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The Double Crush Syndrome

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Compressive neuropathies at the level of the wrist and elbow involving the median and ulnar nerves can cause disabling pain, numbness, and weakness in ambulatory and otherwise high-functioning patients.^{1,2} Frequently, patients with symptoms suggestive of compressive neuropathy will also have coexisting pain in the cervical spine region and a diagnosis of cervical radiculopathy (CR) by electrodiagnostic studies (EDS) or clinical examination.^{3,4} Such patients may have double crush syndrome (DCS) with compression of nerve fibers at 2 distinct sites: 1 proximal in the C-spine, and 1 distal in the cubital tunnel, carpal tunnel, or elsewhere. Discerning which area of nerve compression or irritation, proximal or distal, is most responsible for a patient's symptoms is often challenging for the clinician.

A plethora of controversy exists among surgeons regarding the existence of the DCS and the underlying mechanisms that could be responsible for producing it.⁵ Upton and McComas were the first to describe the potential susceptibility of a distal nerve segment in the setting of a more proximal lesion, citing impaired axonal transport as the mechanism.⁶ Other investigators have noted neural ischemia, inherent elastic characteristics of the nerve, systemic diseases such as diabetes and thyroid disease, and chemotherapeutic agents in creating the "first hit" along a nerve that renders its distal segment prone to injury.^{7–9} In 2011, a panel of 17 international experts was convened to determine the most likely mechanisms producing a peripheral nerve insult that would predispose the nerve to the development of another disorder. Fourteen mechanisms were identified through various rounds of surveying, of which 4 were identified as highly plausible. These were impaired axonal transport, ion channel upregulation or downregulation, inflammation in the dorsal root ganglions, and neuroma-in-continuity.⁵

In 1973, Upton and McComas introduced the hypothesis of the DCS, stating that "neural function is impaired because single axons, having been compressed in one region, become susceptible to damage at another site."⁶ Support for this claim came years later in a case series review by Hurst et al, in which an association of cervical spine arthritis and carpal tunnel syndrome (CTS) bilaterality was made, suggesting a link between the two.¹⁰ Baba et al also reported CR that complicated cases of known CTS in a retrospective study.¹¹ Many more investigations have also found an increased incidence of CTS in patients with diagnosed CR compared to the normal population, suggesting that this coexistence is beyond coincidence.^{12,13}

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Despite the association between CR and CTS, the etiology of the DCS and the pathophysiology of this condition are still questioned.⁵ Multiple studies have failed to recognize an electrodiagnostic correlation between patients with isolated CTS and those diagnosed with DCS.³ Furthermore, physical examination findings, such as the Tinel and Phalen signs, have also been shown to be unreliable as the sole method of evaluation in cases of suspected DCS, although they do correlate well with symptoms in patients with isolated CTS diagnosed by EDS.³ In addition, Kwon et al, in 2006, evaluated the severity of CTS based on the level of confirmed CR (C6, C7, C8) with electrophysiologic parameters of median motor and sensory nerves. The electrophysiologic results revealed no significant correlation between median sensory parameters in C6, C7 cases, and no relationship was observed between median motor responses and C8 radiculopathy.⁴

Regarding ulnar nerve dysfunction, the association of a peripheral ulnar neuropathy and CR as another example of a DCS phenomenon has also been suggested.¹⁴ Galarza et al studied a cohort of patients with both EDS-diagnosed cubital tunnel syndrome and CR diagnosed by cervical magnetic resonance imaging findings. Their objective was to determine whether the timing of the surgical procedure, ulnar nerve release versus anterior cervical discectomy and arthrodesis, had an impact on the patient's outcome. This cohort of 24 patients was compared with another group of 20 patients with ulnar nerve compression at the elbow in the absence of cervical spine pathology. The latter group had isolated ulnar nerve decompression at the elbow. In patients diagnosed with DCS—that is, both cubital tunnel syndrome and CR, several factors seemed to favor a poorer outcome without statistical significance: ulnar nerve symptoms present for more than a year, multiple EDS diagnosed neuropathies and radiculopathies, and anterior cervical discectomy and arthrodesis performed before ulnar nerve release. Outcomes were similar and not significantly different compared to the ulnar nerve release cohort, as well.¹⁴ Smith et al investigated a specific population, cyclists, for an increased incidence of DCS as it pertains to ulnar neuropathies. However, these authors diagnosed proximal nerve compression based on clinical examination alone for both CR and thoracic outlet syndrome, stating that EDS provided a low sensitivity and high false-negative rate. This is not supported by current literature, which recommends confirmatory EDS studies as the standard for the diagnosis of cubital tunnel syndrome and CTS.¹⁵ Nonetheless, in 70 cyclists, they discovered a statistically significant higher rate of ulnar nerve symptoms in patients with positive clinical testing for thoracic outlet syndrome and a greater likelihood of neck pain, elevated first rib, and shoulder pain in this group, also suggesting a predilection for DCS.¹⁶

The theory of the DCS has been reported in the literature for approximately 50 years. In that time, many authors have attempted to clearly define its existence and describe its pathogenesis. To this day, controversy exists regarding the DCS theory. Moreover, treatment strategies to improve clinical outcomes in patients with suspected DCS are not clearly defined. Osterman has commented in an earlier investigation of patients with isolated CTS and those with DCS that surgical outcomes of carpal tunnel release were poorer in the DCS group than the isolated CTS group, suggesting that decompression of both sites is required for an optimal outcome.¹⁷ Baba et al also suggested the role of cervical decompression before peripheral nerve decompression or as the sole treatment in the management of DCS when symptoms and functional limitations were more related to cervical spine pathology.⁹

Regarding cubital tunnel syndrome, Galarza et al suggested that anterior cervical discectomy and arthrodesis performed before ulnar nerve release at the elbow lead to less favorable outcomes.¹⁴ At our institution, physical examination findings and symptoms are combined with EDS in the diagnosis and treatment planning of DCS. Neither element of the diagnosis is considered both necessary and sufficient in and of itself. If a diagnosis of DCS is strongly suspected, the lesion with the least invasive surgical treatment option (usually the most peripheral site) is addressed first. This occurs only after patients are counseled that they might not enjoy complete relief from a simple procedure to decompress a peripheral site of compression. This educational process is critical to appropriately manage patient expectations.

