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## General Outline

- Before carpal tunnel syndrome was well understood, there was significant confusion as to pathophysiology that caused the classic sensory (paresthesias) and motor (thenar atrophy) symptoms. Three main ideas are developed to provide an explanation:
  - 1 = “post-traumatic” median nerve injury, usually seen in distal radius fractures with entrapment of the median nerve at the fracture site.
  - 2 = “acroparesthesia” attempted to explain only the sensory aspect of the syndrome with a lesion at the brachial plexus caused by compression of the plexus by cervical ribs.
  - 3 = “thenar neuritis” attempted to explain only the motor findings of thenar atrophy with a lesion at the motor branch of the median nerve at the border of the carpal tunnel.
- Finally, median nerve injury at the site of the carpal tunnel was determined to be the culprit, and surgical techniques emerged to increase

the volume of the canal to relieve pressure on the nerve.

Carpal tunnel syndrome is a now relatively well-understood clinical phenomenon in which neuropathic symptoms are secondary to median nerve compression within the carpal tunnel. However it was not until the mid-1900s that the pathophysiology was sufficiently understood to provide a strong foundation for clinical care. Prior to this understanding, the symptoms of carpal tunnel syndrome were attributed to several different etiologies resulting in several different diagnoses. Three mechanisms were proposed to explain the symptomatology and physical findings: (1) entrapment of the median nerve at the site of an injury, such as the post-traumatic median neuropathy described in distal radius fractures; (2) compression of the lower trunk of the brachial plexus which was invoked to explain the sensory aspect of the syndrome, i.e., acroparesthesia; and (3) a lesion of the motor branch of the median nerve (e.g., thenar neuritis) as it passed beneath the anterior annular ligament of the wrist, which would explain the motor findings of thenar atrophy. It was only after understanding that the pathophysiology was related to compression of the median nerve at the level of the carpal tunnel that effective operative management emerged. The primary goal of operative treatment is to increase the volume of the carpal tunnel by releasing the transverse carpal ligament.

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## Post-traumatic

One of the earliest reports of post-traumatic median neuropathy was described by Gensoul in 1836 [1]. He reported a case of direct injury to the median nerve by entrapment in an open distal radius fracture in an autopsy of a young girl who died of tetanus. Gensoul's description was followed by several others who described median nerve injury following distal radius fracture.

In 1854, Sir James Paget came very close to anticipating our current understanding of carpal tunnel pathophysiology in his *Lectures on Surgical Pathology* [2]. He described a case of median neuropathy that developed after a compression injury at the wrist. He noted that a "cord had been drawn very tight round this man's wrist seven years before the amputation of the arm. At this time it is probable the median and other nerves suffered injury." The second case that Paget described was a median neuropathy that developed after a distal radius fracture and resulted in ulcerations of his radial digits. He reports that these ulcers were "cured only by so binding the wrist that the parts on the palmar aspect being relaxed, the pressure on the nerve was removed. So long as this was done, the ulcers became and remained well; but as soon as the man was allowed to use his hand, the pressure on the nerves was renewed, and the ulceration of the parts supplied by them returned." It is clear through these descriptions that Paget recognized the pathologic effect of compression of the nerve at the wrist, as well as the therapeutic effect of relieving the pressure on the nerve.

In 1933, Abbott and Saunders published their classic cadaveric study that supported the notion that neuropathic symptoms may be due to increased pressure in the carpal tunnel [3, 4]. In this study, they injected dye into the forearm with the wrist held at different positions and described increased fluid resistance with acute flexion and ulnar deviation. The ability to manipulate the volume of the carpal canal and compression of the median nerve with varying wrist positions was an important discovery. This led to their recommendation to immobilize distal radius fractures with a neutral wrist position after fracture reduction,

rather than the established Cotton-Loder position, which maintained the wrist in a flexed position.

This change in treatment was adopted and supported in the following years as more evidence mounted against immobilization with wrist flexion. In 1949, Meadoff recommended prevention of median neuritis after distal radius fracture with closed fracture reduction, neutral wrist immobilization, and surgical exploration of the median nerve with release of the transverse carpal ligament only after 4 months of failed conservative treatment [5].

