They found that working with elevated arms, especially in overhead positions, caused subjective shoulder-arm fatigue.

By means of quantitative electromyography they identified localized muscle fatigue in the deltoid, trapezius and supraspinous muscles. Experience led to adaptation so that fatigue became localized only in the supraspinous muscle. The study focused on the acute reactions of static muscle load, and no conclusions were drawn on the possible long-term effects of the muscle load, excluding supraspinous tendinitis (29).

Pathophysiology

The cause of tension myalgia is still somewhat speculative. In his recent review of the disorder Simons (88) failed to discover consistent pathological findings. He stated that three of the four cardinal features (palpable hardening, local tenderness of the muscle, referred pain and reflex phenomena associated with muscle pain) may occur alone or in any combination. The symptoms of the patients contrasted with the variable objective signs, and the multiple electrophysiological and pathological reports related poorly to the fairly obvious clinical findings (96).

The most commonly proposed theory for the pathogenetic mechanism of this disorder focuses on localized muscle fatigue due to static, sustained contraction. The basic process has been proposed to be the accumulation of metabolic end products in the muscles or insufficient oxygen sup-

ply (2, 58). The studies, however, have concentrated mainly on the acute effects of muscle exercise, and the correlation between chronic muscle fatigue and myalgia has still not been explained on an experimental basis.

The electromyographic changes associated with acute occupational muscle fatigue have been widely investigated (22, 35, 41, 75). Even if the results are still partially controversial (41), a shift of the myoelectric spectrum to low frequencies during the course of fatigue exercise has been noted (3, 41, 75). The results indicate a linear correlation between the spectral change and the subjective rating of fatigue. In contrast to these acute reactions of muscle fatigue, no consistent electromyographic changes have been noted in connection with chronic tension myalgia (40, 88).

Many of the investigations concerning tension neck syndrome stress its psychogenic aspects. The subjective feeling of localized fatigue and pain is often guided by motivational or social factors (8), and the psychological constitution of the patient partially determines the behavior and prognosis of the disorder (23). Here again, one cannot state whether the recorded psychological disorders, such as hypochondria or depression, are due a neurotic preoccupation or are a consequence of the vicious long-lasting spasm-pain cycle (96).

Conclusions

Occupational cervicobrachial myalgia is a functional disorder with fairly common, but somewhat various, clinical symptoms. The objective clinical, electromyographic, histological, and laboratory findings are, however, inconsistent. Both the psychological and, partially, physiological constitution of the worker may contribute to the development of the syndrome. External work factors, such as static loading of the shoulders and arms, and repetitive, high speed motions in connection with mental stress can lead to muscle incoordination and spasm. The connections between external work load and acute muscle fatigue and ache have been well established, but the occupational basis of chronic myalgia has been only indirectly indicated by epidemiologic investigations. These investigations also promise, however, that by work design and ergonomic improvements of the work environment the morbidity of this syndrome can be decreased (65).

HUMERAL TENDINITIS

Definition

True arthrosis of the glenohumeral joint is rare (13). Most of the radiological changes of the glenohumeral area are secondary, limited to soft tissue inflammations and/or ruptures (108). Arthrosis of the acromioclavicular joint is, however, common, but not related to occupation or dominance (7, 13, 19, 113). It can cause local symptoms or secondary irritation of the subacromial bursa (113), but it is often symptomless (13, 19).

The vast majority of shoulder pains is caused by soft tissue lesions around the shoulder joint. The syndrome has been called "periarthritis glenohumeralis," but several clinical entities can be recognized. Pasila (73) differentiated between degenerative and calcific tendinitis (of the rotator cuff), bicipital tendinitis, rupture of the rotator cuff, rupture or luxation of the bicipital tendon, and idiopathic frozen shoulder. In addition primary subacromial bursitis and acromioclavicular syndrome can be named as independent, although rare, entities (5).

The present review focuses on the tendinitises of the rotator cuff, especially in the area of the supraspinous tendon, and the tendinitises of the bicipital tendon. They form the great majority of all the soft tissue disabilities around the shoulder joint (73) and are important etiologic factors also in the ruptures of these tendons. Occupational and attritional factors have often been considered to be of importance in the etiology of these tendinitises, and they have been discussed in this review, in which the supraspinous and bicipital tendinitises are referred to with the common name "humeral tendinitis" but are discussed as individual afflictions.

