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Peripheral Nerves

Neurosurgery of the Peripheral Nervous System: Entrapment Syndromes of the Brachial Plexus

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Entrapment of the brachial plexus (BP) can be primary or secondary to either trauma or surgery. Most frequently, the BP is entrapped at the "thoracic outlet" resulting in thoracic outlet syndrome (TOS). Hyperabduction syndrome, scalenus anterior (or anticus) syndrome, costoclavicular syndrome, cervical rib syndrome, and long-C7 transverse process syndrome are all entrapment syndromes of the BP. These last terms are used either independently or as part of TOS, although "true TOS" is due to an alteration of the soft tissues at the thoracic outlet and not to bone causes.

In 1956, Peet et al. [4] introduced the term TOS to describe a clinical picture with signs and symptoms due to compression of the BP and subclavian vessels at the thoracic outlet. The etiology and clinical presentation of BP entrapment syndromes, including TOS, are variable; diagnosis may be difficult, and treatment is controversial. Knowledge of the anatomy, clinical presentation, diagnostic tools and therapeutic options is very important for correct diagnosis and therapy.

Anatomy

The thoracic outlet is delimited by osseous and muscular structures. The bones are anteriorly the sternum and clavicle, posteriorly the vertebral column, and laterally the first rib (Figure 1). The muscles are the scalenus anterior (or anticus) and scalenus medius that are inserted on the cervical vertebral column, the first rib forming a triangle that is crossed by the elements of the BP, and the subclavian artery (Figure 2). The BP may be entrapped at the level of the roots, trunks, or cords. The potential sites of BP entrapment are: 1) the interscalene triangle (Figure 2), 2) the costoclavicular space between the clavicle and first rib (Figure 1), and 3) the subcoracoid tunnel that is located under the pectoralis minor muscle tendon insertion on the coracoid process of the scapula (Figure 1). Anatomic anomalies such as cervical ribs, long C7 transverse process, and especially fibrous of ligamentous bands are the most frequent causes of BP entrapment syndromes (70% of cases in a series of strictly selected patients with TOS) [2]. Fibrous or ligamentous bands may be present between: 1) the tip of either a cervical rib or a long C7 transverse process and the first dorsal rib; 2) the scalenus minimus muscle (inconstant muscle) and the first dorsal rib; 3) the scalenus anterior (or anticus) and scalenus medius. The last two muscles may also have fibrotic edges that compress or irritate the BP. The Sibson's fascia or suprapleural membrane at its posterior edge may be another compressive factor, as well as anomalies of the first rib (Figure 2).

Pathophysiology

Compression of the BP causes changes in the neural tissues that are similar to those observed in other nerve entrapment syndromes. At the beginning, a breakdown of the blood–nerve barrier of the perineurium occurs. Then, if compression and/or repetitive trauma persists, fiber demyelination, damage to axolemma and endoneurium, and wallerian de-

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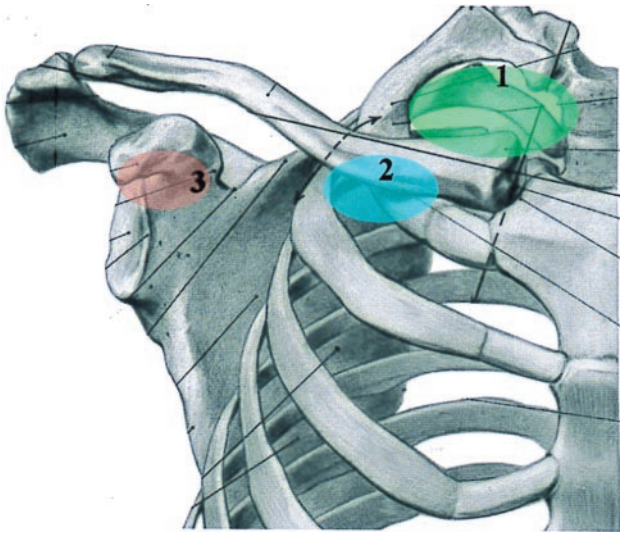
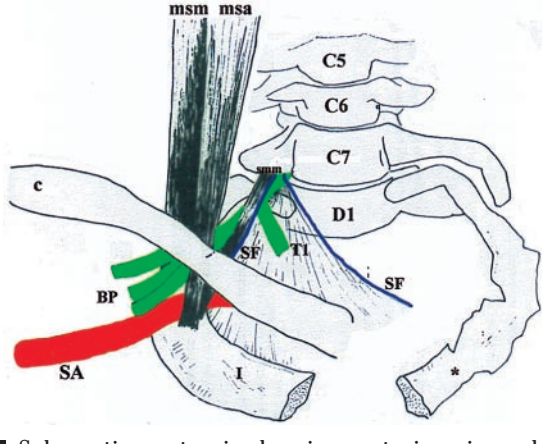


