DIGITAL NERVE COMPRESSION

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It is more than intriguing that the most common instance of irritative (compression, traction, friction, impact, and so on) neuropathy in the body, that of the superficial cutaneous nerves of the distal forearm, wrist, and hand, has received the least attention in print and often in practice. After a decade of submitting abstracts about these problems to national meetings with never an acceptance, I finally concluded that no one was interested. Any physicians who use dressings, anesthesia, casts or external fixation should be interested because the majority of these lesions arise from the contact of the body part and the environment, and a good bit of that etiologic environment is provided by the devices that we use and the devices that are placed on us, including every treatment device known, that is placed in or on that portion of the body.

Such knowledge of these conditions, as is passed from one generation of physicians to the next, seems usually to be in the form of test or trick questions about presumably rare entities with exotic names such as Wartenberg's cheiralgia, manacle wrist, bowlers' thumb, "frisbee finger," and so on. From my perspective of 45 years of examining patients and 40 years with a special interest in neuropathies of the hands, I have concluded that (1) nerve lesions of both the open type (lacerating) and the closed types (compression and other types of physical and chemical trauma) are more common in the hands and feet than anywhere else, and (2) irritation or damage of some nerve or nerve element is present to some degree at some time in almost all of the forearm-wrist-hand conditions that we treat with either external apparatus or internal devices; commonly, of course, we use both and change frequently, giving ourselves exponential opportunities to irritate both nerves and patients. I have frequently played a game with myself, of seeing how long it will take to find a nerve problem in a patient being treated for other than a nerve injury, when reviewing patients in a clinic or office. It never takes long!

ANATOMY

There is probably no nerve anatomy in the body that is better known to more people than the usual pattern of innervation in the wrist and hand. From every anatomy text to the color-coded Netter maps in the Ciba publications to the first article of this volume, this information has been disseminated widely and well. It would be unrewarding to reiterate here these well-documented details of the cutaneous nerve distribution in the forearm, wrist, and hand, but it should be useful to discuss the risk factors inherent in the anatomy concerned, as follows:

(1) Tissue depth of the nerve. Even in this area there are significant differences in the superficial placement and the tissue thickness over the nerve. Almost all of the cuta-
neous nerves can be palpated, but some, such as the branches of the dorsal radial and ulnar sensory nerves, can be pinched because they are so close to the surface and so thinly padded by stretchable skin and minimum fat.

(2) Nearness of the nerve to bone, joint, or hard foreign body. If the external trauma is thought of as the "hammer," then a firm, unyielding, tissue "anvil," against which to impact nerve, constitutes another risk factor. The nerves at the side and dorsum of the wrist and hand are at such risk, as are the nerves passing near sesamoids, near protuberant joint margins, or other bony projections and nerves near foreign bodies, tumors, and other masses.

(3) Nerves that are relatively fixed in position with the normal excursion ability and lateral translational ability not present. This can be due to various abnormalities of the normal anatomic restraints: carpal tunnel and Guyon's canal at the proximal palm, fascial septae in the digits, or to anomalies of nerve, muscle, vessel, musculotendinous unit, or fascia. This also applies to nerve end organs, such as Pacinian bodies, which have relatively small patterns of mobility.

(4) Nerve communication. Another anatomic/physiologic fact about nerves is that they are communication systems, which confuse distally by overlap patterns and confuse at all levels by reporting their perception messages (pain and esthesias) from all parts of the system to all parts of the system. It is not safe, therefore, to assume that even the most obvious and most distal lesion is the whole problem without checking the rest of the system.

