ELECTROMYOGRAPHIC CHANGES IN RHEUMATOID ARTHRITIS

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Muscular wasting is a prominent feature of rheumatoid arthritis. The exact reason for this has never been adequately explained. The idea that it is merely secondary to joint involvement or to disuse does not fit the facts, for the wasting may be out of proportion to the joint involvement, and may be found in muscles around joints that retain a full range of movement. Muscle pain and stiffness are prominent features of rheumatoid arthritis, and, indeed, it is well known that such symptoms may precede joint involvement by many years.

Histological changes in the muscles have been described by several workers, but it is now agreed that these changes are non-specific and certainly not diagnostic of rheumatoid arthritis. Curtis and Pollard (1940) found perivascular infiltration of lymphocytes in the muscles; Steiner *et al.* (1946) performed muscle biopsies on seven cases of rheumatoid arthritis and found inflammatory lesions in every case, calling the lesion "nodular polymyositis." These findings have been confirmed by other workers, and, if the results are analysed, positive biopsies occur in 40–100% of cases, depending on the series. While the histological changes of polymyositis with necrosis are distinct from the nodular polymyositis of rheumatoid arthritis, it must be remembered that Steiner *et al.* also demonstrated various stages of degeneration and atrophy to be present in rheumatoid muscle.

Recently the concept of polymyositis has attracted attention, and, while muscle-wasting and pain are the prominent features of the condition, joint symptoms and signs are frequent and a rheumatoid type of polyarthritis may also be present (Richardson, 1957; Pearson, 1959). Polymyositis may be a primary condition, but it is usually secondary to such conditions as thyrotoxicosis, carcinoma, the collagen diseases, and cachectic conditions. The clinical picture, histology, and electrodiagnostic findings are similar no matter what the cause of the polymyositis. These patients present with proximal muscle weakness, wasting, and pain, with or without a rash. The electrodiagnostic changes are those of a combination of a primary muscle (myopathic) lesion showing a full interference pattern—small-amplitude broken-up short-duration polyphasic action potentials on the electromyograph—and those of a lower-motor-neurone lesion (denervation)—abnormal intensity-duration curves, and long-duration polyphasic potentials on the electromyogram. The myopathic element usually predominates, and motor-nerve-conduction times are normal, showing that the lesion is at the distal end of the axon.

We decided to carry out electrodiagnostic investigations in a large series of patients with rheumatoid arthritis to see if this method would throw any light on the cause of the muscular wasting, and if in fact it could be explained by the occurrence of a "rheumatoid polymyositis."

Method

Patients attending the department of physical medicine of the London Hospital with a diagnosis of definite rheumatoid arthritis (Ropes *et al.*, 1956) were examined in a special electrodiagnostic clinic. Their disease activity was assessed and graded according to the method of Duthie *et al.* (1955). None of these patients had any clinical evidence of a rheumatoid neuropathy, as shown by sensory loss or severe motor weakness (Hart and Golding, 1960; Steinberg, 1960) at the time of the investigation. The activity and range of movement of the neighbouring joint was noted, as was the degree of wasting and power of the selected muscle (power was graded 0–5). The steroid history of the patients was recorded. An agglutination test was performed in every case (sample lost in two). The muscles selected were the small muscles of the hand, the deltoid, biceps, and quadriceps. The muscles were chosen for one or more of the following reasons: (1) they were the site of wasting, (2) they were near affected joints, (3) proximal muscles are a characteristic site of polymyositis, and (4) muscle pain was present.

Intensity-duration curves were plotted on the selected muscles, using an R.A.F. constant-voltage stimulator (Wynn Parry, 1953). The rheobase (intensity at 100 m.sec.), the type of muscular contraction obtained, and the shape of the curve were all noted. Electromyography, using concentric needle electrodes, was then carried out on the same muscle, or muscles, using a GHS electronic double-beam electromyograph. Any spontaneous activity was first studied. The interference pattern on volition was then observed, and the type of unit was measured and classified as normal or polyphasic, the latter being subdivided into normal (5–9 m.sec. duration), long (10 m.sec. or over), or short duration (less than 5 m.sec.). The amplitude of the unit was also measured. Motor-nerve-conduction times were estimated in 10 cases.

