Occupation neuritis of the deep palmar branch of the ulnar nerve : a well defined clinical type of professional palsy of the hand / by J. Ramsay Hunt.

OCCUPATION NEURITIS OF THE DEEP PALMAR BRANCH OF THE ULNAR NERVE

A Well Defined Clinical Type of Professional Palsy of the Hand[1]

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Under the heading "Occupation Neuritis of the Deep Palmar Branch of the Ulnar Nerve," I shall describe a group of cases, which present the following clinical characteristics: An atrophic paralysis of all the intrinsic muscles of the hand innervated by the ulnar nerve; the electrical reactions of degeneration; no objective sensory disturbances in the ulnar nerve distribution. The absence of sensory symptoms and the sharp limitation of the paralysis, atrophy and electrical changes to the muscles of the hand supplied by the ulnar nerve, distinguish this type from all other occupation palsies of the hand hitherto described. The clinical importance of these cases is very much augmented by reason of the absence of objective sensory disturbances, and the consequent resemblance to the Aran-Duchenne type of muscular atrophies of spinal origin. A resemblance which may be still further accentuated by the not infrequent association of progressive muscular atrophy with occupations requiring exces-

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sive use of the hands. Before proceeding to a detailed description of the syndrome, I shall refer briefly to various types of occupation affections of the upper extremities, and more especially of the hand, which are already recognized by systematic writers.

1. A neuritic form, due to stretching or compression of the nerve trunks, or over-activity in a neuro-muscular distribution. The ulnar and median nerves are especially liable and one or both nerves may be involved in some part of their course. The presence of toxic substances in the body, such as alcohol, may act as predisposing factors, thus rendering the nerve elements more vulnerable (toxico-professional palsies). This type is characterized by the neural distribution of the atrophy, and by the presence of subjective and objective sensory disturbances; as cramps, pains, paresthesias, and anesthesias.

2. A pure myopathic form, due to myositis or to pressure atrophy of the muscles.

3. A combination of the neural and myopathic forms.

4. A spinal form may also be mentioned. This is an early localization of progressive muscular atrophy in the intrinsic muscles of the hand, and is characterized by a progressive tendency, fibrillary twitchings, and the absence of sensory disturbances.

5. A special type of occupation atrophy of the hand has been described by Gessler. In this form the terminals of the intermuscular nerves and their motor end-plates are thought to be involved; frequent muscular contractions without sufficient relaxation inducing an anemia of the parts with consequent nutritional disturbances in the motor nerve endings.

In Gessler's type, as in the one which I describe, there are no objective disturbances of sensation; but his cases according to his description are unlike mine, in that the atrophies and paralyses of the intrinsic muscles of the hand are not limited to the ulnar nerve distribution. The etiological factor in the group of cases which I desire to isolate is a compression neuritis of the deep volar branch of the ulnar nerve, as it passes between the tendinous origins of two muscles of the hypothenar eminence, the adductor and the flexor brevis minimi digiti (Fig. 1).

Anatomical considerations.—I would here briefly refer to the distribution and the relations of the ulnar nerve in the hand.

