Electromyography As A Routine Clinical Service

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INTRODUCTION

ELECTROMYOGRAPHY (E.M.G.) has been described as a procedure that "extends the physician's senses," but it is emphasized that the evidence so obtained must be evaluated like any other clinical sign. The value of the EMG depends largely on an association between certain electrophysiological changes and disturbances affecting either muscle fibers or the Lower Motor Neurone. Clinical deductions may be made, largely empirical, on the basis of these associations in a fashion similar to the use made of the electrocardiogram or electroencephalogram.

The object of this paper is to help the physician to decide when an EMG is likely to be of assistance and to appreciate the type of information it can provide in different situations. No attempt is made to cover the fundamentals of electromyography since excellent reviews are available, in addition to text books on the subject.

As a basis for discussion the electromyograms performed here in 1964 have been reviewed and the case histories re-examined with a particular view to correlating surgical findings with the electromyographic diagnosis. The majority of abnormal EMG's had been recorded on tape and if there was disagreement with the clinical findings the EMG was re-examined and re-photographed.

MUSCLE DISEASE

In the investigation of a case of suspected muscle disease the EMG has the advantage that numerous sites may be sampled in distinction to the obvious limitations imposed by a biopsy. The EMG needle records the electrical activity from a distance of about 1 cm. beyond its tip, and a minimum of at least six different areas of each muscle tested are sampled routinely. Interpretation of the characteristics of action potentials seen on an oscilloscope and heard on an amplifier is necessarily subjective to a degree and accounts for some discrepancies between clinical diagnoses and EMG findings. It is possible to render the test more objective by increasing the number of areas sampled and by photographing individual potentials.

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*A Meditron Model 201 A-1 single channel electromyogram with monopolar needle electrodes and a Polaroid camera attachment was used.
and measuring them, but this is so time consuming that it is mainly used as a research tool. Difficulties in interpretation are more likely to occur in the earlier stages of a disease when other tests are also more likely to be equivocal.

Comparisons have been made between biopsy and EMG findings to assess the validity of the investigation by different authors. Where confirmation of the clinical diagnosis has included a positive muscle biopsy there has been a close correlation between the EMG and the clinical picture. It is known that in polymyositis the biopsy is abnormal in only half the cases and characteristic of the disease even less frequently, the diagnosis in these other instances resting on a combination of findings. One of these findings is an abnormal EMG which may indicate only a non-specific myopathy or may have additional features suggestive of polymyositis. It is in these cases of polymyositis with a normal biopsy that the EMG may be of most value; yet, prolonged clinical observation may be needed to confirm the validity of the test.

**INDIVIDUAL MUSCLE DISEASES**

The present clinical diagnoses of the patients on whom EMG's were performed in 1964 are shown (Table I). The cases of myofascial syndrome (fibromyositis, myalgia) were characterized by EMG's that were within normal limits (except in one case which was associated with Raynaud's syndrome and an abnormal creatine excretion in which a long term follow-up will be of interest). These cases occurred mainly in middle aged women and were characterized by myalgia dominating the picture with muscular weakness being more apparent than real because of the pain. Other investigations were mostly normal. The clinical problem is usually that of excluding polymyositis, and a normal electromyogram is of help.

Although no cases of polymyalgia rheumatica have been recognized, this condition may also be confused with polymyositis and is also characterized by a normal electromyogram.

The acquired myopathies can roughly be divided into those that are associated with collagen diseases or neoplastic disorders and those that are unrelated. The former include: (1) Those in which myositis dominates the picture, although other features of connective tissue disorders may also be present. (2) Cases in which other diffuse collagen disorders such as systemic lupus or systemic sclerosis dominate the picture and muscle involvement is less striking and, (3) Least well defined is rheumatoid arthritis with associated muscular wasting and weakness. The other main group includes infective and parasitic causes and the various myopathies found in conjunction with endocrine disturbances.

