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THE AFFERENT VOLLEYS RESPONSIBLE FOR SPINAL PROPRIOCEPTIVE REFLEXES IN MAN

BY DAVID BURKE, SIMON C. GANDEVIA AND BRIAN McKEON

From the Unit of Clinical Neurophysiology, Department of Neurology, The Prince Henry Hospital and School of Medicine, University of New South Wales, Sydney 2036, Australia

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SUMMARY

1. To define the neural volleys responsible for the Achilles tendon jerk and the H reflex, muscle afferent activity was recorded using micro-electrodes inserted percutaneously into appropriate fascicles of the tibial nerve in the popliteal fossa.

2. The response of soleus muscle afferents to tendon percussion consisted of a dispersed volley, starting 3·5–7·0 ms after percussion, increasing to a peak over 6·5–11·0 ms, and lasting 25–30 ms, depending on the strength of percussion. Electrical stimuli to the sciatic nerve at a level adequate to evoke an H reflex but subthreshold for the M wave produced a more synchronized volley, the fastest fibres of which had conduction velocities of 62–67 m/s, and the slowest 36–45 m/s.

3. The wave of acceleration produced by percussion subthreshold for the ankle jerk spread along the skin at over 150 m/s. Midway between the bellies of the gastrocnemii it consisted of a damped oscillation with four to five separate phases and maximum amplitude approximately one-twentieth of that recorded on the Achilles tendon.

4. With ten primary spindle endings, tendon percussion subthreshold for the ankle jerk elicited two to five spike discharges per tap, the shortest interspike intervals being 4–7 ms. Tendon percussion elicited single discharges from two Golgi tendon organs, and altered the discharge pattern of a single secondary spindle ending. The degree of dispersion of the multi-unit muscle afferent volley can be explained by the pattern of discharge of primary spindle endings.

5. Percussion on the Achilles tendon evoked crisp afferent volleys in recordings from nerve fascicles innervating flexor hallucis longus, tibialis posterior, the intrinsic muscles of the foot and the skin of the foot. Electrical stimuli delivered to the tibial nerve in the popliteal fossa at a level sufficient for the H reflex of soleus produced either a volley in muscle afferents from the intrinsic muscles of the foot or a volley in cutaneous afferents from the foot.

6. For comparable stimuli in the two positions, the H reflex was inhibited but the Achilles tendon jerk enhanced when the ankle was dorsiflexed from 105° to 90°.

7. The duration of the rise times of the excitatory post-synaptic potentials (e.p.s.p.s) produced in soleus motoneurones by electrical stimulation, and by tendon percussion subthreshold for the H reflex and the ankle jerk respectively, was estimated from post-stimulus time histograms of the discharge of voluntarily

activated single motor units in soleus. The mean e.p.s.p. rise times were 1·9 ms for electrical stimulation and 6·6 ms for tendon percussion. There was evidence that the duration of the electrically evoked e.p.s.p. was curtailed by an inhibitory post-synaptic potential (i.p.s.p.) of only slightly longer latency than the e.p.s.p.

8. The mechanically induced and electrically induced afferent volleys are not homogeneous volleys in group Ia afferents from triceps surae. The afferent volleys differ in so many respects that it is probably invalid to compare the H reflex and tendon jerk as a measure of fusimotor activity. It is suggested that neither reflex can be considered a purely monosynaptic reflex.

INTRODUCTION

It is a traditional teaching that the tendon jerk and its electrically evoked equivalent, the 'H reflex', are monosynaptic spinal reflexes, which differ only in that the H reflex bypasses receptor mechanisms, being dependent on a group Ia volley set up by direct electrical stimulation of the motor nerve (Henneman, 1980). Comparisons of mechanically and electrically induced reflexes have been used in man to investigate receptor sensitivity and thereby the level of fusimotor activity (for example, Paillard, 1955, 1959; Buller, 1957; Buller & Dornhorst, 1957; Weaver, Landau & Higgins, 1963; Bishop, Machover, Johnston & Anderson, 1968; Dietrichson, 1971; Iles, 1977). However, the afferent volleys responsible for the mechanically and electrically induced reflexes also differ in their degree of dispersion (Gassel & Diamantopoulos, 1966), although this may not be sufficient to invalidate a comparison of the resulting reflex responses as a measure of fusimotor activity.

