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EFFECT OF VIBRATING AGONIST OR ANTAGONIST MUSCLE ON THE REFLEX RESPONSE TO SINUSOIDAL DISPLACEMENT OF THE HUMAN FOREARM

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SUMMARY

1. The mechanical resistance of the human forearm to imposed sinusoidal movements has been determined. By means of a visual monitor, subjects maintained a steady force (typically 100 N) by flexing the elbow so as to pull with the wrist against an isometric force transducer. This was mounted upon a stretcher which displaced the forearm sinusoidally at frequencies of 7–11 Hz with a peak-to-peak amplitude of movement of about 1 mm. The average mechanical resistance over 10–40 sec of stretching was analysed into its vector components at the fundamental of the stretching frequency. Observations were made of both the normal resistance and that obtained while applying continuous vibration at 100 Hz to the tendon of either the biceps (agonist) or triceps (antagonist).

2. In confirmation of Joyce, Rack & Ross (1974), at frequencies around 10 Hz the normal (unvibrated) response sometimes showed a component of '*negative viscosity*' (force increasing during muscle shortening), rather than the simple 'positive viscosity' attributable to muscle visco-elasticity; this effect is attributable to the stretch reflex being appropriately delayed and of sufficient magnitude to over-ride the inherent properties of muscle. Vibration of either agonist or antagonist usually increased the extent of the 'negative viscosity' (negative quadrature component of force), as well as changing the 'elastic' stiffness of the arm (in-phase component of force).

3. More commonly, the component of viscosity was initially positive. It was then normally reduced by vibration; that is, the vibration had (in formal terms) again added a component of negative viscosity.

4. The vibration did not produce these effects by acting directly upon the contractile system of muscle to reduce its 'visco-elasticity'. On increasing the frequency of stretching the effect of vibration systematically shifted from being the addition of a negative viscosity, as above, to being the addition of a positive viscosity. These effects may all be attributed to an action of vibration on the stretch reflex, with the precise action of the reflex determined by the relation between the cycle time and the delays round the reflex pathway.

5. In some experiments the activity of the flexor muscles was sampled by surface electromyograms from biceps and from brachioradialis; these were rectified, smoothed

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and averaged. For biceps, the absolute depth of e.m.g. modulation in relation to the cycle of stretching was sometimes, but not always, increased by vibration; but for brachioradialis the modulation was always reduced. Thus vibration cannot invariably produce its effects on the mechanical resistance of the arm by increasing the size (gain) of the stretch reflex. However, in all subjects the phase of the electromyographic modulation of both muscles was significantly delayed during vibration, whether of biceps or of triceps. In comparison with the normal, vibration introduced a phase lag on average of 18°. In qualitative terms, this can be shown to explain the typical augmentation of 'negative viscosity'.

6. The findings are discussed in relation to the genesis of tremor and to the reflex regulation of muscle contraction. They support the classical idea that afferent activity from the antagonist is as crucially implicated as that from the agonist.

INTRODUCTION

The quantitative study of the stretch reflex in man offers the prospect of achieving better understanding of how we normally control our muscles since the reflex is now widely agreed to be normally in action during movement, as well as during posture (see for example articles in Desmedt, 1978). Sinusoidal stretching provides a convenient method for analysis and its action on various parts of the reflex loop has been extensively studied in animals. Its use is of particular interest in relation to the genesis of tremor, but it can also provide findings of wider significance. In 1974 Joyce, Rack & Ross made an extensive study of the mechanical effects of sinusoidally displacing the forearm while the subject was attempting to maintain a constant force by elbow flexion. When the mean force was high (order of 100 N) and the amplitude of wrist displacement was small (1–2 mm peak-to-peak), then at frequencies around 10 Hz the subject's arm behaved as if it possessed '*negative viscosity*'; hence, averaged over the cycle, the arm did work upon the stretcher instead of work being transferred from the stretcher to the arm. This was attributed to the stretch reflex acting with a significant delay so that it assisted the applied movement, rather than opposing it as it does at lower frequencies of stretching. In other words, because of the phase lags inherent in neural transmission and muscle activation, at a frequency of about 10 Hz the reflex contraction occurs while the muscle is shortening rather than while it is lengthening. Under the appropriate mechanical conditions such 'negative viscosity' must lead to tremor, and Joyce & Rack (1974) saw this as exemplified by the tremor, of some mm extent at the wrist, that develops when a subject pulls against a spring. Using spectral analysis, Matthews & Muir (1980) provided further evidence for the reflex genesis of such tremor by observing the appropriate rhythmic motor firing when the subject forcibly flexed against the resistance of a spring, but which was not present when the subject developed the same force isometrically. Cussons, Matthews & Muir (1980) found that the tremor at a given mean force was enhanced on vibrating either the agonist (biceps) or the antagonist (triceps); remarkably, the effect of vibrating the antagonist was usually slightly greater.

The present experiments extend the analysis of the effect of vibration by studying its action on the reflex response to sinusoidal displacement of the arm. The findings consolidate previous work by showing that for appropriate frequencies of stretching

the vibration has the effect, in comparison with the normal, of introducing a component of 'negative viscosity', whether by increasing that already present or by reducing a pre-existing positive viscosity. Thus vibration would indeed be expected to increase the tremor seen when the arm is suitably free to move, as we have already briefly noted (Matthews, Muir & Watson, 1979). Concomitant electromyographic findings suggest that the essential change produced in the reflex by the vibration is a small but consistent phase lag. In addition the gain of the reflex was sometimes increased. These were both probably attributable to a similar behaviour on the part of the Ia discharge, as recently documented in the cat (Matthews & Watson, 1981). Vibration of the antagonist as well as of the agonist affects the reflex, even though the antagonist is apparently lying passive throughout. Thus the human stretch reflex, occurring *in situ* and in conjunction with ongoing motor activity, would seem to depend not only upon the afferent activity from the agonist, which provides the excitatory contribution, but also upon the afferent activity of the antagonist which provides an inhibitory contribution of apparently comparable power. Because the muscles are arranged in opposition to each other the two actions will regularly provide mutual support, with inhibition decreasing while excitation is increasing, and vice versa.

METHODS

The experiments were performed on thirteen normal human subjects aged 22–38; five were female and eight were male. Most of the methods have already been given in detail (Cussons *et al.* 1980; Matthews & Muir, 1980; Matthews & Watson, 1981) and so are now dealt with only in outline.

Mechanical arrangement. The subject sat with the right elbow supported on a rigid rest so that the upper arm was approximately horizontal. The elbow was flexed and the forearm ran vertically upwards in full supination. The hand was supported in extension by a light plastic splint which was taped along the back of the forearm and across the wrist to the level of the metacarpo-phalangeal joints. The front of the wrist pulled against a flat metal harness which was connected by steel cables to a force transducer. The stiffness of this part of the system, excluding the compliance of the soft tissues at the wrist, was 70 N/mm. The force transducer was mounted on the moving element of a large electromagnetic coil (Ling Dynamics, 409) which was controlled by feed-back to form a positional servomechanism. The stiffness of this 'stretcher' was 80 N/mm and its noise level, including some stray 100 Hz vibration, was 1·5 μm peak-to-peak. It had ample power to displace the arm sinusoidally with peak-to-peak movements of 1–2 mm at frequencies around 10 Hz (the stiffness of the arm was then always below 5 N/mm). In any case, the movement of the coil was monitored continuously by a length transducer thereby permitting compensation for any slight failure of the electromechanical system to produce the desired sinusoid in the face of the resistance of the arm. However, the feed-back system could not pass sufficient current to allow the coil to be used to produce the full steady force (up to 140 N) with which the subject was pulling against it. This bias force was provided by the elastic resistance of pre-stretched thick rubber tubing; when the subject was resting the tubing pulled the shaft of the coil against a mechanical stop.

