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THE 4–6 HZ TREMOR DURING SUSTAINED CONTRACTION IN NORMAL HUMAN SUBJECTS

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SUMMARY

1. Continuous elevation of the middle finger for 15–60 min gave rise to modulation of the e.m.g. at 4–6 Hz. A marked peak (50–150 % of the amplitude of the coexisting 8–12 Hz peak) at 4–6 Hz was produced in sixteen out of twenty-one subjects.

2. The 8–12 Hz peak was also enhanced (2–14 times with respect to its initial amplitude in eighteen subjects) during the course of the prolonged contraction but its frequency did not change.

3. The 4–6 Hz and the 8–12 Hz peaks were present simultaneously; it is concluded that the two phenomena are separate entities.

4. A step-function mechanical perturbation of the finger generates, time-locked to the stimulus, a train of waves at the frequency of the slow tremor, which can be abolished by local ischaemia. It is proposed that this slow tremor is due to an oscillatory process, possibly involving the reflex arc, but entailing a longer neuronal delay than that responsible for 8–12 Hz tremor.

INTRODUCTION

Tremor can be defined as a series of spontaneous oscillatory movements, at a frequency greater than 1 Hz, superimposed on the intended movement or position of a limb. Spectral analysis of normal finger tremor acceleration reveals that, superimposed upon the background distribution of tremor in the 1–30 Hz range, there may be a peak in the 8–12 Hz frequency band and sometimes one between 17–30 Hz. Tremor is thought to be due to (a) oscillations in the force of muscular contraction (Halliday & Redfearn, 1956), (b) mechanical resonances in the limb dependent upon inertia and stiffness (Rietz & Stiles, 1974) and (c) the cardiac impulse transmitted to the limb (Brumlik, 1962). The amount of tremor that originates in each of these three ways is influenced by the experimental conditions, depends on the particular limb concerned, and will also vary with the method used for recording it. The 8–12 Hz peak is likely to arise as oscillation in the feed-back loops controlling muscle length or tension (for a short account of the evidence for this see Lippold, 1973). The peak in the range 17–30 Hz can be shown to be the damped mechanical response of the

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finger to a forcing input (Stiles & Randall, 1967). The ballisto-cardiac impulse may act as one forcing input and may also excite reflex oscillation at 8–12 Hz (Ellaway & Furness, 1977).

A prolonged, moderate, contraction leads to the development of uncontrollable limb movements of large amplitude which have a frequency around 4–6 Hz, a form of tremor that was originally described by Eshner in 1897 and has more recently been studied by Stiles (1976). He showed that it develops if the wrist, for example, is kept continuously raised by extensor action over the edge of a table for about an hour. Should it prove to be the case that a prolonged contraction causes a progressive fall in tremor frequency from 8–12 Hz to 4–6 Hz, as was suggested by his experiments, serious doubt is cast upon the servo-loop hypothesis for normal 8–12 Hz muscular tremor. This is because the frequency of the tremor due to the latter mechanism would be determined by the reflex delay leading to instability and oscillation, and it therefore should be invariant during a prolonged contraction. The work to be described in this paper shows that the 4–6 Hz peak is an entity separate from the 8–12 Hz peak and thus the appearance of slow tremor cannot be interpreted as evidence against the servo-loop hypothesis for 8–12 Hz tremor. A preliminary account has been published already (Gottlieb & Lippold, 1981).

METHODS

Subjects

Subjects were forty male and female volunteers, aged 18 to 58 yr, all of whom gave informed consent to these experiments which were subject to scrutiny by the local ethical committee.