**HISTOPATHOLOGY**

The pathophysiology of the compressive nerve lesion has been dealt with at length elsewhere in this issue, but it is not likely that the bottom line will have changed from the multiple factor–multiple response reports that have filled the literature for several decades. As indicated in the introduction, the hand is a battleground where even such damaging factors as toxic materials and vibration may be etiologic. The characteristic lesions for digital nerve compression have, so far, been restricted to two. Both are often called "neuroma" in the literature, but it is more probable that the two lesions are hyperplastic responses to irritation than "new growths," ie, "neoplasms," the term "neuroma" may be inappropriate. The terms "compressive" or "entrapment" or "traction" neuropathy are also often used in the literature, but these terms imply nothing directly about the pathology. The more commonly recognized of the two conditions at present is perhaps best termed "perineural fibrosis" and demonstrates a proliferation of perineural fibrous elements, which first surround and separate the neural elements and eventually cause the neural elements to atrophy, close, or disrupt (Fig. 1). The fibrosis, of course, is seldom confined...
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the nerve alone. Neighboring tissues also
participate or may even precede the nerve in
fibrotic response. Frequently affected are the
adjacent fibrous tissues, vessels, subcuta-
nous tissues, and even the skin. The second of the two clinically recognized
entities involves a sensory end-organ, the
Pacinian corpuscle, although tiny branches
digital nerve are also involved. The typical
findings are collections of large Pacinian cor-
cuples with fibrosis around them and unus-
ually prominent and numerous small nerves
coursing between them. These nerves show
dendonural and epineural fibrosis. In some
instances, the hyperplastic Pacinian corpus-
cles develop within the epineurium of the
digital nerves. It is apparent that there
might be considerable difficulty on occasion
in differentiating hyperplastic lesions from
neoplastic lesions such as schwannomas, Pa-
cinian schwannomas, or neurofibromas.
Clinical history coupled with sophisticated
histopathology will usually give a satisfactory
determination.

ETIOLOGY

Many references have already been made
to the almost unlimited sources for irritation
of peripheral nerve elements in the forearm,
wrist, and hand. They can be grouped as
follows: (1) normal or anomalous restriction,
constriction or tethering sites for nerves.
Most of the normal sites are well known
because of the clinical syndromes associated
with them. Anomalous sites are highly vari-
able but have been investigated in many
reports; (2) repetitive irritation; (3) peripheral neuropathy of any cause;
(4) physical or toxic irritation other than com-
pression, impact, or traction, such as chemi-
cals, thermal injury, and vibration injury;
and (5) nearby space-occupying or tissue-alter-
ning lesions, such as tumors, masses, foreign bodies, and scar.

In spite of the ubiquitous nature of hand
use and of hand damage, it is amazing how
little information is processed about the
many ways, other than by open wounding,
that nerves are damaged. One exceedingly
common way is by diagnostic and treatment
methods applied to the hands. Damage by
needles and biopsy tools are open wounds
of a sort, but they are often unrecognized at
the time and present as a closed lesion prob-
lem, until and if an appropriate history is
obtained. Damage by tourniquet, ring, wrap,
bagade, tape, splint (even a simple finger
splint and maintenance materials can be
causative), cast, pad, external fixator, pro-
longed positioning of hand against object or
vice versa—all these and more have been
and continue to be culpable in creating nerve
damage. The level of nerve damage can vary
from an imperceptible irritation, at the clin-
ical level, coming to attention only with ad-
ditional insults, to complete closed disrup-
tion of nerve fascicles, ie, from very minor
neuropraxia to neurotmesis. The timing of
the insult may vary from a quick, single
episode (closed ring injury, for instance) to

Figure 2. Subcutaneous fat from
the area lying between the tissue
displayed in Figure 1 and the skin
of a bowler's thumb. There is an
extensive fibrous reaction. (Photo-
graphed from an H & E slide, re-
duced from x 50; from Dobyns JH:
Bowler's thumb: Diagnosis and
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sion.)
constant repetition of the trauma over days, months, or years.