1 Bony elements delimiting the thoracic outlet (sterno-costo-vertebral space), costo-clavicular space, and subcoracoid tunnel.

generation take place. The clinical picture will reflect the histologic changes of the involved components of BP. In the pathophysiology of BP entrapment syndromes, the "double-crash" theory [8] should be taken into consideration. According to this theory, there is a proximal-to-distal combination of two compressive forces acting on two different sites along one nerve (for example, forces compressing the ulnar nerve at both the thoracic outlet and cubital tunnel). Though a single distal compressive force does not reach the "minimal damage threshold" by itself, combination with a second proximal compressive force acting at a different site may cause a nerve degeneration. The term "reverse double crash" indicates a distal-to-proximal compressive force.

In the pathophysiology of BP entrapment syndromes, the concept of muscle imbalance and its functional anatomic basis must also be considered [9]. Anomalous positions of the head, spine and upper limb causes some muscles to be shorter than normal. As time passes, these muscles actually tend to become shorter and when stretched or overused, they become painful and, because of their increased volume, exert further neural compression. At the same time, other muscles become weaker, thus creating a pattern of muscle imbalance.



2 Schematic anatomic drawing, anterior view, showing some of the structures that can be involved in brachial plexus entrapment syndromes. C5, C6, C7, D1 vertebral bodies: a long C7-transverse process and morphological changes of the first rib (*) are seen. msm: muscle scalenus medius; msa: muscle scalenus anterior or anticus; smm: muscle scalenus minimus; SF: Sibson's fascia; T1: T1 root forming with the C8 root the inferior trunk of the brachial plexus; c: clavicle; I: first rib; BP: brachial plexus; SA: subclavian artery.

Clinical Features

Symptoms of BP entrapment syndromes are related to the involved neural components. Compression of the BP must be distinguished from: 1) proximal pathologies, like cervical disc and spondylitic disease; 2) distal pathologies, like ulnar or median nerve entrapment; 3) BP intrinsic of extrinsic tumors; and 4) BP inflammation.

TOS can be neurogenic or vascular, according to the prevalence of neurologic or vascular symptoms, and can be unilateral or bilateral. There is no age prevalence. Cervicoscapular discomfort due to muscle imbalance is frequent; the head and cervical spine are anterior to the thorax in an incorrect posture. Cervical, suprascapular, and infrascapular burning pain irradiating to the upper extremity is also typical. More frequently, the C8-T1 component of the BP is involved, either proximally at the lower trunk or more distally at the medial cord level with associated sensory disturbances along the ulnar nerve distribution. Less frequently, the BP is entrapped at the upper trunk; in this case, sensory symptoms in the area of the median nerve distribution are present. Paresthesias and numbness of the upper limb are often exacerbated by movement. Sensory symptoms may be followed by motor symptoms such as weakness and tiring of the arm that may affect the intrinsic muscles of the hand. Vascular symptoms are less frequent, and include coldness, cyanosis, or edema of the involved limb. In severe cases, thrombosis and, eventually, embolism with distal infarct may be seen [1]. A focal hand dystonia was recently described in a patient with

TOS. It consisted of flexion of the wrist and fingers curled into the palm [5].

Diagnosis

There are no methods to diagnose the precise site of BP entrapment. Diagnosis of BP entrapment is difficult because of the highly variable clinical presentation and the subjective symptoms that are frequently associated. Careful attention to the clinical history noting onset and duration of symptoms and signs is very important. Clinical conditions presenting with symptoms that are similar to those of BP entrapment syndromes should be ruled out. Thus, when a BP entrapment syndrome is suspected, it is necessary to perform all the diagnostic procedures to exclude pathologies affecting the cervical spinal cord and the ulnar and median nerves in the upper extremity.