Conduct of experiment. The subject's task was to maintain a given steady force at the wrist by keeping an oscilloscopic display of the output of the force transducer at a constant level. Mean forces of 60–140 N were used on different occasions. The cyclic component of force at the stretching frequency was eliminated from this display by low-pass filtering (cut-off, 2 Hz). The order for collection of data was usually as follows. After resting for about 1 min the subject was instructed to pull up to the commanded force. Analysis commenced some 5 sec after this had been achieved; in the early experiments, because of the particular requirements of the computer programme, only 2·5 sec of data were collected, but in later experiments 12–15 sec data were collected. The subject then relaxed. The stretcher was left oscillating throughout.

The observations were made in groups of three such trials. First, the subject pulled in the absence of muscle vibration. Secondly, he pulled while a physiotherapy vibrator (Pifco, 1556) oscillating

at 100 Hz was being pressed firmly by the experimenter upon the subject's arm immediately over the tendon of the biceps muscle (peak-to-peak movement about 0·5 mm *in situ*). Thirdly, he pulled while the vibrator was applied over the tendon of triceps, about 10 cm from the point of the elbow. Four or five such tripartite groups of data were collected in immedate succession for each condition studied.

Recording and analysis. The determination of the mechanical resistance of the arm to its displacement was done 'on line' using a PDP 12 computer by a method that enabled us largely to employ programmes that had been developed previously. A 2·5 sec segment both of the force recording and of the simultaneous displacement record from the 'stretcher' was filtered (1·5–17 Hz) and then analysed into Fourier components to give spectra with a bin width of 0·4 Hz. The stretching frequency was then given by the bin with the highest power in the displacement spectrum. The phase and depth of modulation of the force for this frequency were then compared with the corresponding values for the displacement and the computed phase difference and stiffness printed out; the separate in-phase and out-of-phase components of stiffness (cf. description of Fig. 1) were also printed. The results of several such analyses were then averaged to give a mean value for the vector stiffness under a given set of conditions. In the later experiments, with longer periods of recording on each pull, several separate 2·5 sec Fourier analyses were performed on the data from each pull, thus increasing the statistical reliability of the average.

All the data was also recorded on a seven-channel FM tape recorder along with markers indicating the phase of the sinusoidal movement. Cycle averages of the modulation of force were then sometimes constructed using a hard-wired averager (Neurolog, NL 750). The phase and gain of the force modulation could also be determined from these.

Electromyography. In the later experiments the gross electromyogram of both the biceps and the brachioradialis muscles was recorded with surface leads. After amplification (band width, 1 Hz–1 kHz) the signals were tape-recorded. On replay they were high-pass filtered (25 Hz), to remove any artifact at the stretching frequency, then rectified and finally smoothed in two stages (low-pass filters with cut-off at 25 and 17 Hz) to give the average envelope of the electromyographic activity ('demodulated' e.m.g.). The records from several separate periods of flexion were then averaged with regard to the cycle of stretching, and the gain and phase of the fundamental of the response determined. Such processing of the e.m.g. produced an appreciable phase lag. The extent of this was determined by treating similarly a stream of uniform pulses from a voltage controlled oscillator, the frequency of which was modulated sinusoidally.

RESULTS

The principal finding of the present experiments is that muscle vibration alters the mechanical resistance of the arm to imposed sinusoidal movement. An example is illustrated in Fig. 1 which also introduces the method of analysis. The averaged records on the left show the cyclic variations in the force generated at the wrist on displacing it sinusoidally while the subject was endeavouring to maintain a steady force of 120 N. These were obtained both for a normal purely voluntary contraction, and also while a physiotherapy vibrator oscillating at 10 Hz was firmly pressed onto biceps tendon just above the elbow. In both conditions the force can be seen to be modulated by the stretching, but to a slightly greater extent during vibration (thin line). This is further shown on the right where each force record has been analysed into its vector components in relation to the cycle of stretching.

The rationale behind vector analysis has been explained elsewhere (Joyce *et al.* 1974; Rack, Ross & Brown, 1978). Those deflexions in the horizontal axis that go to the right correspond to an elastic resistance (force in phase with stretch), and those to the left correspond to an inertial resistance (force 180° out of phase with stretching). Upward deflexions in the vertical axis correspond to a viscous, frictional, type of resistance (force leading displacement by 90°, and in phase with velocity), and downward deflexions correspond to a 'negative viscosity' (force lagging displacement by 90°, with maximum force at the time of maximum velocity of shortening). A vector with any other phase

angle corresponds to some mixture of the above components. The length of a vector specifies the magnitude of the change in force per unit change in length (mechanical impedance, complex stiffness). In terms of work, the existence of 'positive viscosity' means that the stretcher transfers energy to the limb, whereas the finding of a 'negative viscosity' means that the limb does work on the stretcher.

The wave form of the tension modulation in the experiment of Fig. 1*A* was approximately sinusoidal, but in some experiments the average modulation was clearly non-sinusoidal. Such 'harmonic distortion' tended to be associated with appreciable variation of the modulation from cycle to cycle as described by Joyce *et al.* (1974), and may be attributed to the variability of the stretch reflex. The most nearly sinusoidal modulations seemed to occur when the mechanical

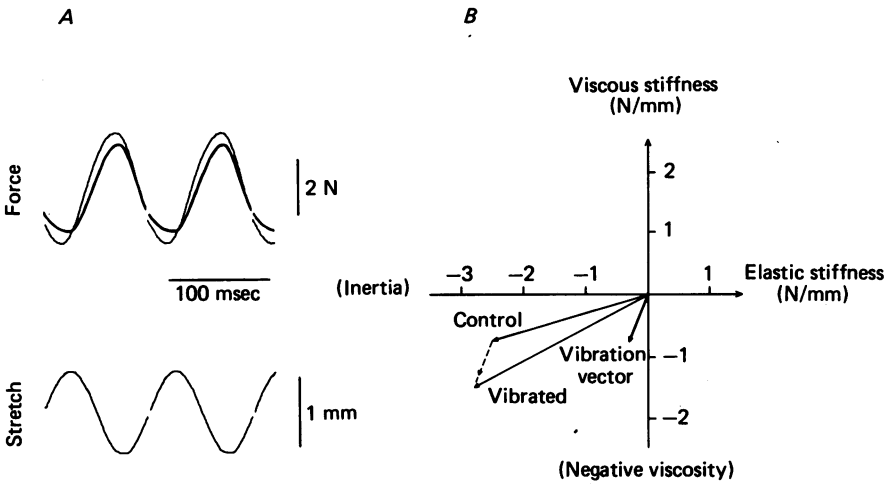

Fig. 1. An example of the effect of vibration on the mechanical resistance of the arm to sinusoidal stretching. Left, averaged cyclic variation in force at the wrist on displacing it by 1·2 mm (peak-to-peak) at 9·8 Hz while the subject was maintaining a mean force of 120 N by flexing the elbow; thick line, normal control; thin line, during vibration of biceps tendon at 100 Hz. Right, vector plot of the same data expressed as stiffness at the wrist. (The averaged record, based on 96 cycles of stretching, was actually computed for fractionally less than a complete cycle, but for ease of inspection the same data have been played out twice in the correct temporal relations to give the impression of two cycles; the gap corresponds to the short segment that was not averaged).

response to stretch was dominated by the passive inertia of the limb. Since the physiologically relevant component of the response, namely the action of the stretch reflex, was variable it was always necessary to average the measurements over a considerable number of cycles of stretching; this was occasionally done by averaging the force recording itself (as in Fig. 1*A*) but normally by performing a spectral analysis over an appreciable period of time to extract the fundamental of the stretching frequency (as in Fig. 1*B*, see Methods).