Recording

The subject was seated comfortably in a chair next to a small table, placed at roughly elbow height. Screwed into the surface of the table was a plaster cast shaped to accommodate the subject's elbow, forearm and hand, padded with cotton wool. The subject then rested his or her arm in a prone position in the plaster cast. When recording, the middle finger was raised (by extension) to a fiducial mark, keeping the hand and forearm firmly fixed in the support. The palm of the hand and the other four fingers were supported by the plaster; care was taken to ensure that the only movement that could occur was a flexion–extension movement of the middle metacarpal–phalangeal joint. The mark was adjusted so that the middle finger made an angle of 20° above the metacarpal plane when raised. This required the exertion of about 10% of maximal contraction strength. It was usual practice to observe the subject throughout the experiment, to check that the hand and forearm were maintained in the correct position. The room temperature was also noted, and care taken to ensure that the subject did not become cold, or shiver, as this can alter both the frequency and amplitude of 8–12 Hz tremor.

Tremor was recorded from m. extensor digitorum communis using the e.m.g. from surface electrodes on the skin over the muscle belly with filters to limit the pass-band to between 80 Hz and 10 kHz (−3 dB points). The signal was amplified and subjected to full-wave rectification followed by demodulation (essentially smoothing by a third-order Butterworth filter set to remove frequencies above 18 Hz). A detailed account of this method for tremor recording can be found in Elble & Randall (1976). It has two considerable advantages as the signal (a) is proportional to the force of contraction (Lippold, 1952) although movement is not limited by the presence of a transducer and (b) is derived solely from the one muscle under study. Finger tremor was also recorded using a Brüel & Kjaer accelerometer (wt. 13 g) and charge amplifier, the output of which was subjected to frequency analysis as described below. In a few experiments tremor force was measured using the strain gauge described by Lippold (1970); the muscles were: first dorsal interosseous, adductor pollicis, abductor digiti minimi brevis and extensor digitorum communis. The slow tremor to be described was found in all these cases.

Mechanical arrangements

The extensor digitorum communis was given brief stretches by displacement of the middle finger with the solenoid device described by Gottlieb (1981) and Gottlieb & Lippold (1980). The applied force produced a displacement of the finger of about 1 cm and a peak acceleration of 44 ms^{-2}. Muscle twitches were recorded using a strain gauge as described by Lippold (1970).

Analysis

Frequency analysis of the rectified and demodulated signal was carried out in real time using the Hewlett–Packard 3582A spectrum analyser, remotely programmed with a type 9528A desk-top computer and automatically plotted on a type 7225 graphics plotter. The frequency span was 0–25 Hz which gave a time record length ($N\Delta t$) of 10 s, and a calculated point spacing (Δf) of 0·1 Hz. A Hann pass-band shape was used, to minimize leakage, giving 0·15 Hz equivalent noise band width. Since the sampling frequency was 102·4 kHz (i.e. Nyquist frequency = 51·2 kHz) alias contamination did not occur within the specified frequency range. Four separate, consecutive 10 s power spectra were averaged to give 90% confidence limits between +4·7 and −2·9 dB for each spectral point.

The experiments involving the application of brief step-function extensions to extensor digitorum communis were analysed using a Biomac 1000 computer to average thirty-two sweeps of 1·28 s duration triggered by a Digitimer pulse. Sweeps were generated every 1903 ms.

Twitches were also analysed in this manner using the Biomac to average thirty-two consecutive responses recorded using the strain gauge described by Lippold (1970).

RESULTS

Development of 4–6 Hz tremor with sustained contraction

Whilst the frequency of tremor in the 8–12 Hz range was little affected by prolonged contraction, it was observed in preliminary experiments using the accelerometer upon a few subjects that a prolonged contraction produced a second peak in the range 4–6 Hz. This peak generally occurred after some 30–40 min of continuous contraction, and quite clearly existed in addition to the activity at 8–12 Hz. As contraction continued, the amplitude of the 'slow' tremor often matched, or exceeded, the amplitude of 'normal' tremor at 8–12 Hz. At this point, the subject experienced difficulty in maintaining a steady contraction with the customary degree of accuracy, due to the large oscillations in tension. The development of this tremor is illustrated in Figs. 1 and 2. At the start of contraction, tremor amplitude was small, and the spectrum was almost flat (lower record). As the contraction continued, the 9 Hz peak amplitude increased and, after about 30 min, a completely different tremor peak appeared, at 4 Hz.