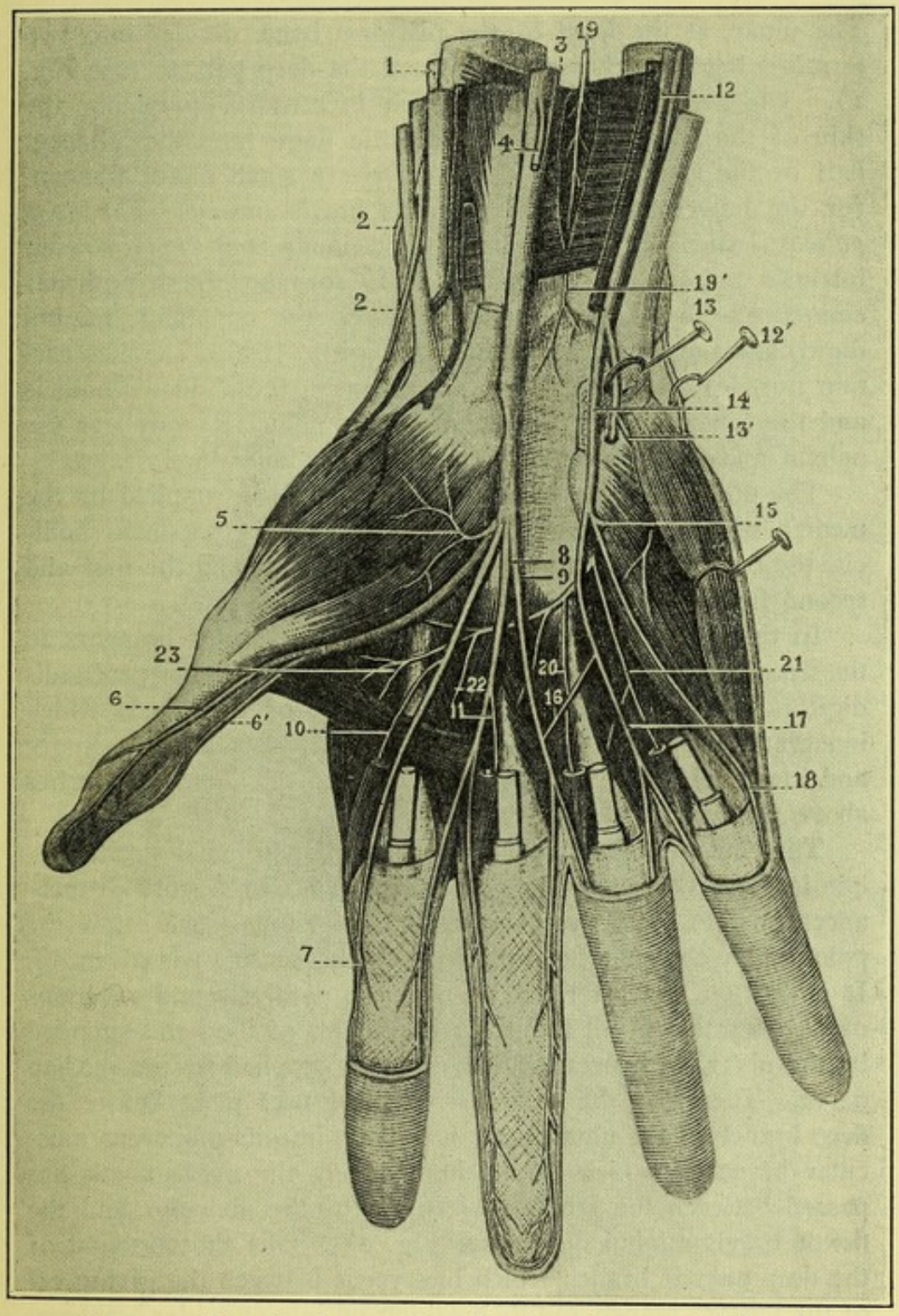

FIG. 1. (Testut's Anatomy.) Showing the superficial and deep palmar branches of the ulnar nerve. 12, ulnar nerve; 12′, dorsal cutaneous branch; 13, deep palmar branch; 13′, a branch to the hypothenar; 14, superficial palmar branch; 15, nerve of the palmaris brevis; 16, anastomosis with the median; 17 digital branch; 18, digital branch; 20, nerve to the third lumbricalis; 21, nerve to the fourth lumbricoids; 22, branch to the interossei; 23, branch to the adductor of the thumb.

The ulnar, at the level of the pisiform bone, divides into two terminal brances—the superficial and the deep palmar (see Fig. 1). The superficial palmar branch is sensory, supplying the skin of the palmar aspect of the little finger and the adjacent half of the ring finger. It also carries a small motor filament for the innervation of the palmaris brevis muscle. *The deep palmar branch is purely motor,* and innervates the following intrinsic muscles of the hand: those forming the hypothenar eminence—the abductor minimi digiti, the opponens minimi digiti, and the flexor brevis minimi digiti. It also supplies certain muscles of the thenar region, namely, the abductor pollicis and the inner head of the flexor brevis pollicis; as well as the palmar and dorsal inter-ossei, and the two inner lumbricales.

The other intrinsic muscles of the hand are supplied by the median nerve. These are the abductor pollicis, opponens pollicis, the outer head of the flexor brevis pollicis, and the first and second lumbricales.

In the upper forearm the ulnar sends muscular branches to the flexor carpi ulnaris and the inner half of the flexor profundis digitorum. The dorsal cutaneous branch of the ulnar, which innervates the skin of the posterior surface of the little finger and inner half of the ring finger, is given off about two inches above the wrist.

