The evidence they quote in support of this statement is from experiments in the cat using brief rapid muscle stretch. Under the rigorous conditions that can be imposed in animal experiments, the parameters of stretch can be controlled to allow selective activation of primary endings, but this cannot be done in man.

5. The authors assume that changes in the evoked ulnar nerve potential produced by reinforcement or eye closure result from changes in fusimotor bias. However, they have not eliminated a change in muscle length. Even if they had immobilised the hand (which they did not do), it is not possible to immobilise human subjects completely. Only an accurate strain gauge will detect the small changes in muscle length that can still occur in an immobilised human hand.

6. On page 229 the authors state that, of the factors contributing to the reinforcement of tendon jerks, “the fusimotor effects are more dominant.” However, nerve block studies have shown that reflex reinforcement still occurs when the muscle spindle contribution has been abolished by local anaesthetics (Clare and Landau, 1964) or by pressure/ischaemia (Bussel et al., 1978). In addition, Hagbarth et al. (1975) have monitored the spindle input during reinforcement manoeuvres and have shown that tendon jerks are potentiated in manoeuvres which did not affect the input from spindles. These studies do not exclude that fusimotor activation contributes to the reflex potentiation seen in the clinical setting, but they do demonstrate that the Jendrassik manoeuvre does not exert its effects predominantly by facilitating fusimotor neurones. This conclusion could also have been reached by Murthy et al. by examining their Fig. 4b and e on page 223. The afferent response in Fig. 4e is smaller and more dispersed than in Fig. 4b (although its onset is earlier), but a reflex response is seen in Fig. 4e (although its onset is earlier), but a reflex response is seen in Fig. 4e and not in Fig. 4b. Assuming the experimental conditions were identical (and that is an assumption), some form of central reflex enhancement must have occurred.

7. In single unit recordings Burg et al. (1974) demonstrated what they considered to be a fusimotor effect on spindle discharge during reinforcement manoeuvres, but their methods did not exclude changes in length of the percutted muscle and, assuming that the effects were of fusimotor origin, they did not establish that the effects were due to selective activation of the fusimotor system. When these studies were repeated with appropriate controls, a selective effect of reinforcement manoeuvres on the fusimotor system could not be demonstrated (Hagbarth et al., 1975).

For too long has the neurological literature been plagued by speculations about the role of the fusimotor system in normal and diseased states, based on techniques which are not adequate to isolate or exclude fusimotor involvement conclusively. It would be of great interest if the promising technique described by Murthy et al. were applied with more rigorous experimental controls.

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References

Sir,—Thank you for providing an opportunity to reply to Dr Burke’s criticism of our papers (Murthy et al. 1978a, b). His points are answered in the same sequence.

1. There was no doubt that the contribution to the afferent waveform was mainly from the ulnar nerve with the indifferent electrode over the radius picking up no detectable activity from the radial nerve. Any such activity in the radial nerve would have resulted in a negative phase of the recorded waveform as observed in Fig. 3a (page 228) which was included to show such a possibility.

2. The qualitative evaluation of recorded waveforms does not sufferwant of more rigorous experimental controls. The variability in the form of the taps would have affected the shape of the response quantitatively rather than alter its qualitative features (latency and duration). For more quantitative evaluation modifications can be made in the experimental method, especially in positioning and immobilising the hand, by using an automated percussion device to eliminate the variability in the force of the taps and by using sensitive transducers to record changes in muscle length.

3. As to the contributions from other receptors in other intrinsic muscles of the hand and in the skin over the ulnar aspect of the hypothenar eminence, it should be borne in mind that the mechanical vibration travels very slowly in the muscles and also should decay in some fashion with distance from the place where the taps are applied. Controls as illustrated in Fig. 4a (page 233 in Murthy et al. 1978a) permitted us to conclude that contributions from other intrinsic muscles would be insignificant.

4. We would like to question the connection between the qualitative observations of other muscle afferents responding to a local prod on the belly of the muscle and a percussion applied to the deep muscle tendon in our study. Dr Burke's...
refers essentially to undocumented evidence in declaring as incorrect the assumption that the phasic tendon tap is a selective and adequate stimulus to the primary endings of the muscle spindle. He probably speaks from personal experience in recording from single afferent fibres in human nerves. The precise time relationships of impulses in various muscle afferents responding to a tap are not available in spite of more than a decade of intraneur recordings from human subjects. For example, it is not enough to say that all types of receptors respond to a tendon tap. We need to know if all of them respond within the 20 ms period after the percussion in which the afferent activity was averaged. The published data of intraneur recordings of human muscle afferents to date are observations made on a much slower time scale, so that it is difficult to determine if all the muscle receptors (pacinian corpuscles, tendon organs, etc.) have the same dynamic sensitivity as the primary endings of the spindle to the brief and light tendon tap. Until such details are available we have to base our assumptions on results of animal experiments. As Dr Burke himself states the tendon percussion "excites primary endings better." It should also be noted that nowhere in our papers has it been stated that the afferent response was only due to spindle primary afferents. We took care to specify that primary spindle endings would be the major contributors.

5. In our study, we ensured that no movement of the hand occurred during the tests. Any "small changes in length" of muscle that can "only be detected by an accurate strain gauge" will not have produced the type of changes in averaged afferent waveform (produced by reinforcement of eye closure) which we have described. We have also considered the following documented features of human muscle spindles (Vallbo, 1974a, b).

i. The muscle spindle afferents discharge normally at a low frequency (less than 50 imp/s) and display a poor position sensitivity in relaxed muscles.

ii. Human spindle endings possess high dynamic sensitivity in relaxed muscles, responding impressively to minute mechanical disturbances.

6, 7. The arguments on the contribution of fusimotor efferents to the reinforcement of tendon reflexes reflect the continuing controversy on this subject. Our own analysis of the differences in fusimotor effects obtained in various laboratories has given us the impression that different levels of fusimotor effects may be obtained during reinforcement manoeuvres depending upon the posture of the subject during the manoeuvre, the strength of the manoeuvres, and the strength of tendon percussion itself. It is, then, possible that the relative central effects on alpha and gamma motoneurones are variable, with gamma effects being prominent at times. It seems the timing of the reflex response (in relation to the onset of the excitability changes produced in a reinforcement manoeuvre) is also an important consideration (Kawamura and Watanabe, 1975). In addition, it should be stressed here that the fusimotor effects observed by us are more in reducing the latency of afferent waveform than in increasing its size. As to the latency and duration of the afferent waveform, these correlate very well with intraneur multiunit recordings (Fig. 6 in Hagbarth and Vallbo, 1968; Fig. 6 in Hagbarth et al., 1975). The longstanding controversy of the relative amounts of alpha and gamma effects in reinforcement manoeuvres can only be resolved by obtaining sufficient data with both single unit and multiunit recording techniques.

The differences in the size of the afferent response between Fig. 4b and 4e on page 223 is explained simply by the fact that the response in Fig. 4e was obtained after the test muscle itself had been infiltrated by xylocaine. Obviously the experimental conditions between the two cases cannot be identical since Fig. 4b was recorded when the subject was relaxed and Fig. 4e when the subject was performing a reinforcement manoeuvre.

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References


Notice

The Fourth International Symposium on Wilson's Disease will be held in Buenos Aires, Argentina from 13-15 November 1978. The deadline for submission of abstracts is 15 September. Special typewriting forms for papers and registration forms, and any other information can be obtained from the General Secretariat, Congressos Nacionales SA, Reconquista 533, 6th Floor, Buenos Aires, 1003 Capital Federal, Argentina.