Although sufficient evidence is lacking to support a single mechanism producing the DCS phenomenon, professionals must consider multiple causes of a primary nerve insult to better understand dual nerve disorders.³ Controversy remains surrounding the mechanisms responsible for, and even the existence of, the DCS. Patients with multiple complaints related to compressive neuropathies should be evaluated for a more proximal lesion potentially contributing to the patient's presentation. Future research will likely identify those mechanisms responsible for the DCS and treatment strategies to help alleviate patients' symptoms.

References

1. Cranford CS, Ho JY, Kalainov DM, et al. Carpal tunnel syndrome. *J Am Acad Orthop Surg.* 2007; 15(9):537–548. [PubMed: 17761610]
2. Elhassan B, Steinmann SP. Entrapment neuropathy of the ulnar nerve. *J Am Acad Orthop Surg.* 2007; 15(11):672–681. [PubMed: 17989418]
3. Lo SF, Chou LW, Meng NH, et al. Clinical characteristics and electrodiagnostic features in patients with carpal tunnel syndrome, double crush syndrome, and cervical radiculopathy. *Rheumatol Int.* 2012; 32(5):1257–1263. [PubMed: 21259009]
4. Kwon HK, Hwang M, Yoon DW. Frequency and severity of carpal tunnel syndrome according to level of cervical radiculopathy: Double crush syndrome? *Clin Neurophysiol.* 2006; 117(6):1256–1259. [PubMed: 16600675]
5. Schmid AB, Coppieters MW. The double crush syndrome revisited - A Delphi study to reveal current expert views on mechanisms underlying dual nerve disorders. *Man Ther.* 2011; 16(6):557–562. [PubMed: 21646036]
6. Upton AR, McComas AJ. The double crush in nerve entrapment syndromes. *Lancet.* 1973; 2(7825):359–362. [PubMed: 4124532]
7. Lundborg G, Dahlin LB. The pathophysiology of nerve compression. *Hand Clin.* 1992; 8(2):215–227. [PubMed: 1613031]
8. Morgan G, Willbourn AJ. Cervical radiculopathy and coexisting distal entrapment neuropathies: double-crush syndromes? *Neurology.* 1998; 50(1):78–83. [PubMed: 9443461]
9. Hebl JR, Horlocker TT, Pritchard DJ. Diffuse brachial plexopathy after interscalene blockade in patient receiving cisplatin chemotherapy: the pharmacologic double crush syndrome. *Anesth Analg.* 2001; 92(1):249–251. [PubMed: 11133638]
10. Hurst LC, Weissberg D, Carroll RE. The relationship of the double crush to carpal tunnel syndrome (an analysis of 1,000 cases of carpal tunnel syndrome). *J Hand Surg Br.* 1985; 10(2):202–204. [PubMed: 4031604]
11. Baba H, Maezawa Y, Uchida K, et al. Cervical myeloradiculopathy with entrapment neuropathy: a study based on the double-crush concept. *Spinal Cord.* 1998; 36(6):399–404. [PubMed: 9648195]

12. Richardson JK, Forman GM, Riley B. An electrophysiological exploration of the double crush hypothesis. *Muscle Nerve*. 1999; 22(1):71–77. [PubMed: 9883859]
13. Flak M, Durmala J, Czernicki K, et al. Double crush syndrome evaluation in the median nerve in clinical, radiological and electrophysiological examination. *Stud Health Technol Inform*. 2006; 123:435–444. [PubMed: 17108465]
14. Galarza, Gazzeri R, Gazzero G, et al. Cubital tunnel surgery in patients with cervical radiculopathy: double crush syndrome? *Neurosurg Rev*. 2009; 32(4):471–478. [PubMed: 19685252]
15. Keith MW, Masear V, Chung KC, et al. American Academy of Orthopaedic Surgeons Clinical Practice Guideline on diagnosis of carpal tunnel syndrome. *J Bone Joint Surg Am*. 2009; 91(10): 2478–2479. [PubMed: 19797585]
16. Smith TM, Sawyer SF, Sizer PS, et al. The double crush syndrome: a common occurrence in cyclists with ulnar nerve neuropathy—a case-control study. *Clin J Sport Med*. 2008; 18(1):55–61. [PubMed: 18185040]
17. Osterman AL. The double crush syndrome. *Orthop Clin North Am*. 1988; 19(1):147–155. [PubMed: 3275922]

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Learning Objectives

- Recognize the mechanisms responsible for double crush syndrome.
- Establish the clinical diagnosis of double crush syndrome.
- Recognize the role of electrodiagnosis in double crush syndrome.
- Discern the common clinical patterns of double crush syndrome.
- Establish a treatment plan for double crush syndrome.

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