FINGER TREMOR IN TABETIC PATIENTS AND ITS BEARING ON THE MECHANISM PRODUCING THE RHYTHM OF PHYSIOLOGICAL TREMOR

BY

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In a recent paper, Marshall and Walsh (1956) have considered various possible explanations of the tremor with a characteristic peak frequency of 8 to 10 c./sec. which is associated with muscular contraction in healthy subjects. Among other theories, they consider but reject the hypothesis that tremor may be due to oscillation round the " servo-loop " from muscle spindle to muscle (Halliday and Redfearn, 1956; Hammond, Merton, and Sutton, 1956). Three reasons are given for rejecting this hypothesis: (1) The similarity of the frequency of tremor in muscles having short neural pathways to the cord with that in muscles of the distal parts of the extremities; (2) the fact, reported by them, that young children show a tremor rhythm of low frequency, whereas reflex time increases with growth and might be expected to produce a slower rhythm as it did so; (3) evidence which is taken to show that tremor persists following deafferentation.

The present paper is concerned with the third of these reasons. Under this heading, two authorities are cited (Lashley, 1917; Altenburger, 1937), but neither appears to offer sufficiently good grounds for settling the matter one way or the other. Lashley's paper deals not with a deafferented limb but with a leg rendered anaesthetic from the knee downwards following an injury to the cord at a higher level. It is true that in this case the knee jerks were absent, and this might appear to indicate additional damage to the reflex arc, but, as Kuhn (1951) has pointed out, permanent arreflexia is a not uncommon sequel to spinal transection at a higher level. It cannot therefore be regarded as conclusive evidence of damage to the reflex arc itself. The relevance of the clinical assessment of the tendon jerk to the activity of the stretch reflex will be considered further in the discussion. In any case, Lashley only mentions the 10 c./sec. rhythm in a single sentence at the end of

his paper. Altenburger's recordings of arm movements in a subject with posterior rhizotomy were made with a mechanical system and it is by no means clear that the record does show the 10 c./sec. rhythm.

The present paper presents the results of recording and analysing the frequencies of finger tremor in eight patients suffering varying degrees of deafferentation due to tabes dorsalis. It will be shown that the tabetic patients can be divided into two groups, those without a 10 c./sec. rhythm and those with an increased rhythm, and that the absence of the rhythm is very clearly correlated with the degree of deafferentation as determined by clinical testing. In the case of the less severely deafferented tabetic patients, who showed an increase in the rhythm compared with healthy subjects, it will be argued that such an increase on partial deafferentation is by no means a surprising observation on the " servo-loop " hypothesis. The evidence from these patients is therefore regarded as supporting this hypothesis.

Method

Tremor was recorded photo-electrically from the right index finger, and a continuous analysis was made automatically of frequencies between 1·5 and 30 c./sec. The apparatus has been fully described elsewhere (Cooper, Halliday, and Redfearn, 1957), and the method of recording and analysis of the records was the same as for the analysis of tremor in healthy subjects (Halliday and Redfearn, 1956). The subjects were out-patients attending the National Hospital. In each case tremor was recorded for 50 seconds, first with the finger unloaded and then for two further periods with a 50 or 100 g. weight strapped beneath the terminal phalanx with cellulose tape. Results are presented as force spectra for frequencies between 1·5 and 30 c./sec., calculated from an analysis of the angular velocity of the finger about the metacarpophalangeal joint.

Results

Assessment of Degree of Deafferentation.—Since the object of investigating tremor in tabetic patients

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was to determine the effect of a lesion interrupting the reflex arc, the assessment of the relative degree of deafferentation in different patients was of great importance. A full clinical examination was carried out in each case and sensory damage was assessed by the appreciation of pain (pin-prick), light touch (cotton wool), and position sense; the level of activity of the tendon jerks was also recorded. As tremor was recorded from the right index finger in each case, interest is directed towards the degree of deafferentation of the right upper limb. Sensory data are therefore presented for the right upper limbs only. In fact, however, all limbs were tested and there is little difference in the final assessment if the whole picture is taken rather than the localized one. It is not of course practicable to limit testing to the particular muscles producing finger tremor, as many muscles may be concerned and the eliciting and measurement of the tendon jerks in each of them would be difficult and laborious.