Proprioceptive reflexes can also be elicited in man by abrupt stretch of a contracting muscle or by muscle vibration. The former produces a complex response which contains reflex activity of spinal origin, reflex activity of long latency, possibly of long-loop origin, reaction time processes, and volitional activity. Muscle vibration produces a slowly incrementing tonic reflex contraction with inhibition of the tendon jerk and H reflex of the vibrated muscles. The afferent activity underlying these complex motor phenomena has been analysed in detail (Burke, Hagbarth, Löfstedt & Wallin, 1976*a*, 1976*b*; Hagbarth, Hägglund, Wallin & Young, 1981; Roll, 1981; Eklund, Hagbarth, Hägglund & Wallin, 1982). Paradoxically, there has been no systematic study in man of the afferent volleys responsible for such elementary proprioceptive reflexes as the tendon jerk and the H reflex. The present study was undertaken as part of a reassessment of the mechanisms underlying what have traditionally been considered simple monosynaptic spinal reflexes. It has become apparent that these reflexes are not as simple as has been assumed and that they are probably not exclusively monosynaptic.

METHODS

Experiments were performed on ten healthy adult volunteers, all of whom gave informed consent to the procedures. Each subject was studied on two to five occasions. Direct recordings of muscle afferent activity using micro-electrodes were obtained from each subject at least once. The subjects lay prone on a comfortable bed with the limb being tested supported so that the knee was in 20–30° flexion and the ankle at 105° (plantar flexed 15°).

Neural recordings. Direct recordings of muscle afferent activity (and, on six occasions, cutaneous afferent activity) were made using micro-electrodes inserted percutaneously into appropriate fascicles of the tibial nerve in the popliteal fossa (Vallbo, Hagbarth, Torebjörk & Wallin, 1979; Burke, 1981). The nerve fascicles were identified as innervating only muscle by (i) twitch contractions of the innervated muscles in response to weak stimuli delivered through the micro-electrode; (ii) absence of cutaneous paraesthesiae with such stimuli; (iii) multi-unit neural discharges in response to percussion on the muscle belly or tendon, to muscle stretch and to isometric voluntary contractions; (iv) absence of neural activity in response to light tactile stimuli; (v) the presence of sympathetic efferent activity occurring in pulse-synchronous bursts within the same fascicle (see Vallbo *et al.* 1979). The particular muscle innervated was identified by the same stimuli. Cutaneous fascicles were identified using comparable tests. The extent of the cutaneous innervation zone was mapped for each fascicle using weak tactile stimuli. Sympathetic efferent activity was recorded in each fascicle and was characteristic of that seen in fascicles innervating skin (Vallbo *et al.* 1979).

Much of the experimental data was obtained from multi-unit recordings, the micro-electrode being positioned in the fascicle so that the recorded neural activity was dominated by dynamically responding mechanoreceptors. Recordings were also made from thirteen single afferent fibres. The receptors were classified as muscle spindle endings (eleven endings; see Figs. 4 and 5) or Golgi tendon organs (two endings) by the typical responses to strong twitch contractions of the receptor-bearing muscle, produced by electrical stimuli delivered through the recording microelectrode (see McKeon & Burke, 1980). The spindle afferents were further classified as probably of primary ending origin (ten afferents) and probably of secondary ending origin (one afferent) by the presence or absence, respectively, of a dynamic response to muscle stretch and a pause in discharge on muscle shortening (Vallbo *et al.* 1979; Burke, 1981). The neural activity was amplified (gain 20000), filtered (300 Hz–3 kHz), and stored on tape for subsequent analysis.

Experimental procedures. The Achilles tendon was percussed using a tendon hammer which closed a circuit on skin contact, so providing a trigger pulse, or a light-weight vibrator (Ling Altec type 201). The vibrator was driven by a rectangular pulse of 1 ms duration. The rate of percussion was usually about 1 Hz, occasionally 3–4 Hz (see below). Percussion with the vibrator proved to be relatively weak and did not evoke a tendon jerk in all subjects, but it was reproducible in any given series, and was therefore used in studies of the rise time of the composite excitatory post-synaptic potential (e.p.s.p.) in soleus motoneurones (Fig. 9*B*). Manual percussion using the tendon hammer was not perfectly reproducible. However, the site of percussion on the Achilles tendon was marked, and the experimenters could deliver consistent tendon taps when provided with visual feed-back of the intensity of percussion (from an accelerometer on the tendon, see below). In any one series using the tendon hammer, there may have been some variability of percussion intensity but, because skin contact provided the trigger pulse, there was little if any variability in timing. Hence, variability in manual percussion would not have contributed to the marked temporal dispersion of the percussion-evoked afferent volley. The reproducibility of percussion was checked using an accelerometer (BBN Instruments type 501) strapped to the Achilles tendon 2 cm proximal to the site of percussion. The propagation and damping of the percussion wave as it travelled through the limb were measured using an identical accelerometer secured to the limb at various levels, most commonly over the belly of medial gastrocnemius. The accelerometers measured the component of the acceleration wave at 90° to the skin surface.