It can be seen from the control vector of Fig. 1 that the subject's normal resistance was mainly inertial, as might be expected from the appreciable force needed to move the mass of the arm backwards and forwards (and which outweighed the direct elastic resistance of the contracting muscles). In addition, there is a finite component of 'negative viscosity' during this unaided voluntary contraction, as already described by Joyce *et al.* (1974). During vibration of biceps the vector was rotated slightly

anti-clockwise, and the components of both the apparent inertia and of the 'negative viscosity' increased. The effect of vibration can be more succinctly described by taking the vector difference between the two experimentally determined responses; we have termed this difference measure the '*vibration vector*'. It is shown by the thick arrowed line in Fig. 1*B* and provides a direct indication that vibration has had the effect of adding a component of 'negative viscosity' to the mechanical impedance of the arm. Joyce *et al.* (1974) have shown that the normal degree of 'negative viscosity' must be attributed to phased reflex action, presumably of the stretch reflex,

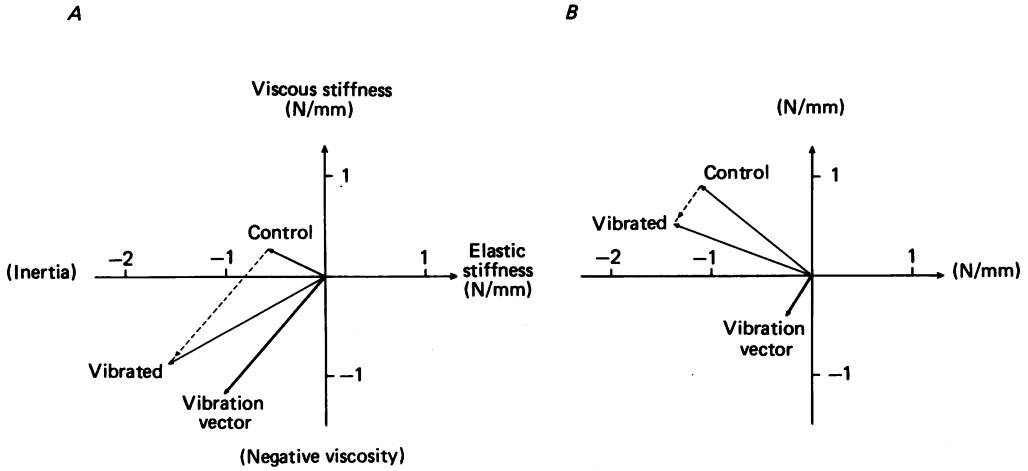

Fig. 2. Vector plots for two further subjects showing negative going vibration vectors. As was usual, one or both of the directly measured vectors ('control', 'vibrated') was upwards going, indicating positive rather than negative viscosity. For both, the vibration was applied to triceps tendon. Frequency of stretching, 8·2 Hz. Mean force, *A*, 100 N; *B*, 80 N. Period of analysis, *A*, 40 sec; *B*, 10 sec.

since there is no other way that the limb could be brought to do cyclic work on the external world. Accordingly, the addition of a further component of 'negative viscosity' on vibration seems likely to be due to some change in the stretch reflex, such as of its gain or phase relations.

Fig. 2 shows negative-going vibration vectors from two further subjects, but in this case produced by vibration of the flaccid antagonist (triceps) of the steadily contracting flexor muscles. In Fig. 2*A* the normal voluntary response showed a component of 'positive viscosity', rather than of 'negative viscosity' as in Fig. 1, but on vibration the 'viscosity' component reversed in sign and became negative. In Fig. 2*B*, however, the response showed a 'positive viscosity' both in the normal state and during vibration. Nonetheless, the underlying effect of vibration was the same as before, since the vibration once again caused an anti-clockwise rotation of the experimentally determined vector and, in formal terms, added a component of 'negative viscosity'; this is again shown by the downward-going vibration vector. It should be noted that an upward-going experimental vector need not necessarily mean that the stretch reflex is no longer contributing 'negative viscosity' to the

over-all response; a net component of positive viscosity would arise when a small amount of 'negative viscosity', due to a weak stretch reflex, was less than the normal positive viscosity of the contracting muscles. The findings of Fig. 2, with some of the experimental vectors being positive, were not dependent upon the vibration being applied to the antagonist rather than the agonist; vector plots similar to both those of Fig. 2 were obtained on vibrating biceps. Likewise, when the experimental vector for the purely voluntary contraction was already downward-going, as in Fig. 1, then vibration of triceps like that of biceps added a further component of 'negative viscosity'.

The level of statistical significance of the observed differences between the 'control' and the 'vibrated' vectors could be calculated on the basis of the variation between the separate 2·5 sec segments of original data used for spectral analysis (essentially by a multivariate analysis; see Watson, 1981, for detailed application of Wilks' criterion as described by Marriott, 1974). For the last five of the thirteen subjects the vectors were uniformly derived from fifteen rather than four segments of data. In these cases high levels of statistical significance were achieved: values of P (probability of difference between vectors being due to chance) of far below 0·001 were usual as was the case for Fig. 2*A*. When the earlier experiments were tested retrospectively, the values of P were generally not so significant. For Fig. 1*B* the value of P was below 0·05, whilst for Fig. 2*B* the value was 0·07; both these results were based on four segments of data. Higher values were obtained for some observations for some subjects. However, for eleven of the thirteen subjects the differences with vibration, both of biceps and of triceps, was significant at the 5% level or better for one or more frequencies of stretching. Full statistical analysis was performed on only a portion of the data, but a running check was kept on the variability of all the vectors by routinely determining the standard deviations as well as the means of the in-phase and out-of-phase components of the averaged vector.

Effect of frequency of stretching. In an early experiment we were initially puzzled by a subject who showed an upward-going vibration vector, indicating an additional component of positive viscosity, rather than the downward-going vector to which we were becoming accustomed. At that stage we were using only the single frequency of sinusoidal stretching of 9·8 Hz; it had been chosen to fall in the middle of the range for which tremor had already been observed on pulling against a spring (Cussons *et al.* 1980). This brought home to us that at a particular frequency the action of the stretch reflex on the mechanical properties of the limb might vary from subject to subject, since whether or not a 'negative viscosity' is produced will depend crucially upon the relationship between the temporal delays round the stretch reflex (including that of muscle contraction) and the period of the sinusoidal stretching; these may well vary between subjects. At low frequencies of stretching the reflex lags will be relatively unimportant and so the tension developed will tend to be in phase with the stretching and the reflex will act as an elastic resistance; it may even be phase-advanced on the stretch because of the properties of the muscle spindle. As the frequency increases, the lag in the development of reflex tension will become steadily more important and progressively delay the reflex relative to the phase of the stretching cycle so that the reflex produces in turn a 'negative viscosity', an inertial resistance, and then a 'positive viscosity' (see also Rack *et al.* 1978).

Thus if vibration produces some specific change in the stretch reflex, such as on its gain, then the precise mechanical effect of vibration should alter with the frequency of stretching, and the vibration vector should progressively change in direction. As illustrated in Fig. 3 this has indeed been found to be so. For both

subjects, at low frequencies the vibration vector was downward-going (added negative viscosity), but for high frequencies it was upward-going. Similar clockwise rotation of the vibration vector with increase of frequency of stretching was seen for eight of the eleven subjects, in each case both for vibration of triceps and for vibration of biceps. The rotation, however, was not always quite as systematic as in Fig. 3, presumably simply because of random variation. In fourteen of these sixteen frequency scans the final vectors were upward-going as in Fig. 3. The frequency at which the 'viscosity' component reversed from negative to positive varied between 8·4 and 11 Hz for different subjects.