**DIAGNOSIS**

As in all diagnoses, the first principle is awareness. So available are the hands for review by the patient and for examination by the physician that it is startling how much is overlooked by both parties. Much pathologic information can be obtained by merely doing nerve status examination on patients with other complaints. Of those whose complaints are related to the nerve pathology, most have pain or esthesias (hyp-, hyper-, or par-) as their presenting complaint. Many of these patients have noted a thickening or a mass; in a few instances, the mass may be the only complaint. The mass is usually related to the nerve pathology directly, although there are associations such as ganglia, foreign body granulomas, and tumors of other sorts. If the lesion is not in the terminal branches, the nerve can usually be located proximal and distal to the lesion level, and differential mobility of those segments compared with the lesion level can be demonstrated. Such manipulation of the nerve, or pressure, stretch, percussion, flipping, vibrating, and so on, will often result in tingling or other paresthesias and a Tinel type of radiation. Sensory disturbance is often present to contact, two-point discrimination (2PD), fiber stimulation, or vibration testing and may be severe to the point of anesthesia. Local fibrosis of the skin and subcutaneous tissue is often present; nutritional and sweat changes of the skin distal to the lesion may be noted. Most useful of all is reproduction of the pain by pressure or other stress applied to the nerve at or near the lesion level. Cold, vibration, or other stimulus may also be useful; if a provocative cause can be identified, utilization of this mechanism to reproduce the symptoms is also confirmatory.

Differential diagnosis includes the following: (1) All causes of masses and tumors in the hand; on occasion, some of these may coexist with the nerve lesion. (2) Peripheral neuropathy from any cause; this may also coexist with the lesion. (3) More proximal nerve lesions. These may coexist with the digital lesions but are more likely to be producing the symptoms themselves. It is particularly common for neuropathy of the radial sensory nerves at the wrist or the dorsal sensory branch of the ulnar nerve to mimic digital neuropathy, because they do normally supply the dorsum of the hand and the dorsum of digits 1 and 5, thumb, and little finger, as far as the dorsal tip and sometimes part of the palmar aspect and sometimes part of the palmar aspect. (4) Muscle compartment syndromes in the hand or tissue compartment syndromes in the digits. Classic presentations give no problems, but the early or mild compartment syndrome with its vague discomfort and mild firmness may suggest a nerve problem. (5) Vascular thrombosis, particularly of a vessel accompanying a nerve in the hand or digit, will often give similar symptoms because of irritation of the adjacent nerve; lesions of both structures may exist together. (6) Tenosynovitis, of the flexor tendons in particular, but sometimes even joint synovitis, may irritate nearby nerves enough to produce similar symptoms.

**CLINICAL MANIFESTATIONS**

Interestingly, the best known instance of digital nerve compression to the majority of physicians is not in the hand but in the foot, and it is called Morton's neuroma. It too is almost certainly a repetitive insult and device-related lesion, the device in question being the shoe. Because there is an anatomic predisposition risk factor, Morton's neuroma might develop even in a barefoot population, but I have never heard of it. In the shoe-wearing population it is all too common, as might be expected of an organ whose complaints are studiously ignored, whose support device, the shoe, is often poorly fitting, and whose functional requirements are prolonged and invariably bilateral. The plantar nerve or nerves involved are tethered by their anatomy and abused by their neighbors, the metatarsal heads. The pathology is identical to that described in this article, ie, perineural and endoneural fibrosis resulting in atrophy, then disruption of nerve fascicles. In the hand the most commonly reported lesion is that of bowler's thumb, although there have been miscellaneous reports about a number of other similar lesions, such as frisbee finger, jeweler's thumb, cheerleader's hand, harpist's hand, guitar finger, pen pusher's paresthesia, batter's thumb, tennis player's finger, and so on. From my experience, I can add the following to this list: violinist's finger, pipettist's pulp.
surgery to mimic a normal finger normally used and the use of the right hand, and little or no swelling. Sometimes
(4) Muscle and/or tissue
around the digits, causing problems, but
(5) Vascular compromise accom-
panied by swelling of the digit, will
cause of irritation of both the ulnar and radial surfaces of the digit.
Tenosynovitis, a common, but
insignificant, complaint may irritate the median nerve and produce similar
trouble.

As mentioned many times before, the most
common group of forearm-wrist-hand neuropathies that I have encountered have been
in patients being treated for some condition
in those areas. Other contributors to this
issue discuss those problems associated with irritation of the forearm and wrist and dorsal hand sensory nerves, which are the most
frequently seen in my experience. I will con-
tent myself with giving the data on three
instances in which digital nerves were in-
volved.