The H reflex was elicited by electrical stimuli of 1 ms duration delivered to the tibial nerve in the popliteal fossa. The stimulating and recording conditions were as recommended by Hugon, Delwaide, Pierrot-Deseilligny & Desmedt (1973). The anode was a dispersive lead plate secured to the skin on the anterior aspect of the thigh immediately above the patella. The cathode was a gauze-covered Ag/AgCl electrode of approximately 1 cm diameter. The precise position of this electrode in the popliteal fossa was adjusted so that weak stimuli produced a painless twitch of triceps surae without radiating cutaneous paraesthesiae. In four experiments, the H reflex was also elicited by stimuli delivered to the sciatic nerve in the thigh, immediately below the buttock crease, while recordings were made at popliteal fossa level using a micro-electrode in the nerve fascicle innervating soleus. For these experiments electrical stimuli of 0·2–0·5 ms duration were delivered through needle electrodes insulated to within 3 mm of the tip, the positions of which were adjusted so that weak stimuli produced a twitch of soleus.

Using standard nerve conduction techniques, the conduction velocities between the ankle and popliteal fossa were determined for the mixed nerve action potential of the posterior tibial nerve,

the sensory action potential of the digital nerves of the hallux, and the sensory action potential of the sural nerve. The compound nerve action potentials were recorded using a pair of Ag/AgCl surface electrodes with a fixed spacing of 40 mm. Similar electrodes were used for stimulation, except with the digital nerves of the hallux for which ring electrodes were used. The conduction velocity of the fastest motor axons innervating abductor hallucis was determined for the same segment using surface electrodes over the muscle belly and tendon. The amplifier band width was 1·6 Hz–1·6 kHz.

The potentials of single motor units in soleus were recorded in studies of e.p.s.p. duration using standard concentric needle electrodes, the amplifier band width then being 32 Hz–8 kHz. In these studies, the subjects maintained a voluntary contraction of sufficient force to sustain a steady motor unit discharge; they were given auditory and visual feed-back of the motor unit potential. No particular discharge rate was required, but the contractions were weak (up to 5% maximum voluntary power), so that the motor units studied were of low threshold. While a steady motor unit discharge was maintained, the tibial nerve was stimulated in the popliteal fossa as for H reflex studies (see above) but using a level of stimulus just subthreshold for the H reflex. In a second sequence with the same motor unit, the Achilles tendon was percussed using the vibrator, the strength of percussion being subthreshold for the ankle jerk. The driving pulses for the vibrator and for the electrical stimulator occurred at a mean rate of 3–4 Hz, the precise timing being randomized to avoid a spurious interaction between the motor unit discharge rate and the stimulus rate.

Analysis. Multi-unit neural activity was full-wave rectified and averaged on a microprocessor against the trigger pulse from the tendon hammer, the driving pulse to the vibrator or the driving pulse to the electrical stimulator, as appropriate. Between 16 and 128 responses were averaged in different experiments. The computer sampling rate was 5 kHz. Latencies were measured using a cursor. The response of single afferents to tendon percussion was assessed directly from oscilloscope sweeps triggered by the percussion-induced contact pulse, with both the afferent potential (or a Z-modulated transformation – see Figs. 4 and 5) and the accelerometer wave delayed in real time by 20–25 ms. The effects of weak tendon percussion and of weak electrical stimulation of the tibial nerve on the discharge patterns of voluntarily activated single motor units were assessed from post-stimulus time histograms (p.s.t.h.s) constructed using the microprocessor. For this purpose the motor unit potentials were converted into standard pulses using an amplitude discriminator. The time of occurrence of each pulse was logged to the nearest 256 μs. The latencies of the increased probability of discharge of the motor units following these stimuli were corrected for the difference between the onset of the motor unit potential and the trigger site, since this could be as long as 5 ms (see legend to Fig. 9).