Amplitude of 8–12 Hz peak during sustained contraction

The 8–12 Hz peak also increased in amplitude during the prolonged contraction although the frequency did not change. For ten subjects, after 30 min continuous contraction the amplitude of the 8–12 Hz peak, as measured using the e.m.g., increased by 2 to 13·9 times (mean 5·48 times ± S.D. 3·5) with respect to the initial amplitude. In two subjects increases of 10- and 14-fold had occurred after 18 and 28 min respectively.

The variation in peak frequency with sustained contraction has been plotted for both peaks in Fig. 3 (from a subject with some slow tremor at the start of the contraction) and shows an obvious separation of the two peaks.

From these results, it is obvious that normal tremor at 8–12 Hz has not 'slowed

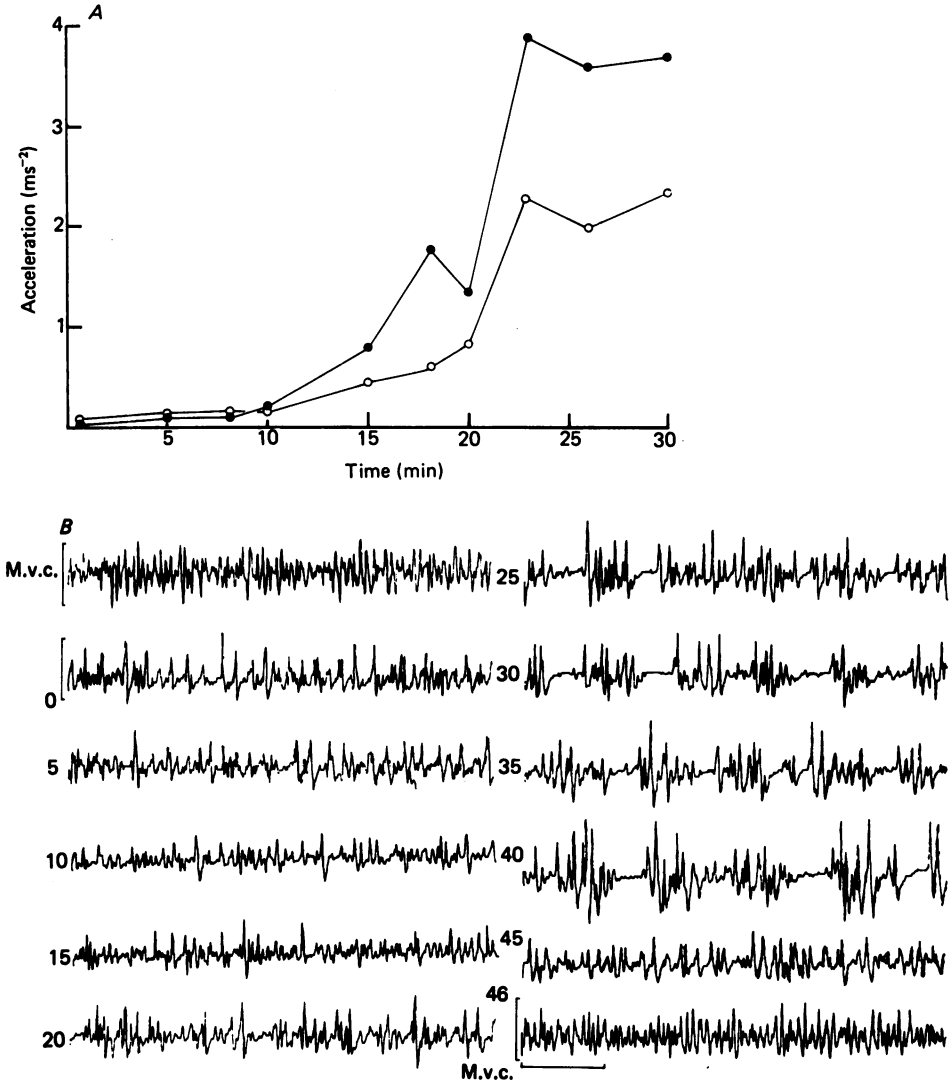

Fig. 1. *A*, the effect of sustained submaximal voluntary contraction of extensor digitorum communis on 8–12 Hz tremor (○) and 4 Hz tremor (●). A piezoelectric accelerometer (type 4367) on the middle finger produced a signal that was subjected to Fourier analysis and the amplitudes of the respective peaks were plotted for 0–30 min after the beginning of the contraction. *B*, records of the e.m.g. from surface electrodes over extensor digitorum communis during sustained submaximal contraction (10% maximal: m.v.c.). Top left: m.v.c. before start of contraction. Left column: recordings taken during contraction at the times stated after start. At 42 min an arterial cuff was inflated and the records at 45 min and 46 min were obtained with the cuff still inflated. The record at 46 min was a further m.v.c. Note that from the 25th min the e.m.g. records exhibit a periodicity at about 4 Hz that is abolished by the 3 min application of the cuff.

