The changes in nerve conduction in acute idiopathic polyneuritis

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In acute idiopathic polyneuritis weakness of relatively sudden onset sometimes progresses swiftly to severe paralysis. And yet even severely affected patients with this disease may survive with the aid of artificial respiration and apparently recover complete motor and sensory function within several weeks. The nature of the disturbance of nerve conduction underlying this reversible loss of function is not understood and is of considerable interest in view of its relatively rapid time course. There have been reports of slowing of motor conduction velocity in some patients with 'neuritis' (Hendriksen, 1956; Redford, 1958). Lambert (1960) showed a proximal failure of excitability and/or blocking of conduction in the majority of motor fibres, with subsequent recovery, but we are not aware of any detailed serial electrophysiological studies of sensory function in patients with acute idiopathic polyneuritis. In this paper we describe serial electrophysiological studies of both motor and sensory nerve function in such a patient in the hope that they may throw light on the nature of the reversible disturbance of nerve conduction in acute idiopathic polyneuritis. Investigations were started within 12 days of the onset of the illness while the patient required artificial respiration, and they were repeated at intervals subsequently as he recovered.

METHODS

The serial electrophysiological investigations included routine electromyographic examination, measurement of the conduction velocity of motor fibres to the small muscles of the hand and of the fibres of the digital nerves, and the response of the digital nerve fibres to mechanical stimulation. All the investigations were made in a warm laboratory except for the first records which were taken in a ward while the patient was on artificial respiration. The measurement of the conduction velocity of motor fibres to the small muscles of the hand was made after routine electromyographic examination with a coaxial needle electrode. For stimulating the median nerve at the wrist or at the elbow the cathode was a saline pad E.E.G. type electrode and the anode a metal plate 2.5 x 6.0 cm. placed over the flexor surface of the forearm. The stimuli were either condenser discharges of 130 \( \mu \text{s} \, \text{sec} \) time constant with a maximal voltage available of 170 volts, or they were square wave voltage pulses of up to 670 \( \mu \text{V} \, \text{sec} \) duration, 75 volts maximal. In both instances the stimuli were applied through an isolation transformer. The conduction velocity was calculated from the difference in conduction times when the nerve was stimulated at two levels; the length of nerve was estimated by surface measurement.

Two procedures were used to measure the conduction velocity of sensory fibres in the digital nerves. The digital nerves were stimulated electrically through ring electrodes on the fingers and the orthodromically conducted volley of impulses recorded from surface electrodes placed over the median nerve or ulnar nerves at the wrist or at the elbow (Gilliatt and Sears, 1958). The other procedure consisted of stimulating the median or ulnar nerve at the wrist, and recording the volley of impulses conducted antidromically in the digital nerves from ring electrodes on the fingers (Sears, 1959). The method used for stimulating the digital nerve fibres at the wrist was the same as for stimulating the motor fibres.

For recording digital nerve action potentials from the fingers, the hands were washed with soapy water to remove grease. Electrode jelly was then rubbed into the finger from which recording was to be made and the excess jelly removed. Clean silver wire (gauge 26) was twisted around the finger to form a ring electrode, care being taken not to occlude the circulation through the fingers. The electrode was then immobilized with a band of sticky tape. Usually three electrodes were applied, one each on the proximal, middle, and distal phalanges. In normal subjects the antidromic volley of impulses is recorded as a prominent diphasic wave with a latency of 2-0 to 3-5 m.s.c., the greatest amplitude compound action potential being recorded from the two most widely spaced electrodes (see Fig. 2b). However, when the proximal electrode is used as one of the pair and the stimuli applied to the median nerve above the threshold of the motor fibres, the record may show a late wave with a latency of 8 to 10 m.s.c. This late component is not present in the record made from electrodes on the middle and distal phalanges and is due to action currents arising in the distal portions of the lumbrical muscles covering the proximal phalanx. When recording from abnormal nerves, care was always taken to exclude this muscle artifact by recording only from the more distal pair of

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electrodes or by ascertaining whether the presence of a late component was associated with a twitch of the lumbrical muscles.