Interpretation of p.s.t.h.s. The increased probability of discharge in the p.s.t.h.s has been taken to represent the time differential of the rising phase of the composite e.p.s.p.s, in line with Noguchi, Homma & Nakajima (1979). This interpretation may appear to be at variance with the results of Kirkwood & Sears (1982), who have demonstrated that their p.s.t.h.s reflect both the time differential of the rising phase and the duration of the e.p.s.p. In their situation the e.p.s.p. produced by a single afferent (100–200 μV) was small relative to background synaptic noise (several millivolts). In the present study, the responses recorded were to a population of afferents, the stimulus (tendon percussion or electrical shock) being just below threshold for a reflex response. Hence it is likely that the composite e.p.s.p. was large relative to the background synaptic noise, such that motoneurone discharge would be confined to the rising phase of the e.p.s.p. These conditions probably correspond more closely to those of Fetz & Gustafsson (1980) than to those of Kirkwood & Sears (1982).

RESULTS

Dispersion of the afferent volley from triceps surae. In the ten subjects, the muscle afferent response to tendon percussion was recorded from fascicles of the tibial nerve innervating soleus (nine recordings), medial gastrocnemius (three recordings) and lateral gastrocnemius/soleus (two recordings). The afferent volley lasted 25–30 ms, merging with the reflex efferent volley when present (Figs. 1*A*, 2*B*). The latency from the onset of percussion as detected by an accelerometer on the Achilles tendon to the

increase in afferent activity was 3·5–6·9 ms and the rise time to peak of the afferent volley was 6·5–11 ms, both dependent on the strength of percussion (Fig. 1*A*, *B*). When percussion was below threshold for the tendon jerk, the neural volley was similarly dispersed (Fig. 1*B*).

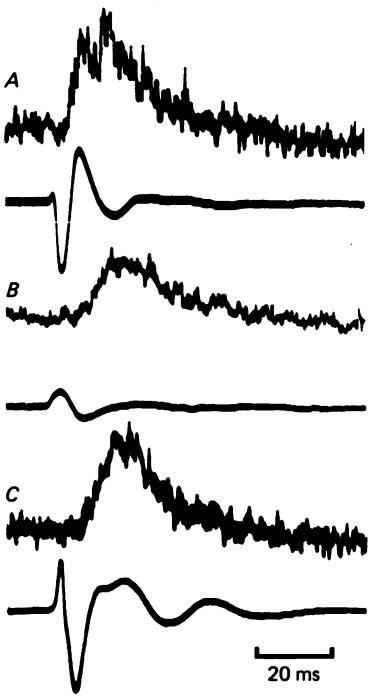

Fig. 1. Muscle afferent response to percussion on the Achilles tendon. *A* and *B*, response of afferents from soleus, percussion being adequate (*A*) and subthreshold (*B*) for the Achilles tendon jerk. *C*, response of afferents from abductor hallucis in the same subject. Upper traces: the averaged full-wave rectified muscle afferent response (using sixteen, sixty-four and thirty-two sweeps in *A*, *B* and *C*, respectively); lower traces: output of an accelerometer on the Achilles tendon. The three neural and the three accelerometer traces are each at equivalent gains; the traces are aligned at the first deflexion of the appropriate acceleration wave.

In four of the ten subjects, neural volleys were also recorded when the tibial component of the sciatic nerve was stimulated immediately below the buttock (Fig. 2*A*). The fastest fibres in the neural volley had a peak conduction velocity of 62–67 m/s, and there was little dispersion (up to 4 ms dependent on stimulus strength). If the stimulus produced no direct motor response (M wave), being therefore subthreshold for motor axons (as in Fig. 2*A*), the neural volley was an antidromic volley in afferent fibres, the degree of dispersion attributable to the range of conduction velocities for low-threshold muscle afferents (largely group I muscle afferents). Thus the slowest fibres in the antidromic afferent volley had conduction velocities of 36–45 m/s. These last figures underestimate the true values slightly because they are based on the cessation of the afferent volley and, hence, the end rather than the onset of the slowest constituent potentials.

The percussion-induced stimulus. Using percussion subthreshold for the ankle jerk,

the spread of the percussion wave to triceps surae was recorded in four subjects using two identical accelerometers firmly secured to the skin, one (the 'control' accelerometer) on the Achilles tendon 2 cm proximal to the percussion site, the second (the 'exploring' accelerometer) at standard distances along the leg. In non-contracting muscles the transmission of the disturbance along the skin was rapid, exceeding 150 m/s, the precise value dependent on the degree of muscle stretch (Fig. 3*A*). The amplitude of the acceleration wave decreased as distance from the percussion site

