بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

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لم ترد بالأصل

Entrapment Neuropathies

Essay Submitted for partial fulfillment
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Content

Page

Introduction

Entrapment neuropathy is a group of peripheral nerve disorders characterized by paraesthesia, pain, or loss of function in the distribution of a nerve resulting from extrinsic anatomical constraints in the course of the nerve ***" Spinner and Spencer 1974 "***.

Entrapment of nerves generally occurs they pass beside a joint such as the wrist, the elbow, or the hip . It is uncommon in other areas of the extremities . This coincide with the fact that entrapment neuropathy rarely occurs in the head for trunk, suggests that repetitive motion is a major factor that precipitates entrapment in an anatomically constrained segment ***"Campbell et al., 1991"***.

Persistant symptoms following surgical release for entrapment neuropathy are a source of great frustration for both surgeon and patient . Fundamentals of management require review of the patient`s history and examination in an attempt to confirm the initial diagnosis and to rule out elements of the differentail diagnosis . Persistent symptomatology may be a reflection of an incorrect diagnosis or double crush syndrome . If the initial dignosis can be reconfirmed, then it is reasonable to reinstitute conservative management and to objectively re – evaluate the enptapment neuropathy by electrodiagnostic testing .
This electrodiagnostic testing must be comprehensive in order to evaluate all potential entrapment neuropathies within the differential diagnosis . Individuals who demonstrate a transient response to conservative management or evidence of further deteioration on electrodiagnostic testing may be considered candidates for revision surgery . These individuals may be found to have had an incomplete release, error of technique, or iatrogenic compression . Persistence of symptoms on the basis of end-stage disease must be recognized to avoid further surgery that is unlikely to be of benefit to the patient .If surgical intervention is chosen, the procedure must address issues of residual compres-

sion, preservation of nerve vascularity, prevention of neurodesis, and protective padding in the presence of nerve hypersensitivity. ***"Idler, 1996".***

Anatomy Of The Peripheral Nerve

A peripheral nerve as visualized at the operating table is composed of many different axons of diverse diameters .The axon is a long projection from the soma, which can extend for a distance of several feet . they tend to be grouped together forming tracts in the C.N.S. and nerves out side the C.N.S . It is bounded by a semipermeable membrane called the axolemma . This is surrounded by a basement membrane , which in turn, is encircled by the myelin sheath laid down by schwann cells . Nerve fibers may be myelinated or unmyelinated in myelinated fibers the regions where longitudinally adjacent schwann cells join each others are called nodes of Ranvier . The entire axon is then covered by endoneurium, the inner most layer of the peripheral nerve connective tissue structure . A number of axons are grouped together in a bundle called fascicle, which is invested in another mesenchymal sheath called the perineurium . The fascicle is the smallest segment of the nerve that is generally visible with the operating microscope .The perineurium is itself a semipermeable membrane and, as such, is the primary regulator of the intrafascicular environment . The fascicles are grouped together to form a peripheral nerve, and this structure is covered by the outer most connective tissue layer, the epineurim, which is often used to hold sutures in peripheral nerve repairs. The individual axons, as mentioned, are of different sizes, varying in diameter and nerve conduction velocity is proportional to the square root of fiber diameter (fig.1) . They are classified to three groups, Group A up to 20 micrometer in diameter and subdivided into – α: motor and proprioception (I a and I b) .
β: touch, pressure and proprioception (II) . γ : fusimotor to muscle spindles (II) . δ : touch, pain and temperature (III) . Group B up to 3 micrometer in diameter. myelinated preganglionic autonomic. Group C up to 2 micrometer in diameter unmyelinated postganglionic autonomic, (IV) . Peripheral nerve trunks in the limbs are supplied by branches from local arteries . The sciatic nerve in the buttock and the median nerve at the elbow have

each a large branch from inferior gluteal and common inteross-eous arteries respectively . Elsewhere , regional arteries supply nerves by a series of longitudinal branches which anastomose freely within the epineurium ***(Worth, 1996)*** .