Case 1 in this group was a 54-year-old woman
with a comminuted Colles fracture, reduced else-
where and treated with a snugly fitting cast,
which was molded around the index metacarpal and head to maintain an ulnar deviation of the wrist and hand. At 2 days after reduction, there
was mild swelling and moderate pain. One of the
pain sites was along the molded area around the
index metacarpal, and it was further noted that she
had tingling and numbness in the distribution of
both the radial palmar sensory nerve and the
dorsal radial sensory nerve to the index finger.

With fingertrap traction and light countertraction
applied, the cast was removed and a new, more
pliable cast applied with plaster only to the
midlevel of the index metacarpal and without so
much molding. Luckily, position was maintained,
pain and swelling were controlled, and fracture
healing occurred. The recovering cutaneous
nerves went through a period of hyperesthesia,
with special treatment measures required for a
few weeks, but recovery was complete in 3
months.

Case 2 of this group was a 60-year-old woman
who had a giant cell tumor of tendon sheath
removed from the distal portion of a finger with
palmar digital nerve block for anesthesia and a
rubber glove ring at the base of the digit for
tourniquet (in place about 45 minutes). She com-
plained of numbness and tingling in the distri-
bution of the digital nerve opposite to the side of
the tumor, when sensation returned the following
day. No treatment was required, but a Tinel's sign
developed at the site of the tourniquet and slowly
moved distally over the next few months.

Case 3 of this group was a 22-year-old man who
was seen about 1 week after a PIP dislocation had
been reduced and a snug dorsal, digit-based,
montaricul splint had been applied to the proxi-
mal phalanx area, but extending out over the PIP
tumor, when sensation returned the following
day. No treatment was required, but a Tinel's sign
developed at the site of the tourniquet and slowly
moved distally over the next few months.

The final group of cases will consist of
occupational causes of digital neuropathy.

Case 1 of this group was a 58-year-old gyneco-
logic surgeon who had numbness and tingling of
all fingers of his nondominant hand, present for about 2 years and increasing to the point that it was interfering with his surgery (he liked to retract for himself and held a retractor tightly in that hand for most of the case). He had a mild generalized peripheral neuropathy, but there were localizing signs at the digital nerves that held the retractor. So it was believed that the many years of retractor holding represented the principal cause of neuropathy. With some difficulty, he was persuaded to allow an assistant to hold the retractor and he had improved when seen 1 year later.

Case 2 of this group was a dental hygienist who began to note pain and numbness in several digits, but particularly the index finger of her left hand, which was the holder for a new vibrating tool that she had begun to use about 18 months previously. She had pulp tenderness in several fingers, along with hypesthesia and paresthesias in the distal half of the index finger. She stopped using the vibrating tool and returned to manual instrumentation, but she was unable to continue full-time dental-hygiene work, even though she improved. She later sued the dental instrument company and was awaiting a worker's compensation appeal.

Case 3 in this group was a 28-year-old woman who was first violin and assistant conductor of a municipal symphony orchestra. In addition to her performance duties, she practiced about 6 hours per day and had done so for about 20 years. She was finding it increasingly difficult to practice and to play, however, because of a painful mass at the tip of the ring finger of her fingering hand. She had first ignored it, then used a small bandage, similar to a Dr. Scholl's bunion pad, over it. However, the mass continued to increase in size and discomfort and was interfering badly with her technique. The mass was subcutaneous, very near the tip of the pad, and her pain was reproduced exactly when pressure was applied. She did not feel able to alter her work or schedule, and no protection could be devised that did not interfere with her performance technique. Treatment is presented in the next section.