Six cases of confirmed polymyositis were seen, all of which had abnormal EMG's. It is of interest that in three of these the multiple areas sampled showed a patchy distribution of changes, frankly abnormal areas being adjacent to muscle with a completely normal pattern. It is this abrupt and frequent variation of the
EMG changes within small areas of the same muscle that emphasizes how much a positive biopsy result is a matter of chance — and it is perhaps surprising that as many as 50 per cent are abnormal. It is due to the lack of any consistently positive specific test for polymyositis that an abnormal EMG pattern, particularly one showing the additional features of myotonic discharges and fibrillation-like potentials, can be of help in the difficult case. While the negative biopsy may be frustrating, it is well to recall that a biopsy taken from along a needle track — including that of the EMG needle — can produce an inflammatory reaction that may be confused with polymyositis. It is a wise precaution to biopsy a muscle that has not been needled, or, alternatively, to ensure that the biopsy is carried out immediately following the EMG.

Weakness and wasting of voluntary muscle in patients suffering from rheumatoid arthritis in whom there is no evidence of peripheral nerve compression is a frequent clinical finding and the reason for this is still in doubt. Because the changes are maximal in relation to affected joints, particularly those in the hands, it has been suggested that disuse atrophy may be the cause. The association between rheumatoid arthritis and polymyositis together with positive EMG findings has led others to conclude that myositis frequently complicates rheumatoid arthritis. Nodular myositis as a histological finding occurs more frequently in cases of rheumatoid arthritis than normal subjects, and it is possible but unlikely that this nodular “myositis” is responsible for the EMG changes described. Abnormalities are also seen more often in rheumatoid patients receiving cortico-steroids, but this treatment may simply reflect the increased severity of the underlying disease. Only five patients with this

Table I

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<th>Muscle Disorders.</th>
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<td>Disorders of the Lower Motor Neurone</td>
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<td>Root and proximal nerve compression syndromes</td>
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Clinical diagnosis of 158 consecutive patients.
problem were seen here in the past year and two of them showed myopathic changes from both the deltoid and biceps muscles; the value of electromyography is to confirm the presence of myopathic changes in the face of rheumatoid disease, even if the reason for this remains in doubt.

Other collagen diseases may have accompanying myositis, as occurred with five other cases including three confirmed instances of systemic lupus erythematosus.

Acquired myopathies of other causes include those associated with thyrotoxocosis which may clinically resemble polymyositis and rarely with thyroid deficiencies. Myopathies may also be seen with Addison's disease and Cushing's syndrome, and various steroids, particularly Triamcinolone and Decamethasone, have been incriminated. Hyperparathyroidism gives rise to muscular weakness in some cases, but the histologic and EMG findings are normal.

In the hereditary dystrophies, including that associated with myotonia, the place of electromyography is largely to confirm the diagnosis, although clinical differentiation from a subacute neuropathy or chronic polymyositis may be difficult without electromyography. Since the outlook is so gloomy it is reasonable to confirm thoroughly the diagnosis and in occasional cases a muscle biopsy may be equivocal.

While the diagnosis of myasthenia gravis is typically confirmed by the use of a short acting anticholinesterase (Tensilon) in instances where the response is doubtful, electromyography may allow objective assessment. A falling out of the number of action potentials and progressive reduction in the duration and amplitude of the remainder may be seen in the fatigued muscle which can be reversed by Tensilon. In addition, in doubtful cases, other pharmacologic methods of diagnosis are used. Rooke has also described a myasthenia-like syndrome associated mainly with lung carcinoma with suggestive clinical and electromyographic features. In this condition superamaximal stimulation with a single stimulus to a rested muscle produces a response that is smaller than usual: at frequencies of over 10 per second, the amplitude is enhanced. These findings differ from myasthenia gravis where the initial response is normal and potentiation does not occur at higher frequencies. Recognition is valuable since an intensive search for an intrathoracic tumor is then indicated, and also the condition responds poorly to conventional treatment of myasthenia.

**DISEASES OF THE LOWER MOTOR NEURON**

Electromyography, together with nerve conduction studies, finds its other main application in diseases of the lower motor neuron. Evidence of weakness of neurogenic origin, sometimes with features suggesting that the origin is in the proximal part of the lower motor neurone, may be seen. Consideration of the anatomical distribution and the clinical picture frequently allows inferences to be drawn regarding either the etiology or prognosis.

