2. Electromyography as an Aid to Diagnosis

By A. T. RICHARDSON, M.B., B.S., D.PHYS.MED.
Registrar, Department of Physical Medicine, St. Thomas's Hospital

Electromyography is a diagnostic procedure by which the electrical activity (action potentials) of voluntary muscle can be studied either at rest or during contraction. The alterations in these action potentials, which occur in association with lesions of the lower motor neurones or of the muscle fibres themselves, are readily detectable by the modern electromyograph. Its use is therefore a valuable adjunct in the clinical diagnosis of diseases of the neuromuscular system.

Recent technical developments in the field of electronics have made available clinical electromyographic equipment which is simple and yet accurate (Fig. 1). The apparatus consists of two types of detection electrodes (surface and needle), a high gain amplifier and a display unit. The display unit contains a meter measuring the average voltage of the detected muscle action potentials and a cathode ray tube to enable visual interpretation or photography of them. The output from the amplifier is also applied to a loud speaker to allow recognition of the potentials by sound. It is expedient to use apparatus incorporating a nerve stimulator and allowing estimation of the duration of the muscle action potentials, conveniently, by comparison with a standard calibration wave.

Electromyography with the use of surface (skin) electrodes allows estimation of the integrated electrical activity derived from the ill-defined mass of muscle underlying the electrodes. By placing these electrodes in comparable positions on suspected and normal contralateral muscles and measuring the electrical activity on maximal voluntary contraction, supramaximal nerve trunk stimulation and tendon jerk response, an index of the number of intact motor units in the suspected muscle can be obtained. Serial measurements of motor unit activity on nerve trunk stimulation may be used to follow recovery in peripheral nerve injuries (Hodes, *et al.*, 1948). Similarly the progressive decline in muscle electrical activity on repeated stimulation of the nerve in myasthenia gravis offers a quantitative measurement of the effects of the disease and of any response to treatment (Fig. 2). Electrical activity accompanying spontaneous muscle action such as that occurring in motor neurone disease (fasciculation) and in motor nerve irritation by prolapsed intervertebral discs, etc., is detectable by surface elec-

trodes but is more accurately investigated by the use of needle electrodes.

Of more value in diagnosis is the detection, localization and analysis of individual muscle electrical potentials by means of needle electrodes. Of the several varieties of needle electrodes available the coaxial or concentric hypodermic needle electrode is very suitable for general use. This needle with its limited detection range, allows localization of the potentials and consequent accurate localization of any pathological lesions. When using needle electrodes in the analysis of action potential wave form characteristics, care should be taken to move the needle into a position which detects the potential changes maximally. This minimizes distortion of the wave by passage through the tissues before pick-up. Care should also be taken to analyse only repetitive potentials and thus avoid the analysis of complex wave forms produced by fortuitous interference by two or more simple potentials. Because the individual potentials constituting the complex wave forms generally have different frequencies they soon pass out of phase and the interference complex wave form is not repeated (Fig. 3).

The action potentials which may be detected in muscle are conveniently divided into those occurring on voluntary effort and those occurring spontaneously or on mechanical stimulation of the muscle, e.g. by movement of the needle electrode in the muscle.

FIG. 1.—A modern double-channel electromyograph.
(Photograph by permission of Stanley Cox Ltd.)

Fig. 2.—Muscle potential curves obtained on supramaximal stimulation of the nerve trunk in myasthenia gravis at the start of contraction and after 1 min. (stimulation rate, 10 per sec.).

Fig. 3.—Complex wave form (X) produced by simultaneous detection of fibrillation potential (Y) and motor unit potential (Z).

Fig. 4.—The normal interference pattern detected by needle electrode on maximal voluntary contraction of a normal brachioradialis muscle (milliseconds time scale).