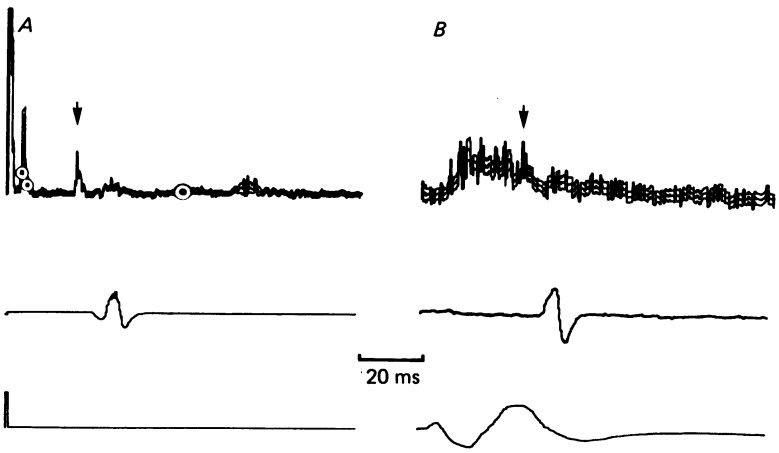

Fig. 2. Comparison of the muscle afferent volleys evoked electrically (*A*) and mechanically (*B*) in the same subject. In *A* and *B*, the top trace is the averaged rectified muscle afferent volley from soleus (with the 95% confidence limits of the average); the middle trace is averaged e.m.g. of triceps surae, showing the H reflex response in *A* and the tendon jerk response in *B*; and the third trace represents the stimulus (electrical pulse in *A*; accelerometer output in *B*). Each trace is the average of sixteen responses. The neural trace in *A* contains a large stimulus artifact, followed by a synchronized low-threshold muscle afferent volley, and a small reflex efferent volley (indicated by the vertical arrow). In *B*, the afferent neural response is highly dispersed and merges with the reflex efferent volley (indicated by the vertical arrow).

increased (Fig. 3*C*). When the exploring accelerometer was positioned between the bellies of the gastrocnemii, there was a 20-fold difference in the amplitudes of the disturbances recorded by the exploring and control accelerometers. At the popliteal fossa there was an increase in the amplitude of the recorded displacement, presumably due to closer proximity to bone. The disturbance reaching the hamstrings muscles was only slightly less than that reaching the bellies of the gastrocnemii. In addition, in all four subjects, the acceleration wave became dispersed and more complex as the site of recording moved proximally along the calf (Fig. 3*B*), presumably reflecting an interaction between the disturbance set up in muscle and skin, that transmitted through bone and that reflected from the proximal insertion onto bone. The frequency of the resulting mechanical oscillation was 25–45 Hz, dependent on subject, and its magnitude varied with muscle stretch and contraction. A detailed study of these changes was not made. The transmission through bone was estimated by securing the exploring accelerometer to skin overlying the tibia. The percussion wave was

again significantly dampened at proximal sites, but the degree of attenuation along bone was much less than with transmission over the calf muscles.

The amplitude of the disturbance recorded over tibialis anterior was approximately 50 % of that recorded from equivalent positions on triceps surae. Over the motor point of abductor hallucis it was of similar amplitude to that recorded between the bellies of the gastrocnemii.