The findings are summarized in Table I, where the patients are arranged somewhat arbitrarily in what appears to be the order of decreasing deafferentation. Thus the first two patients (Nos. 1 and 2) showed severe sensory loss combined with complete absence of deep reflexes, while the last three (Nos. 6 to 8) showed little or no sensory loss together with tendon jerks which were still present and active (the actual level of activity varied and in one case (No. 6) some of the jerks were only elicitable on reinforcement). Between these two extremes there is a group of patients (Nos. 3 to 5) in whom the signs were more equivocal, in that absent tendon jerks were associated with minimal sensory changes (Nos. 3 and 4), or marked sensory loss was combined with active reflexes (No. 5).

Examination of the past notes of these three equivocal patients added some further information. Patient 3 had shown severe sensory loss on his original admission two and a half years before, with impairment and delayed response to pin-prick over the cuirasse area (D3-D8), over the ulnar border of the forearm and little finger, and

also in the lower limbs. Light touch and temperature sense had also been impaired over these areas, although position sense had appeared normal. He had made a very marked clinical recovery following a course of penicillin, but was still markedly ataxic in his gait and was unable to stand with feet together and eyes closed without falling. At the time of the tremor recording, pain, touch, and temperature were normally appreciated in all areas. Vibration sense was normal in both hands but impaired at the ankles. Two-point discrimination was good in the upper limbs except over the little fingers, but was very poor in the feet, only a separation of 9-10 cm. being appreciated. Thus the whole picture was one of an originally severe sensory loss, now greatly improved, but with residual damage, most markedly manifest in the ataxic gait and Rombergism. The latter features might suggest that the muscle and joint afferents were still fairly severely affected.

Patient 4 had also been originally admitted two and a half years before the tremor was recorded, but his sensory loss at this time was much less severe than that of Patient 3 and was limited to a variable slight diminution to pin-prick and temperature over the chest and ulnar border of both arms and in both legs below the knee. There was no diminution to light touch except over both feet. Joint position sense and two-point discrimination were accurate in all the fingers of both hands, and both stance and gait had been specifically reported on at the time as being normal. There thus seemed little or no subsequent change in a picture which suggested definitely milder sensory damage than in the case of Patient 3, but in which, nonetheless, no tendon jerks could be elicited.

Patient 5, on the other hand, showed a marked but " patchy " type of sensory loss, with a variable and often delayed response to pin-prick on the face, trunk, and lower limbs, and in the upper limbs, particularly along the ulnar border. Patches of complete analgesia alternated with areas of hyperalgesia. Of the other modalities, light touch was defective on the trunk, lower limbs, and ulnar border of the upper limbs but was normally appreciated over the face. Position sense was grossly diminished in all joints of the lower limbs and in the fingers, where movement but not direction could be appreciated, but was normal at the wrists, elbows, and shoulders. His gait was still ataxic and wide-based, but had improved

TABLE I

DEGREE OF DEAFFERENTATION

			Deep Reflexes of Right Upper Limb				Sensory Loss to		
			Biceps Jerk	Triceps Jerk	Supinator Jerk	Finger Jerk	Pin-prick	Cotton Wool	Position Sense
Patient 1	..	..	—	—	—	—	Severe	Slight	Severe
Patient 2	..	..	—	—	—	—	Severe	Severe	Slight
Patient 3	..	..	—	—	—	—	No loss*	No loss*	No loss*
Patient 4	..	..	—†	—	—	—	Slight	No loss	No loss
Patient 5	..	..	—†	++	—	—	Marked	Marked	Marked
Patient 6	..	..	+R	+	+R	++	No loss	No loss	No loss
Patient 7	..	..	++	+	+	±	Marked	No loss	No loss
Patient 8	..	..	+++	++	++	++	No loss	No loss	No loss

*Had been severe, but recovered following penicillin (see text).
†Present and active at last previous examination 14 months previously.
+R = present on reinforcement only.