*Motor Neurone Disease.* In degenerative disease of the motor neurone in its early stages, particularly if fasciculations or wasting of the small muscles of the
hand are the only obvious features, diagnosis may be difficult. Involvement of the lower motor neurone at numerous different cord levels may be shown by EMG with a fallout of action potentials and evidence of denervation together with abnormally large motor units. Marked slowing of the motor nerve conduction velocity is not found in motor neurone disease and this if present tends to make the diagnosis unlikely. Providing root compression at multiple levels (as in extensive disc disease), can be excluded, these features may suggest the diagnosis and save the patient multiple and prolonged investigations. It must be emphasized that other less common degenerative disease of the anterior horn cell such as Jakob Creuzfeldt disease can produce an identical EMG picture. Benign fasciculations are characterized by an electromyogram that is normal in all other respects.

Three “floppy infants” were examined. Two had infantile spinal muscular atrophy (Werdnig-Hoffman syndrome) and both showed evidence of denervation; both have since died. One further infant was thought to have weakness of myogenic origin, and both the EMG and muscle biopsy failed to show evidence of denervation. This clinical problem has been reviewed in detail by Walton and others; the distinction between the two causes of muscular weakness is important because of the difference in prognosis, that due to neurogenic weakness usually being steadily progressive.

**ROOT AND PROXIMAL NERVE COMPRESSION SYNDROMES**

Mechanical compression of a nerve root will, after an interval of approximately three weeks, produce electromyographic evidence of denervation in the muscles supplied. This principle has lead various authors to perform EMG’s on patients with root compression syndromes, mainly due to disc disease and also to compare the results with those of myelography, using the operative findings as a criterion of accuracy.

Both methods are found to have about the same margin of error and the EMG is found to be accurate in 75-90 per cent of cases. By either method a minority of cases will be misleading, but it is of interest that a few cases with negative myelograms may have evidence of denervation by electromyography. It is not suggested that one method should replace another, but rather it is emphasized that in problem cases the EMG is a valuable complementary test. It has the draw back that there is a time lapse before it becomes positive and also that thorough sampling of the muscles of an affected limb involves extensive needling. For these reasons, in the straight forward case, particularly if a myelogram is to be carried out routinely prior to surgery, mere additional documentation is of little value.

**PERIPHERAL NEUROPATHY**

The essential feature of a neuropathy is the failure of the normal transmission of a nerve impulse. A delay in transmission may be suggested electromyographically by the increased number of polyphasic potentials and by delayed conduction velocities
following nerve stimulation. Evidence of interruption in the transmission of a nerve impulse is the presence of spontaneous denervation activity, and a fallout in the total number of action potentials and in addition a complete block to electrical stimulation of the nerve can sometimes be demonstrated.

These changes very roughly parallel the severity and course of the disease. In cases of the Guillain-Barré syndrome the majority show decreased motor conduction velocities, at least after the first few weeks of the illness together with accompanying EMG changes; these changes may occur when objective clinical signs are slight or confusing. A few cases, however, that are clinically severely affected may have normal conduction velocities. In these a possible explanation is that the main lesion is a radiculopathy rather than a diffuse neuropathy. The converse has been reported in which patients with suspected mononeuritis may show involvement electromyographically of apparently unaffected limbs. Slight slowing of nerve conduction velocity in the absence of overt neurological changes has also been described in alcoholism and uremia.

In diabetes slowing of both motor and sensory nerve conduction velocities is reported, although this change is less marked than with the Guillain-Barré syndrome. While the reduced motor conduction velocity is statistically apparent, it is often not enough in an individual case to fall below the limits of normal variation. As might be expected the slowing is greater in diabetics with a clinically apparent neuropathy and the EMG features common to a peripheral neuropathy will be present when motor nerve fibers are involved.

The syndrome of diabetic amyotrophy was initially thought to be due to involvement of the spinal cord, producing an asymmetric limb girdle weakness with wasting, accompanied by pain and occasionally by pyramidal tract signs. Rarely the condition may mimic amyotrophic lateral sclerosis and demonstrate the fasciculations that are occasionally described in diabetic neuropathies. Electromyographically the findings suggest a myopathy; in addition there also may be evidence of a coexistent neuropathy. This paradox of an apparent “myopathy” in a condition that otherwise is not associated with primary disease of muscle is the basis of the electromyographic diagnosis of the syndrome. Although the existence of diabetic amyotrophy as a clinical entity is disputable, the histological appearance of atrophy of scattered single muscle fibres suggests a disturbance at the nerve terminals leading to a fallout of individual muscle fibres within the motor unit.