Calibrations: time bar = 250 ms. Voltage bars = 10 mV for first and last records (m.v.c.s) and 2 mV for remainder of records (see opposite time 0).

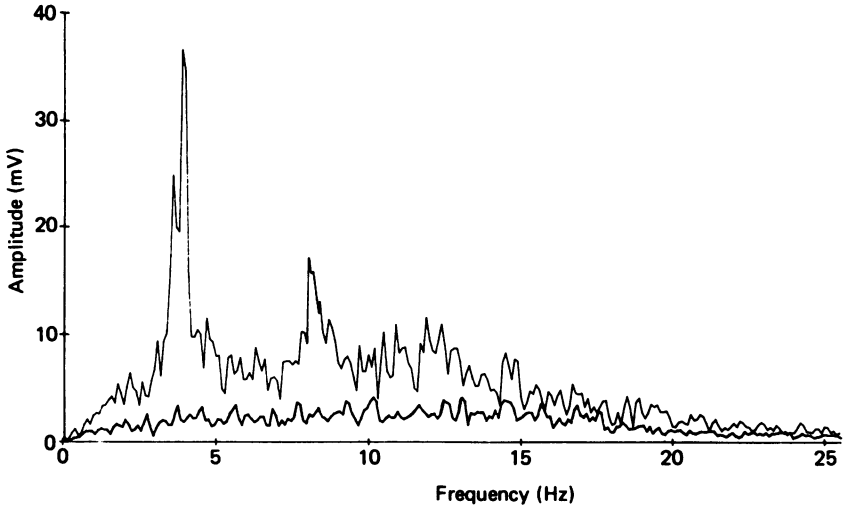

Fig. 2. The development of slow activity at 4 Hz during a sustained contraction of extensor digitorum communis at approx. 10% m.v.c. Upper amplitude spectrum: after 28 min, lower: after 9 min from start of contraction. Spectra were obtained by rectification and demodulation of the e.m.g. signal from the muscle and analysis for frequency components.

(Note that the Fast Fourier Transformation produces a real and an imaginary spectral component at each analysis frequency. These are squared and added, and a cumulative sum maintained for each of 256 bins. For a K-sized average, each bin contains 2 K squared values at the end of the processing. The sum is divided by K, for each bin, giving a power spectrum average. Before display, the square root of each sum is extracted to produce the amplitude spectrum, the units of which are mV).

Calibration of the amplitude in mV refers to the voltage input to the analyser. At the time the second sample was taken, direct displacement measurement gave a rotation of the finger about the metacarpal–phalangeal joint of 10° peak to peak.

down' with increasing amplitude, but that slow tremor at 4 Hz is an entity separate from normal tremor at 9 Hz, since the two peaks coexist.