The clinical type of occupation neuritis which I desire to isolate is characterized by the absence of objective sensory disturbances, so that the nerve compression must take place below the point where the superficial palmar branch (sensory) is given off. It is further characterized by atrophic paralysis and reactions of degeneration in all the intrinsic muscles of the hand supplied by the ulnar, with preservation of those supplied by the median nerve. Therefore the compression must take place before the deep branch of the ulnar nerve breaks up into its numerous muscular branches, which begins immediately the nerve trunk has passed between the tendons of origin of the abductor and the flexor brevis minimi digiti (see Fig. 2). This short section of the deep palmar branch which intervenes between the giving off of the sensory branch and its breaking up into muscular branches, represents the seat of the compression.

This short section of nerve lies on the outer side of the pisiform bone and passes downward, backward, and outward, wind-

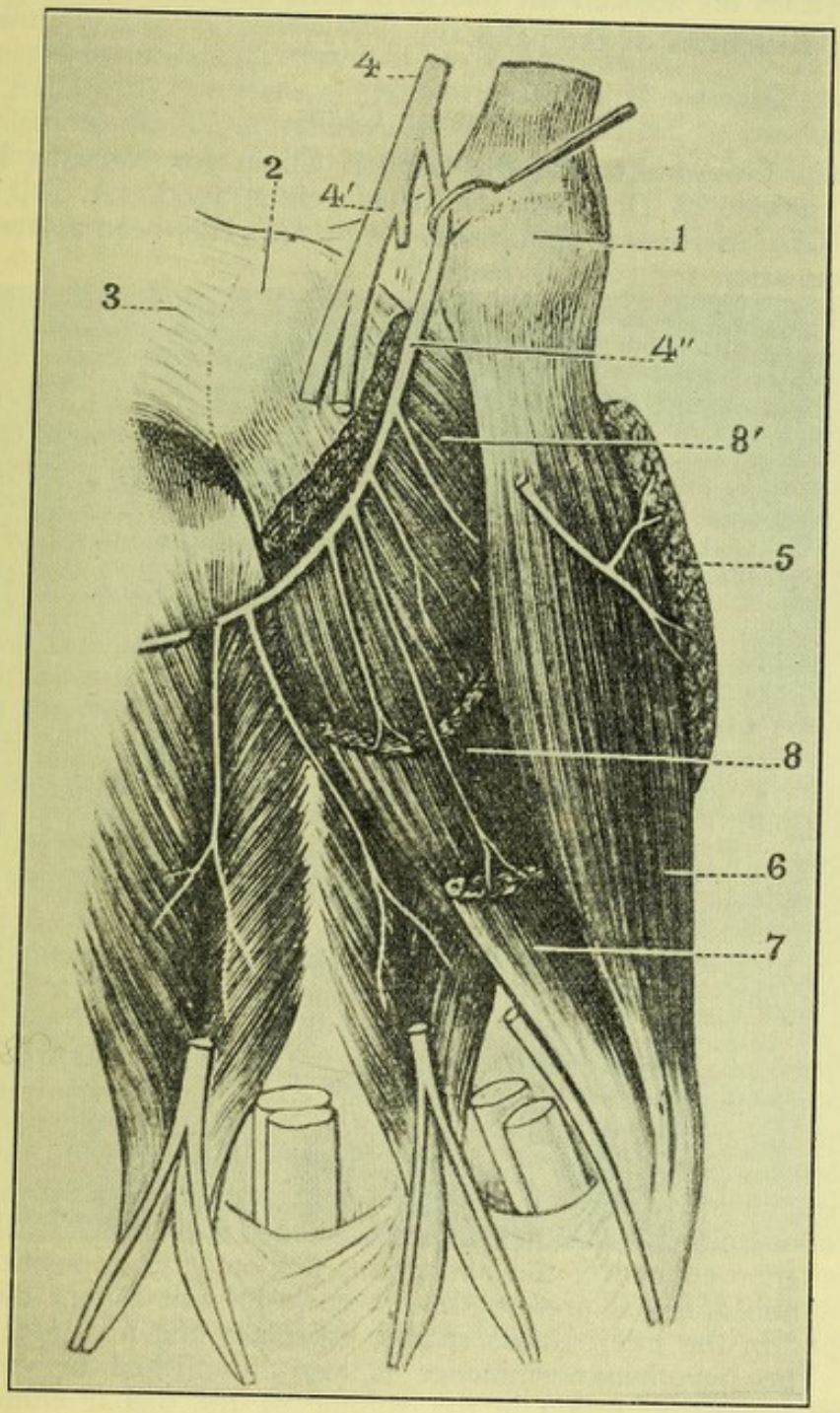

FIG. 2. (Testut's Anatomy.) The deep palmar branch of the ulnar and its relations to the abductor and flexor brevis minimi digiti. 1, pisiform bone; 2, unciform process of the unciform; 3, anterior annular ligament; 4, ulnar nerve; 4′, superficial branch; 4″ deep palmar branch; 5, palmaris brevis muscle; 6, abductor minimi digiti; 7, Flexor brevis minimi digiti; 8, 8′, Opponens minimi digiti.

ing beneath the hook of the unciform bone and thus reaching the deeper structures of the palm.