Lynch and Lipscomb provided further support for this approach in their retrospective review of 600 patients with distal radius fractures followed for 10 years at the Mayo Clinic. In this cohort they found a 3.3% rate of associated carpal tunnel syndrome [6]. The majority of patients that developed carpal tunnel syndrome did so within 3 months of the inciting trauma. Two patients that were initially immobilized in full wrist flexion and ulnar deviation (Cotton-Loder position) immediately developed carpal tunnel syndrome requiring remanipulation of the wrist to a neutral position. Based upon their retrospective review and their surgical experience, these authors recommended conservative management with observation and neutral wrist positioning to treat median nerve symptoms, with surgical decompression reserved for severely symptomatic patients. As an alternative to incising the transverse carpal ligament, they noted the benefit of nonoperative treatment with steroid injections to decrease swelling. In their work, they were undoubtedly influenced by their Mayo colleagues P. S. Hench and E. C. Kendall who were awarded the Nobel Prize in Physiology or Medicine in 1950 for discovering the anti-inflammatory effects of corticosteroids [7].

Subsequent anatomical and physiological studies provided more evidence to support the surgical observations that wrist position was important in carpal tunnel pressures. An anatomical report in 1959 by Tanzier described compression of the median nerve with wrist and digit flexion as the long flexor tendons were displaced anteriorly [8]. In 1984 Gelberman quantitatively confirmed the effect of wrist position on carpal tunnel pressure [9]. Gelberman reported on the carpal tunnel pressures

of 22 patients with distal radius fractures. He found that the pressure increased 2.6-fold from neutral to 40° of wrist flexion and doubled with 20° of wrist extension. He concluded that with Colles' fractures the surgeon should avoid immobilization in significant flexion and that patients with acute carpal tunnel syndrome require operative decompression.

In 1987 Paley and McMurtry described nine case reports of distal radius fractures with volar displacement, eight of which developed acute or subacute median neuropathy [10]. They found the volar fragment 1 cm proximal to the transverse carpal ligament and displaced 1 cm volarly causing compression of the median nerve as it becomes more superficial to run through the carpal tunnel. They recommended treatment with reduction or removal of the volar fragment in addition to carpal tunnel release.

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## Acroparesthesia and Thenar Neuritis

Early reports described the sensory and motor findings of median nerve compression as separate entities: acroparesthesias and thenar neuritis. In the late 1800s, "acroparesthesia" became an accepted diagnosis to describe sensory alterations of the hand in the absence of motor dysfunction [11]. However, it would not be until the mid-1900s that acroparesthesia would be attributed to median nerve dysfunction and become synonymous with carpal tunnel syndrome.

In 1862 Raynaud attributed acroparesthesia to vasomotor dysfunction, although his description of the symptoms could be consistent with carpal tunnel syndrome: "a depressing sense of numbness and tingling... the tactile sense may be so much impaired that it is difficult for the fingers to retain small objects" [12]. In his initial thesis, Raynaud did not differentiate between vasospasm (what we call today Raynaud's disease) and fixed arterial occlusions (i.e., causing Raynaud's phenomenon). Each of these (vasospasm or fixed arterial occlusions) can reduce nutritive flow sufficiently that neurological function can be impaired. In either case, the impaired sensation in the fingers should also be associated with cool-

ness, pallor, and/or cyanosis. Generally the acroparesthesia due to vasospasm is episodic (i.e., precipitated by stress or cold), although there are patients with persistent color changes (e.g., acrocyanosis) and numbness of the fingers in more severe cases of Raynaud's disease.

In Raynaud's era there were descriptions by others, including Gamberini, Romberg, Martin, Putnam, Schultz, and Handfield-Jones, of cases with a constellation of symptoms that we would recognize as idiopathic carpal tunnel syndrome today. In his 1855 case series of six female patients with nocturnal hand paresthesias, Handfield-Jones incorrectly postulated that the culprit was "brachial neuralgia" [11]. Similarly, "cervical rib syndrome," compression of the brachial plexus by a cervical rib, was popularized by Farquhar Buzzard, Physician Extraordinary to His Majesty the King, as a syndrome secondary to C7 nerve root compression leading to both thenar atrophy and sensory changes of the digits. This concept persisted and led to the recommendations of Farquhar Buzzard in 1913 that these patients should be treated with rib resection to relieve pressure on the brachial plexus [11, 13].