To assess how commonly this rather unexpected variety of tremor occurred, prolonged contraction experiments were repeated upon a randomly selected group of twenty-one subjects, aged from 19 to 30 using the smoothed e.m.g. for analysis. Amongst this group, six subjects quite clearly developed slow modulation of the e.m.g., the maximum amplitude of which exceeded the coexisting 8–12 Hz peak. A further seven subjects developed a slow peak that reached an amplitude 80–99% of the 8–12 Hz peak. Another three subjects developed a slow modulation with a maximum amplitude 60–79% of the 8–12 Hz peak.

The time taken to reach maximum amplitude (or for the peak to become distinct) was typically 30–45 min, although two subjects displayed some slow activity from the start of contraction. The frequency of the slow activity lay in the range 3·6–7·9 Hz (mean value 5 Hz).

In the case of the remaining five subjects, the results were equivocal. Although the activity in the 4–6 Hz range increased as contraction continued, it could not be distinguished with any certainty from the general increase in background activity, and no distinct peak developed. It is possible that contraction was not continued for long enough in these subjects. These results are summarized in Table 1.

TABLE 1. Occurrence of slow activity

Subject no.	Time to development of peak (min)	Frequency (Hz)		Amplitude (% 8–12 Hz max)	Ratio L/H
		Low	Physiol		
1	39	5·4	9·6	100	1·7
16	16	6·5	11·1	135	1·7
3	45	3·6	10·7	108	3·0
9	42	3·6	10·6	109	2·9
29	18	4·2	9·5	172	2·3
2	45	4·5	9·9	268	2·2
8	0	3·6	10·8	86	3·0
13	17	5·5	10·1	91	1·8
10	28	3·8	11·8	81	3·1
5	30	6·8	10·0	97	1·5
4	48	4·5	9·4	84	2·1
7	24	7·9	12·0	94	1·5
22	53	7·0	10·3	95	1·4
14	20	4·2	10·7	77	2·5
12	34	3·0	11·0	61	3·6
28	40	5·5	10·4	70	1·9
18	60	3·6	10·4	40	2·9
17	49	4·4	10·1	48	2·3
15	36	3·6	10·5	48	2·9
6	55	5·5	10·7	38	1·9
19	33	6·5	10·0	59	1·8

Twenty-one subjects were investigated and are arranged in four blocks according to rank order of low frequency tremor amplitude. Column 2 gives the time elapsing from the start of finger elevation until slow activity developed. Columns 3 and 4 show the frequency of the peak measured at the time when that peak reached its maximum amplitude. Column 5 is the maximum amplitude of the 4–6 Hz peak attained throughout the experiment expressed as a percentage of the maximum 8–12 Hz peak. Column 6 is the ratio of the peak frequencies of low to high frequency (L/H).

Step-function inputs

The behaviour of a negative feed-back loop is commonly tested by injecting into it a signal that will cause the output to oscillate at a particular frequency related to the loop delay. If the oscillation is produced in response to the input signal, then it would be expected that input and output bear a constant phase relationship. The existence of this relationship can be tested for by repeatedly injecting an input signal and superimposing the resulting output oscillations. If the responses summate then this would indicate that the input–output phase relationships are constant. This principle has been exploited in these experiments, in attempting to discover whether the 4–6 Hz peak behaved like an oscillation in a negative feed-back loop, and could be excited in response to a peripheral input signal. Obvious summation of 4–6 Hz was obtained in all subjects tested ($n = 16$). Fig. 4 shows the results of this procedure applied at intervals during the course of a submaximal continuous contraction. The traces were obtained during sustained contraction by averaging thirty-two sweeps, 1·28 s duration each, of the rectified and demodulated e.m.g. signal from the extensor digitorum communis. At the arrow in each trace, the solenoid delivered a brief (31 ms) extension to the middle finger, resulting in a short summated wave form lasting 2 or 3 cycles. Record (A) was taken at the beginning of the contraction, (B)