The response of digital nerve fibres to mechanical stimulation was determined by tapping the finger nail. The excitation of mechano-receptors in the finger tip by this rapidly acting stimulus evokes an afferent volley of impulses of which the action currents are sufficiently synchronous to be recorded from ring electrodes on the fingers (Sears, 1959). Recording was made from a pair of wire ring electrodes of the pattern described above, the distal electrode being placed on the middle phalanx, close to the terminal interphalangeal joint, the proximal electrode on the proximal phalanx. To tap the finger nail, a pen unit of an electroencephalograph was mounted so that when the pen was deflected by feeding an electric pulse into the E.E.G. amplifier, the pen struck the finger nail. The force of stimulation was kept constant throughout the investigation. The finger tip rested on a crystal microphone which was fixed to a perspex platform. The output from the crystal, after amplification, was mixed with the time scale and displayed on one beam of the cathode-ray oscillograph, thus providing a stimulus marker for the mechanical event and a rough indication of the pressure transmitted through the finger tip.

Nerve and muscle action potentials were amplified with a condenser-coupled amplifier and displayed on one beam of a twin-beam cathode ray oscillograph. The overall time constant of the amplifier was reduced to 0.03 sec. Records were made directly on 70 mm. bromide paper. The records made in the ward on the patient who was on artificial respiration were taken on 35 mm. film.

CASE REPORT

The patient was a naval engineer of 61. Seven days before his admission to hospital he had noticed weakness of his legs on climbing stairs, and also some 'numbness' of his toes. The following day he complained of weakness of both hands and some 'numbness' of the fingers of both hands. His legs and arms then grew progressively weaker until the time of his admission when he had mild bilateral facial weakness and loss of all voluntary limb movement except for slight power of flexion of the fingers of both hands, minimal power of flexion of both knees, and slight toe movements. All tendon reflexes were absent. He complained of numbness of his hands and feet and he had a moderate impairment of light touch, pin prick appreciation, two point discrimination, and hot and cold appreciation over a 'glove' and 'stocking' distribution more marked distally and fading proximally. Joint position sense was moderately impaired in all extremities. Investigations included examination of the cerebrospinal fluid which contained 500 mg./% of protein but no cells. The patient developed a chest infection and the day after his admission he became dyspnoeic at rest owing to intercostal weakness and so a tracheostomy was performed. He was placed on intermittent positive pressure artificial respiration, which was necessary for 10 days. There was no further neurological progression. Twenty days after the onset of the illness recovery of the motor power started and was followed within four days by an improvement in sensory function which was accompanied by paraesthesiae in all extremities which later subsided. Both motor and sensory function then steadily improved and were apparently normal to clinical testing 55 days after the onset of the illness but deep tendon reflexes were then still absent.

RESULTS

The first electromyographic examination was 12 days after the onset of the illness while the patient was on the respirator. Although motor power was almost absent on clinical testing, a few motor units could be activated in the abductor pollicis brevis muscle by voluntary effort, although the interference pattern was markedly reduced. The conduction time was 5 m.sec. from wrist to muscle (6 cm.) for the fastest surviving motor fibres in the median nerve at the wrist, as illustrated in Fig. 1a. Moving the stimulating cathode from the wrist to the elbow increased the latency of the response to 10.5 m.sec. (Fig. 1b). The conduction distance from the wrist to the elbow was 27 cm. which gives a conduction velocity of 49 m./sec. over the wrist to elbow segment of the median nerve. The value falls

![FIG. 1. Co-axial needle electrode recording from the left abductor pollicis brevis muscle. Electrical stimulation of median nerve at wrist (a) and elbow (b) to show latency of surviving motor fibres. Note the abnormal polyphasic form of the compound muscle action potential with late components. Latencies of 5 m.sec. and 10.5 m.sec. for stimulation at the wrist and elbow respectively. Cal. 400 μV Time scale 1 and 5 m.sec.](http://jnnp.bmj.com/)

"R. G. Bannister and T. A. Sears"
The changes in nerve conduction in acute idiopathic polyneuritis

just outside the normal range for the fibres innervating the abductor pollicis brevis muscle (57.2 ± 4.2 m./sec., Thomas, Sears, and Gilliatt, 1959), and the conduction time from the wrist to the abductor pollicis brevis muscle was also a little prolonged. These records also show the polyphasic form of the compound muscle action potential due to the great reduction in number of motor fibres. Some of the late components of the potential show a latency of 15 m.sec. from the wrist which is greater than would be expected for the slowest of the motor fibres innervating the small muscles of the hand (Thomas et al., 1959). On five subsequent occasions, the last being at 50 days when motor power was normal, the median nerve was stimulated at the wrist and elbow and recordings made from the abductor pollicis brevis. On none of these occasions was the latency as short as that obtained on the first occasion, the shortest latency subsequently obtained being 6 m.sec. This suggests that those few fibres of near normal conduction velocity, which alone survived at an earlier stage of the illness, subsequently also succumbed to the disease process.