Results

A total of 93 patients were examined and classified according to the above methods. The electromyograph was thought to be the most sensitive test, and 79 (85%) of these patients showed electromyographic evidence of polymyositis in one or more muscles. For the purpose of this paper the group of patients in whom evidence of polymyositis was found are called the "positive" group, and those with a normal E.M.G. the "negative" group. The composition of the groups is shown in Table I, and, as can be seen, the two groups are comparable regarding age, disease duration, and sex ratio. In the negative group either the sheep-cell-agglutination test or the latex test was positive in nine and negative in five. In the positive group these tests were positive in 61, negative in 16, and the test was

TABLE I.—*Composition of the Groups*

	E.M.G. Positive	E.M.G. Negative	Total
No. of patients	79	14	93
Male	25	4	29
Female	54	10	64
Average age (years)	53·8	54·8	54
Disease duration (years)	8·4	9·6	8·7

not performed in two of the cases (Table II). It seems that the agglutination tests are positive more frequently in the cases with polymyositis. The occurrence of polymyositis bore no constant relationship to the activity of the neighbouring joints or to the range of movement. In the positive group, 39% of the neighbouring joints were not active, and in the negative group nine of the joints were active (50%).

Relationship of Intensity Duration Curve to E.M.G. Findings.—Negative Group: 18 muscles were tested in 14 patients. In 16 muscles the rheobases and curves were completely normal, but in two the curves showed a slight kink with normal rheobases. In both cases the muscles were weak (power 3, different patients) and the joints were affected. *Positive Group:* 100 muscles showed E.M.G. evidence of polymyositis. The curves showed partial denervation in 37, of which 10 had high rheobases. The curves were normal in 63, of which 21 had high rheobases. The rheobase was regarded as high if above 50 volts in the deltoid or biceps muscles, and 60 volts in the quadriceps or small muscles of the hand. These values are conservative (Wynn Parry, 1953). It would appear that just over one-third of the E.M.G.-positive group show abnormal curves, and, in addition, a large number show high rheobases.

Quality of Response.—Of the negative cases, only one muscle showed a brisk-sluggish response with a normal curve. All the other muscles showed a normal brisk response, including the two partially denervated muscles. The quality of the response was analysed in the 100 muscles of the patients showing changes of polymyositis. When the curve showed partial denervation 22 muscles showed a brisk response, 12 a brisk-sluggish response, and only 3 a definitely sluggish response. Of those with a normal curve, 52 showed a brisk response and 11 a brisk-sluggish response. The quality of the response is not merely difficult to assess, but it is of no help (Wynn Parry, 1953).

Wasting and Weakness.—In the E.M.G.-positive cases, 81 of the muscles were graded as weak (power 3+ or less) and 19 showed normal power. In the E.M.G.-negative group 10 of the muscles were considered to be weak and 8 had normal power. 80 (80%) of the muscles in the E.M.G.-positive group were wasted, and in 60 of these the wasting was considered to be moderate or severe. There was no constant relationship between the degree of wasting and the weakness. In the E.M.G.-negative group six of the muscles were not wasted and 12 were.

Muscles Examined.—In the E.M.G.-positive group these changes were found in the following muscles: deltoid (50), biceps brachii (6), first dorsal interosseus (12), abductor pollicis brevis (10), and quadriceps (22). 15 other muscles in the E.M.G.-positive group showed no evidence of polymyositis; six of these were small muscles of the hands and the others were proximal muscles. In the negative group six of the muscles were the muscles of the hand; the others were proximal muscles. Thus 100 out of 133 muscles showed evidence of polymyositis.

Conduction Times.—Motor-nerve-conduction times were estimated in 10 of the E.M.G.-positive cases. This was normal in every case.