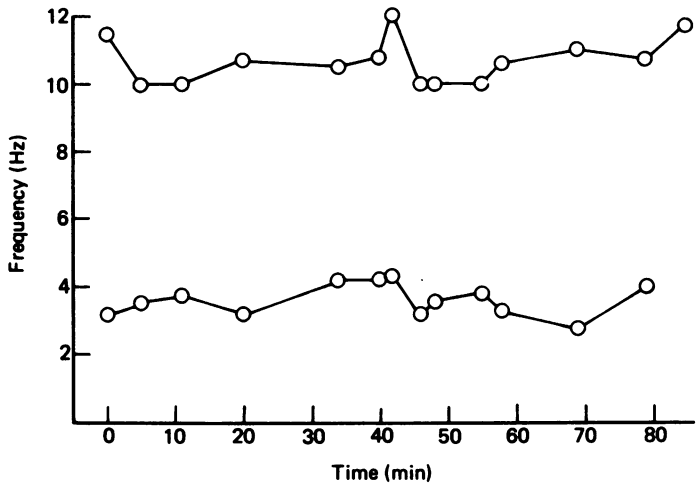

Fig. 3. Variation in tremor frequency with sustained contraction. Upper points correspond to normal tremor in the 8–12 Hz range, lower points correspond to slow tremor. Slow tremor was present at the start of contraction, and maximum tremor amplitude (both peaks) was attained after 42 min. There is a negligible change in tremor frequency with time ($r = 0{\cdot}16 \pm 0{\cdot}293$ S.E. of the mean for normal tremor, $r = 0{\cdot}069 \pm 0{\cdot}276$ S.E. of the mean for slow tremor), and the two forms of tremor are clearly distinct.

after 5 min, (*C*) after 32 min and (*D*) after 35 min. The lower two records, (*C*) and (*D*) show waves in the 3·6–4·6 Hz range, indicating that summation of the slow wave form is taking place.

A direct comparison between the 4–6 Hz peak amplitude of the sum of thirty-two sweeps and that of a single sweep showed the former to be between 10 and 15 times greater than the latter. This indicates that between a third to a half of the individual waves had truly summated, a value that is good evidence that the wave form is phase-locked to the brief mechanical input to the system.

Arterial occlusion

A brief period of ischaemia is known to abolish 8–12 Hz tremor, both during the arterial occlusion and for a short time subsequently (Halliday & Redfearn, 1954; Lippold, 1973). Whilst the mechanism underlying this is unclear, it is probably a result of opening the servo-loop responsible for the 8–12 Hz peak, because ischaemia rapidly affects muscle spindle sensitivity to stretch (Matthews, 1933). Regardless of the mechanism, it is an example of a peripheral manoeuvre affecting tremor that can be used to determine whether it depends on afferent activity. In twelve experiments on ten subjects, a 3 min period of ischaemia greatly reduced or abolished the 4–6 Hz peak in the electrical activity from the muscle (see Fig. 1(*B*)). In the twelve experiments the amplitude of the 4–6 Hz peak declined to 1·5 to 50·5% of its pre-cuff value (mean 28% ± S.D. 3·8).