On the day of the first electromyographic studies, attempts were made to record sensory nerve action potentials using both of the procedures previously described. At this time the threshold for two-point discrimination was 2.5 cm. over the finger tip of the right second digit. The records illustrated in Fig. 2 were made from the right second digit following electrical stimulation of the median nerve at the wrist. The stimulus intensity, which was the maximal tolerated by the patient, was approximately three times that giving a maximal antidromic volley of digital nerve impulses in a normal subject and was adequate to excite the surviving motor fibres in the median nerve. It is emphasized that in a normal subject the electrical threshold of the digital nerve fibres at the wrist is lower than that of the motor fibres (Dawson, 1956). Following the stimulus artefact there is no evidence of a diphasic action potential such as would occur in a normal nerve nor could such a potential be recorded from any of the other fingers. A typical response of a normal subject is shown for comparison in Fig. 2b. After the stimulus artefact there occurs a diphasic wave with a latency

![Figure 2](http://jnnp.bmj.com/)
of 2.3 m.sec. and of some 60 μV peak-to-peak amplitude. The response shown is not a maximal one, and in fact was obtained with the stimulus intensity below threshold for the motor fibres. This comparison serves to illustrate the profound reduction in the number of conducting digital nerve fibres at an early stage of acute polyneuritis. Consistent with this finding was the failure to record with surface electrodes at the wrist any evidence of orthodromically conducted impulses following electrical stimulation of the digital nerves in the fingers.

The disturbance of sensory nerve conduction was also present in proximal as well as distal segments, as was shown to be the case for the motor fibres. Thus all attempts to record just above the elbow a compound nerve action potential following high-intensity stimulation of the median nerve at the wrist (several times supra-threshold for the surviving motor fibres and of painful intensity) were unsuccessful. According to Dawson and Scott (1949) the compound action potential thus recorded in a normal subject is due to activity in both sensory and motor fibres. It is evident therefore that not only were the surviving motor nerve fibres too few to produce a registerable action potential (or conducting too asynchronously) but also that an identical change had probably affected the majority of the sensory nerve fibres.

Attempts at recording digital nerve action potentials evoked by nail tapping 12 days after the onset of the illness were also unsuccessful. Subsequently, attempts at recording the antidromic volley of impulses in the digital nerves were made 19, 25, 32, and 41 days after the onset of the illness, all without success, although during this time the patient had made a striking recovery, to the extent that by the forty-fifth day his motor power was only mildly reduced. He no longer complained of numbness and sensation to clinical testing was only slightly impaired, the two-point threshold being 1.0 cm. for the finger tip of the right second and third digits. Records made on the fiftieth day showed for the first time, as illustrated in Fig. 3a, electrical evidence of recovery of sensory nerve conduction, but the conduction velocity was grossly slowed, and the form of the response abnormal. In Fig. 3a it may be seen that following the stimulus artefact there is an ill-defined wave with a latency to the first negative peak of 8.0 m.sec. This recording was made from electrodes on the middle and distal

FIG. 3. Antidromic digital nerve action potentials right middle finger; recording electrodes on middle and distal phalanges. (a) 50 days after onset of illness; (b) same (different time base); (c), (d), and (e) effects of spraying ethylchloride on proximal phalanx for 30 seconds; record f, 69 days after onset. Cal. 10 μV. Time scale 1, 5, and 20 m.sec. Stimulation intensity 75 V, square wave pulses of 200 μ sec. duration. Twenty sweeps superimposed.
The changes in nerve conduction in acute idiopathic polineuritis

phalanges thus excluding the possibility that the wave was due to action currents from lumbrical muscles (see Methods). As the conduction distance was 20.5 cm, the conduction velocity of these recovered fibres was approximately 26 m./sec., accurate estimation being impossible owing to the grossly abnormal form of the compound action potential. In contrast, in normal subjects the conduction velocity of the group of digital nerve fibres which caused the compound action potential, as illustrated in Fig. 2b, measured to the peak of the negative wave, is 54 m./sec. ± 4.7 (Sears, to be published). The slow conduction rate of these abnormal fibres was also associated with an abnormally high electrical threshold. Thus condenser discharges of approximately three times the intensity and of time constant twice the duration normally used were wholly inadequate to excite the fibres and only square voltage pulses of long duration were effective.