since his original admission, following penicillin treatment. The muscular reflex responses were also affected in a somewhat " patchy " and variable way. Of the tendon jerks in the right upper limb only the triceps was present at the time of the tremor recording. However, in the left arm the biceps, triceps, and supinator could all be elicited and even the finger jerk was obtained on reinforcement. None of the jerks could be evoked in either of the lower limbs. Examination of the past notes revealed that the right biceps jerk had not only been present, but active, at the last previous examination fourteen months before, at which time the right triceps jerk (now quite active) had been unelicitable. Tendon jerks had been consistently absent in the lower limbs since his first admission six years before. In his case, de-afferentation appeared to have affected cutaneous sensation and tendon reflexes severely, but in an incomplete and variable distribution.

Characteristics of Tremor Spectrum.—Tremor in healthy subjects shows a base line of activity at all frequencies, but there is an increase between about 5 and 15 c./sec. with a distinct peak at 8 to 10 c./sec. (Halliday and Redfearn, 1956). It is increased in amplitude by loading the finger with weights, while the peak frequency is not greatly changed. Figs. 1, 2, and 3 show frequency spectra obtained from 46 healthy subjects (Halliday and Redfearn, 1956) compared with the present group of tabetic patients. Fig. 1 presents spectra for the unloaded finger, Fig. 2 for the finger with a 50 g. weight strapped beneath the terminal phalanx with cellulose tape, and Fig. 3 shows the same with a 100 g. weight attached. In contrast to the frequency, tremor amplitude varies greatly between different healthy individuals. The frequency spectrum from the healthy individual with the greatest amplitude in each case has therefore been plotted along with the average spectrum for the healthy group.

For clarity of presentation, the tabetic patients have been divided into two groups, namely, patients 1 to 3 and patients 4 to 8. It was found impracticable to include data from all eight patients on one graph, and in any case the spectra seem to fall naturally into these two groups.

It is clear from the figures that on the whole the tabetic patients show a higher general level of activity than normal. This increase is quite apart from any effect on the 5 to 15 c./sec. waveband, and is seen as a generalized raising of the base line throughout the spectrum; it seems particularly marked at the lower frequencies. This higher general level of random activity seems to affect the tabetic group as a whole and is not confined to the most severely deafferented.

However, it is with the 8 to 10 c./sec. rhythm of tremor, rather than with the random element, that this paper is mainly concerned. Whatever the finger

loading, this peak is seen to be completely absent in patients 1 to 3. Patients 4 and 5 show little or no peak in the case of the unloaded finger, but a normal or high peak when the finger is loaded. Patients 6 to 8 always show a well-marked or abnormally high peak in all cases and especially when the finger is loaded.