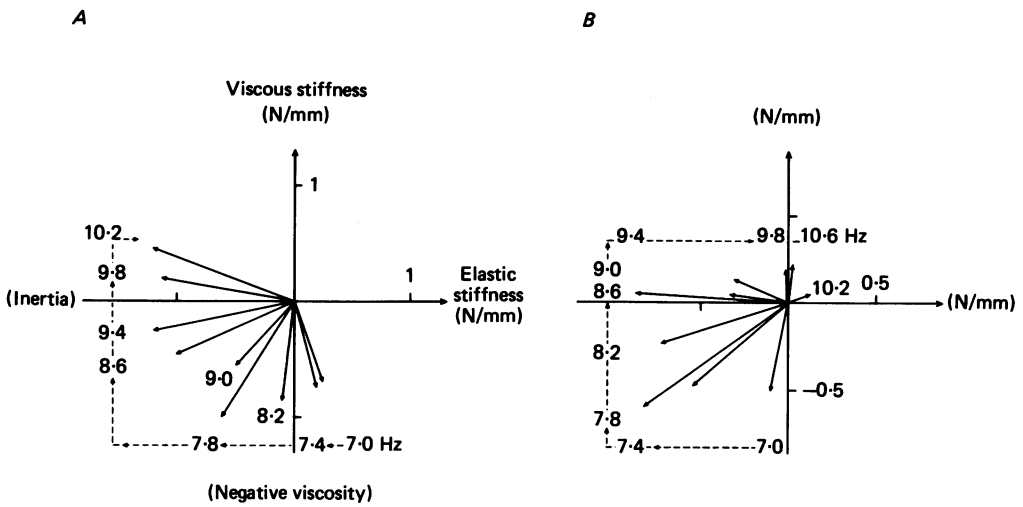

Fig. 3. The effect on the vibration vector (cf. Figs. 1, 2) of altering the frequency of sinusoidal stretching, shown for another two subjects. Increasing frequency is associated with clockwise rotation of the vibration vector and eventually with conversion of its viscous component from negative to positive. Vibration: *A*, biceps; *B*, triceps. Mean force, 80 N. Period of analysis, *A*, 40 sec; *B*, 10 sec.

Regression analysis of the relation between frequency and phase for the eight subjects showing clockwise rotation gave a mean of −34°/Hz (S.E. ±4°) with individual values of the correlation coefficient that were significant at the 5% level or better for all but two of the sixteen frequency scans (the value was not statistically significant once each for biceps and for triceps vibration, in different subjects). Two other subjects showed on average an anti-clockwise rotation of the vector with increasing frequency, but the variability was such that this could have arisen by chance ($P > 0{\cdot}4$). One subject showed the usual clockwise rotation for triceps vibration, but an anti-clockwise rotation for biceps vibration.

The repeated observation that with small changes of frequency of stretching the vibration vector can become upward-going is particularly important because it excludes an otherwise plausible possibility for the mode of action of vibration. This is that vibration acts directly on the contractile machinery of muscle to reduce its normal viscous resistance to stretching, as might happen by the rupturing of cross-bridges in the way that is thought to occur with large amplitudes of vibration (Matthews, 1966; Rack & Westbury, 1974). This would have the effect, in formal terms, of adding a component of 'negative viscosity' on vibration, but in fact the

change would be no more than the reduction of the pre-existing positive viscosity. It seems unlikely that vibration has any appreciable such action in the present situation, since the actual longitudinal vibratory movement in the muscle is likely to be too small (cf. Matthews & Watson, 1981). Moreover, even if the vibration did have some such slight effect, this could not explain the production of a 'positive viscosity'. This requires a quite different explanation, such as an action on the stretch reflex, which is then equally applicable to the production of a 'negative' viscosity at a lower frequency of stretching (see Discussion).

Comparison of responses between subjects. Eleven of the thirteen subjects were studied with a range of frequencies of stretching (usually 7·4–10·6 Hz in steps of 0·4 Hz). All showed negative-going vibration vectors for lower frequencies of stretching with vibration, whether it was applied to biceps or to triceps. The subject's steady target force was normally 80–100 N; the particular force was chosen either so as to optimize the appearance of a negative experimental vector in the absence of vibration by increasing the mean force (for the strong subjects), or to minimize fatigue by decreasing the mean force (for the weaker subjects). Control observations showed that for a given frequency of stretching the magnitude of the vibration vector tended to increase with increasing mean force, but not on such a scale as to vitiate the comparison between subjects (on average a 10 N increase in mean force produced a 15% increase in the size of the vibration vector). The results of Joyce *et al.* (1974) indicate that the size of the underlying stretch reflex increases with the mean level of force. The phase of the vibration vector, however, showed no consistent change on varying the mean force.

Fig. 4 shows the range of vibration vectors found for the main subjects at two frequencies of stretching (7·4 and 9·4 Hz). The points replace the lines of Figs. 1–3 and show the individual values of vibration vector, with vibration of each muscle indicated by separate symbols. The thick arrowed lines give the vector average for all the points, including those with each type of vibration. For the lower frequency the average vibration vector was strongly negative-going, but it was less so for the higher frequency. The magnitude of the vibration vector is in both cases about 0·6 N/mm, corresponding to a force modulation of the order of 0·7% of the ongoing contraction. It is obvious from the effect of varying the frequency of stretching for individual subjects (Fig. 3) that these average measures conceal as much as they show, since for a given frequency of stretching there appear to be real differences between subjects.

Triceps versus biceps vibration. Statistical analysis showed that there was no systematic significant difference between the effects of triceps and biceps vibration. On pooling the data for all frequencies for the eleven main subjects the mean size of the vibration vector for triceps vibration was 17% above that for biceps vibration, but the mean phase angles were the same (within 2°). The value of 17% is not significant at the 5% level in the t test, nor was a significant difference found when the same data were tested by the sign test (i.e. whether at each frequency for each subject the vibration vector was larger for biceps or for triceps vibration). It may be concluded that vibration of the antagonist is at least as powerful in its action on the reflex contraction of the active muscles as is vibration of one of the main agonists, namely biceps.

It should be emphasized that the antagonist, triceps, was normally lying flaccid

throughout, whether or not it was being vibrated. On the estimates made by Goodwin, McCloskey & Matthews (1972) this means that it was developing below 2% of its maximal contraction, equivalent to below 2 N, if it were to have been contracting at all. It must therefore have been having a negligible effect on the steady development of force. Equally, it seems most unlikely that the triceps could have been contributing to the cyclic variations in the force at the wrist by virtue of its own contraction.