Volitional Muscle Action Potentials

(a) Normal Motor Unit Action Potentials

In normal muscle in full relaxation no electrical activity is detectable electromyographically by a stationary needle electrode. Insertion of the needle electrode into normal muscle provokes an outburst of motor unit potentials; these have been called, by Weddell, motor unit insertion potentials. On voluntary contraction repetitive normal motor unit action potentials occur at increasing frequencies up to an average maximum of 20 per sec. (Weddell, 1944), and in increasing numbers with developing tension. On full voluntary contraction the many asynchronously contracting motor unit potentials form the normal interference pattern. This is characterized by a low rumbling sound in the loud speaker and a completely disturbed base line on the cathode ray tube screen (Fig. 4). The individual motor unit potential has a duration in the average limb muscle of 5 to 10 milliseconds, and an amplitude of 100 microvolts to 1 millivolt (Weddell, 1944). As detected by a coaxial needle electrode the motor unit potentials are generally diphasic or triphasic in form (Fig. 5a, b). Triphasic forms when detected at a distance appear monophasic on account of failure to differentiate the small first and third phases from the base line at low amplitudes. The normal polyphasic motor unit action potentials (Fig. 5c) are detected infrequently in limb muscles but more frequently in the facial muscles. The formation of normal motor unit action potentials is apparently due to the polarization changes accompanying the almost synchronous contraction of the muscle fibres of one motor unit (i.e. all the muscle fibres supplied by one lower motor neurone).

Failure to obtain the normal interference pattern on maximum volition almost always indicates a myelopathic (cord) or neuropathic (peripheral nerve and root) lesion. These are in contrast with myopathic lesions which are confined to the muscle fibres themselves in which, although the individual motor unit action potentials may be reduced in duration and there is an increased incidence of polyphasic potentials, an interference pattern is obtainable except in the late stages of the disease (Kugelburg, 1949; Buchthal, 1941). The detection of volitional motor unit action potentials after peripheral nerve injuries, indicating an incomplete nerve lesion, is of obvious importance in diagnosis and prognosis.

In neuropathic lesions the failure to obtain an interference pattern may be due to disturbance of nerve conduction (neuroparaxia) or to degeneration of the nerve. Lower motor neurone degeneration gives rise to spontaneous muscle fibre activity which is detected electromyographically as 'fibrillation action potentials' (*vide infra*).

(b) 'Spike' and Complex Volitional Potentials

Regeneration of a lower motor neurone is often detectable electromyographically long before clinical signs of recovery are apparent. In neuronal regeneration the muscle fibrillation potentials indicative of denervation become reduced in number, and highly polyphasic (complex) potentials may be detected on attempted voluntary contraction (Weddell, 1944) (Fig. 6). These complex volitional potentials are often of long duration and produce a characteristic rough sound in the loudspeaker by which they are readily distinguished. As well as complex potentials, short

Fig. 5.—Normal motor unit action potentials. (a) Diphasic. (b) Triphasic. (c) Polyphasic. (Milliseconds time scale.)

duration (1 to 2 milliseconds) 'spike' potentials may also be detected on attempted voluntary contraction.

Spike and complex potentials detectable on voluntary contraction during regeneration of nerve are due to the contraction of the recently re-innervated motor units. The complexities and temporal dispersion are probably due to variations in the conduction rate of the pre-terminal fibres of the recovering axon or variations in the transmission at the myoneural junctions (Weddell).

Loss of muscle fibre activity within a motor unit, such as may occur in myopathic lesions and on fatigue in myasthenia gravis, is responsible for the higher incidence of polyphasic motor unit potentials in these conditions (Fig. 7).

Spontaneous Muscle Action Potentials

(a) Fibrillation Potentials

These small (amplitude less than 100 microvolts) potentials of 1 to 2 milliseconds duration may be detected in muscle about three weeks after denervation (Fig. 8). They occur spontaneously when they are heard as regular 'clicking' sounds at a frequency of 2 to 20 per sec., or in showers on insertion of the needle electrode into the denervated muscle (Weddell, 1944). They are most numerous when the muscle is warmed, after the injection of prostigmine and during active physiotherapeutic treatment (e.g. interrupted galvanism). They are detectable for long periods after denervation and their detection, denoting as it does denervated muscle tissue with contractile elements still present, is a most valuable sign. Fibrillation potentials are considered to be due to the spontaneous contraction of individual muscle fibres.