Compared with the mean spectrum of the healthy subjects, in which the peak mean frequency shows little change on loading (Figs. 1 to 3), the peak tremor frequency of those tabetics with the less severe deafferentation is markedly slowed when the weight on the finger is increased, especially from 50 g. to 100 g. Data for peak frequency and load are given for each patient in Table II and show a change of as much as 4 c./sec. in all save one of the tabetics, to be compared with a change of 1 c./sec. in the healthy group.
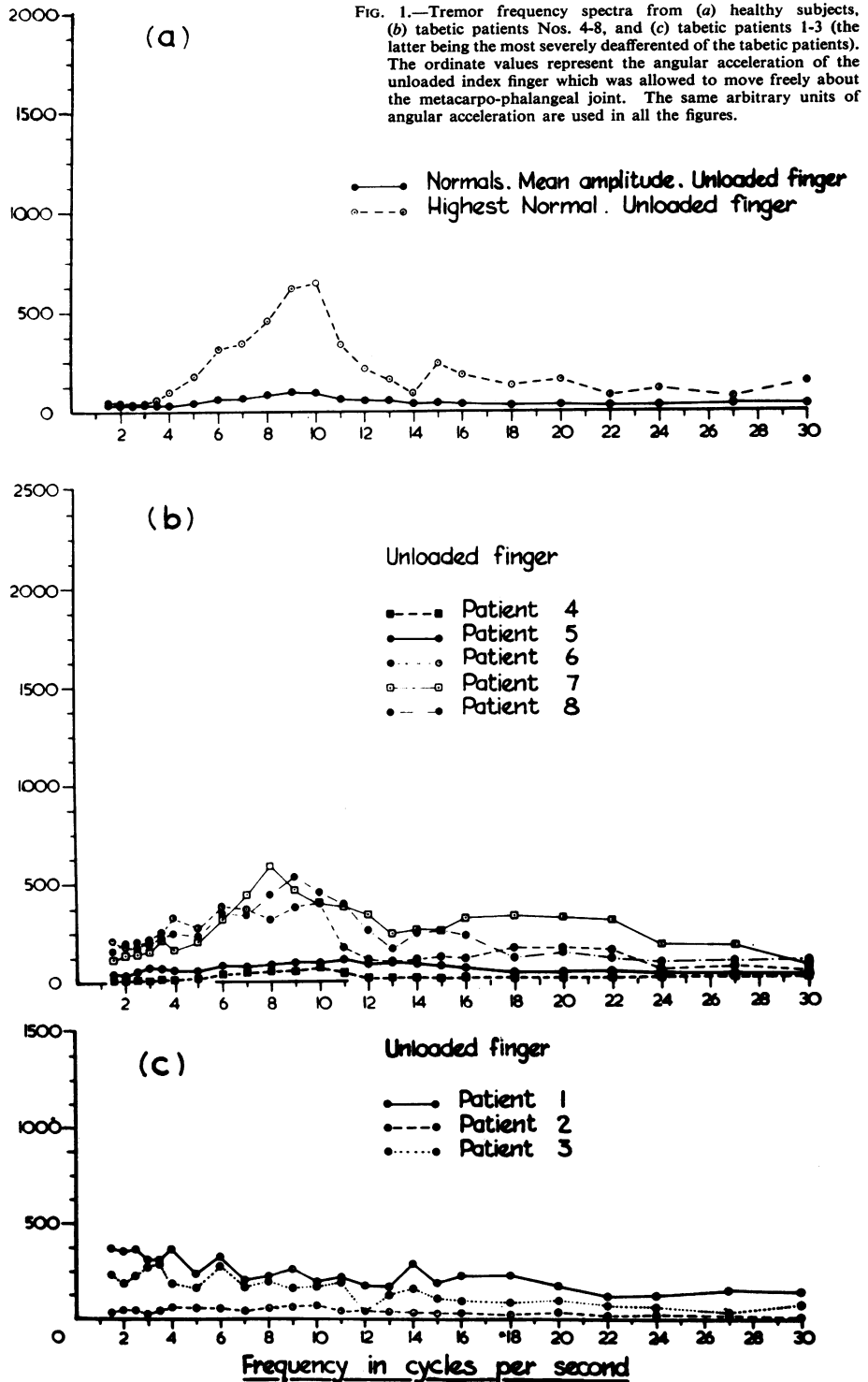
Figs. 1 to 3 (*a*) show an apparent slowing of 3 c./sec. in the peak frequency of the " highest normal " spectrum after loading. This effect is purely accidental and results from the fact that the " highest normal " curves come from a different individual for each load. Thus the normal subject showing the most tremor of the unloaded