Clinical value of nerve conduction studies

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IT IS MORE than 20 years ago since Hodes, Larrabee and German (1948) measured the motor nerve conduction velocity for clinical diagnosis. Since then, nerve conduction studies are performed routinely in conjunction with clinical electromyography as they have been shown to be of proven value in the differential diagnosis of neuromuscular disorders. The procedure is simple and rapid. The result is also objective. Facilities for carrying out such studies are a recent introduction to this country. It is felt that a report of the examinations personally performed during a year's fellowship in the Department of Clinical Neurophysiology, Rikshospitalet, Oslo, Norway, will help to illustrate the usefulness and principles of these tests.

In 1852, Helmholt with his ingenious mechanical apparatus measured the conduction velocity of the human median nerve and obtained values corresponding to present day figures. Animal experiments by Erlanger and Gasser (1927) demonstrated the relationship between the conduction velocity and the diameter of the peripheral nerve and that different nerves with different nerve fibres vary in their conduction velocities. Erlanger (1927), Gasser and Grundfest (1939) showed that the conduction velocities in warmblooded animals were almost directly proportionate to the axon diameters of the nerve fibres.

Hodes, Larrabee and German (1948) proved the values of motor conduction studies in patients with peripheral nerve injury and hysterical paralysis. By stimulating a given nerve at two points and finding the latency difference to the muscle response, they were able to measure the conduction velocity. Later workers like Lambert (1956), Gilliatt and Thomas (1960), Gilliatt and Sears (1958) showed that nerve conduction velocity is slowed in localised nerve lesions, in polyneuropathies and peroneal muscular atrophy. They helped to establish the technique as a practical diagnostic tool. Dawson and Scott (1949) showed that it is possible to detect nerve potentials in the median and ulnar nerves evoked by distal percutaneous stimulation at the wrist. The evoked potentials were picked up by bipolar electrodes placed at the appropriate nerve at the elbow. The evoked response in this case is made up of an orthodromic sensory and antidromic motor response. Dawson (1956) showed that by stimulating the digital nerves in the fingers, a purely sensory potential could be obtained from the median or ulnar at the wrist.

In 1952, Norris, Shock and Wagman demon-

strated slowing of the conduction velocity with increasing age in adult life. Wagman and Lesse (1952) showed that in persons over 60 years old, there is a reduction of about 10% in the conduction velocity. Gamstrop (1963) has shown that in motor fibres at birth, the conduction rate increases from only about half that of adults to reach a maximum in early adolescence. The conduction rate is also affected by temperature. Henriksen (1956), Buchthal and Rosenfalck (1966) showed that the conduction velocity slows with cooling by 2-2.4 metres/sec. per degree centigrade.

Conduction velocity of normal human peripheral nerves varies from 40 to 70 metres per second. In newborn infants, the conduction velocity is about 27 metres per second and usually reaches adult values when the child is 2 to 5 years of age.

Method

Satisfactory apparatus is now readily available commercially. The principal components and their connections are represented by the block diagram, fig. 1.

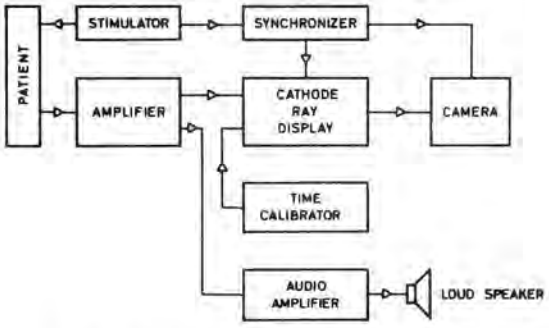

Fig. 1. BLOCK DIAGRAM OF AN ELECTROMYOGRAPH

The stimulator is designed to give a brief condenser shock, a square wave type of stimulus, with a duration of 0.1 msec. to 0.5 msec. Stimulus artifact is reduced either by a transformer or radio-frequency isolating unit.

The stimulation may either be bipolar or monopolar. The stimulators have round padded electrodes moistened with saline. For stimulating the digital nerves of the fingers, Dawson recommended small pliable silver strips two to four mm. wide covered by lint, moistened with saline and firmly applied to the fingers.

For motor nerve conduction, either a surface or concentric needle recording electrode may be used. The former electrode usually consists of silver or solder disc placed over the muscle and is especially advantageous in children. However, for distinguishing damage to one of the smaller terminal branches of the median or ulnar nerve, a needle electrode is needed. For sensory nerve conduction, surface electrode or a needle electrode may be used.