Why electromyography is found in some instances to anticipate clinical changes but in others only to follow later is poorly understood. Simpson emphasizes that the problem is fundamentally biochemical. He has presented some evidence that metabolic disturbances in the axons, particularly electrolyte changes, are less likely to alter the speed of transmission of nerve impulses compared with disorders of the myelin sheath. It is probable that multiple segments of the sheath need to be affected before the conduction velocity is reduced.
ELECTROMYOGRAPHY

Peripheral, Mechanical and Traumatic Nerve Lesions

For purposes of prognosis nerve injuries may be divided into those with a physiological block to the transmission of a nerve impulse and those with an anatomical block. Electromyographically the former is characterized by the absence of spontaneous denervation activity and, on nerve stimulation a complete block to conduction is not present. In cases with an anatomical block, after an appropriate interval, neuronal degeneration will lead to the electromyographic features of denervation and a conduction block on stimulation of the nerve.

While in a given situation a combination of the two types of injury may exist, one will usually predominate. In general, recovery in a matter of weeks is the rule for injuries characterized by a physiological interruption of the nerve impulse. If recovery is mechanically possible following anatomical interruption of nerve fibres, it will only proceed at a rate of regeneration of those fibres.

If any nerve fibres are functioning under voluntary control, even when not apparent clinically, this can be demonstrated by electromyography. Such evidence that at least part of the nerve is intact may influence a surgeon to await further recovery rather than perform an exploratory operation. The presence of Wallerian degeneration in a nerve can be demonstrated, and if the site of nerve injury or compression is known, the earliest possible time for recovery may be calculated. Evidence of regeneration of a nerve may be anticipated electromyographically before clinical recovery is apparent.

The symptoms resulting from peripheral nerve entrapment can largely be anticipated from a knowledge of their anatomy and the site of compression, but acroparesthesiae may present a particularly difficult diagnostic problem. Cervical root pain, brachial neuritis, the thoracic outlet syndrome and Raynaud's disease are the more common causes of confusion with the syndrome of median nerve compression at the carpal tunnel.

It has been established that abnormal motor conduction latencies are present in two-thirds of patients with carpal tunnel syndromes. The percentages of abnormal findings may be increased by using threshold stimulation techniques or concomitant routine electromyography and possibly by sensory conduction velocity estimations. A few cases, however, particularly the mild ones, will have no demonstrable EMG changes, and it is in these that the diagnosis still has to be made on clinical grounds. The extent of the problem is illustrated by the fact that of the last 10 patients referred because of acroparesthesiae only two had electromyographic evidence of median nerve involvement. Two others had surgical decompression of the median nerve carried out on clinical grounds but at operation, the nerve did not appear swollen and there was no relief of the symptoms following surgery.

A special form of compression neuropathy is Bell's palsy in which the facial nerve within the bony canal is subjected to pressure with no history of trauma. The natural history of the condition is that 75-85 per cent recover spontaneously. Attempts
at treatment must therefore be directed towards the minority who do poorly. Electromyography is of value in selecting those cases with a poor prognosis. It has been shown that a complete block to stimulation of the facial nerve at its exit from the sternomastoid foramen is associated with only a 20 per cent recovery rate if carried out after an interval of over five days from the onset. A raised threshold to stimulation compared with the unaffected side is associated with an intermediate prognosis. Other factors including the presence of any potentials under voluntary control and fibrillation activity are also related to the eventual outcome. Steroids and cervical sympathetic block have been tried but not shown conclusively to be of value in treatment. Decompression of the facial nerve within the bony canal may be valuable in those patients who are unlikely to recover, although it must be admitted that no controlled trial of this form of treatment has yet been carried out and the optimal time for surgical intervention is still not settled. However, in the light of present opinions regarding the appropriate time and criteria for surgical intervention, EMG should at least be performed once between the first and fourth weeks after onset of the palsy, particularly if spontaneous improvement is not occurring. This will allow a probable prognosis to be given, and patients suitable for operative treatment to be chosen.

CONCLUSION

An attempt has been made to place electromyography in perspective. Emphasis has been placed on its practical application as a clinical aid to diagnosis or management when it is relevant and its position in relation to other established tests has been discussed.

REFERENCES