Incidence

The occurrence of rotator cuff tendinitis and rupture rapidly increases with increasing age, starting from 30 years (109, 110). Calcification of the rotator cuff, as seen in radiographs, has been noted in 8 % of the population over 30 years of age (9, 100) and 35 % of all calcific deposits produce symptoms at some time (86). Similarly, histological changes of rotator cuff tendinitis and degeneration were determined for between 70 and 100 % of the persons over 40 years of age, and no association was found between different types of degenerative changes and shoulder pain (67). The likelihood of rotator cuff ruptures, however, increased with the severity of the degenerative changes (67). Cuff ruptures were found in 20 to 40 % of the autopsies of 30-year-old or older persons (38, 109), and their prevalence increased from 25 % at the mean age of 51 years to 50 % at the mean age of 81 years (27).

Degenerative changes of the rotator cuff were equally common in both shoulders, and there were no differences between the sexes (16, 38, 47, 64, 67). Clinical ruptures of the rotator cuff are usually more common in the male right shoulder (7, 73, 110).

Bicipital tendinitis has been found in 10 to 20 % of routine autopsies (20, 31, 56, 110), often combined with rotator cuff tendinitis or rupture, but it can manifest itself also as an isolated entity (30, 48, 73, 82) in as many as 60 % of all cases of shoulder pain (17).

Etiology

The literature presents three main causes for rotator cuff tendinitis, namely, age-dependent degeneration of the hypovascular cuff area, attritional usage of the cuff, and traumatic disruption of the tendon bundles (38, 64).

The degeneration of the rotator cuff starts in the "critical zone" of the supraspinous tendon, where the end arterioles derived from the bone attachment and muscle side terminate (47, 59, 76, 79). The head of the humerus enlarges during growth, and the tendinous part of the supraspinous muscle elongates (110). Angulation and pressure of the rotator cuff over the humeral head further increases the ischemia (76, 79), occluding the longitudinal vessels in the tendon. The healthy tendon is the strongest part of the muscle-tendon-bone system, and tensile stress leads to bony avulsion or to muscle rupture. With ischemic degeneration the tendon loses tensile strength and a rupture occurs (52, 54).

Recently, Herberts and Kadefors (29) postulated another possible factor for an increase in tendon ischemia, namely, the static contraction of the supraspinous mucle in overhead activities. By means of clinical examination, soft tissue radiography, and electromyographic spectral analysis they found rotator cuff tendinitis in elderly welders and attributed it to prolonged overhead work. A study with different control groups must, however, be undertaken before any definite conclusions can be made concerning these occupational aspects of supraspinous tendinitis.

The theory of attritional friction between the humeral head and the acromion was suggested by Meyer (57) and Howard (32). They thought that attrition and microtraumas of the collagen bundles of the tendon may cause an invasion of vascular granulation tissue that erodes the tendon like a rheumatic pannus. The trauma responsible for tearing in the rotator cuff tendon is, in most cases, indirect muscular violence exerted by the shoulder muscles. During abduction in internal rotation the acromion impinges against the rotator cuff and may lacerate it. Anterior dislocation or subluxation are known to result in the rupture of the supraspinous and subscapular tendon (15). Although symptomatic tendinitis is more common in working males, the condition is also found in sedentary patients and females without a history of trauma (7, 16, 64).

Rotator cuff tendinitis is often associated with a secondary involvement of the bicipital tendon (16, 17, 67, 109). Meyer (56) was the first to notice bicipital tendinitis as an isolated entity. Anatomic variations in the depth and angle of the bicipital groove, a supratubercular ridge and impingement under an elongated acromion may increase the likelihood of tendinitis (30). Repetitive abduction-rotation move-

ments are thought to be conducive to the syndrome (17, 48, 56, 82), but no conclusive data on their association have been found as yet.