finger was not the same subject as the one who showed most tremor with a 50 g. weight attached, and again was different for the 100 g. weight. The three individuals had different peak values, as can be seen in Table II, but none of the individuals showed more than a 1 c./sec. change of peak frequency with loading.
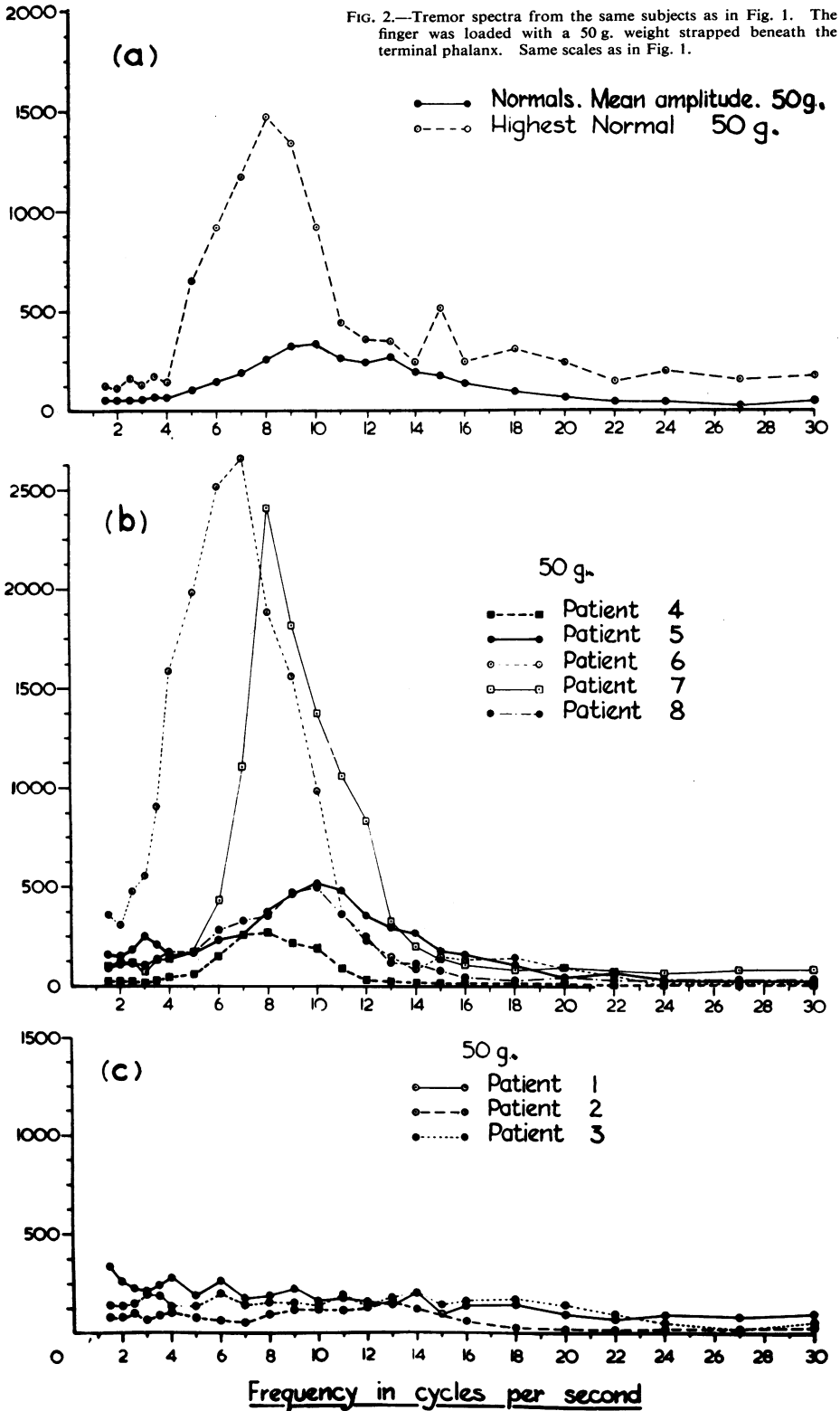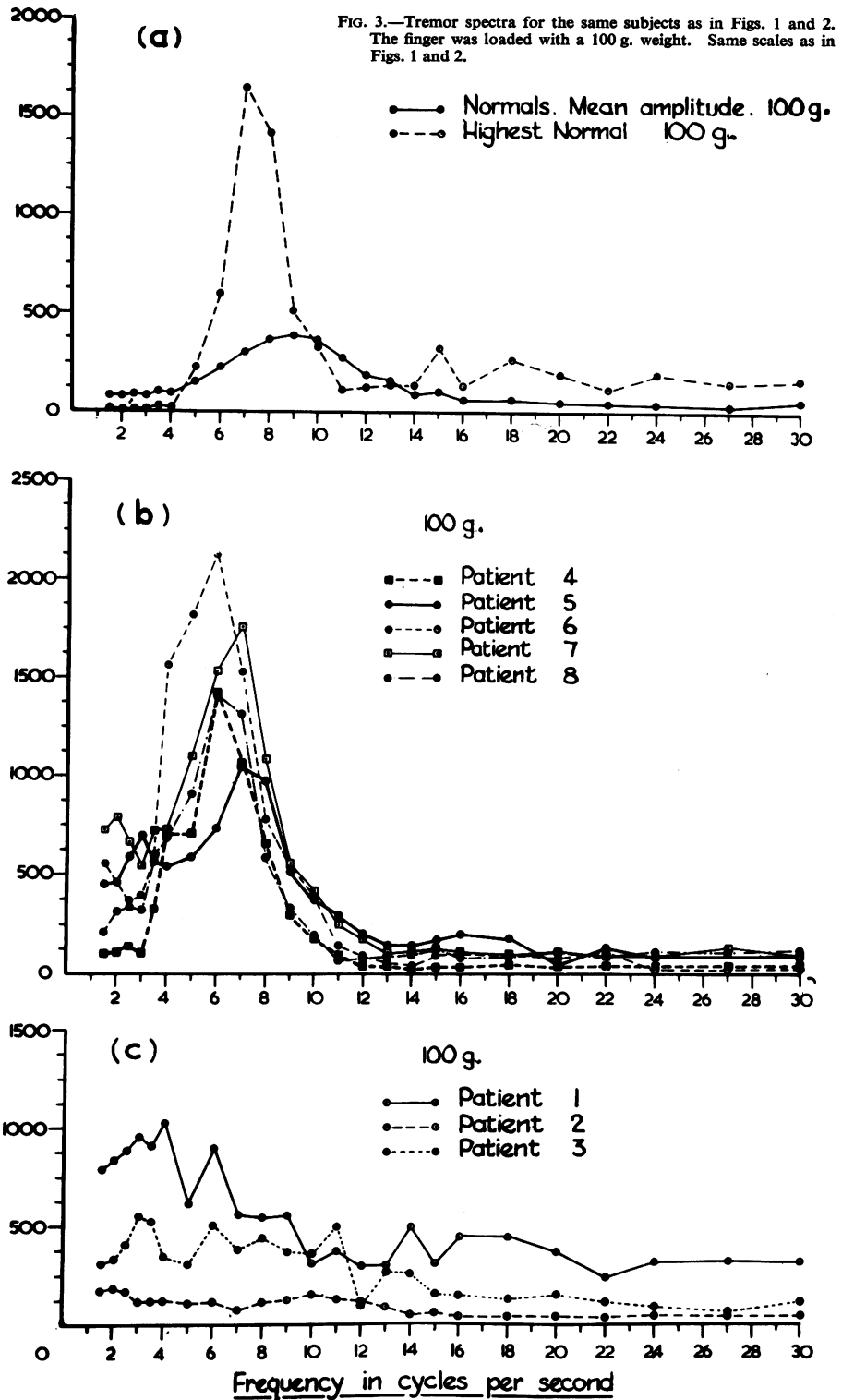
TABLE II
CHANGE OF PEAK FREQUENCY WITH LOADING