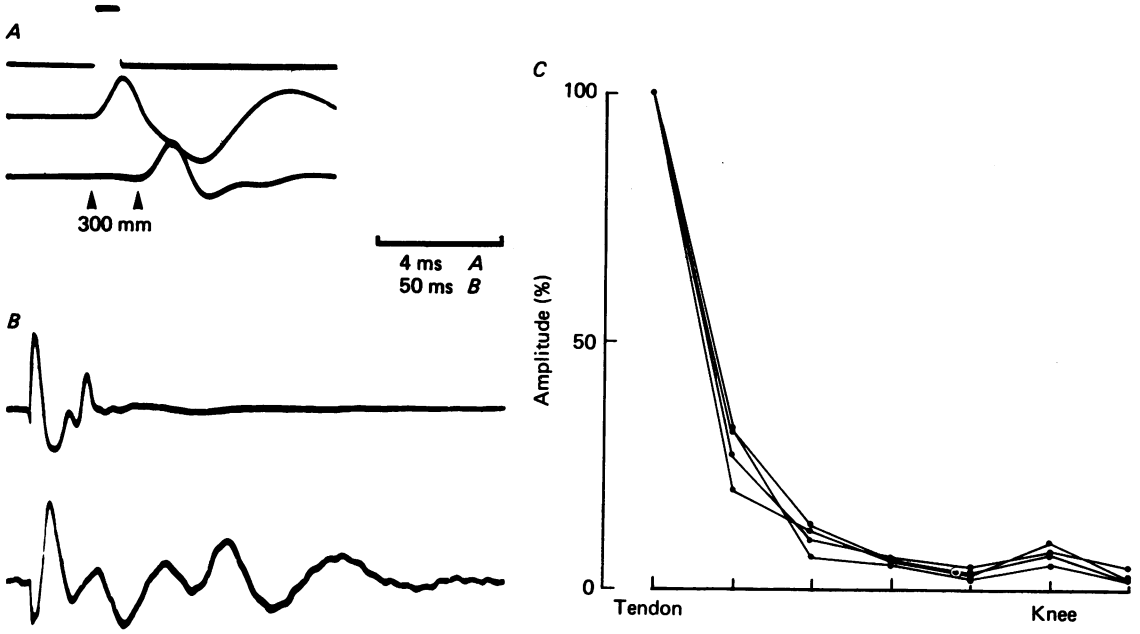

Fig. 3. Spread of the percussion wave. *A*, the outputs of the control accelerometer on the Achilles tendon (second trace) and of the exploring accelerometer secured to the belly of lateral gastrocnemius 300 mm more proximally (third trace) in response to percussion with the vibrator on the Achilles tendon (drive pulse, first trace). Five sweeps superimposed. *B*, outputs of control accelerometer on Achilles tendon (upper trace) and exploring accelerometer at the mid-calf level (lower trace) in response to percussion on the Achilles tendon using a tendon hammer. In *A* and *B*, the outputs of the exploring accelerometers were increased to sizes comparable to those of the control accelerometers. *C*, the relative amplitude of the percussion wave at standard positions between tendon (far left) and hamstrings muscles (far right) for four subjects.

It may be concluded that asynchronous activation of mechanoreceptors contributes little to the total dispersion of the percussion-evoked afferent volley (see Figs. 1 and 2*B*), but that the stimulus reaching different receptors will still vary, dependent on receptor location. The complexity of the degraded stimulus could result in the activation of some endings many times (see Fig. 4), so prolonging the duration of the evoked afferent volley. Comparable conclusions have been reported for contracting human forearm muscles by Eklund *et al.* (1982), although a much lower propagation velocity (40 m/s) was found.

The responses of single mechanoreceptors in the calf muscles. Single unit recordings were made from ten muscle spindle afferents which responded dynamically to muscle stretch and were therefore presumed to be of primary ending origin. Six endings were

in triceps surae, three in tibialis posterior, and one in flexor hallucis longus. All were capable of at least two discharges in response to weak percussion on the Achilles tendon, subthreshold for the ankle jerk, although one ending did so only occasionally. With weak percussion, five endings regularly discharged three times, one four times and one five times (Fig. 4). Repetitive activation of some endings is to be expected given the complexity of the degraded percussion wave reaching the bellies of the

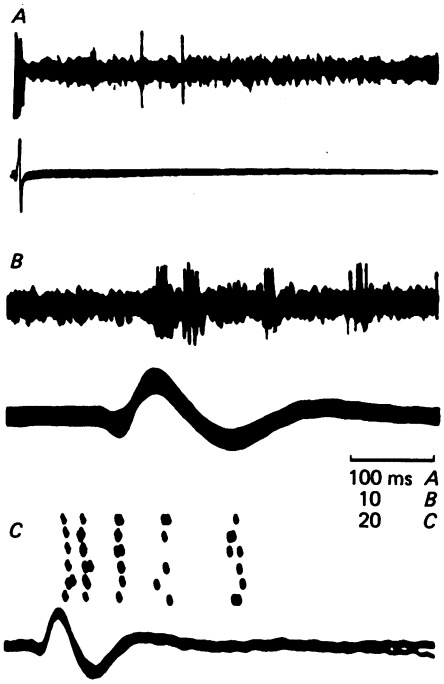

Fig. 4. The response of a presumed primary spindle ending in medial gastrocnemius to weak tendon percussion. *A*, electrically induced twitch test, the afferent discharging twice, at 170 ms and 220 ms, latencies appropriate to the falling phase of twitch force (not recorded). Upper trace: raw neural activity; lower trace: e.m.g. of triceps surae. *B*, the response to tendon percussion subthreshold for Achilles tendon jerk. Upper trace: raw neural activity; lower trace: output of accelerometer on Achilles tendon. Multiple sweeps superimposed. The fifth discharge (see *C*) is not seen on this time base. *C*, Z-modulated raster of the responses to individual tendon taps with up to five spike discharges in response to any one tap. Lower trace: superimposed accelerometer tracings for these taps.

gastrocnemii (see Fig. 3*B*). All primary endings tested responded to percussion on the tendon of tibialis anterior (as also did the secondary ending; see Fig. 5*C*).