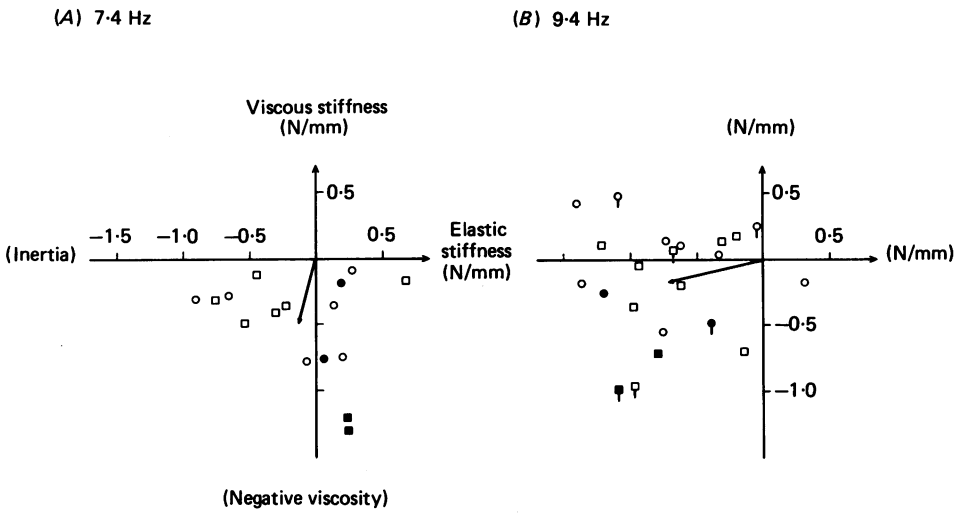

Fig. 4. Scatter diagrams of the vibration vectors (shown as points rather than as full lines) from 11 subjects for two different frequencies of stretching. The arrowed vectors indicate the mean value for all subjects. ○, ● biceps vibration; □, ■ triceps vibration. The filled symbols indicate that during vibration the directly measured vector (cf. Figs 1, 2) was downwards going (negative viscosity); the open symbols indicate that this measured vector was upwards going. The tails on some symbols in *B* indicate that these subjects were not tested at 7·4 Hz as in *A*.

Cyclic triceps activity would have been associated with a readily visible modulation in surface e.m.g. recordings from triceps as it was for biceps (as next described). In a control experiment in which the matter was specifically tested a just detectable modulation was found on averaging the triceps signal, which was about 10% of that found concurrently for the biceps e.m.g. This seems likely to have been due to electrical 'cross-talk' from the flexor muscles to triceps as described by Dietz, Noth & Schmidtbleicher (1981). The efficacy of the recording for detecting the activity of triceps itself was shown by the finding that the mean level of the rectified triceps e.m.g. on developing a given force at the wrist in extension was slightly greater than was that for the biceps e.m.g. on developing the same force in flexion. It should be noted, moreover, that because of the tetanic fusion properties of muscle the development of some 0·5 N of rhythmic contractile force at 8–10 Hz by triceps would have had to be accompanied by an appreciable steady contraction.

Electromyographic responses

In the last five experiments surface electromyograms were systematically recorded for a range of frequencies of stretching from the biceps and from the brachioradialis muscles. This was done to provide further information on the nature of the change in the stretch reflex produced by vibration; no attempt has been made to analyse

the equally interesting effect of altering the frequency of stretching on the stretch reflex *per se*, as recently done for the human jaw (Cooker, Larson & Luschei, 1980). For present purposes the e.m.g. has the advantage over force recording that no account has to be taken of the mechanical properties of the limb and muscles, thus simplifying interpretation. However the surface e.m.g. has the disadvantage that it provides a spatially limited sample of the muscular activity responsible for flexion; three separate muscles are involved in this (biceps, brachioradialis and the inaccessible

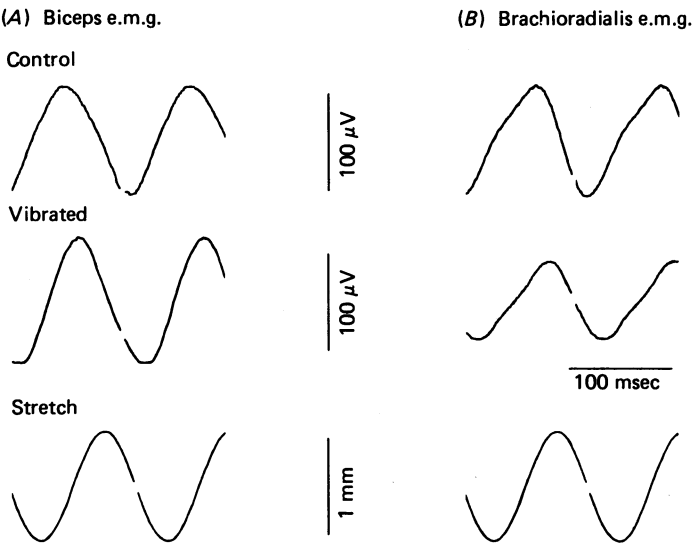

Fig. 5. Cycle averages of the rectified and smoothed surface electromyograms from biceps and from brachioradialis, recorded simultaneously. Top, control; middle, during vibration of biceps. 330 cycles averaged; as in Fig. 1, for ease of inspection, the average is displayed as if it covered nearly two cycles. Mean force 100 N. Frequency of stretching, 8·2 Hz. Bottom trace positioned to compensate for phase shifts in analysis. (Same subject as in Fig. 2*A*).

brachialis); moreover, the electrodes over a given muscle will have picked up the activity of an indeterminate and possibly small proportion of its active motor units, and thus provided a sample with an unknown degree of bias. Probably related to this, the recordings proved to be rather 'noisy' so that extensive averaging was required. None the less, certain e.m.g. findings stand out and assist understanding of the mechanical response.

Fig. 5 illustrates the consistent effect on the electromyogram, at all frequencies of stretching, of vibrating biceps in one particular subject. The records consist of the average of the rectified and smoothed e.m.g. for 330 cycles of stretching at 8·2 Hz, derived from five separate 8 sec periods of recording. As was usual, the averaged e.m.g. records were well modulated in synchrony with the stretching cycle. In the absence of vibration (top) the peak-to-peak modulation was 30 % of the mean level of activity for the biceps e.m.g., and 33 % for the brachioradialis e.m.g. (zero level not illustrated) The wave form of all four responses is approximately sinusoidal,

though fortuitously in this example less so for brachioradialis than for biceps. On occasion the cycle averages for either muscle might show a marked deviation from a simple sinusoid, such as a definite hump on the rising phase, a grossly flattened peak or occasionally even a slightly bifid peak. Such 'harmonic distortion' has not been studied currently. The main response has been assessed numerically by fitting a pure sine to the fundamental at the stretching frequency and determining its amplitude and phase, and then comparing the values obtained in the presence and absence of vibration.

Effect of vibration on the depth of the e.m.g. modulation. The middle traces of Fig. 5 show the e.m.g. responses during vibration of biceps. The mechanical resistance of the arm then had a clear component of 'negative viscosity', in comparison with a slightly positive value of 'viscosity' in the absence of vibration (the vector plot was very similar to that of Fig. 2*A* which shows the effect of triceps vibration on the same subject). In association with this the depth of modulation of the biceps e.m.g. can be seen to have increased relative to the normal, while that for brachioradialis decreased. Similar e.m.g. changes were seen for this subject for all ten frequencies of stretching studied. The mean increase of modulation of biceps e.m.g. across all frequencies was 37 % ± 6 % (S.E. of mean), and the mean decrease for brachioradialis was 19 % ± 4 %. Vibration of triceps had the same effect (mean increase for biceps e.m.g., 38 % ± 6 %; mean decrease for brachioradialis, 11 % ± 4 %). Such pooling of the effects over a range of frequencies of stretching seemed valid since no systematic relation could be detected by regression analysis on relating the change of gain to the frequency.

Of the five subjects studied in detail (including the one of Fig. 5), all showed a reduction of the absolute modulation of brachioradialis e.m.g. with vibration, whether of biceps or of triceps. The reduction ranged from 5 to 35 %. The effect of vibration on the modulation of the biceps e.m.g. was, however, very variable. On vibrating biceps the average modulation increased for two subjects (by + 37 % for the subject of Fig. 5, and by + 109 % for another), was effectively unchanged for two subjects (+ 6 % and + 2 %) and was slightly decreased for the fifth subject (− 15 %). On vibrating triceps the modulation of biceps was clearly increased only for the subject of Fig. 5 (+ 38 %), was possibly increased for another (+ 8 %), and was decreased for the three other subjects (by 16–30 %). For all the values just cited the standard error was below 10 % (median value 5 %). Another four subjects were studied earlier in the series, with shorter periods of recording and this from biceps only; both increases and decreases of its modulation were again seen. What was happening to the equally important flexor muscle brachialis was quite unknown.