Aetiology Of Entrapment

Peripheral nerve entrapment syndromes may be caused by inflammation, trauma, or congenital deficits . Some entrapment neuropathies are overdiagnosed, particularly carpal tunnel syndrome . Other nerve entrapment syndromes, such as the thoracic outlet syndrome , posterior interosseous nerve syndrome, and anterior interosseous nerve syndrome, are rare .
In most cases of an entrapment neuropathy, the diagnosis and management are straight forward, but much remain to be learned about the natural history, course, and prevention of these clinical conditions . ***" Nakano, 1997 ".***

Two types of anatomic constraints predispose to entrapment neuropathy . The first type is a fibrosseous tunnel and the second type is a fibrotendinous arcade .

Constriction in the first type is static one due to :
1) Contents of the tunnel become larger or hypertrophic .
2) The walls of the tunnel encroach upon the tunnel (Fig. 2)

Constriction in the second type is dynamic one as it involves compression of the nerve as it passes through a fibrotendinous arcade (Fig. 3) ***"Rengachary , 1996".***

Risk Factors In The Pathogenesis :

1- Inflammatory and autoimmune disorders :-
* Rheumatoid arthritis .
* Dermatomyositis .
* Scleroderma .
* Polymyalgia rheumatica .

2- Increased susceptibility of nerves to pressure :-
* Alcoholic or diabetic polyneuropthy .
* Hereditary neuropathy with liability to pressure palsies .
* Amyloidosis .

* Proximal lesion of the nerve "double crush syndrome"

3- Metabolic disorders : -

* Mucopolysaccharoidoses .

Mucolipidoses . *

Chondrocalcinosis * .

* Gout .

4- Factors unique to women :-

* Pregnancy and lactation .
* Post partum state .
* Menstrial cycles .
* Contraceptive pills .
* Menopause .
* Toxic shock syndrome .
* Eclampsia .

5- Other systemic factors :-

* Myxedema .
* Acromegaly .
* Obesity .
* Raynaud`s disease .
* Hematologic disorder .

6- Space occupying lesions :-

* Lipomas .
* Post traumatic edema .
* Hematomas .
* Abnormalities of the surrounding bones .

"Hudson A, et al. 1982"

Pathophysiology Of Nerve Compression

The pathophysiologic changes following nerve compression are dependent on degree and duration of compression . Lose of nerve function due to compression is manifested clinically by motor paralysis paraesthesia and numbness. Histologic examination of the cross section of a peripheral nerve that has undergone chronic compression shows that the fibers are not affected uniformly . The superficial fibers tend to bear the burnt of compression, whereas the central fibers are relatively spared .
Large diameter, heavily myelinated fibers are more sensitive to compression than are poorly myelinated fibers . Thus, fibers subserving light touch and motor fibers are more likely to be involved in compression neuropathy than are unmyelinated pain fibers . Also, nerve fibers having a greater proportion of nerve fascicles than epineurium are more susceptible to compression than those having scanty nerve fascicles and a large amount of epineurium (Fig. 4) ***"Ochoa, 1980"*.**

The pathophysiologic changes following nerve compression depend upon two critical factors, the degree and duration of compression . The physiologic and structural response tend to be graded according to the severity of these two factors . Mild and brief compression produces a transient conduction block in the nerve, which normalized soon after the pressure is relieved .
There is no major structural changes in the nerve, but the axoplasmic flow is interrupted because of extrinsic pressure .
There is impairment of the centrifugal and axonal flow with blockade of metabolic products and enzymes at the margins of compression . Such impediment to axoplasmic flow is thought to lead to impaired membrane excitability and conduction block .
With chronic compression, segmental demyelination occurs in the compressed segment (Fig. 5) . This accounts for the clinically observed slowing of the conduction velocity of the nerve . In the early phases, the nerve fibers distal to compression show normal morphology . However, but with sustained compression, axolysis occurs in the compressed segment and Wallerian degeneration