Reports of Cases

Case I.—Admitted to the Cornell Clinic for Nervous Diseases, service of Dr. C. L. Dana, March 2, 1906. A. R., Hebrew, born in Russia, unmarried. Age 24. Has been a jeweler by occupation for several years.

He is not addicted to the use of alcohol, and enjoys good general health. Lues is denied. In his work as jeweler, the object upon which he is working is held firmly in the left hand, and is grasped chiefly by the thumb and fingers. The filing process is performed by the right hand. He works from eight to ten hours a day, and experienced no inconvenience until about four months before he came under observation. At that time he observed a weakness and awkwardness in the movements of the fingers and thumb of the left hand, which was soon followed by distinct evidences of muscular wasting in the interosseous spaces. The atrophy was especially noticeable in the first interosseous space. He had no pain in the hand at the time of onset and has had none since, nor have paresthesias been present at any time. The weakness and consequent disability were of such a character that he was forced to abandon his occupation, although retaining a good grasping power in the hand.

Status Præsens.—On attempting to straighten out the left hand, it is found that the fingers cannot be fully extended, but remain slightly flexed. This tendency to flexion is more apparent in the little and ring fingers, although slightly present in the middle finger. The little finger is held somewhat abducted. On testing the various movements of the fingers, it is found that the basal phalanges cannot be flexed upon the metacarpal bones while the fingers are held extended. Abduction and adduction of the fingers are practically abolished. The movements of abduction and apposition of the little finger, and the adduction of the thumb are also abolished. In contrast to the paralysis of the adductor pollicis, the interossei, and the muscles of the hypothenar eminence, the abduction, flexion, and apposition of the thumb are well preserved. There is present a very distinct atrophy in the interosseous spaces, especially the first (see Fig. 3). The hypothenar eminence is also diminished in volume. There is no diminution in the volume of the thenar eminence. No fibrillary twitchings. The movements of the wrist are well preserved, the ulnar flexion of the wrist showing no impairment. The common extensors of the thumb and fingers are also well preserved, as are the deep and superficial flexors. The dynamometer on the right registers 50; on the left 40. There is no tenderness along the nerve trunks of the upper extremity, and

no vaso-motor disturbances. *The touch, pain and thermic sensibility of the left hand and forearm is perfectly normal.* The general neurological examination was negative.

Electrical Reactions.—Strong faradic and galvanic currents applied to the ulnar nerve at the elbow failed to produce any muscular response in the intrinsic muscles of the hand supplied by this nerve; a good contraction, however, is elicited in the flexor carpi ulnaris and the ulnar portion of the flexor profundus

FIG. 3. Case I. Occupation neuritis of the deep palmar branch of the ulnar nerve, showing atrophy of the intrinsic muscles of the left hand.

digitorum. The faradic current directly applied to the intrinsic muscles of the hand, produces a slow and sluggish contraction in the muscles of the hypothenar eminence and the interosseous muscles. The direct galvanic current produces a slow vermicular response with reversal of the normal electrical formula in the muscles of the hypothenar eminence and the interossei. (Note: Qualitative changes were also present in the

muscles of the thenar eminence. These changes, however, were referable to the deeper muscles of this region supplied by the ulnar nerve, the abductor pollicis, and the inner head of the flexor brevis pollicis.) Faradic and galvanic currents, when applied to the median nerve at the elbow and at the wrist produce good contraction in the muscles of the thenar eminence corresponding to the distribution of this nerve.

June 11, 1906. The hand is free from pain or paresthesias, no fibrillations. Strong galvanic and faradic currents to the ulnar nerve at the elbow still fail to elicit any contraction in the intrinsic muscles of the hand supplied by this nerve. Strong faradic currents applied directly to the intrinsic muscles of the hand, produce only a sluggish response, and the galvanic response is still vermicular, with the CACLC = ACLC.