Twitch time

A possible explanation for the appearance of slow, 4–6 Hz activity after a prolonged, voluntary contraction is that mechanical properties of the neuromuscular system are altered. The required changes in stiffness and inertia would have to be

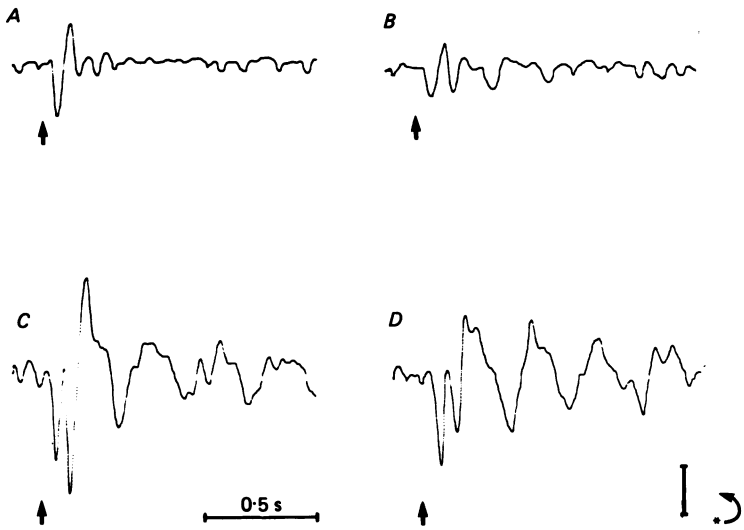

Fig. 4. Oscillatory response of extensor digitorum communis to stretch during sustained contraction. The muscle was repeatedly stretched (at arrows) by delivering brief downward jerks to the extended finger, at a repetition rate of 1·903 s. Each stretch triggered a 1·28 s sweep of rectified and demodulated e.m.g. activity. Thirty-two successive sweeps were summed, at various times throughout the contraction, to obtain the above figure. The finger was kept continuously raised by the subject and the nail was kept as near as possible to a fiducial mark. *A*, taken at start of experiment, *B*, after 5 min; *C*, after 32 min and *D*, after 35 min. These records should be compared with Fig. 4 in Lippold (1970). In the lower two records, the wave forms have summated, indicating that the oscillatory response is phase-locked to the stimulus and the predominant frequency response is in the range 3·6–4·6 Hz, corresponding to slow tremor. Vertical bar (*) is 10 × peak amplitude of single first wave in series of thirty-two, and was approximately equivalent to a rotation at the metacarpal–phalangeal joint of 9°.

large and would be reflected in a considerable slowing of the muscle twitch. To check this possibility, muscle twitches were elicited with just supramaximal direct shocks to the skin over extensor digitorum communis (30 V; 50 μs) at various times during a sustained contraction in the presence of 4–6 Hz tremor, the developed tension being recorded with a strain gauge. It can be seen from Fig. 5 that little change in the time course of the twitch resulted. It seems unlikely that an alteration in the mechanical properties of the neuromuscular system can be responsible for the 4–6 Hz peak.

Visual feed-back

The eyes remained open throughout the experiment and no systematic attempt was made to assess the role of visual feed-back in the generation of the slow activity. However, it was noted that the 4–6 Hz waves continued without alteration if the subject looked away from the finger, provided it did not then hit any part of the apparatus.

DISCUSSION

If normal 8–12 Hz muscular tremor is to be explained in terms of an oscillating servo-loop, it follows that in a particular individual, tremor frequency in a muscle would be expected to vary only within narrow limits. This is a consequence of the