On this first occasion on which there was electro-physiological evidence of recovery in the sensory fibres, the effect of local cooling of the nerve proximal to the recording electrodes was investigated. Figure 3b shows the control response, as in Fig. 3a, but recorded on a different time basis. Ethyl chloride spray was then applied to the proximal phalanx (proximal to the proximal recording electrode) for 30 seconds. Records 3c and 3d were taken 20 and 45 sec. after ending cooling and it may be seen that there was a slight but definite dispersion of the wave and reduction in amplitude, particularly in record 3d. Record 3e, taken one minute after record 3d, shows a complete recovery.

Record 3f was taken 69 days after the onset of the illness, that is, a further 19 days after the record shown in Fig. 3a. The improvement is in fact greater than immediately apparent, for it should be noted that record 3f was made on a faster time base and at a lower amplification than record 3a. Thus the peak-to-peak amplitude of the wave has nearly doubled and the latency diminished, especially of the second phase of the compound potential.

Records made on the fiftieth day also showed, for the first time, definite evidence of a small response to nail tapping (Fig. 4a). The onset of the action potential evoked by nail tapping may be seen occurring as the upward-going wave with a latency of 3.5 m.sec. from the onset of pressure pulse recorded by the crystal as indicated by the upward elevation in the lower sweep. The record of a response to nail tapping in a normal subject is illustrated in Fig. 5; the action potential arises from the baseline roughly coincident with the peak of the pressure pulse with a latency of approximately 1 m.sec.

The records shown in Fig. 4b, c, and d prove that

FIG. 4. (a) Upper sweep, digital nerve action potential (orthodromic) evoked by nail tapping; lower sweep, time scale; upward deflection denotes moment of mechanical stimulation. 'Collision' experiment; (b) response to nail tapping; (c) antidromic digital nerve action potential evoked by electrical stimulation at wrist; (d) nail tapping precedes electrical stimulus by 5 m.sec. (Note that antidromic response was occluded.) Cal. 10 μV. Time scale 1, 5, and 20 m.sec. Twenty sweeps superimposed.
It is convenient to consider the electrophysiological and clinical findings at the two different phases of the illness.

**ACUTE STAGE: SENSORY**  
At the height of the illness the sensory disturbance present on clinical examination was associated with a complete absence of action potentials from sensory nerves of the forearm, hand, or digits, following electrical stimulation, and absence of the response to natural (mechanical) stimulation of the digital nerves. This association of clinical and electrophysiological data is most simply explained on the basis of a failure of conduction in at least the large, myelinated fibres. The loss of the response to mechanical stimulation suggests a similar involvement of the small myelinated fibres as, in the normal subject, the small fibres also contribute action currents to the compound action potential evoked by nail tapping (Sears, unpublished). These conclusions require some qualification. The possibility cannot be excluded that at this acute stage of the illness some sensory nerve fibres of near-normal conduction velocity were present, as was found to be the case for the motor fibres, but that they were too few to produce a registerable action potential. Even if this were so, the number of such fibres could only have been a small proportion of the normal complement as judged by the total absence of the response.

A further possibility is that at the time no responses were obtained the electrical threshold of the damaged fibres was so high that they were not excited by the shocks, which were approximately three times the threshold in normal subjects. Nevertheless the effects of a raised electrical threshold were not restricted to the applied electrical stimulus, for the response to natural stimulation was also abolished. The latter finding also makes it improbable that temporal dispersion of the action potentials of individual nerve fibres, due to slowing of conduction velocity, accounted for the loss of the responses to nail tapping. This is because the recording was made only a few centimetres from the point of initiation of the impulses whereas the conduction distance for the tests using electrical stimulation were of necessity some five times greater.

Collectively then, the electrophysiological data from a study of conduction in sensory fibres are consistent with an actual failure of nervous conduction in myelinated fibres, probably extending at least to the elbow, and this is compatible with the clinical impairment of sensation.

**ACUTE STAGE: MOTOR**  
At the time in the acute stage of the illness when there was clinically almost complete paralysis, there must have been a failure of conduction in nearly all the motor axons. It should be appreciated that the concomitant, nearly complete deafferentation would also be expected to contribute to the loss of voluntary power according to present concepts of the regulation of movement (Eldred, Granit, and Merton, 1953; see also review of the effects of deafferentation in man, Nathan and Sears, 1960).

The level of the conduction disturbance included at least the elbow-to-wrist segment of the nerve as judged by the complete loss of the compound nerve action potential which may be recorded over this stretch of nerve in normal subjects (Dawson and Scott, 1949). Evidently the surviving motor nerve fibres which were shown to be present were too few to produce a registerable action potential. It should be noted that the conduction velocity of the surviving motor fibres was below the lower limit of the normal range. Furthermore, the procedure used to measure the velocity measured that of the fastest fibre present and there were some indications that still slower conducted components were present.