The responses of the four group I a afferents from tibialis posterior and flexor hallucis longus were similar to those of the six from triceps surae. Multi-unit recordings were also obtained from these fascicles. Percussion on the Achilles tendon above and below threshold for the tendon jerk evoked crisp neural afferent volleys with the same latencies and duration as seen with volleys from triceps surae.

With the ten primary endings the precise timing of spike discharge varied slightly with identical taps. This was best seen with four afferents which maintained a steady background discharge: their responses to the same percussion varied in latency and

number from trial to trial. This phenomenon was also apparent with the secondary ending (see Fig. 5).

When the tap was subthreshold for the ankle jerk the mean latencies from the onset of the mechanical disturbance to the first afferent spike ranged from 5 to 7 ms for nine endings, and was 13 ms for the tenth. With stronger percussion, the latencies shortened to 4–5 ms for some endings. The intervals between the first and the second spikes were 4–7 ms (Fig. 4*B*, *C*), and subsequent spikes occurred at intervals of 6–26 ms. Comparison of these figures and of the Z-modulated raster of Fig. 4*C* with

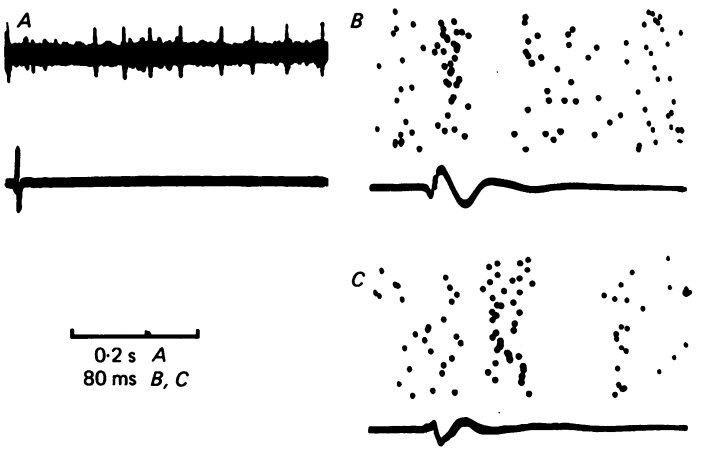

Fig. 5. Response of a presumed secondary spindle ending in lateral gastrocnemius to weak tendon percussion. *A*, twitch test, showing a pause in afferent discharge lasting 145 ms before resumption of a steady stable discharge. Upper trace: raw neural activity; lower trace: e.m.g. of triceps surae. *B* and *C*, Z-modulated raster displays of the response to percussion on the Achilles tendon (*B*) and the tendon of tibialis anterior (*C*), much as in Fig. 4*C* but using a slower time base.

the multi-unit afferent responses of Figs. 1 and 2*B* suggests that the patterns of discharge of individual Ia afferents can probably explain the time-course of the multi-unit afferent volley. However, mechanoreceptors other than the primary spindle ending are also activated by percussion and their discharge will contaminate the afferent volley.

The discharge of the secondary ending (in lateral gastrocnemius) was modulated by percussion but was not tightly locked to it. Percussion elicited a spike discharge only if the ending had not recently discharged (Fig. 5*B*), a phenomenon which was sometimes seen with actively discharging primary endings. An 'extra' spike was recorded only in response to some taps, and this could occur anywhere within 20 ms of percussion. The two Golgi tendon organs (both in triceps surae) had no background activity and discharged single impulses in response to percussion on the Achilles tendon at a level below threshold for the ankle jerk. However, the recordings were not sufficiently stable to allow detailed analysis of their responses.

Muscle afferent activity from the foot. Recordings were obtained at popliteal fossa level from three different tibial nerve fascicles innervating the intrinsic muscles of the foot (predominantly abductor hallucis in two recordings, predominantly abductor

digiti quinti in the third recording). With each recording, percussion on the Achilles tendon at an intensity sufficient for an ankle jerk produced a strong burst of multi-unit neural activity (Fig. 6*A*). The time course of this activity in one subject is seen in the averaged traces of Fig. 1*C*, where it can be compared with the multi-unit afferent responses recorded later in the same experiment from a fascicle innervating soleus. At popliteal fossa level, the onset of neural activity from abductor hallucis had a latency of 8·2 ms (4·1 ms for soleus) and a rise time of 9·0 ms (6·5 ms for soleus).