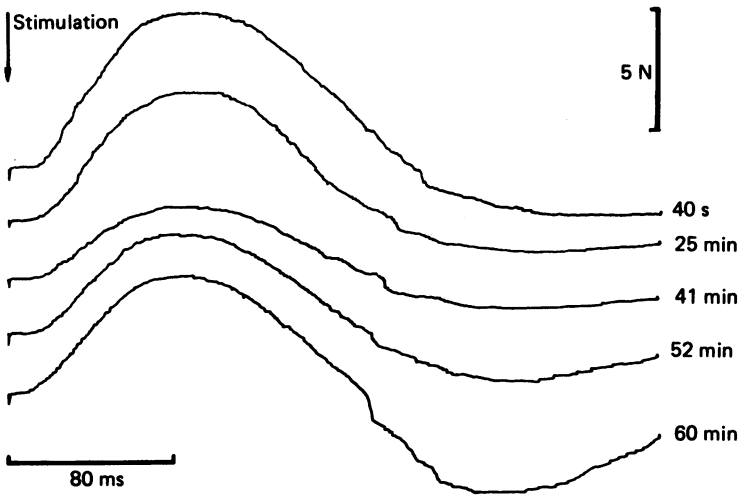

Fig. 5. Change in muscle twitch properties with sustained contraction. The extensor digitorium communis was stimulated at various times during the 60 min contraction (30 V, 50 μs), and the resultant twitch recorded as an extension of the middle finger upwards against a strain gauge. Time to peak tension decreased from 74 to 72 ms; relaxation time decreased from 72 to 60 ms. Times after start of contraction are shown at right; 4–6 Hz tremor was present from 25 min onwards.

delay in the loop (which determines frequency) being relatively invariant. Stiles (1976) and Stiles & Rietz (1977) have attacked the servo-loop theory for tremor on the basis that a prolonged contraction of the hand or ankle muscle leads to a progressive, considerable slowing of tremor. From our results it is clear that a prolonged contraction of extensor digitorum communis, while it does lead to the appearance of 4–6 Hz tremor, does not progressively lower the peak frequency of 8–12 Hz tremor until it is transformed into slow tremor. Moreover, both peaks are present at the same time and there is no evidence that either the 8–12 Hz or the 4–6 Hz tremor is changed in frequency appreciably during the contraction once it has appeared. It may be concluded therefore, that the two phenomena are separate entities. The appearance of 4–6 Hz tremor does not invalidate the servo-loop hypothesis.

The origin of the 4–6 Hz tremor can be sought in a number of possible mechanisms including (a) a visco-elastic-mass determined oscillation of the finger (Stiles & Randall, 1967), (b) a longer delay developing in the muscle itself, resulting from altered muscle properties or (c) oscillation in a long loop that may be spinal (Hagbarth, Hägglund, Wallin & Young, 1981) or includes the cerebral cortex (Phillips, 1969) for example. The possibilities (a) and (b) seem to be ruled out by the finding that the prolonged voluntary contraction that we have been using does not change the time course of a muscle twitch. A common index of muscle fatigue is prolongation of relaxation time (Mosso, 1915) and the lack of effect of the contraction upon twitch times underlines the fact that the development of the slow tremor is not necessarily associated with muscular fatigue.

An indication as to the origin of the slow tremor can be found in the results of the experiments with brief perturbations delivered to the finger. It is assumed that a

mechanical displacement of the finger excites muscle stretch receptors and acts as a step-function input to the reflex loop. If this is correct, the production of 4–6 Hz tremor by such an input must indicate the involvement of afferent feed-back. Oscillation would be characteristic of the behaviour of an under-damped servo-loop. We therefore propose that the slow tremor has a mechanism similar to that underlying 8–12 Hz tremor, but that it must involve a longer loop delay.

This dependence on activity in a feed-back loop, and also the fact that the slow activity is greatly reduced by ischaemia, incidentally rules out the possibility that this tremor arises in an autonomous central pace-maker (cf. Walsh, 1976).

Explanations for the slow tremor that implicate an oscillatory process i.e. category (c) above, also include the possibility that the 4–6 Hz peak is the first sub-harmonic of a fundamental at 8–12 Hz. Support for this hypothesis would be furnished if it could be demonstrated that the two frequency peaks were harmonically related. We do not find the frequency of the slow component in a given subject to be precisely half that of the 8–12 Hz peak frequency (see Table 1).

It is also of interest to note that the tremor characteristic of Parkinson's disease has a predominant frequency of 4–6 Hz. At present we do not know the mechanism whereby it is generated but further investigation of the slow tremor may show that the latter is a useful model for Parkinson's disease as suggested by Stiles & Pozos (1976), although our results lead us to conclude that it is unlikely to be a 'slowed-down form' of normal tremor.

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