Pathophysiology

The literature concerning pathological changes in the supraspinous muscle is abundant (15, 47, 48, 54, 56, 64, 67, 76, 79, 109). The changes begin microscopically with hyaline and fibrinoid degeneration and fragmentation of the collagen bundles, loss of tenocytes, and deposition of calcium salts in the necrotic debris. Surrounding the necrotic area is an inflammatory reaction with the presence of giant cells and neovascularization. An autoimmune response resulting in diffuse capsular involvement has been suggested (54). In the presence of definite tears of the rotator cuff, also other signs of acromiohumeral attrition may occur (64, 108).

Changes in the tendinitis of the long head of the biceps has been compared to those of de Quervains' stenosing tenosynovitis (86). In the acute stage there is edema of the synovial sheath and tendon with cellular infiltration and filmy adhesions between the tendon and its sheath. In the chronic stage the adhesions organize and lead to fibrous tenodesis in the sulcus, or the tendon erodes and ruptures as a result of muscular exertion. The intraarticular tendon adheres to the articular capsule or totally disintegrates (17, 30, 48, 67, 86).

Conclusions

Bicipital and rotator cuff tendinitis form the majority of all the shoulder joint disabilities, and they are contributing factors to other soft tissue lesions, namely, tendon ruptures and subacromial bursitis.

Tendinitis of the rotator cuff, especially in the area of the supraspinous tendon, seems to be caused by the untoward anatomic relationships leading to local ischemia and degeneration in middle age. The role of external factors, such as repetitive horizontal activities of the arm (causing tendon attrition and impingement) or static shoulder muscle contraction (diminishing further the tendon blood supply) must be determined by additional controlled studies.

Muscular overexertion and overt trauma can lead to rupture of a degenerated, but not a healthy, tendon and can markedly exacerbate the symptoms of tendinitis, thus partly accounting for the increased morbidity in heavy work.

Bicipital tendinitis can be found as an isolated entity, but is often secondary to lesions in the rotator cuff. Primary bicipital tendinitis has been related to direct and indirect traumas to the shoulder and to overexertion and repetitive activity of the arm, and thus its etiology resembles that of traumatic tenosynovitis in the wrist. Primary degeneration of the bicipital tendon has not been reported in the literature, but constitutional variations in the passage of the bicipital sulcus can be contributory factors in bicipital tendinitis.

THORACIC OUTLET SYNDROME

Definition

Conditions causing pain, numbness, and fatigue in the upper extremities are too many to list in their entirety. Neuralgic pain in the cervicobrachial area may be caused by lesions in the cervical cord or root area, e.g., herniated cervical disc or spinal cord tumors, or by lesions compressing the brachial plexus, such as tumors of the superior pulmonary sulcus or trauma. Distal entrapment neuropathies, including radial and ulnar nerve compression at the elbow, and enthesiopathies or humeral epicondylitides, among others, can be ruled out by systematic clinical examination.

Thoracic outlet syndrome is an important, and often neglected, cause of cervicobrachial pain, weakness and sensory disturbances (4, 112). The various forms of neurovascular compression at the superior thoracic aperture have been recently described by, e.g., Tyson and Kaplan (101). These syndromes can be grouped into compression syndromes at three levels, namely, the scalenus syn-

drome, including also the skeletal anomalies of the cervical and first thoracic rib, the costoclavicular compression syndrome, and the hyperabduction compression syndrome of the pectoral minor tendon.

Incidence and diagnosis

The diagnosis of these syndromes is made on the basis of typical history and physical findings, especially test maneuvers to provoke symptoms of the outlet syndrome.

The neurologic symptoms of numbness, pain and paresthesia, generally in the distribution of the ulnar nerve, dominate (4). Various stages of arterial compression produce arm weakness and claudication, Raynaud phenomenon and, in grave stages, thromboembolic episodes with gangrene in the digits. Symptoms of venous occlusion are rare but can lead to effort thrombosis of the subclavian vein.

The three aforementioned thoracic outlet syndromes are tested by different maneuvers. The Adson maneuver (deep inspiration, neck extension and head turn toward the examined side) tenses the scalene muscles to provoke symptoms of the scalenus syndrome. The costoclavicular maneuver, with bracing of the shoulders backwards and downwards, narrows the costoclavicular space. The Allen hyperabduction test with the arm abducted 90° results in compression of the neurovascular bundle under the pectoral minor tendon insertion to the coracoid process.