**RECOVERY STAGE: SENSORY**  
Clinically improvement was detected as early as the twenty-fourth day and the patient had completely recovered sensation seven weeks after the onset of his illness. However, his apparently normal sensibility was subserved by digital nerve fibres conducting at approximately half the normal velocity. To appreciate this fully it is necessary to consider the fibre-calibre spectrum of a normal digital nerve. According to Ranson, Droegemueller, Davenport, and Fisher (1935) the fibre calibre spectrum of a digital nerve (fifth proper volar) displays two peaks at 8 to 10 μ (27.2% of all fibres) and

50 days after the onset of illness the nerve fibres conducting impulses antidromically into the fingers, although conducting at half their normal rate, were also excited by the natural stimulus of nail tapping. Record 4c shows the antidromic volley evoked by electrical stimulation as described previously. The upper record (4b) shows the response to nail tapping. When the electrical stimulus evoking the antidromic volley was preceded by an appropriately timed mechanical stimulus (stimulus interval 5 m.sec.) the antidromic volley was abolished (4d). This effect is due to orthodromic impulses evoked by nail tapping colliding with the antidromic impulses evoked by electrical stimulation preventing the latter from reaching the recording electrodes (Sears, 1959). The response to nail tapping in these records is unfortunately partly obscured by an artefact which was derived from the stimulator.
at 2 to 4 μ (40-2%) respectively. Assuming that the factor 6 relating fibre diameter and conduction velocity (Hurst, 1939) applies to human myelinated nerve fibres then it would be expected that the group of large diameter digital nerve fibres would conduct at between 48 and 60 m./sec. In a group of 20 normal subjects the mean conduction velocity over the proximal phalangeal segment of nerve was 54 m./sec. (S.D. ± 4.7) (Sears, to be published). A typical compound action potential of these fibres is shown in Fig. 2 of this paper for comparison. Even with intense electrical stimulation no trace is seen in the normal subject of the action potential of small diameter fibres, due presumably to the greater temporal dispersion and also to the smaller action currents to be expected from small fibres.

Thus, the compound action potential conducting at approximately 25 m./sec. which appeared during recovery must have arisen in recovering, previously rapidly conducting nerve fibres. Furthermore these fibres had established functional connexion with the periphery as was shown by the interaction between mechanical and electrical stimulation.

**Recovery stage: Motor**  The fact that motor power to clinical testing became normal again indicated that a large proportion of the previous non-conducting fibres had functionally re-innervated muscle fibres. However the conduction velocity of the fastest fibres present was slightly slower than that of the fibres which were still conducting at the height of the illness. Hence, although there was increased innervation in recovery, there was also some evidence that those fibres which had survived the first stage of the illness subsequently were affected, most probably in the same way. This continuing manifestation of the disease process was not apparent from clinical examination.

**Conclusions**

At the height of the illness as judged clinically there was a failure of conduction in the majority of motor and sensory nerve fibres. Some of the fibres which were still conducting in the early stage of the disease probably themselves subsequently succumbed; this was suggested by the progressive increase in minimal conduction time of the motor fibres. The extent of the failure of conduction at this early stage of the disease was further shown by the total absence of the response to nail tapping. As fibres of small diameter also contribute action currents to the compound action potential evoked by nail tapping in normal subjects, the absence of any potential suggests that conduction in the small sensory fibres was also affected. We have no comparable evidence for any except the fastest motor fibres though involvement of gamma fibres would contribute to the motor weakness.

The completeness of the recovery, as judged by simple clinical tests, was misleading for it obscured a persisting grossly abnormal state of the nerve fibres. This was most evident for the sensory nerve fibres which were conducting at approximately half their normal velocity over the palmar segment of the nerve. These partially recovered fast conducting fibres must also have been affected over the forearm (elbow to wrist) segment. This can be stated because it is known from the work of Dawson (1956) that the compound action potential recorded at the elbow following electrical stimulation of the median nerve at the wrist is due in part to the large digital nerve fibres. Thus the failure to record any compound action potential over the forearm segment shows that the digital fibres must have been affected as well as the motor fibres, as already discussed.