It is essential for the sweep of the cathode ray tube (oscilloscope) to be triggered by a stimulus after a suitable delay. The action potential, picked up by the recording electrode, is displayed on the oscilloscope and is also heard via a loudspeaker arrangement. The action potential on the oscilloscope screen is also photographed for permanent record and further analysis. A time marker with 1 msec. divisions is superimposed on the trace.

The time from the stimulus to the response is measured from the photograph. Alternatively, a special device which causes a vertical deflection of the horizontal axis of the oscilloscope trace, available commercially, permits direct measurement of the time latency on the oscilloscope. This value is read off from a digital counter, which records as the deflected baseline is moved from the stimulus to the response.

In the procedure, the patient is suitably earthed with a plate type electrode; where possible, it is placed between the site of stimulation and the pick up electrodes, so as to decrease the stimulus artifact.

In determining the motor conduction velocity of the median or the ulnar nerve, the recording surface electrodes are placed with the active electrode over the belly of the small muscle of the hand, with the reference electrode over the tendon of the muscle. With a needle electrode, it is inserted into the muscle. Stimulus is delivered to the nerve at the wrist, elbow, and axilla, usually at the rate of one per second and the latency time recorded accordingly. The distance for each latency is then measured.

The conduction velocity is calculated as follows. (See Fig. 2.)

In the sensory nerve determinations, the author determines the antidromic sensory velocity. The recording surface electrodes are silver strips placed firmly round the fingers at a suitable distance apart. Stimulus is again applied on the nerve on the sites used for determining motor conduction velocity. When the sensory response is small, better visualisation can be achieved by superimposition of many sweeps on a photograph. Other devices for increasing the signal to noise ratio could be obtained by electronic averaging of multiple sweeps.

The method of measuring the conduction velo-

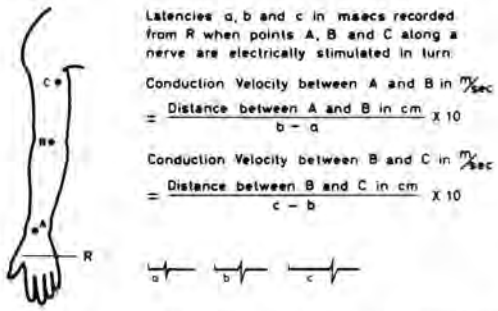

Fig. 2. DIAGRAM OF MOTOR STIMULATION AND RESPONSE AND METHOD OF CALCULATION OF NERVE CONDUCTION VELOCITY.

city of the lower limbs follows a similar technique.

Results

Out of a total of 110 clinical electromyographic examinations performed personally, 70 of the examinations were accompanied by nerve conduction studies.

In all the examinations, the motor nerve conduction velocity was measured. In about 20% of these, antidromic sensory conduction velocity was tested as well. A total of 112 determinations were performed on the 70 patients. 26 patients had abnormal conduction velocities.

Nerves	*Number*	*Number Abnormal*	*Percentage*
Median	29	10	34
Ulnar	33	6	18
Lateral Popliteal	28	10	35
Posterial Tibial	5	3	60
Facial	17	7	41
Total	112	36	32

In this series of examinations, the majority with abnormal conduction velocity correlated with clinical findings.

Case Reports

M.G., a 61-year-old married woman, was found to have cancer of the left breast and undergone a radical mastectomy 3 years ago. Six months prior to being seen, she started to complain of numbness and paraesthesia of the left hand, especially over the radial aspect. Examination revealed hypalgesia over the thumb. There was no evidence of metastatic recurrence of cancer. Electromyography of the hand muscles showed a few fibrillation potentials localised to the abductor pollicis brevis and a reduced interference pattern. Motor conduction velocity study showed a prolonged distal delay of 5.2 msec. (normal not more than 4.2 msec.) for the left median nerve, which had a normal proximal velocity. The conduction velocity of the left ulnar nerve was normal. These findings pointed to a Carpal Tunnel syndrome rather than a metastatic involvement of the plexus as originally feared.