The prevalence of the positivity of the various outlet maneuvers is not exactly known. Wright (115) noticed that in 82 % of his test persons the hyperabduction test was falsely positive, whereas Winsor and Brown (111) stated the number to be 15 %. Similarly, Telford and Mottershead (98) reported that in 38—68 % of normal persons the provocative tests were positive. There exists, then, a sliding scale of positivity for the test, and additional criteria of symptoms or, preferably, persistent clinical findings are necessary for the diagnosis.

The symptoms are, however, often bizarre (112), and no diagnostic procedure is purely objective. Cervical radiographs are helpful in detecting the bony anomalies. Cervical ribs occur in 6 out of every 1,000 persons and are twice as common in women as in men (62, 69); 50—90 % of the cases are however symptomless (18, 93).

Similarly, cervical radiographs are negative for half of the patients (4, 102, 112). Angiography, nerve conduction studies and electromyography are reported to be of value, especially in the exclusion of other causes of nerve compression syndromes (42, 102, 112).

Etiology

At the level of the thoracic outlet the neurovascular bundle passes through three narrow spaces of potential compression. Anatomic anomalies and variations of the superior thoracic aperture such as cervical ribs or elongated transverse processes with associated fibrous bands, anomalies of the first thoracic rib or in the origin of the brachial plexus, and variations of the interscalene triangle can cause such compression. The costoclavicular triangle may be narrowed by an abnormally flattened or bifid clavicle (63, 101).

The descent of the anterior chest wall and the shoulders during growth decreases the width of the outlet canal. This descent is greater in women and explains their increased affection, the ratio being from 1:4 up to 1:10 (69). Similarly, the right side has been claimed to be involved more often because it is lower in most individuals (69).

In addition to the aforementioned musculoskeletal abnormalities, the neurovascular bundle can be compressed by acquired varieties of posture. Scalenic muscle spasm with an accompanying outlet syndrome has been claimed to occur after hyperextension injuries of the neck (12, 78, 112), secondary to nerve root irritation due to cervical spondyloarthrosis (12), or as a result of exertional or postural fatigue or emotional stress (62).

Costoclavicular and hyperabduction syndromes especially have been combined with habitually poor posture involving sagging shoulders and with occupational factors such as carrying heavy shoulder loads or working overhead (e.g., painting a ceiling) (69). Recently, the thoracic outlet syndrome was identified in male

athletes who showed unilateral hypertrophy of the upper extremity (97). The absolute number of occurrences of the syndrome, e.g., in different occupations, is not known.

Pathophysiology

Most cases of thoracic outlet syndrome are mild, with intermittent neuralgic pain and paresthesia. A reduction of the ulnar nerve conduction velocity due to brachial plexus neuritis may occur, causing reduced sensation and muscle weakness in the ulnar area. Permanent muscle wasting is relatively late and rare (101, 102, 112).

Compression of the subclavian artery is usually transient and mild. A poststenotic dilatation of the subclavian artery, with distal thromboembolization, may occur, but is usually rare (4, 101).

A compression of venous circulation is unusual because of its route anterior to the scalene muscles. Incidental cases of effort thrombosis of the subclavian vein have been reported (1, 77, 101, 102).

Conclusions

Thoracic outlet syndrome results from compression of the neurovascular bundle between the first rib, clavicle and the accompanying muscles. Local abnormalities and constitutional factors are the prime causes of the syndrome. External postural and occupational factors may initiate the clinical symptoms, but cannot be regarded as being important in the etiology of the syndrome. The clinical syndrome occurs relatively seldom, but mild forms of it can also contribute to cervicobrachial pain and should be taken into consideration in the differential diagnosis.

In this review of cervicobrachial pain syndromes related — or unrelated — to work, I have tried to approach the different etiologic factors and most important causes of the symptom complex. The complaints of cervicobrachial pain are bizarre, and no one specialist is capable of mastering all the aspects of the syndromes in this area. Information on the different causes of these complaints should be collected with the cooperation of many different specialists, e.g., occupational physiatrists, orthopedists and neurologists, so that morbidity in work life can be decreased. Also, in future studies on the occupational relationship of these syndromes, multidisciplinary collaboration is mandatory.

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