To aid in the clinical diagnosis of TOS, tests like that of Adson have been proposed. This test is positive when the radial pulse is obliterated following a deep breath, with the patient having the arm down and the head turned toward the affected side. These tests, however, give a high number of false positive results. In general, a diagnosis of TOS is probable when a positive vascular test is associated with neural symptoms.

Electrodiagnostic studies are more useful for the diagnosis of distal nerve compressions than for BP compression. However, low ulnar compound muscle and sensory nerve action potential amplitudes, low median compound muscle action potential amplitude, and normal median sensory action potential amplitude are said to be characteristic of TOS [3]. Recently, it has been concluded that the abnormal medial antebrachial cutaneous nerve (derived from C8-T1 and the inferior trunk) sensory response is of diagnostic value in patients with neurogenic TOS [3].

Computed tomography (CT) scans or X-ray plain films may show bony abnormalities like cervical ribs, long C7 transverse processes of degenerative diseases involving the fibrosseous structures of the thoracic outlet region. Spiral CT is able to simultaneously visualize osseous and soft-tissue structures; it has been recommended as a valuable tool for the diagnosis of TOS [6]. Peripheral angiography, echo color doppler studies, and phlebograms may be useful in vascular TOS.

BP entrapment syndromes are complex clinical conditions in which the psychologic aspects are important in both pathogenesis and prognosis. The pain of organic origin can be reinforced by unresolved psychologic conflicts and the derived conversion reaction is an additional obstacle to the right diagnosis and treatment. Psychologic assessment with a Minnesota Multiphasic Personality Inventory test has been proposed, before surgery, in patients with TOS to plan preoperative psychotherapy as well as a program for chronic pain management before and after surgery [7].

Treatment

In suspected BP entrapment syndromes conservative therapy should be attempted as a first step. Correction of posture and position, appropriate physiotherapy, and modification of patient behaviour are the key points of such non-surgical therapy. Examination of single muscles reveals patterns of muscle imbalance that dictate the program of correction of posture and position.

Surgery should be considered after failure of appropriate conservative treatment of six months' duration. Demonstration of anatomical and/or electrical abnormalities is of crucial importance to select patients for surgery. Two main surgical trends exist. The first one advocates either sectioning of the muscle scalenus anterior (or anticus) or resection of the first rib to obtain BP decompression. From a technical point of view, the sectioning of the scalenus anterior or anticus muscle via a supraclavicular approach is easier; after identification and dissection of the phrenic nerve, the muscle can be sectioned. The resection of the first rib via a transaxillar approach is more difficult because the BP, pleura, and vascular structures might be inadvertently injured. A second surgical trend advocates a more rational approach with an exploratory operation, usually via an anterior supraclavicular approach, less frequently via a posterior subscapular approach or an infraclavicolar approach, to look for possible causes of BP compression. With this kind of approach, the surgeon has several options, such as removal of cervical ribs, long C7 transverse processes, abnormal edges of muscles (scalenus anterior or anticus, scalenus medius, scalenus minimus), compressing fascial borders, and abnormal ligaments. In our experience, the disarticulation of cervical ribs or gross section of long-C7 transverse processes are not necessary; skeletonization and simple reduction of the bony mass using a drill or rongeurs under microscopic control is enough to relieve the neural compression. We have never operated on a patient with a BP compression at the subcoracoid tunnel.

Conclusions

BP entrapment syndromes remain complex and challenging conditions involving the interest of different specialists like neurologists, orthopedics, vascular and general surgeons, neurosurgeons, physiatrists, and psychiatrists. The best treatment can be achieved after careful evaluation of clinical and instrumental data. Surgery for these complex syndromes should avoid axiomatic standard procedures, such as resection of the first rib, and promote a more rational approach based on the anatomy found at surgical exploration. Elimination of real compressing factors are the best guaranties of good clinical results.

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Most major types of disease will be virtually eliminated by 2050, thanks to a combination of improved diet, lifestyle, and environmental factors, and advances in gene therapy and drugs. Health-care costs will fall, as expensive procedures such as surgery will be restricted to treating accidents and traumas.

—"Outlook 2000"
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The number of centenarians worldwide will increase 16-fold by 2050, reaching 2.2 million persons, up from 135,000 currently. Women will far outnumber men in that age group, but men who survive into their 100s—avoiding Alzheimer's disease in their 80s and 90s—will retain sharper mental fitness, researchers predict.

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