Watson (1981) enlarges upon these findings. It should be noted, *inter alia*, that if vibration were to produce a significant degree of synchronization of the firing of different motor units, then the arithmetic of the electrical summation of their activity might be different in the presence and absence of vibration (cf. Matthews & Watson, 1981) and so affect the depth but not the phase of the measured response. Whether any appreciable synchronization occurs was not currently investigated. Cussons *et al.* (1980) earlier argued on indirect evidence that any that occurs does not have a major effect.

Effect of vibration on the phase of e.m.g. modulation. In marked contrast to the variable effects of vibration on the depth of the e.m.g. modulation, vibration was

regularly found to delay the phase of the e.m.g. response. For example, in Fig. 5 the modulation both for biceps and for brachioradialis during vibration (middle) can be seen to lag behind the normal responses (top). In this case the phase lag produced by vibration, obtained by fitting a pure sine to the responses, was 30° for biceps and 50° for brachioradialis. Fig. 6 shows the change of phase angle with vibration, for every frequency of stretching, for the five subjects studied systematically with

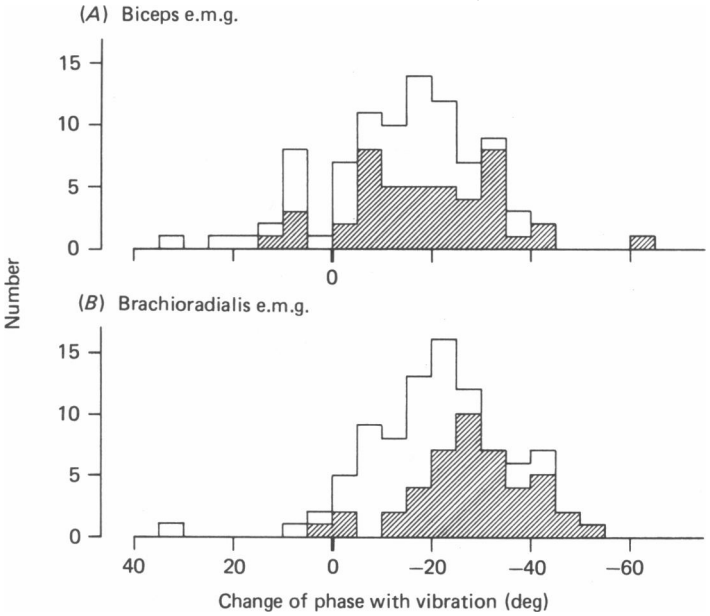

Fig. 6. Histogram of the change in phase of the averaged e.m.g., produced by vibration. A negative value indicates that during vibration the response to the stretching lagged behind the normal. Top, e.m.g. recorded from biceps; bottom, e.m.g. recorded from brachioradialis. Cross-hatching, biceps vibrated; clear, triceps vibrated. Data from 5 subjects for all frequencies of stretching employed for each (not the same for all subjects); each value is derived from a pair of cycle averages, as in Fig. 5, for a given frequency of stretching and for about 40 sec recording. There was no systematic relation between the change in phase and the frequency (see text).

recording both from biceps (above) and from brachioradialis (below), on vibrating in turn both agonist and antagonist. The few phase advances that were observed in such individual averages seem likely to have been due to random variation. The mean phase lag with vibration was 14° (±15° S.D.) for the biceps e.m.g., and 21° (±14° S.D.) for the brachioradialis e.m.g. Calculation of regression lines for each subject individually showed that there was no systematic relation between the magnitude of the change of phase with vibration and the frequency of stretching (a small effect might perhaps have been lost in the noise). However, the absolute phase of the e.m.g. response in relation to the stretching lagged progressively as the frequency increased. For each subject the change of phase of the e.m.g. modulation was averaged across frequencies. This showed a lag with vibration, for each muscle considered on its own and for each type of vibration. Equally, on pooling all the data for a given subject

the average phase change was also always a lag. The mean value on pooling all 180 observations from the five subjects, as in Fig. 6, was 17·6° with a standard deviation of the population of the points of ±15°. The four additional subjects studied only with biceps recording gave confirmatory results.

Extensive statistical analysis would be required to determine confidence limits for the data. The variability in the whole sample will depend partly upon random variation in the collection of data and in the behaviour of the reflex, but it will probably also depend upon real variations between

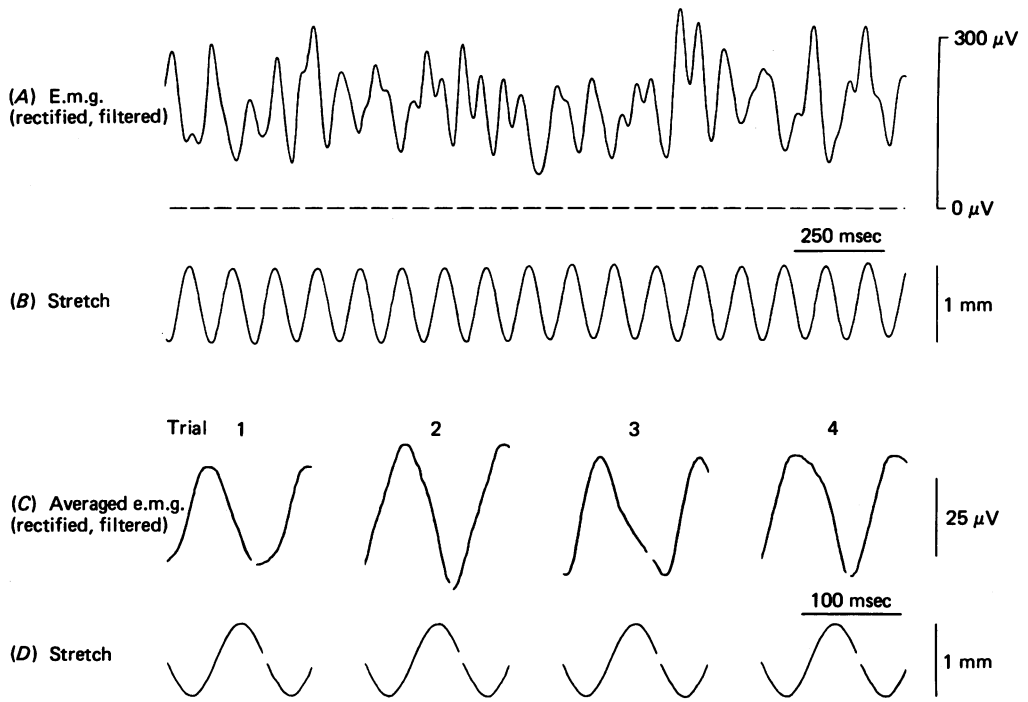

Fig. 7. Records illustrating the variation in the e.m.g. recordings, even after a certain amount of averaging. *A*, short segment of original e.m.g. after rectifying and smoothing. *C*, four separate cycle averages of the e.m.g., each based on 69 cycles of 8·6 Hz stretching and obtained from a separate trial in which the subject maintained the target force of 100 N for some 8 sec; the averages of Fig. 5 were obtained by pooling data from 5 such trials. No vibration applied. (Averaging procedure as in Fig. 5. The traces in *C* have been aligned to have approximately the same mean level, and do not have quite the same zero level).

subjects and the effects of recording from, or of vibrating, different muscles (cf. bottom histogram of Fig. 6). Moreover, the values obtained for the change of phase with vibration of biceps and of triceps are not independent, since they depend upon the same control observation of the unilateral value of phase.