December 31, 1906. The hand is much stronger, the atrophy is less marked, and the inter-osseous spaces have filled out; at no time since the last note has he had any pain or paresthesias in the hand. The grip is practically as strong on the left as on the right side. He says that the hand now feels strong and well, and he has resumed his old occupation.

Electrical Reactions.—The intrinsic muscles of the left hand now respond both to the faradic and galvanic currents when applied to the ulnar nerve at the groove of the elbow. Strong faradic and galvanic currents are required to produce good contractions in the intrinsic muscles of the hand, but apart from this the response is normal in character and shows no qualitative changes.

Case II.—Admitted to the Cornell Clinic for Nervous Diseases, August, 1901, service of Dr. C. L. Dana. H. St. P. An Italian aged 24, married, and a machinist by occupation. Denies lues and is moderate in the use of alcohol. Had typhoid fever in March, 1901. Is of a nervous and excitable temperament, but enjoys good general health. On August 1, 1901, he commenced work with a machine, which necessitated his grasping a handle firmly with the right hand, and at the same time making some pressure against it. This occupation not only required constant flexion and relaxation of the hand and fingers, but there was also associated a pressure against the deeper structures of the palm. He worked nine hours on the first day. At the end of the second day he noticed a weakness of the right hand, but he has continued his occupation up to the present time, although with difficulty. There has been during this period, occasional sensation of pins and needles in the tips of all the fingers, and toward the end of the day, a dull aching pain in the whole right arm.

Status Præsens, August 30, 1901.—On holding out the right hand, a slight tremor in the hand and fingers becomes apparent. In the interosseous spaces there are distinct signs of wasting,

which is more conspicuous in the first interosseous space. The muscles of the forearm and upper arm are normal in appearance and functions. There is a complete paralysis of the muscles of the hand supplied by the ulnar nerve, *i. e.*, the hypothenar, the interossei, and the adductor pollicis. The functions of the muscles of the thenar eminence, which are supplied by the median nerve are well preserved. The dynamometer on the right registers 40, on the left 50. There is no tenderness of the nerve trunks or of the muscles, and *the sensations of touch, pain and temperature of the hand are perfectly normal.* The general neurological examination was negative.

Electrical Examination.—Faradic and galvanic stimulation of the ulnar nerve at the elbow induces a good contraction in the flexor carpi ulnaris, and the ulnar portion of the flexor profundus digitorum, but no response in the intrinsic muscles of the hand supplied by this nerve. Faradic and galvanic stimulation of the median nerve at the bend of the elbow produces a good contraction in the flexors of the wrist and fingers, as well as in the muscles of the thenar eminence supplied by this nerve. The direct faradic excitability of the hypothenar, the interossei, and the adductor pollicis is completely abolished, and the galvanic current in the same muscles produces a slow vermicular response with reversal of the poles. (Note: The patient did not return to the clinic and the subsequent course of the case is unknown.)

CASE III.—Admitted to the Cornell Clinic for Nervous Disease, April 17, 1902. Service of Dr. C. L. Dana. J. M., single, 38 years of age, a brass polisher by occupation. Denies lues; is fairly moderate in the use of alcohol, with occasional excesses. Three weeks before he came under observation, the right hand commenced to feel weak and awkward, and at times numb. It was found impossible to straighten out the little and ring fingers; there was no history of pain, and no tenderness of nerve trunks or muscles; *the sensation of the hand is undisturbed.* As in the previous cases, there was present a paralysis of the intrinsic muscles of the hand supplied by the ulnar nerve, with complete reactions of degeneration. (Note: This case also passed from observation, and the subsequent course is unknown.)

Remarks.—The symptoms in all three cases just described are practically the same. All have a common etiological factor, an occupation requiring flexion or grasping movements of the hand. One hand only was affected, and the weakness made its apearance without pain or cramps. In cases I and III the onset was apparently insidious, while in case II the symptoms made their appearance on the second day. Case I terminated in recovery; cases II and III passed from observation, and the eventual outcome is unknown. Careful tests were made of the objective sensibility of the hand in all of the cases, and it was found perfectly normal. The paralysis was sharply limited to

the intrinsic muscles of the hand supplied by the ulnar nerve. In these muscles the typical electrical reactions of degeneration were present. The flexor and extensor muscles of the forearm were normal, both in their voluntary innervation and electrical responses, as were also the muscles of the thenar eminence supplied by the median nerve. A characteristic attitude of the hand resulted when the attempt was made to extend the hand and fingers. This consisted in a persistence of slight flexion of all the fingers, but more especially of the little and ring fingers, the little finger assuming the position of abduction. The atrophy in case I was quite marked, and was also evident in a lesser degree in cases II and III which were more recent.