TREATMENT

The most effective treatment is prevention. Next is protection and modification or even cessation of the provocative activity. Surgery is useful in some instances, but it is a sign of failure because it means the diagnosis was delayed too long or that protective treatment has been inadequate, usually owing to non-compliance. Because many of the problems discussed here are iatrogenic, it is obvious that greater care in the use of positioning, the application of external materials, and the post-treatment counseling of patients is required. Knowledge of anatomy, materials, and techniques are all important, but probably most important is ensuring that the patient is so comfortable in the bandage or support that little or no pain medication is required. If such medication is required, vigilance must be redoubled. In occupational and avocational risk areas, it also should be possible to anticipate and avoid the problems, if all who are involved are alert for risk situations. In retrospect, it is nearly always quite obvious what should have been avoided. So many of the digital compression lesions are of the repetitive stress variety that the usual rules for controlling repetitive stress lesions apply, ie, modify or stop the aggravating activity. The effect of both prevention and protection has been seen in bowling, not because of any sustained, urgent, industry-wide action but simply because the problem is well known to all concerned and the measures to avoid it are practiced with fair consistency. These measures consist of stopping the provocative activity until symptoms improve. Then, a graduated return to bowling is attempted with preliminary readjustment of the thumb insert hole or an alteration of other features of the technique, such as digit insert depth or number, hole placement, ball weight, and so on. For other activities that may also aggravate the damage area, the use of a digit-based, short, thumb shell (from carpometacarpal to interphalangeal levels) is adequate protection. If symptoms cannot be controlled in these ways, complete cessation of the provocative activities and/or surgery are to be considered.

SURGICAL TREATMENT

As already indicated, surgery is usually a last resort, if the diagnosis is known with near certainty. A good history of either significant single or repetitive episode trauma plus the findings of nerve symptoms, nerve dysfunction, and findings suggestive of a localized nerve lesion is not always obtained. The hand is the locale of so many injuries and so many masses and tumors, some of which are of life-threatening import, that surgical examination and biopsy may be the only foolproof way of establishing a certain diagnosis. Furthermore, granulomas, ganglions, and other masses may be found in

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Figure 3. Surgical view of the enlarged, thickened, elongated ulnar digital nerve of a bowler's thumb after mobilization and before transposition. The normal-sized radial digital nerve is also displayed, lying in the curve of the dental probe. (From Dobyns JH: Bowler's thumb: Diagnosis and treatment. J Bone Joint Surg 54A:752, 754, 1972; with permission.)

an epineural release may be considered if the intraneural scar seems very firm. Electrical conductivity testing at the table may well be the best method of selecting nerves for any type of internal lysis, but the crude instrumentation traditionally available has led me to choose the old technique of subepineural injection, using a No. 30 needle and Tissu-sol as the liquid. Where internal resistance was easily overcome by gentle fluid pressure, nothing further was done; where fluid block occurred, epineural incision was done. Once released from scar, including excision of any associated masses/tumors, the nerve is somewhat, often much, longer than previously, presumably owing to stretch and rebuilding of the structural units. A good example of this is case 1 of the "bowler's thumb" group discussed also in this article. The surgical view (Fig. 3) of the damaged nerve and the normal paired digital nerve of the thumb shows the impressive differences. The thicker and longer ulnar digital nerve of that thumb, once mobilized, would have lain in serpentine loops on the scarred bed from which it was freed. It is considered better to transpose such nerves to the most normal tissue plane available. In this case that was the adjacent web and ulnar...
midlateral aspect of the thumb, maintaining the new position by fascial flaps obtained from the thenar fascia and the adductor aponeurosis. In other cases it has been more feasible to place the damaged nerve in the same compartment as the other neurovascular bundle in the digit. In some areas such as the distal palm, lumbricals or other muscles are available to be transferred over the nerve segments. The ingenious surgeon can nearly always find a site to which to transpose the nerve and a method by which to maintain it.

As previously noted, excision is occasionally the preferred treatment, particularly when the lesion is in terminal branches or involves Pacinian corpuscle hyperplasia. In case 3 in the occupational neuropathy group, the violinist/conductor had such a lesion, i.e., a combination of terminal branch nerve hyperplasia and Pacinian body hyperplasia at exactly the spot where finger tip met violin string. The mass was excised and she did lose sensibility in that area, 10 mm in diameter, but gradually diminishing to half of that. The numbness and the eventual callus in the area were somewhat bothersome also, but not nearly as much as the prior lesion. At some 10 years postoperatively, she was still pursuing her profession, although in a teaching capacity.

