ISOLATED NEURITIS OF THE ANTERIOR INTEROSSEOUS NERVE

BY

LESLIE G. KILOH, M.D., B.Sc., M.R.C.P., D.P.M.

AND

SAMUEL NEVIN, M.D., B.Sc., F.R.C.P.

(From the Department of Neurology, King's College Hospital, London)

Lesions of the anterior interosseous branch of the median nerve have been recorded so infrequently that this description of two cases of isolated neuritis of the nerve seems justifiable. The deep position of the nerve protects it from trauma, and when damage does occur it is to be expected that the median nerve—and perhaps the long flexor tendons—will also be affected. Puncture wounds of the forearm are the most likely cause of injury to the nerve. It is probable that examples of neuritis of the nerve occur more commonly than is generally recognized, for the resulting disability is not severe, accommodation occurring quickly in most patients, while recovery follows in many.

Case 1

A man aged 52 noticed suddenly on February 7, 1949, that he was unable to pick up small objects with his right hand. He found that the thumb and radial two fingers were weak. He had no paraesthesiae or numbness and no pain in the arm or hand, though subsequently he noticed an aching pain along the medial border of his right forearm when grasping objects. After three or four days there was some improvement of the fingers, but not of the thumb. His general health had been good and he made no other complaints. There was nothing relevant in his previous medical history.

When seen on February 21, 1949, there was complete paralysis of the right flexor pollicis longus, moderate weakness of the head of the flexor digitorum profundus to the index finger (M.R.C. scale, 3), and less marked weakness of the head to the middle finger (4−). The remainder of the flexor digitorum profundus, the flexor digitorum sublimis, and all the small muscles of the hand were normal. There was no sensory impairment, and in all other respects the nervous system was normal. The electrical reactions of the muscles of the right forearm showed complete reaction of degeneration in the flexor pollicis longus and the radial half of the flexor digitorum profundus. The reactions of the medial half of the flexor digitorum profundus, the pronator teres, and the flexor carpi radialis were normal. The blood Wassermann reaction was negative.

In January, 1950, the power of the long flexors of the index and middle fingers began to improve, and in March a feeble contraction of the flexor pollicis longus could be appreciated. By January, 1951, considerable recovery had occurred (flexor pollicis longus 3+ ; flexor digitorum profundus to index finger 4+, and to the middle finger 5).

Case 2

A man aged 28 awakened one morning in January, 1950, with pain along the outer aspect of the left forearm and a feeling of stiffness in the left thumb. He found that he was unable to bend the terminal joint of his thumb. The pain disappeared after two weeks. He continued at work as a glazier, but had difficulty in picking up small objects with his left hand. He denied any weakness of his index finger.

He was not seen until November, 1950, when examination revealed a complete paralysis of the left flexor pollicis longus. No other muscles were affected, and there was no sensory loss. The nervous system was otherwise normal. The left flexor pollicis longus showed complete reaction of degeneration. No other muscles were tested. The blood Wassermann reaction was negative.

The patient was treated with galvanism, and when seen in January, 1951, active flexion of the terminal joint of the left thumb was again possible (M.R.C. scale, 3). By April further improvement had occurred (4−).

Discussion

The anterior interosseous branch of the median nerve is given off a short distance below the elbow and runs distally on the volar aspect of the interosseous membrane, accompanied by the anterior interosseous artery. It lies between the flexor pollicis longus and the flexor digitorum profundus, overlapped by both, and passes deeply beneath the pronator quadratus, its terminal twigs supplying the wrist-joint. It innervates the flexor pollicis longus, the radial portion of the flexor digitorum profundus, and the pronator quadratus. It contains no fibres subserving superficial sensation.

In Case 1 the weakness was precisely in the distribution of the anterior interosseous nerve. The fact that the paralysis of the heads of the flexor digitorum profundus to the index and middle fingers was not absolute does not necessarily indicate that the nerve lesion was incomplete, for, as has been shown by Sunderland (1945), there is considerable individual variation regarding the proportions of this muscle supplied by the median and ulnar nerves. In some, the slip to the middle finger is entirely supplied by the ulnar nerve. No assessment of the function of the pronator quadratus was made, for it belies its name in being a very feeble pronator of the forearm. Its main action is to prevent separation of the radius and ulna when pressure is exerted on the carpus, a function not readily susceptible of clinical evaluation. Unfortunately the electrical reactions of this muscle were not investigated.