The clinical picture just described must be attributed to a lesion of the ulnar nerve; and from the complete absence of objective sensory symptoms the site of lesion must be placed below the giving-off of the superficial palmar branch, which conveys sensory fibers to the palmar surface of the fingers. While it is true that in pressure lesions of the peripheral nerves, motor fibers are more vulnerable than sensory, it is inconceivable that motor fibers alone should suffer without any objective evidences of sensory involvement. I would also emphasize the fact that not one muscle or one group of muscles was paralyzed, but that all the muscles supplied by the deep palmar branch of the ulnar were involved. Therefore the lesion of the deep palmar branch must have taken place before this nerve breaks up into its various muscular branches which begins immediately after this nerve has passed between the muscles of the hypothenar eminence.

These muscles of the hypothenar which I believe are chiefly concerned in the compression of the nerve trunk and consequently in the production of the lesion are: the flexor brevis minimi digiti, and the abductor minimi digiti. The short flexor takes its origin at the tip of the unciform process of the unciform bone, and from the anterior surface of the annular ligament. It is inserted into the base of the first phalanx. The abductor minimi digiti takes its origin from the pisiform bone and is inserted into the outer side of the base of the first phalanx. The deep volar or palmar branch of the ulnar nerve, according to some authorities, passes between the origins of these two muscles; according to others, it traverses the flexor brevis minimi digiti muscle just below its point of origin. Testut describes the course of this branch of the ulnar nerve as follows: "Aris-

ing at the external surface of the pisiform bone, sometimes a little higher, it is directed obliquely downward, backward and outward, traversing the insertion of the flexor brevis minimi digiti; passing beneath the unciform process and then reaching the deep palmar region." It would seem to me probable that the essential factor in the production of this form of neuritis, is one of muscular contraction or pinching as the nerve passes between the abductor and short flexor muscles of the hypothenar eminence near their origin. It cannot be denied that direct pressure may also play a role as well as traction of the nerve, as it passes beneath the hook-like process of the unciform bone.

Diagnosis.—The affections with which this condition may be confused are: the Aran-Duchenne type of progressive muscular atrophy and Gessler's type of occupation atrophy of the hand. Both conditions may give rise to atrophic paralyses in the intrinsic muscles of the hand without the objective disturbances of sensation. The separation of progressive muscular atrophy, beginning in the hand should not present great difficulty. The early involvement of the muscles in the thenar eminence (corresponding to the median nerve distribution) the presence of fibrillary twitchings, the tendency to progression, and the character of the electrical reactions should make the differentiation comparatively easy. The Gessler type of occupation atrophy, which he attributes to an involvement of motor nerve terminals and motor nerve endplates, would present greater difficulties; in fact, it seems to me not improbable that the two cases described in his paper should be classed with the group of cases which I have described rather than be utilized as the basis of a new pathological entity. All other neuritic forms of occupation atrophy of the hands are characterized by definite subjective and objective sensory disturbances.

From the diagnostic standpoint I would emphasize this fact, indeed the most important of all, in attempting to establish a diagnosis. That is to demonstrate the *limitation* of the paralysis to the intrinsic muscles of the hand supplied by the ulnar nerve. In order to do this it is necessary to show the preservation of functions of the muscles of the thenar eminence which are supplied by the median nerve. Usually these are the abductor pollicis, the opponens pollicis, the outer head of the flexor brevis pollicis, and the two outer lumbricales muscles. It must be borne in

mind that variations in this innervation may occur. Both the adductor pollicis and the third lumbricale are occasionally innervated by the median. Furthermore, in palsies in which the intrinsic muscles of the hand are alone concerned, the separation of these various movements of the fingers is by no means easy, because of the preservation of the long flexors and extensors of the thumb and fingers. These in themselves give a very considerable power and variety of movements to the hand and fingers.

Care must also be exercised in the interpretation of the electrical reactions in the thenar region. In an ulnar neuritis, the presence of the reaction of degeneration in the adductor pollicis and inner head of the flexor brevis pollicis which fill in the deeper portions of the thenar region, may mask the otherwise normal reaction in the more superficial muscles of the thenar eminence of median nerve innervation, from the well-known tendency to diffusion of the electrical currents. So that in case of doubt a more crucial test would be the response of these muscles by the indirect current through the ulnar and median nerves respectively.