It may be noticed in Fig. 5 that the response for brachioradialis lags behind that for biceps. Such behaviour was characteristic for this subject and one other, while for another two the difference was the other way round, and for the fifth there was no appreciable difference. This may perhaps reflect small differences in the precise posture of the subjects and the way they pulled, and seemed immaterial for present purposes.

Variability of response. The description of the results so far has been based on the implicit assumption, now common to much physiology with the advent of simple averagers, that sufficient smoothing and averaging to permit the emergence of the statistical average will also preserve everything of physiological interest. We are concerned, however, that in doing this we may have allowed features of the individual responses to escape attention. Thus it seems desirable to emphasize the large amount of variability that may occur between successive cycles of stretching, some of which is probably due to moment-to-moment variations in the behaviour of the stretch reflex rather than simply to the limited e.m.g. sampling. The top record of Fig. 7 shows a period of raw biceps e.m.g. after rectification and smoothing but without averaging. For part of the time there appear to be two waves per cycle rather than just one. Complex responses to stretching have already been noted by others (Rack, Ross & Walters, 1979; Agarwal & Gottlieb, 1977). The bottom of Fig. 7 shows four averages of the e.m.g. obtained under identical conditions, each based on 8 sec recording approximately 3 min apart; our usual 40 sec averages were produced by combining five such trials. The gain and the phase of the 8 sec averages can be seen to vary appreciably, as well as the extent of harmonic distortion. The maximum phase advance of the electromyographic modulation in relation to the point of maximum stretch was 121° and the minimum value was 111°. The maximum depth of modulation was 1·35 times the minimum value. Some such variability must have persisted in the 40 sec averages used hitherto, and been responsible for some of the scatter in the data.

DISCUSSION

The present observations on the effect of vibration on the mechanical resistance of the arm to stretching provide an immediate explanation for the earlier finding that vibration augments the tremor of the compliantly loaded arm (Cussons *et al.* 1980). When the 'viscosity' is negative, as averaged throughout the cycle, the arm with its reflexes is able to do work on the external world and can thus maintain a continuous tremor movement if the arm is suitably free to move. This occurs on pulling against a spring which also allows for the development of the high mean level of contraction that is essential for the gain of the stretch reflex to be appropriately high (Joyce *et al.* 1974). The considerable moment-to-moment variability of the stretch reflex that was seen both in the present work and in that of Joyce *et al.* means that even when the average value of the 'viscosity' is positive there will still be short periods when it becomes negative, thereby permitting short runs of tremor, as is commonly seen. Thus the regular effect of vibration in adding a component of 'negative viscosity' to the mechanical resistance of the arm should regularly enhance any pre-existing tremor, whether or not the 'viscosity' averaged over a long period was already negative. It is essential, of course, for the development of appreciable such tremor that the mechanical tuning of the mass-spring system comprised by the arm and its attachments should have a suitable resonant frequency, namely one which falls within the range for which the lags in the stretch reflex cause it to contribute to the development of 'negative' rather than of 'positive' viscosity.

It bears reiteration that the change in 'viscosity' with vibration must be attributed

to some action on the stretch reflex rather than any direct effect on the mechanical properties of muscle or joint, since with increasing frequency of stretching the effect of vibration on viscosity sometimes reversed in sign. With 7–8 Hz stretching, vibration normally added a component of 'negative viscosity' to the pre-existing response, whereas with 10–11 Hz stretching it might add 'positive viscosity', as in Fig. 3.

How and why does the stretch reflex change during vibration?

On the balance of evidence, the earlier finding of an enhancement of tremor with vibration was suggested to arise peripherally rather than centrally, and to depend upon a change in the pattern of the Ia response to stretching (Cussons *et al.* 1980). The actual effect of vibration on the response of Ia afferents to sinusoidal stretching has since been studied on de-efferented muscle spindles in the anaesthetized cat and the following changes found (Matthews & Watson, 1981). With small amplitudes of vibration the depth of Ia modulation induced by the stretching was increased; the afferents then altered their firing between the frequency of the vibration during the rising phase of the stretching, when 1:1 driving occurred, and virtual silence during the releasing phase of the sinusoid. But with larger amplitudes of vibration the modulation decreased progressively because the Ia afferent responded at the vibration frequency and its sub-harmonics throughout more and more of the cycle, until eventually its discharge was 'clamped' continuously at the vibration frequency. Thus, depending upon its amplitude, vibration may either increase or decrease the 'gain' of the spindle and its response to sinusoidal stretching. However, whatever the amplitude of the vibration it invariably caused the phase of the fundamental of the averaged envelope of the Ia response to stretching to be delayed with respect to the normal. The lag increased progressively with the amplitude of vibration up to a maximum of about 30°. The lag arose because the spindle continued to respond maximally to the vibration at around the peak of the stretch, at which time its frequency would normally have been decreasing, rather than to a uniform delay in the spindle response throughout the cycle.

In the present experiments the electromyogram was found to be cyclically modulated by the stretching, presumably because of a modulation of Ia firing. For the biceps e.m.g. the depth of modulation was sometimes increased and sometimes decreased (see Results), while that for brachioradialis was uniformly decreased. However, the phase of the averaged e.m.g. responses for both muscles regularly showed a lag (average, 18°). Thus there is a general similarity between reflexly elicited e.m.g. modulation in man and that earlier described for Ia firing in the cat. However, there is no guarantee that the central gain of the reflex remains unchanged during vibration and it would be premature to conclude that a change in the depth of e.m.g. modulation directly corresponds to the change in the depth of Ia modulation. But the phase lag produced in the e.m.g. by vibration seems reasonably explained by the phase lag that occurs regularly for the cat spindle, and which may be presumed to have occurred equally in the present situation in man.

It should be noted that on the basis of rough estimates we earlier suggested that the Ia modulation in the present human situation would commonly be increased by vibration (Matthews & Watson, 1981), whereas this has been the exception for the e.m.g. in the present experiments;

nor does there seem any reason to revise the estimates. The difference might be suggested to arise from the human spindles being under tonic fusimotor drive and so showing less augmentation of their sinusoidal modulation with vibration than occurs in the de-efferented state (see the Discussion in Matthews & Watson, 1981). But the spindles in the flaccid triceps muscle in the present experiments seem unlikely to have been under appreciable tonic fusimotor drive, since this is normally coupled with alpha motor activity (Vallbo, Hagbarth, Torebjörk & Wallin, 1979). Thus this explanation would only be applicable if the reduction of e.m.g. modulation were to have occurred solely with vibration of biceps and not with that of triceps; if anything the tendency was the other way round. A more likely reason for the discrepancy is that, as for the leg (Delwaide, 1973; Iles & Roberts, 1981), presynaptic inhibition of Ia afferents is reflexly initiated by the vibration (whether by means of spindle or cutaneous afferents) and that this then has a significant effect in reducing the synaptic efficacy of a given Ia discharge. If so, the e.m.g. could be reduced during vibration even though the Ia modulation was increased.