G.S., a 50-year-old married male, a carpenter, complained of weakness of his left hand for the last five months. He had been in the habit of using his palm to drive chisels. There was no sensory disturbance. Physical examination revealed wasting and weakness of the right first dorsal interosseous muscle. No sensory changes were detected. Routine investigations, including radiological examination of the cervical spine, were normal. A provisional diagnosis of injury of the deep branch of the ulnar nerve was made. Electromyography showed evidence of neurogenic lesion affecting the muscles innervated by the deep branch of the ulnar nerve. Conduction velocities of the median and the ulnar nerves were as follows:

Nerves	*Median*	*Ulnar*
Distal Delay to 1st. Dorsal Interosseous	—	6.2 msec.
Distal Delay to Hypothenar Group		3.6 msec.
Distal Delay to Abductor Pollis Brevis	3 msec.	
Conductor velocity		
Axilla to Elbow	56m/sec.	55m/sec.
Elbow to wrist	70m/sec.	60m/sec.

Only the distal delay of the ulnar nerve to the first dorsal interosseous muscle was prolonged. It confirmed the diagnosis of injury of the deep branch of the ulnar nerve, a benign condition compared to amyotrophic lateral sclerosis which was the other diagnosis suggested.

K.K., a 74-year-old female, since one year ago complained of numbness and paraesthesia of the fingers of both hands, which she was unable to localise. Later the symptoms became worse. She also began to have vague aches and pains of the shoulders and elbows, especially at night. She dropped things at times. Examination revealed wasting and weakness of the thenar muscles. There was slight impairment of sensation over the thenar eminences and the lateral three fingers of both hands. X-ray of the cervical spine showed moderate cervical spondylosis. Electromyography showed fibrillation and positive dener-

vation potentials in both abductor pollicis brevis. Nerve conduction velocity study showed a prolonged distal delay of 7.5 msec. and 9.6 msec. for the left and right median nerves respectively as compared to a normal distal delay of not more than 4.2 msecs. A firm diagnosis of bilateral carpal tunnel syndrome was made and surgical treatment performed with good results.

W.S., a 40-year-old male working as a labourer, noticed gradual weakness of his left leg 6 months ago. His left foot began to catch the ground recently. As a result, he walked with a limp. There was no sensory symptoms. Five years ago, he had a car accident, but there was no serious injury, except he began to have pains on the lumbar spine now and then. Clinical examination revealed a left foot drop. There was weakness and wasting of the muscles innervated by the lateral popliteal nerve. No sensory loss was detected Myelography showed no abnormality. Electromyography and conduction study showed partial palsy of the left lateral popliteal nerve with a prolonged conduction velocity for the ankle-knee segment of the nerve, while the distal delay and the conduction rate for the popliteal fossa-knee segment of the nerve were normal. These findings were more in favour of a lateral popliteal nerve palsy instead of root compression syndrome from disc protrusion of the lumbar spine.

S.A., a 16-year-old schoolgirl, in 1968 began to have progressive weakness of the lower extremities. She found it difficult to walk. There were no sensory symptoms. No other members of the family had a similar illness. Clinically, she had bilateral atrophy and weakness of the foot muscles and the anterior tibial and posterior tibial muscles of the legs. The small muscles of the hands, especially the first dorsal interosseous muscles, were atrophied and weak. No objective sensory loss was detected. Laboratory investigations, including C.S.F. and enzyme studies for muscle disease, were normal. Electromyography showed evidence of a neurogenic lesion affecting the distal group of muscles of the lower extremities and the muscles of the hands. Conduction velocity study performed showed the following:

Nerves		
Left Lateral Popliteal (motor)	Knee to Ankle	30m/sec.
	Distal Delay	15.2 msec.
Left Posterior Tibial	Knee to Ankle	23m/sec.
	Distal Delay	8.4 msec.
Sensory	Distal Delay	11.2 msec.
Right Ulnar (Motor)	Axilla to Elbow	56m/sec.
	Elbow to wrist	44m/sec.
	Distal Delay	4.8 msec.

The result showed that the conduction rates of the left lateral popliteal, the posterior tibial and the right ulnar nerve were prolonged. The findings indicated a diagnosis of a chronic peripheral neuropathy which affected the lower limbs more than the upper limbs.

Discussion

The motor unit is a physiological concept, which consists of the motor neuron, its axon, the neuromuscular junction, and motor fibres, which the nerves innervate. The number of muscle fibres per motor unit varies from a few in the extraoccular muscles to about two thousand in some of the muscles of the lower limbs. (Feinstein, Lindergaard, Nyman and Wohfart, 1955) Fig. 3.

