# POSTRAUMATIC DOUBLE-CRUSH SYNDROME

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**ABSTRACT**

Patients affected by a preipheral nervous system entrapment, such as TOS or cervical discarthrosis, when suffering a blunt injury will develop a double-crush syndrome.
Fibrotic process will grow through the pathologic area, and will entrape the nerves in the second site.
The treatment of both the problems is needed to solve the clinical picture.

**INTRODUCTION**

The double-crush syndrome was initially described by Upton and McComas in 1973. They postulated that non symptomatic impairment of axoplasmic flow at more than one site, along a nerve, might summate to cause a symptomatic neuropathy. Lundborg has demonstrated that the basic pathophysiology of nerve compression injuries can be related to changes in intraneural microcirculation and to nerve fiber structure: slow-down of blod circulation, and alterations in vascular permeability will cause the formation of edema, and from this hischemia which will grow poportional to the complexity of the intraneural microanatomy.
Disturbances in axonal transport, induced by compression, will evolve in functional and morphological changes in axons and thereby in the nerve cell bodies. The presence of a second, minor, site of compression will give place to a major nerve dysfunction, immediately evident for its clinical manifestation.
Several Authors, after Upton andMcComas, have underlined that the primary crush may be anatomical or methabolic in origin, since a methabolic disease source of nerve compromise will render the nerve more susceptible to compression. MacKinnon and Dellon have expanded the description of the syndrome to include
a)multiple anatomical regions along a peripheral nerve
b) multiple anatomical structures across a peripheral nerve within an anatomic region,
c) entrapments superimposed on a neuropathy, and
d) combinations of the above.

We report here our experience with a series of patients suffering from a traumatic lesions to peripheral nerves which came in superimposition to a former diagnosis of an entrapment neuropaty, or radicular pathology, or methabolic disease.

**PATIENTS AND METHODS**

Out of a series of 271 peripheral nerve traumatic lesions, (1996-2000) we have extrapolated a group of 45 patients ( 16.6%) who presented with a previous diagnosis of neuropathy or methabolic dysfunction.
The distribution of cases is reported in the table:

double crush syndrome: 45 cases

| pre-existing pathology | | nerve injury | |
|---|---|---|---|
| -tos | 24 | ulnar n. in the elbow | 7 |
| | | median n. in the elbow | 6 |
| | | median n. in the wrist | 8 |
| | | median & radial n. in the arm | 3 |
| -cervical radiculopathy | 12 | praclavicular brachial plexus | 12 |
| -diabetes | 6 | ulnar n. in the elbow | 1 |
| | | median nerve in wrist | 2 |
| | | radial nerve in the arm | 1 |
| | | ulnar & median in the arm | 1 |
| | | supraclavicular brachial plexus | 1 |
| -rheumatic disease | 3 | supraclavicular brachial plexus | 1 |

| | | | |
|---|---|---|---|
| | | ulnar nerve in the arm | 1 |
| | | radial nerve in the arm | 1 |

*Table 1: distribution of blunt traumatic lesions in patients with preexisting neuropathy.*

**TREATMENT**

In face of complex clinical pictures, where the patient's complaint was given as a consequence of a blunt injury, we have had problems in the tentative of defining a correct diagnosis and hence offering a complete treatment.

Previous nerve pathology had not been diagnosed in a relevant percentage of cases: only 3 out of 24 TOS were aware of the cause of their recurrent complaints before the injury.
The same consideration is valid for 10 out of 12 cervical radiculopaties.

Blunt injuries to an arm or to the region of the thoracic outlet may appear minor events, with no consequence in the first moments. Symptomatology may develop slowly in the days or in the weeks or even in the months following the event.

This is due to the fibrotisation which will progressively develop.

In these cases we have performed the surgical treatment of the local problem of the peripheral nerve lesion, and successively we have treated the problem of the thoracic outlet syndrome, by microsurgical decompression of the plexus from specific pathology.

Cervical pathology due do discarthrosic problems has been treated by conservative therapy, either conventional or by ozone and vasoactive drugs.

Methabolic diseases have been treated.

**CONCLUSIONS**

We wish to underline the need to treat always both the pathological elements which come to compose the double-crush syndrome, since the two fact come togrther to build a single clinical picture, more severe than the individual one might be by itself.