An increase in the e.m.g. modulation, when it occurs, provides an immediate explanation for an increase in 'negative viscosity', since if the stretch reflex is already producing 'negative viscosity' the effect will increase *pari passu* with the size of the reflex. When at the outset of the present experiments such e.m.g. increases were found for biceps it was initially supposed that this would be all that there was to it; biceps is so much larger and stronger than brachioradialis that its behaviour can be expected to dominate the over-all mechanical response. But as the data accumulated this view became untenable as it was found that vibration might produce an increment of 'negative viscosity' at the same time as reducing the e.m.g. modulation both for biceps and for brachioradialis. Apart from suggesting that the important flexor brachialis was acting in the opposite way, which would be unsupported special pleading, there thus seems no possibility that the development of 'negative viscosity' with vibration can be attributed solely to an increase in the *size* of the stretch reflex. It must next be asked whether the changes in viscosity can be ascribed to the slight lag produced by vibration in the phase of the reflex, although at first sight such small changes in timing (*ca.* 6 msec) might seem unimportant. In qualitative terms the answer is: *yes*, they could be responsible; but no attempt has been made to test the matter quantitatively.

Fig. 8 illustrates diagrammatically how the phase lags produced by vibration could, in principle, add 'negative viscosity' to the system. The vectors in Fig. 8*A* show the reflex responses both in the e.m.g. and in force generation, in the presence and absence of vibration. The e.m.g. leads the stretch very appreciably, as seen experimentally (the value of 100° used for the 'control' e.m.g. vector was found on stretching at frequencies around 8 Hz). The lead must arise partly from the behaviour of the Ia afferents, but quite likely also from action within the spinal cord, as suggested for the cat (Jansen & Rack, 1966; Westbury, 1971). Because of the slowness of muscle contraction the reflexly developed force lags well behind the e.m.g., and so slightly behind the stretch, thereby contributing a component of 'negative viscosity' along with elastic resistance (the lag of 125° shown is that for the human masseter at about 8 Hz; Cooker *et al.* 1980). The dotted lines show the same responses on the assumption that the vibration acts solely to cause a slight lag in the Ia response, without change of reflex gain (a lag of 20° has been used in accordance with our own e.m.g. data). The electromyographic and reflex responses are then similarly delayed so that the vibration adds a component of 'negative viscosity'. This is further shown by the vibration vector, thickly drawn in the third quadrant (i.e. the vector difference

between the force responses in the presence and absence of vibration). It should be noted that the phase of the vibration vector bears no direct relationship to the phase of the reflex responses themselves. If the vibration were to have simply increased the size of the reflex without changing its phase then, of course, the phase of the vibration vector would have been the same as that of the reflex.

Fig. 8*B* shows how the reflex responses would interact with the passive mechanical properties of the arm to give the force vectors, as observed experimentally. The

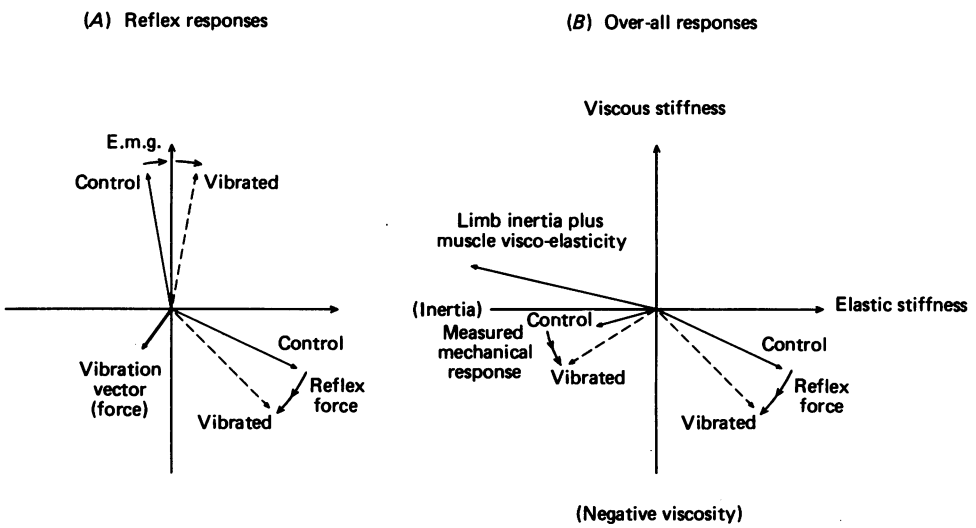

Fig. 8. Speculative diagram to explain how a lag in the phase of the electromyogram on vibration might lead to the addition of a component of 'negative viscosity' to the overall mechanical response of the arm to sinusoidal displacement. *A*, vector plot of the reflex responses during a voluntary contraction (control, solid) and during vibration (dotted) shown for the electromyogram and, with a lag of 125°, for the reflexly evoked force; the difference between the two force vectors gives the 'vibration vector' of the earlier illustrations. *B*, vector combination of passive mechanical response of the limb and the contracting muscles with the reflex responses of *A* to give the experimentally measured mechanical response. Further explanation in text. (The e.m.g. vectors in *A* are arbitrarily drawn at the same size as the reflex force vectors).

difference between these two directly determinable resultants is, of course, the vibration vector as we originally defined it. From Fig. 8*A* it can readily be appreciated that quite small changes in the parameters of the system would produce large changes in the phase and magnitude of the vibration vector. A rigorous analysis would require the collection of a great deal more quantitative information.

Fig. 8 also helps to illuminate the effect of increasing the frequency of stretching on the direction of the vibration vector. In terms of phase angle both the delay between the Ia discharge and the resulting e.m.g. activity (not illustrated), and the delay between the e.m.g. activity and the development of reflex force can be expected to increase with frequency. Moreover, these additional lags with frequency outweigh any increased phase advance of the Ia firing, since we observed that the phase advance of the e.m.g. on the stretch decreased progressively with increasing

frequency. The e.m.g. and, more particularly, the force vectors thus both rotate clockwise with increasing frequency, thereby also causing the vector difference between the latter (the vibration vector) to rotate clockwise. Hence it is to be expected that at some frequency the viscosity component of the vibration vector should reverse from negative to positive with increasing frequency of stretching.

It may be concluded that the observed lag in the e.m.g., and thus also in the reflex response, can contribute to the development of negative viscosity with vibration. It should be emphasized, however, that the present study has confined itself to the analysis of the fundamental response at the frequency of stretching and important matters may thereby have escaped attention, such as the roles of reflex pathways of different lengths. The present analysis with its emphasis on 'negative viscosity' brings out those features of response which are important for determining whether or not tremor occurs when the limb is working into a suitably tuned load.

Action of antagonist

Vibrating the triceps, the antagonist of the contracting flexor muscles, had just as much effect on the reflex response of the flexor muscles to the imposed arm movement as did vibration of biceps itself. For various reasons the effect seems most unlikely to have been due to spread of vibration to the flexor muscles (see Cussons *et al.* 1980, especially for the use of modulated vibration). Rather, it supports the idea that the antagonist is normally playing a significant part in the development of a 'negative viscosity' of the arm in the absence of vibration. However, in the present situation triceps does this by its reflex effects on the contraction of the flexors rather than by contracting itself; triceps was believed to remain flaccid throughout. Because of the double inversion of sign involved, the patterning of the Ia discharges from the antagonist muscle will, in functional terms, directly support the Ia discharges from the agonist, although at first sight such synergy might seem paradoxical. First, the antagonist Ia discharges have an inhibitory action on the agonist motoneurones. Secondly, these antagonist discharges are excited in push–pull with the agonist Ia discharges, by virtue of the mechanical arrangement of the joint so that one muscle is stretched while the other is released.

An important general point follows from the above. Assuming that vibration produces much the same change in spindle firing for agonist and antagonist muscles, then their reflex actions under normal conditions must be similarly potent, since vibration of the opposing muscles has similar effects. In other words, the reflex response to joint movement of an active muscle depends as much upon the changing pattern of afferent activity from its antagonist as upon that from itself. The present experiments thus help to illustrate for man the importance of reciprocal innervation and its contribution to the myotatic reflex as seen *in situ*; these classical ideas arose largely from work on the cat.

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