(b) Spontaneous Motor Unit Activity

The spontaneous muscle fasciculations of motor neurone disease (amyotrophic lateral sclerosis and progressive muscular atrophy) result in electrical disturbances characteristic of motor unit twitches. The activity is apparently due to spontaneous impulses in abnormal motor units (Denny-Brown, 1938), and there is evidence to indicate a peripheral origin for these impulses.

Irritation of nerve roots or nerve trunks by cervical ribs, etc., may give rise to spontaneous motor unit activity. This finding is, however, inconstant even in the presence of marked neurological signs. In the cervical region, particularly, it may be of value in localizing the point of irritation.

(c) Trains of Oscillations

Trains of high frequency potentials may be obtained on insertion of the needle electrode into affected muscles in dystrophia myotonica, less commonly in motor neurone disease and rarely in other conditions. The duration of the potentials is variable; in dystrophia myotonica some are of the duration of motor unit potentials; others are much shorter. These electrical discharges are remarkable for their change in pitch which causes them to sound like a diving aeroplane, their frequency may be 100 to 150 per sec., dropping to 20 per sec. (Buchthal, 1941). In dystrophia myotonica they have been obtained after curare has been given to block neuro-muscular transmission; their origin is unknown. In dystrophia myotonica, in addition to these trains of potentials, voluntary contraction is followed by a fibrillary after-discharge. Spontaneous fibrillation which occurs

Fig. 6.—Complex motor unit potential. Associated with reinnervation (facial palsy) (milliseconds time scale).

Fig. 7.—Motor units in myasthenia gravis after fatigue (milliseconds time scale).

Fig. 8.—Fibrillation action potentials (milliseconds time scale).

in motor neurone disease, etc., is not detectable in dystrophia myotonica.

Synchronization of Motor Unit Activity

In normally innervated muscle the contraction of motor units is asynchronous, thus providing for the smooth contraction of voluntary muscle. Thus if two needle electrodes of a double channel electromyograph with differential amplifiers are inserted into two different motor units the timing of the motor unit potentials appears out of phase. In myelopathic lesions (notably acute anterior poliomyelitis) Buchthal and Clemmesen (1943) described synchronous activity of motor units detectable by three needle electrodes. This, they suggested, was due to the spread of nervous impulses to contiguous neurones. An alternative explanation has been suggested by Denny-Brown (1944), who has suggested that apparent synchronization is due to the detection by two or more needle electrodes of the same, often large, motor unit due to uncovering of this unit by loss of the small motor units. Whatever may be the explanation the occurrence of 'synchronization' in myelopathic lesions and its rarity in normal muscles or in peripheral nerve lesions is undoubted.

To sum up, therefore, clinical electromyography, although in its infancy, is of proven value in peripheral nerve lesions in that it can provide evidence of nerve damage, recovery and retention of any function before these phenomena can be estimated clinically. Although less is known about the electromyographic appearance in other diseases, the detection and recognition of potentials and their relative preponderance yields information unobtainable by other methods. It is usefully

combined with the use of intensity-duration curves described elsewhere in this issue.

I wish to thank Dr. P. Bauwens, physician in charge of the Department of Physical Medicine, St. Thomas's Hospital, for permission to publish the pictures of muscle potentials illustrating this article, which were taken from departmental records. They were obtained by photographing cathode ray tube traces of the muscle potentials after recording on magnetic tape.

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3. Case Report:

Facial Nerve Palsy

By B. I. EAMES, M.B., CH.B., D.L.O.

Royal National Throat, Nose and Ear Hospital,
Golden Square

A man, aged 61, was first seen in May 1948, giving a history of discharge from both ears since 1917, when he was torpedoed, with recent pain in the right ear, unsteadiness and a right facial palsy in the lower half of the face which had been present