A possible explanation of the rapid recovery in acute polynoeritis is that only terminal portions of the sensory and motor fibres are severely affected and regeneration in these is correspondingly rapid. However, this study shows that nervous conduction was affected over the palmar and phalangeal segments of the digital nerves, over the palmar segments of the motor nerves, and over the elbow-to-wrist segment of motor and sensory nerve fibres. As there were clinical signs of recovery 20 days after the onset of the illness it is clearly evident that the failure to conduct impulses over some 50 cm. of nerve could not have been due to Wallerian degeneration. These facts allow a further conclusion to be drawn, namely, that the nerve fibres which showed early recovery and on which clinical recovery was clearly dependent retained a structural integrity such that Wallerian degeneration did not take place. This conclusion does not exclude the possibility that Wallerian degeneration occurs, but it is evident that any fibres thus affected are in a different category from those considered above.

The physiological mechanism of the conduction block in this patient seems then to reduce it to two major possibilities. First, it might be due to primary involvement of the polarization of the electrically excitable membrane of the nerve. This, however, is not likely to occur, at least as a direct effect exerted on the actual mechanism of impulse generation. In polynoeritis there does not appear to occur any of the phenomena such as tetany that are produced by such a disturbance of electrolytes.

The second more likely possibility is that the electrical excitability of the membrane is not lost but that in some way the ‘local’ circuit currents on which conduction is dependent are prevented from
acting. One possibility which suggests itself is that the myelin sheath loses its insulating properties or that there is actual loss of myelin. The effect of this would be to increase the 'leakage' current through the internode (Huxley and Stämpfli, 1949) thus preventing adjacent nodes from being excited. The sort of process envisaged here may be likened to the effects of experimental 'demyelination' caused by saponin applied to the myelin sheath of single isolated nerve fibres by Tasaki (1953). Such an explanation has the advantage that it explains why there were no obvious signs of impaired conduction in unmyelinated fibres. (Electrical stimulation was painful although no nerve action potentials of myelinated fibres could be recorded.)

The explanation of the disturbance of nerve conduction in acute polyneuritis proposed here on physiological grounds receives indirect support from both clinical and experimental neuropathological observations. Acute idiopathic polyneuritis lacks precise clinical and pathological definition, has been given various names, and has no proven aetiological factors. However, the clinical features of the case described—the acute onset of ascending flaccid paralyisis, with sensory impairment and elevation of the cerebrospinal fluid protein, without pleocytosis—are typical of the largest group of cases included under the name acute idiopathic polyneuritis. In the most extensive pathological study of this disease (Haymaker and Kernohan, 1949) there was insufficient material to determine the relative degree of involvement of the peripheral nerves. However, the sequence of changes observed was oedema during the first three or four days, followed by swelling and irregularity of the myelin sheaths beginning on the fifth day. Adams (1959) commented on a variable degree of degeneration of the medullated nerve sheaths both of the Wallerian and segmental or periaxial types, axis cylinders being usually less severely affected than myelin sheaths. It is of interest that in experimental diphtheritic polyneuritis caused in animals by injections of incompletely neutralized toxin antitoxin mixture, some 30% of the fibres in certain peripheral nerves showed segmental demyelination six or seven days after the injection (Majno, Waksman, and Karnovsky, 1960). The process appeared to start near the nodes of Ranvier, the myelin sheath swelling to twice its normal diameter before disintegration occurred. Then there was phagocytosis of the fragmented myelin by histiocytes but the axon remained intact.

Though Wallerian degeneration may occur in some cases of acute idiopathic polyneuritis, particularly those ending fatally, from the electrophysiological studies presented here it seems likely that, especially in milder cases with recovery, the myelin sheath is damaged but the nerve retains a structural integrity along its entire length. In recovery a greater disturbance of nervous conduction persists than might be expected from the apparently complete clinical recovery.

SUMMARY

Detailed serial electrophysiological studies are described in a patient with acute idiopathic polyneuritis with profound motor and sensory involvement at the height of the illness, followed by apparently complete clinical recovery.

When the clinical disability was maximal there was a failure of conduction in the majority of motor and sensory fibres in the forearm, hand, and fingers, probably sparing unmyelinated fibres.

The completeness of the recovery clinically obscured a persisting grossly abnormal state of the nerve fibres, the sensory fibres conducting at approximately half their normal velocity over the palmar segment of the nerve.

It was concluded that in view of the length of nerve affected and the speed of recovery of nervous conduction, the failure could not have been due to Wallerian degeneration but probably resulted from damage to the myelin sheaths, the structural integrity of the nerve being retained through its entire length.

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The changes in nerve conduction in acute idiopathic polyneuritis

R. G. Bannister and T. A. Sears

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