Results, in general, have been good with both closed and open treatment of digital nerve compression lesions, except in those few in which “pain-dysfunction syndromes” developed. Even in these, the neuroma pain was nearly always controlled; other aspects of these syndromes seemed responsible for the continued problems. Nevertheless, there are often permanent residua, usually in terms of sensitivity to the provocative activity or similar activities. The bowler described in this article did not return to bowling, but others did, knowing that it involved a risk. The violinist continued the provocative activity but did modify her activities to diminish the amount of provocation. Only those persons in whom the diagnosis is made very early and adequate protection and relief are obtained immediately seem to recover without residual sensitivity to similar abuse. Little can be done about the incidence of single-episode accidental injury to digital nerves, except to point out the risk element in such activities as opening soft-drink-can opening rings (the consumer should be wary, but the manufacturer should be induced to change the device). There is much that professionals can do in medicine and in trade and special interest journals to alert each other to the dangers inherent in the use of certain devices and certain techniques, particularly medical treatment techniques, and particularly techniques permitting or requiring repetitive compression, traction, or impact. I renew my offer to act as a clearinghouse for information about the clinical incidence of distal upper limb compression neuropathy. I am certain that the reported incidence of such problems is but the tip of a very large iceberg.

**SUMMARY**

A review of the relatively small body of literature about digital nerve compression injuries has been supplemented by the author’s experience and interest of over 40 years. The principal lesions involved are of two types: (1) fibrosis about, around, and in the nerve; and (2) reactive hyperplasia of the nerve support elements and sometimes of special end organs, Pacinian corpuscles, and even of the nerve fibers themselves. Nerve signs and symptoms often accompanied by a mass in chronic cases are diagnostic. The most important features of the topic are the recognition by all of the risk elements, embodied in many personal and professional devices and techniques. These risk elements are present in all parts of our society, including home and occupational, avocational, and medical situations. Treatment is best provided by awareness of risk and avoidance of risk activities, particularly repetitive risk activities. Surgery is preferably avoided, but when indicated it is effective. It consists principally of lysis and protective positioning of the damaged nerve or, on occasion, of excision of terminal or end-organ lesions. More information must be gathered about these exceedingly common but notably under-reported problems.

**References**

Ann obvious occupational problems involving the upper extremity, and in particular the hand and forearm, are of over 40 million employed persons. The overuse injuries described are of a mechanical nature and, in general, are associated with overuse and overloading of the muscles, tendons, nerves, and joints. These repetitive trauma injuries are called overuse injuries and are emphasized here in the context of occupational practices, particularly technical repetitive trauma injuries. Thus, it is the overuse exposure that causes the injury and not the occupational exposure itself. The repetitive nature of the overuse injury makes it a common problem in the industrial setting. The repetitive trauma injuries are usually caused by activities which are performed repetitively, such as typing, repetitive lifting, or repetitive motion. The repetitive nature of these activities results in cumulative trauma injuries, which are injuries that occur over time due to the repetitive nature of the activity. These injuries are often referred to as cumulative overuse injuries. They may affect any part of the body, but they are particularly common in the upper extremity. The repetitive trauma injuries are often associated with cumulative trauma disorders, which are a group of musculoskeletal disorders that are caused by repetitive stress on the body. They are characterized by pain, swelling, and weakness in the affected area. The cumulative trauma disorders include conditions such as carpal tunnel syndrome, tendinitis, and muscle strains. The repetitive trauma injuries and cumulative trauma disorders are often caused by activities that require repetitive motion or repetitive lifting. These activities may occur in a variety of settings, including the workplace, home, and leisure activities. The repetitive trauma injuries and cumulative trauma disorders are often difficult to treat, as they may require a combination of rest, medication, physical therapy, and surgery. The repetitive trauma injuries and cumulative trauma disorders are a significant problem in the workplace, and they can result in significant disability and decreased productivity. The repetitive trauma injuries and cumulative trauma disorders are often under-