In Case 2 only the flexor pollicis longus was affected. When seen nearly a year after the onset of the paralysis, the patient denied any other weakness, and there is no reason to doubt his statement. Any pathological process capable of picking out the anterior interosseous nerve might as easily select a subdivision of it, and in the great majority of individuals the flexor pollicis longus is innervated by a single branch of this nerve (Sunderland, 1945).

In view of the sudden onset and the delayed partial recovery of these two cases, they would appear to be examples of an interstitial neuritis analogous to the series of cases of acute brachial neuritis described by Spillane (1943). The onset and course of the condition are unlike those of acute anterior poliomyelitis.

References to lesions of the anterior interosseous nerve in the literature are remarkably scanty. Involvement by penetrating wounds of the upper forearm has been reported by Borchardt and Wjasmenski (1917). These authors, together with Ranschburg (1917), suggest that in some cases showing isolated weakness of the long flexors of the thumb and index finger the lesion may affect the entire median nerve, the other muscles normally innervated by the median nerve receiving an aberrant supply from either the ulnar or musculo-cutaneous nerve. This view is repeated by Kinnier Wilson (1940). It is possible that this is the explanation of an occasional case, but in most—including those described above—there is no doubt that the lesion is restricted to the anterior interosseous nerve.

In a review of 136 cases of acute brachial neuritis (neuralgic amyotrophy) Parsonage and Turner (1948) described five patients in whom weakness of the long flexors of the thumb and index finger occurred in addition to shoulder-girdle weakness. In one the condition was bilateral, the thumb and index finger being affected on one side, the thumb alone on the other. A further case is described in which weakness of the left thumb and

index finger occurred as an isolated lesion. They conclude that such a "localized paralysis cannot anatomically be of peripheral-nerve or nerve-root distribution, and is only explicable by an anterior-horn-cell lesion." This conclusion is to be doubted in view of the fact that the weakness is in the distribution of the anterior interosseous nerve.

In both the cases described above it was approximately a year after the onset of the weakness that recovery began. In the absence of any recovery after a prolonged period, the question of a tendon transplant into the thumb would have to be considered. No decision should be made until at least 18 months after the onset. In the meantime the function of the denervated muscles should be maintained so far as possible by means of galvanism. Splinting is unnecessary.

Summary

Two cases of acute interstitial neuritis of the anterior interosseous nerve are described, one complete, the other affecting the branch to the flexor pollicis longus. Other examples of this condition found in the literature are reviewed.

We should like to express our thanks to Mr. H. L.-C. Wood for permission to report the second case.

REFERENCES

Borchardt, M., and Wjasmenski (1917). *Beitr. klin. Chir.*, **107**, 553.
Parsonage, M. J., and Turner, J. W. A. (1948). *Lancet*, **1**, 973.
Ranschburg, P. (1917). *Neurol. Zbl.*, **36**, 521.
Spillane, J. D. (1943). *Lancet*, **2**, 532.
Sunderland, S. (1945). *Anat. Rec.*, **93**, 317.
Wilson, S. A. K. (1940). *Neurology*. p. 329. Arnold, London

ON THE VERBAL USAGE OF THE CDE NOTATION FOR THE RH BLOOD GROUPS

BY

SOL HABERMAN, Ph.D.

AND

JOSEPH M. HILL, M.D.

(From the J. K. and Susie L. Wadley Research Institute and Baylor Hospital, Dallas, Texas, U.S.A.)

Since Fisher and Race published their theory of the triple allelomorphic nature of the Rh–Hr blood antigens and antibodies (see Race, 1944) there has been considerable discussion on the usefulness of their CDE notation as compared with the Wiener (1949) notations. One of the most frequent criticisms that have been made of the Fisher–Race notation is that it is difficult to use verbally. In spite of such an objection, we have found that the Fisher–Race system is complete, clear, and versatile enough to allow for the inclusion of new antigens and antibodies as they are discovered.

Method

In our laboratories there has evolved a verbal usage of the Fisher–Race notation that seems useful to the laboratory worker as well as to the clinician who is faced with an iso-immunization problem. In this system the genotype or "probable genotype" (Hill, 1947) is taken into account rather than the phenotype. Although our method is not offered as a panacea for this controversial subject, we feel that it has certain virtues, such as: (1) using the CDE notations verbally for both the antigens and the antibodies; (2) elimination of double symbols and double thinking in writing the CDE genotype and presenting it verbally; (3) consideration of the antigens present in an erythrocyte on a genetic basis; and (4) a presentation of usable information for clinical problems.