Relationship to Steroids.—Negative Group: 12 of the patients were not and never had been on steroids; only 2 had. Both of these patients had been on prednisolone, 10 mg. daily, for over one year. *Polymyositis Group:* Of the 79 patients with E.M.G. findings of polymyositis, 63 were not on steroids, and these agents had never been employed. In 15, A.C.T.H. or one of the steroids was currently being given at the time of examination, and, in one, prednisone had been discontinued 18 months previously. The steroid history of these patients is summarized in Table III. In only one patient had triamcinolone been employed, and then only for seven days. It was therefore unlikely to be responsible for the findings. There is thus no relationship between the finding of polymyositis and the use of steroids.

TABLE II.—*Clinical Details of the Groups*

Steroids

	Steroids	No Steroids	Total
E.M.G. negative ..	2	12	14
,, positive	16*	63	79

* 1 stopped 16 months previously.

Disease Activity

	Grade 1 Very Active	Grade 2 Moderately Active	Grade 3 Inactive	Total
E.M.G. negative ..	0	11	3	14
,, positive ..	19	56	4	79

Joint Activity

	Active	Not Active	Total
E.M.G. negative ..	9	9	18
,, positive	61	39	100

Agglutination Tests

	Test Positive	Test Negative	Total
E.M.G. negative ..	9	5	14
,, positive	61	16	77*

* Test not performed in two cases.

TABLE III.—*Details of the 16 Steroid-treated Patients*

Name	Sex	Steroid	Dose	Duration	Currently
M. M. ..	F	Prednisone	10 mg. daily	2 years	Yes
E. R. ..	M	,,	10 ,, ,,	18 months	No*
W. S. ..	F	Dexamethasone	2 ,, ,,	7 ,,	Yes
M. M. ..	F	Triamcinolone	4 ,, t.d.s.	7 days	..
D. H. ..	M	A.C.T.H. gel.	30 units daily	3 months	,,
F. R. ..	F	,,	30 ,, ,,	4 years	,,
J. R. ..	M	,,	40 ,, ,,	7 days	,,
K. W. ..	F	Prednisone	10 mg. daily	4 months	,,
E. B. ..	M	,,	15 mg.	2½ years	,,
C. C. ..	M	A.C.T.H. gel.	40 units daily	5 days	,,
A. C. ..	F	Prednisone	10 mg. daily	5 months	,,
N. Evans	F	,,	15 ,, ,,	1 year	,,
C. F. ..	F	A.C.T.H.	40 units daily	2 weeks	,,
B. H. ..	F	Prednisone	15 mg. daily	7 months	,,
A. G. ..	M	,,	10 ,, ,,	2 ,,	,,
W. F. ..	M	,,	10 ,, ,,	7 ,,	,,

* Discontinued 18 months previously.

Relationship to Disease Activity.—The patients were classified into three grades of disease activity according to the method of Duthie *et al.* (1955). The relationship of the disease activity to the E.M.G. findings is shown in Table II. It can be seen that not one of the negative group was in grade I, but 19 of the positive were; 11 of the negative and 56 of the positive were in grade 2; 3 of the negative and 4 of the positive were in grade 3. There is thus some relationship between disease activity and E.M.G. findings, although the findings, if positive, did not vary significantly in the different grades.

Discussion

Characteristic E.M.G. changes of polymyositis were found in 79 of the 93 patients examined. If other muscles had also been examined in the negative group the figure for the positive group might have been even

higher, for in the positive group the changes were not always found in all the muscles examined. These findings confirm the work of Graudal and Hvid (1959), who found changes of polymyositis in 60% of 31 rheumatoid patients, not all of whom were regarded as typical. They detected these changes only in the first dorsal interosseus muscle. In 17 other patients they examined 19 other muscles and found no such changes. In six of these muscles, however, an insufficient number of measurable potentials were registered. In the present series the changes of polymyositis were often found in the proximal muscles. Amick (1960), who studied 25 rheumatoid patients with wasting of the intrinsic muscles of the hand, found no electrodiagnostic abnormality to account for the wasting. He equated the atrophy with disuse. As he states, a disuse atrophy shows normal electrodiagnostic findings. The E.M.G. changes in this series could not, therefore, be due to wasting, and indeed there was no such relationship—several muscles with wasting showing a normal E.M.G., and several without wasting showing changes of polymyositis. A mixed group of patients with wasting of limb muscles due to osteoarthritis of hips and knees and post-operative atrophy were examined electrodiagnostically. All showed a normal E.M.G.