	Peak Frequency (c./sec.)			Maximum Change of Peak Frequency
	Unloaded	50 g.	100 g.	
Healthy subjects (mean)	9	10	9	1
Healthy subject with highest tremor level for unloaded finger (Fig. 1)	10	10	10	0
Healthy subject with highest tremor level with 50 g. attached (Fig. 2)	8	8	8	0
Healthy subject with highest tremor level with 100 g. attached (Fig. 3)	8	8	7	1
Patient 4	10	8	6	4
Patient 5	11	10	7	4
Patient 6	10	7	6	4
Patient 7	8	8	7	1
Patient 8	9	10	6	4

Discussion

In considering the implications of these results it should be realized that in none of the hundred or more healthy subjects so far examined have we failed to demonstrate the characteristic peak in the tremor spectrum in the neighbourhood of 9 c./sec.,
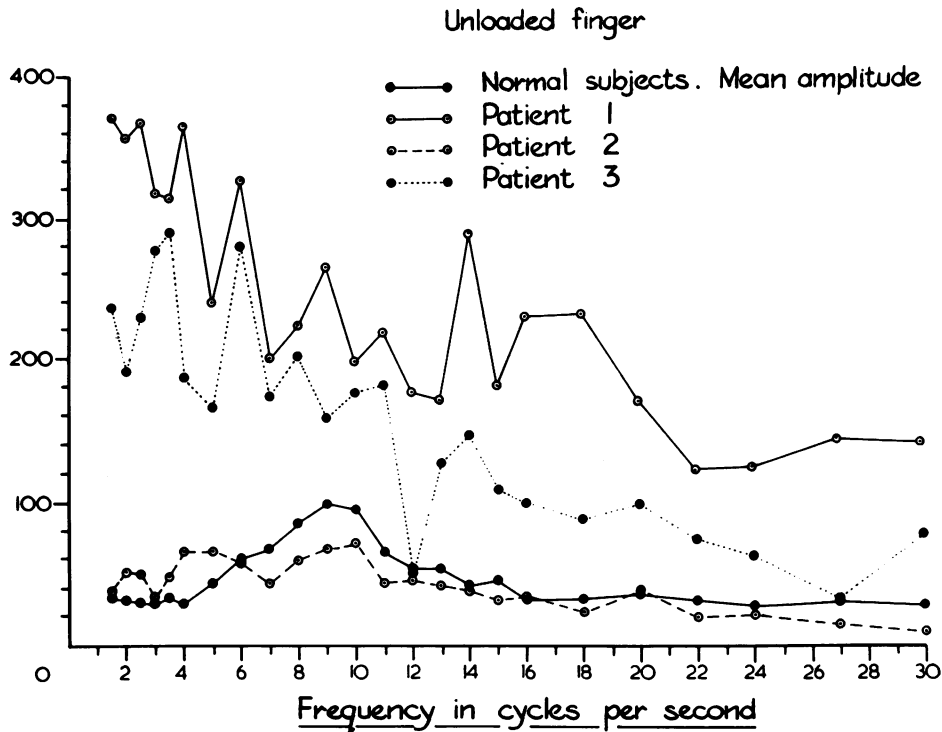
FIG. 1.—Tremor frequency spectra from (*a*) healthy subjects, (*b*) tabetic patients Nos. 4-8, and (*c*) tabetic patients 1-3 (the latter being the most severely deafferented of the tabetic patients). The ordinate values represent the angular acceleration of the unloaded index finger which was allowed to move freely about the metacarpo-phalangeal joint. The same arbitrary units of angular acceleration are used in all the figures.

Fig. 2.—Tremor spectra from the same subjects as in Fig. 1. The finger was loaded with a 50 g. weight strapped beneath the terminal phalanx. Same scales as in Fig. 1.

Frequency in cycles per second

Fig. 3.—Tremor spectra for the same subjects as in Figs. 1 and 2. The finger was loaded with a 100 g. weight. Same scales as in Figs. 1 and 2.

FIG. 4.—Tremor spectra for normal and severely deafferented tabetic subjects compared. The mean spectrum for a group of normal subjects is the same as in Fig. 1 (*a*), and the tabetic spectra are the same as in Fig. 1 (*c*). The same units are used as in Fig. 1, but the ordinate scale has been lengthened so that the contrasting shapes of the normal and of the deafferented spectrum may be seen more clearly.

although in some subjects it only becomes obvious on loading the finger in the manner described. The first three tabetic patients of the present series are quite distinct in this respect, as appears even more clearly if the ordinate scales of Fig. 1 are lengthened (Fig. 4). Though the mean level in the healthy subjects is lower than that shown by the tabetics, the latter do not show any signs of the clear and consistent rise to a maximum amplitude at 9 c./sec. characteristic of the normal subjects. The absence of this peak, of course, becomes even more evident when the amplitude of tremor in the healthy group is increased by loading (Figs. 2 and 3).