It is interesting to note in relation to this group of occupation cases, that the deep palmar branch of the ulnar nerve may be severed by the injury. In one case recorded by Gortz, the prong of a pitchfork passed between the third and fourth metacarpal bones, entering the dorsal aspect of the hand; and in another case recorded by Bregmann, the nerve was injured by a spicule of glass which entered the palmar aspect of the hand near the pisiform bone. In both cases with paralysis and atrophy of typical distribution, there was an entire absence of sensory involvement.

Remarks on the Gessler Type of Occupation Atrophy of the Hand, and Its Relation to the Occupation Neuritis of the Deep Palmar Branch of the Ulnar Nerve

Hermann Gessler[2] in 1896 described a type of muscular atrophy of the hand, which he termed "a peculiar form of progressive muscular atrophy in gold polishers." The atrophy was stationary and there were no objective sensory disturbances. The

[2] "Eine Eigenartige Form von progressiver Muskelatrophie bei Goldpoliririnnen." Hermann Gessler. Medecinisches Correspondenz-blatt des Würtembergschen Arzlichen Landes Vereins, Bd. LXVI, No. 36.

non-progressive nature of the affection was sufficient to exclude a spinal type of atrophy. As sensory disturbances were absent, it could not be a neuritic atrophy, in the usual acceptation of the term.

It was also clearly not a myopathy. The distribution and limitation of the paralysis to the intrinsic muscles of the hand, the very considerable degree of atrophy and the accompanying reactions of degeneration, suggested very strongly a neuritic origin. The neuritis must however be limited to the motor nerves.

In order to meet the requirements of this clinical picture, Gessler evolved a fourth form of muscular atrophy. This new form of muscular atrophy was to have as its pathological basis, a degeneration of the terminations of the intermuscular nerves, inclusive of their end plates.

Such degenerative changes in the motor nerve terminals he attributed to the effects of persistent and long-continued muscular contractions, with insufficient relaxation. An anemia of the parts was induced with nutritional disturbances, and as a consequence, motor nerve end degeneration.

The delicacy and fragility of the motor nerve endings were supposed to render them peculiarly liable to such changes.

He refers to experimental work on the motor end plates,[3] carried out on warm and cold blooded animals; and as a result of which he became convinced that such changes must also furnish the pathological basis for some of the muscular atrophies in man.

It may be remarked that no pathological studies have been reported on cases of this character, which might throw light on this important subject. So much for the pathological evidence, on which Gessler's theory was based.

His clinical evidence consists of two cases which were practically identical. The following is, as far as possible, a literal translation of his case report.

I would emphasize their resemblance to the group which has been described as "occupation neuritis of the deep palmar branch of the ulnar nerve."

Gessler's Case Report.—"As the clinical picture is the same in both cases, I will confine myself to the description of the latter.

[3] Gessler, "Die motorische Endplatte und ihre Bedeutung für die periphere Lähmung." Habilitationsschrift, Munschen, 1885.

"Gertrude B., 21 years old, a gold polisher. Has worked for the past 8 years in the Pforzheimer gold ware fabrik.

"She is well developed and in good general health. Up to one and one half years ago, she had experienced no inconvenience from her occupation. At that time there appeared a numbness in the little and ring fingers of the right hand, and she experienced a difficulty in the use of these fingers, and a difficulty in extending them completely.

"Later, she found it more and more difficult to approximate the fingers, spreading movements were better preserved, except in the index finger, which lay in constant apposition to the middle finger. The little finger was fixed in the position of abduction.

"The right hand was colder to the touch and showed a tendency to become cyanotic on slight exposure to cold.

"During the past few months she has found it almost impossible to hold the object which was to be polished, and the hand became almost powerless. About the same time, there developed a curious flexion position of the ring finger, and a marked atrophy of the intrinsic muscles of the hand.

"The patient was forced to abandon her occupation, and after five weeks treatment by the family physician, she was admitted to Ludwig's Spital for treatment.

"*Status Præsens, Feb. 18, 1896.*—Large, well developed girl of healthy appearance. Internal organs are normal. No signs of any affection of the central nervous system. Left upper and both lower extremities are normal.