Mueller and Mead (1952) performed E.M.G.s on 25 patients with rheumatoid arthritis and found disorganization of the pattern and potentials of low height. Some of the potentials were neurogenic, but were also of low amplitude. There was no spontaneous activity. Morrison *et al.* (1947) in their series found normal units on volition but spontaneous activity in 50% of 34 rheumatoid patients. They concluded that the neuromuscular system was directly involved, and this could explain the muscular wasting. Spontaneous activity was observed in 18 of the patients in the series of Graudal and Hvid (1959). Wramner (1950) also detected spontaneous activity in 38 out of 50 rheumatoid patients that he examined. Newman *et al.* (1953) found no such activity. Spontaneous activity was not detected in the present series except in two cases where fibrillation was found in the abductor pollicis brevis attributable to the carpal-tunnel syndrome, due to compression by a rheumatoid bursitis. It is clear that there is little agreement between the various series regarding the presence of spontaneous activity and the type of units present. Only Amick (1960) has found a completely normal E.M.G. The intensity-duration curves that he plotted were normal, whereas several in this series showed evidence of partial denervation. Harris (1950) found changes in the curves characteristic of partial denervation in 25% of atrophied muscles in rheumatoid arthritics.

That electromyographic changes occur in the proximal muscles is supported by the work of Horwitz (1949), who found evidence of nodular polymyositis in 40% of biopsies of deltoid muscle in rheumatoid arthritics. The fact that joint activity bore no constant relationship to the presence of E.M.G. evidence of polymyositis is again supported by biopsy work. Desmarais *et al.* (1948) found that histological changes bore no relation to wasting of the muscle, to the nearness of affected joints, or to the activity of the disease. It did bear some relationship to the duration of the disease. In the present series there appeared to be a definite relationship of positive findings with disease activity, for, although these changes could be found in inactive cases, this was rare (Table II).

Only a few of the patients in the positive group were on steroids, and only one of these was on triamcinolone for a few days. Thus steroids could not have been responsible for the changes causing a steroid myopathy (Williams, 1959).

Despite Amick's negative findings, we feel convinced by the unequivocal electrical changes of polymyositis in so high a proportion of our patients, and in other series reported, that this neuromuscular abnormality is common in rheumatoid arthritics and is yet one more systemic manifestation of the disease.

It is well known that proximal muscular weakness and wasting with the same E.M.G. changes occur in systemic lupus erythematosus, dermatomyositis, polyarteritis nodosa, and scleroderma, and it is not really surprising to find the same changes in rheumatoid arthritis, particularly as rheumatoid arthritis can merge imperceptibly into any of these collagen disorders. Whatever causes rheumatoid arthritis attacks the distal end of the axon and the muscle fibre—a region especially vulnerable to disease, as shown by the same characteristic changes in carcinoma and thyrotoxicosis as well as the collagen disorders.

Summary and Conclusions

Electromyograms and intensity-duration curves were performed on 93 patients with "definite" rheumatoid arthritis.

Electromyographic evidence of polymyositis was found in 79 patients.

Intensity-duration curves showed partial denervation in 37 of the muscles showing polymyositis and in 2 muscles with a normal E.M.G.

These changes bore no constant relationship to wasting and weakness of the muscles and activity of the neighbouring joints.

Steroid therapy was not responsible for these changes. There was some relationship between disease activity and these findings.

It is concluded that electromyographic changes of polymyositis are common in rheumatoid arthritis, and this confirms biopsy work that muscles are frequently affected.

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