Of the three patients having no 9 c./sec. peak, two are certainly the most severely deafferented of the whole series; the third was either the third or the fourth most severely deafferented, and clinical evidence has already been adduced for rating him third. All three patients were without detectable tendon jerks in the right upper limb. There was a fourth patient without demonstrable tendon jerks who nevertheless showed the peak. In this series, therefore, an absent 8 to 10 c./sec. rhythm was always accompanied by absent tendon jerks, although the

absence of jerks was not in every case associated with the absence of the rhythm.

In considering the anomalous patient (No. 4), the fact that the sensory deficit was limited to slight loss to pin-prick, while light touch and position sense were unimpaired, suggests that the damage to afferents cannot have been so severe as the absence of jerks might have suggested. Some allowance may also be due for the inaccuracies and approximations implicit in clinical testing. Moreover, if the hypothesis is maintained that the tremor rhythm is due to oscillation in the stretch reflex of the muscles concerned, the tendon jerks of other muscles, even though of neighbouring ones, cannot be taken as a reliable measure of the state of the reflexes in the actual muscles concerned. Hence, perhaps, the higher correlation when other sensory phenomena are taken into account. In any case it is not yet clear whether the tendon jerk is a measure of that aspect of the stretch reflex which is relevant to tremor. It is well known that the tendon jerk and the stretch reflex do not always behave in the same way. Indeed, in the cat the tendon jerk may be highly active when the stretch reflex has been depressed, and it may

come and go quite independently of the level of spindle firing (Granit, 1955). Granit, Henatsch and Steg (1956) have produced evidence that the phasic and tonic components of the muscle's response to stretch may be mediated by two distinct groups of motor neurones. It is not yet clear what role these different mechanisms play in the production of tremor, but the existence of such a differentiation emphasizes how inadequate the tendon jerk may be as an index of stretch reflex activity.

The disappearance of the 9 c./sec. rhythm in the deafferented cases is consistent with the hypothesis that the tremor is due to an oscillation in the reflex arc. However, this is not the only possible explanation of this observation. Rhythmical activity fed down from the central nervous system through the gamma efferent fibres to the muscle spindle (and only reaching the alpha motor neurones indirectly via the afferent fibres) might disappear in much the same way after deafferentation. But the behaviour of the tremor rhythm in those cases with only partial deafferentation provides some further evidence which seems to be in favour of the " servo " hypothesis. The increased sensitivity of the frequency to loading in these cases compared with the normal is strongly suggestive of the behaviour of a failing " servo " mechanism with insufficient energy at its disposal to cope with the increased load. If the rhythm were of central origin but transmitted via the gamma efferents, increasing the load in a partially deafferented case would be expected to diminish the amplitude and leave frequency unaltered, whereas in fact amplitude is increased and frequency diminished.

The high levels of tremor amplitude at 7 to 9 c./sec. reached by the partially deafferented tabetic cases poses an interesting problem. One possibility might be that, with the destruction of some of the stretch reflex arcs, the remainder were being run at a high loop gain by increased gamma activity as a compensation. This hypothesis may receive some support from the fact that tendon jerks are erratically brisk in some of these cases. Further, the fact that the peak amplitude is not increased, but diminished, in the two most tremulous patients when the load is increased from 50 to 100 g. suggests that the available loops may even have reached saturation in

these cases and may be oscillating to the full extent of their power. Such a failure to increase tremor amplitude by loading at these relatively low tensions has not been seen in any other cases.

Summary

It has been suggested in a recent paper that the characteristic tremor rhythm with a peak frequency of 8 to 10 c./sec. is unaffected by deafferentation, and this has been put forward as evidence against the " servo loop " theory of tremor.

In this paper the results of recording and automatically analysing the frequency of tremor in eight tabetic patients showing varying degrees of deafferentation is presented. In the most severely deafferented patients there is no sign of the normal tremor rhythm. In the other patients, who showed less severe sensory loss, the tremor rhythm was present and of greater amplitude than normal, and the peak frequency was abnormally lowered by loading.

These results suggest that the integrity of the reflex arc is essential to the presence of the tremor rhythm and they provide some support for the " servo " hypothesis. Moreover, the behaviour of the tremor frequency on loading in the less severely deafferented patients showed characteristics which would be expected if it was produced by oscillation in a failing " servo " but not if it was a centrally determined rhythm.

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