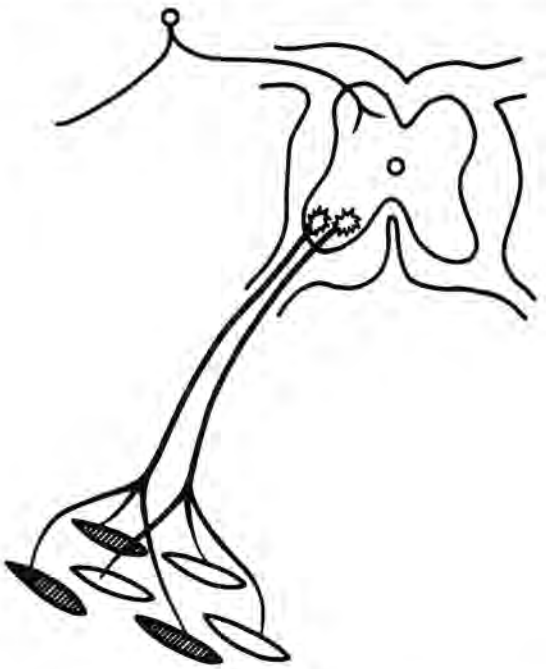

Fig. 3. TWO MOTOR UNITS

Neuropathic lesions of the motor unit can affect either the anterior horn cell or the nerve fibre, anywhere along its course. Myopathic lesions involve the neuromuscular junction or the muscle fibres. In the majority of patients, clinical electro-

myography easily distinguishes the myopathic lesion from the neurogenic lesions. When the peripheral neuropathy affects mainly the motor components of the nerve fibres, it may be difficult to distinguish this from a chronic myopathic lesion clinically and with electromyography. Motor nerve conduction study may be of great help on such occasions. With electromyography alone, it is often difficult to separate those neuropathic conditions affecting primarily the nerve fibres (Case 5) from those affecting the lower motor neuron. Conditions such as poliomyelitis, amyotrophic lateral sclerosis and other spinal cord lesions do not alter the nerve conduction velocity appreciably. Infective polyneuropathy, diabetic neuropathies, pressure and traumatic neuropathies causing damage to the peripheral nerves decrease the nerve conduction velocities.

Measurement of the nerve conduction velocity is a relatively simple and quick method of differentiating the two groups of neurogenic lesion. Localised lesions along a nerve can be frequently identified and localised with this technique. Examples of such localised lesions are the compression of the median nerve beneath the flexor retinaculum at the wrist producing the carpal tunnel syndrome, and pressure of the ulnar nerve at the wrist or at the elbow. At the level of the wrist, if the deep branch of the ulnar nerve is affected, the sensory conduction velocity to the little finger is not affected. In carpal tunnel syndrome (Cases 1 and 3) or in a lesion of the deep branch of the ulnar nerve (Case 2), the distal delays from the wrist to abductor pollicis brevis and the first dorsal interosseous muscle respectively are prolonged. In localised neuropathies at the sites, such as the pressure of the ulnar nerve at the elbow or pressure of the lateral popliteal nerve at the neck of the fibula (Case 4), stimulating proximal to the lesion will give reduced conduction times. Stimulation, distal to the lesion, will reveal a normal nerve conduction velocity.

In lesions of the brachial plexus and the cervical roots, differentiation is more difficult. The presence of normal evoked sensory potential of the median and ulnar nerve after a traction injury of the brachial plexus is suggestive of the roots being avulsed (Bonney and Gilliatt, 1958). Diseases of the spinal cord generally do not decrease the nerve conduction velocities, (Henriksen, 1956; Gilliatt, 1961; Ertekin, 1967).

In polyneuropathy of varying etiology such as infective, toxic, nutritional, metabolic and hereditary neuropathies, the nerve conductions may be prolonged. Chronic neuropathies, like peroneal muscular atrophy and hypertrophic polyneuropathies, may have marked slowing of the nerve conduction, (Dyck, Lambert and Mulder, 1963; Thomas and Lascelles, 1967). In diabetic neuropathy, studies by Mulder, Lambert, Bastron and Spraque (1961), in 108 diabetic patients, showed that nerve conduction velocities are slowed not only in those with clinical neuropathy but also in some without clinical evidence of the neuropathy. Others, like Lawrance and Locke (1961), Downie and Newell (1961), have confirmed these results.

Conclusion

Studies of the nerve conduction velocity have become an integral part of clinical electromyography. The technique is simple and rapid. It is reliable and useful to physicians and surgeons alike. The studies provide objective values and they therefore facilitate the management of patients with neuromuscular disease. They may also be of help in functional disorders seen in clinical practice and in medico-legal situations.

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