In Table I are presented some CDE genotypes, the Race short symbol for verbal usage, the Wiener (1949) terminology, and the method we have used in verbal presentations. In this latter system of verbal usage, only the CDE (Rh) antigens are given, with the cde (Hr) antigens left out but understood to be present. With this system the CDE antigens are regarded as closely linked genes inherited in groups of three. Consequently, the genotypes are given with consideration to both sides of the chromosomes—for example, CDe/CDe is called homozygous CD, whereas CDe/cde is called heterozygous CD, and Cde/Cde is called homozygous C, while Cde/cde is heterozygous C. Although the examples presented in Table I do not cover all of the CDE/cde (Rh–Hr) genotypes, a sufficient number are presented for an adequate demonstration of the system.

TABLE I

Genetic and Antigenic Constitution	Race Short Symbols	Wiener Terminology, 1949	Suggested Verbal Usage
cde/cde	rr	rr	Rh negative
CDe/CDe	R_1R_1	R^1R^1	Homozygous CD
CDe/cde	R_1r	R^1r	Heterozygous CD
CDe/cDE	R_1R_2	R^1R^2	CDDE or CD over DE
cDE/cDE	R_2R_2	R^2R^2	Homozygous DE
Cde/Cde	R′R′	r′r′	Homozygous C
Cde/cde	R′r	r′r	Heterozygous C
C^{w}de/cde	R′^{w}r	r″^{w}r	Heterozygous C^w
C^{w}de/C^{w}de	R′^{w}R′^{w}	r″^{w}r″^{w}	Homozygous C^w
cDe/cDe	R_0R_0	R°R°	Homozygous D
cDe/cde	R_0r	R°r	Heterozygous D
cdE/cdE	R″R″	r″r″	Homozygous E
cdE/cde	R″r	r″r	Heterozygous E
CDe/cDe	R_1R_0	R^1R^0	CDD or CD over D
CDe/cdE	R_1R''	R^1r''	CD over E
CDe/Cde	R_1R'	R^1r'	CDC or CD over C
CDE/cde	R_2r	R^2r	Heterozygous CDE
CdE/CdE	R_yR_y	r^yr^y	Homozygous CE
C^{w}De/CDe	$R_1{}^wR_1$	R^3R^1	C^{w}DCD or C^{w}D over CD
C^{w}De/cDE	$R_1{}^wR_2$	R^3R^2	C^{w}DDE or C^{w}D over DE
CdE/C^{w}de	$R_yR'^w$	$R^yr''^w$	CEC^w or CE over C^w

It should be noted that the verbal usage of the CDE notation has already been successfully applied to the antisera for the Rh–Hr subgroups, using the same notation without special consideration for multiple specificity sera. Table II presents the already used antisera terminology as compared with that previously offered by Wiener.

TABLE II

Fisher–Race		Wiener	Fisher–Race		Wiener
Anti-C	..	Anti-rh′	Anti-D^u	..	Anti-Rh^u
Anti-D	..	Anti-Rh$_0$	Anti-E^u	..	Anti-??
Anti-E	..	Anti-rh″	Anti-CD	..	Anti-Rh$_1$
Anti-c	..	Anti-hr′	Anti-DE	..	Anti-Rh$_3$
Anti-d	..	Anti-Hr$_0$	Anti-C^{w}D	..	Anti-Rh$_0$‴
Anti-e	..	Anti-hr″	Anti-C^{w}C	..	Anti-rh‴rh′
Anti-C^w	..	Anti-rh‴	Anti-D^{u}D	..	Anti-Rh$_0$Rh^u

By comparing the Wiener notations of antigens (Table I) with antisera (Table II) one can readily see the changes in designations that are made between antigen and antibody. For example, the Rh antigen combinations are given without the h, such as r, r″, R′, while the corresponding antibody is designated as anti-rh′, anti-rh″, and anti-Rh$_1$. In addition to the deletion of the h, antigens have the subgroup designations above the r or R, and antibody subgroup designations are positioned below the rh or Rh.

In contradistinction to the Wiener usage, the Fisher–Race designations specify that the same letter used for the antigen be used for the antibody—that is, anti-c, anti-e, anti-CD, etc.

In view of the above verbal usage of the Fisher–Race nomenclature in what we feel is a successful manner, there is no longer that serious objection to the use of this system.

BIBLIOGRAPHY

Race, R. R. (1944). *Nature, Lond.*, **153**, 771.
Hill, J. M. (1947). *Amer. J. clin. Path.*, **17**, 494.
Wiener, A. S. (1949). *Hereditas, Lund.*, Suppl. Vol., p. 419.