"RIGHT UPPER EXTREMITY.—The upper arm and shoulder are normal in motility and sensibility, occasionally there are shooting pains in the shoulder.

"The muscles on the extensor surface of the forearm are slightly atrophic; those on the flexor surface are normal in volume.

"*Hand at Rest.*—On the dorsal aspect of the hand the tendons of the extensor communis digitorum stand out prominently, and between them are well-marked depressions in the interosseous spaces. The slightly abducted thumb shows no atrophy, its phalanges are extended and the end phalanx is hyper-extended. The index finger lies in close apposition to the middle finger and is diverted towards the ulnar side, so that it forms an angle with the first metacarpal bone. There is a marked atrophy of the first interosseous muscle. The terminal phalanges of the index and middle fingers are slightly flexed, the basal phalanges are extended. The ring finger is abducted and presents a high degree of flexion, and the little finger is strongly abducted and flexed.

"On the palmar surface of the hand there is apparent, in addition to the flexion deformities of the fingers, *a marked atrophy*

of the hypothenar eminence and a slight atrophy of the thenar eminence.

"The sensibility of the right hand *shows no objective disturbance;* the right hand is colder than the left.

"MOVEMENTS OF THE HAND.—*The movements of the thumb may be carried out normally in all directions,* with the exception of flexion of the distal phalanx.

"The index finger has neither abduction nor adduction, which is also true of the middle finger. The ring finger may be slightly abducted but not adducted. The little finger on an attempted adduction becomes still more abducted. Flexion of the end phalanges is preserved and is accompanied by hyper-extension of the basal phalanges.

"The holding of objects is very difficult and the grip is very much diminished. Extension of the basal phalanges is normal; but is absent in the middle and distal phalanges. Flexion of the basal phalanges of the fingers is impossible.

"The *muscles of the hypothenar show no movement* and the little finger is held permanently abducted. Flexion and extension of the wrist joint, as well as abduction and adduction, are unaltered; although the extensor movement of the wrist seems weaker than normal.

"ELECTRICAL REACTIONS.—The electrical reactions of the muscles of the forearm are normal.

"Electrical stimulation of the ulnar and median nerves produces no contractions in the hand muscles.

"The interossei and lumbricales show complete reactions of degeneration.

"The muscles of the hypothenar show an increase of the galvanic excitability with reversal of the poles, and a diminished faradic excitability.

"*The muscles of the thenar eminence show a diminution to both currents.*"

Remarks.—In the report of Gessler's case just cited, I would emphasize the following points of resemblance to the group of cases which I have described.

1. At the onset there were paresthesias, definitely limited to the distribution of the ulnar nerve; no objective sensory disturbances however.

2. While the muscles of the hypothenar eminence and the interossei, all of which are innervated by the ulnar nerve, were paralyzed, the movements of the thumb were undisturbed.

3. The atrophy, which had reached a high degree in the interosseous spaces and the hypothenar eminence, was only slightly present in the muscles of the thenar eminence.

4. In the description of the electrical reactions, while the muscles of the hypothenar eminence and the interossei show the typical reactions of degeneration with polar changes, the muscles of the thenar eminence show only a diminished faradic and galvanic response.

It is therefore apparent that the distribution of the paralysis, the atrophy and reactions of degeneration, are all suggestive of ulnar nerve involvement.

It will also be noted that the paresthesias noted were in the distribution of the ulnar nerve alone. The median nerve in the hand innervates the abductor pollicis, the opponens pollicis and the outer head of the flexor brevis pollicis; and as it is stated that the movements of the thumb were normal, the muscles of the thenar region only slightly atrophic, as compared with the hypothenar, and the electrical changes consisted of only a diminution in the response, not a degenerative reaction, one would be justified in assuming that the thenar muscles of median innervation were not involved.

This being true, the remaining paralysis and atrophy must be referred to the ulnar nerve alone. The occupation was one requiring frequent, almost constant flexion and grasping movements of the fingers and thumb, and assuming the lesion to be a degeneration of motor terminals from anemia, there is no reason why the paralysis should be limited to the ulnar nerve distribution, nor why the small muscles of the thumb should escape.

For the reasons just detailed, I feel justified in regarding the cases described by Gessler as belonging in all probability to the syndrome which I have outlined; and resulting from compression of the deep palmar branch of the ulner nerve.

Furthermore an attitude of skepticism towards the pathological type of muscular atrophy introduced by Gessler is more than justified. The clinical facts on which it was based are insufficient and pathological confirmation is wanting.

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