While "acroparesthesia" was used to describe the sensory symptoms of median nerve symptoms, James Ramsay Hunt coined the term "median thenar neuritis" in 1909 to denote the neuromuscular manifestations, e.g., thenar weakness and atrophy. He posited that this condition was caused by isolated compression of the motor branch of the median nerve at the border of the transverse carpal ligament [14, 15]. Hunt was followed by several others who described wasting of the thenar muscles. One of which was Harris who in 1926 attributed the cause of compression to work-related activities, like pressing a tool's handle into the palm [14]. In 1944, Barker and Hines would be the first to describe occupational arterial occlusive disease of the hand, associated with pallor, pain, paresthesia, and occasionally ulcerations of the fingers, an entity that can mimic carpal tunnel syndrome [16].

Although Marie and Foix described median nerve compression as the etiology for both sensory and motor dysfunction in 1913, it was not widely accepted until decades later. In 1938, Moersch described a case of spontaneous median

nerve compression, though he suggested that there were two lesions, one at the motor nerve branch and a second at the carpal tunnel [15]. In 1941 Woltman was the first to describe spontaneous median nerve compression by crowding of soft tissue structures in the carpal tunnel [15]. Zabriskie deduced that the sensory and motor findings were likely part of the same pathology, implicating median nerve compression at the transverse carpal ligament [15]. This directed the goal of treatment to relieving carpal tunnel pressure by releasing the transverse carpal ligament. This approach was anticipated in 1913 by Marie and Foix who were among the first to suggest surgical release of the transverse carpal ligament for resolution of symptoms. They were followed shortly by Cannon and Love who published a report of nine patients that had resolution of median neuropathy after surgical resection of the transverse carpal ligament [11, 17].

Despite this work, brachial plexus compression, usually from a cervical rib, was still the most common diagnosis for carpal tunnel syndrome. Finally, in 1947, Brain and Wilkinson published the first paper describing the pathophysiology of spontaneous median nerve compression in the carpal tunnel. They further clarified that the sensory changes and associated thenar atrophy “can be caused only by a median nerve lesion and not a lesion involving the brachial plexus” [15]. Moreover, they proposed treatment with early operative release of the transverse carpal ligament. While Brian is often acknowledged for this landmark paper, Phalen popularized our current understanding of the pathophysiology of carpal tunnel syndrome in several clinical descriptions in the early 1950s [18]. The term “carpal tunnel syndrome” was first used in print by Kremer in 1953.

## Conclusion

Astute observations of early physicians anticipated our current understanding, which was based upon anatomical and physiological studies of the carpal tunnel, and careful surgical series that linked anatomy and physiology to the symp-

tom complex. Once the pathophysiology of carpal tunnel syndrome had been elucidated, improved diagnostic aids and therapeutic approaches were developed. Currently, electromyography and nerve conduction studies may be used to confirm the diagnosis and quantify the severity of the pathology. Metabolic causes of carpal tunnel syndrome (e.g., hypothyroidism) should be considered, and other conditions that can mimic carpal tunnel syndrome including occupational arterial occlusive disease and Raynaud’s disease should be excluded.

With an understanding of the pathobiology, surgical treatment has improved. Herbert Galloway performed the first carpal tunnel release in 1924. Open carpal tunnel ligament release was first described as a 4–5 cm curved longitudinal inter-thenar incision with release of the transverse carpal ligament under direct vision. Several modifications have since been made, including the length, location, and shape of the incision. Endoscopic carpal tunnel release was popularized in 1987 by Okutsu. Modifications to endoscopic techniques include single-portal (Agee) versus two-portal (Chow) surgical techniques. Jimenez published a description of six different endoscopic techniques for the carpal tunnel release. More recently, a Cochrane database study revealed no significant evidence favoring endoscopic release over open release [19]. With respect to conservative treatment strategies, in addition to night splints, other adjunctive strategies include steroid injections and anti-inflammatories.

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