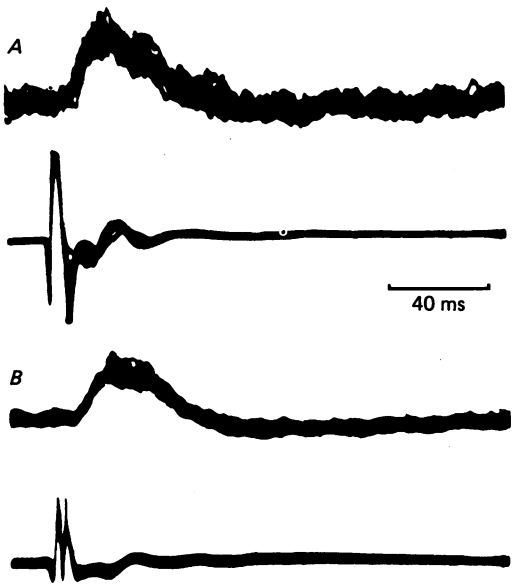

Fig. 6. Responses of muscle mechanoreceptors in abductor hallucis (*A*) and of cutaneous mechanoreceptors in the skin of the hallux and medial aspect of the sole (*B*) to weak percussion on the Achilles tendon. Percussion was above threshold for the tendon jerk. The limb was supported to avoid direct muscle stretch and skin contact. In *A* and *B*, the upper traces are neural activity (rectified and smoothed, time constant 0·01 s); the lower traces, accelerometer outputs. Ten responses superimposed.

The electrically induced afferent volley responsible for the H reflex was also found to be contaminated by afferent activity from the small muscles of the foot. In five subjects, electrical stimuli were delivered in the popliteal fossa at a level adequate for the H reflex but subthreshold for the M wave. The position of the stimulating cathode was adjusted in an attempt to eliminate radiating cutaneous paraesthesiae. In one subject, a stimulus adequate for the reflex could not be given without producing cutaneous paraesthesiae radiating to the hallux. In the remaining four, appropriate stimuli set up a rapidly conducting neural volley in the posterior tibial nerve at the ankle. This volley was, in each subject, an antidromic volley in muscle afferents from the intrinsic muscles of the foot (Fig. 7). Its conduction time between popliteal fossa and ankle was equal to that of the fastest axons in the posterior tibial nerve (compare Fig. 7*A* and 7*B*) – shorter than the conduction time for cutaneous afferents (Fig. 7*C*) or for efferent axons (Fig. 7*D*).

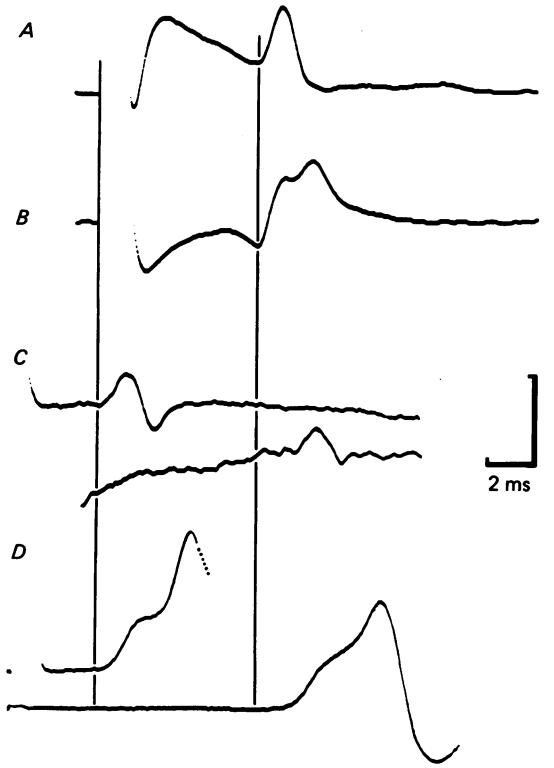

Fig. 7. Activation of muscle afferents from the small muscles of the foot by electrical stimuli to the tibial nerve in the popliteal fossa at a level adequate for an H reflex in triceps surae but subthreshold for the M wave. *A*, neural volley recorded by surface electrodes over the posterior tibial nerve at the ankle in response to the above stimuli; *B*, mixed nerve action potential recorded at popliteal fossa on stimulation of the posterior tibial nerve at the ankle; *C*, cutaneous sensory action potential recorded at the ankle (upper trace) and at the popliteal fossa (lower trace) on stimulation of the digital nerves of the hallux; *D*, compound muscle action potential of abductor hallucis following supramaximal stimulation of motor fibres at the ankle (upper trace) and at the popliteal fossa (lower trace). The vertical lines indicate the conduction time over the ankle-popliteal fossa segment for the volley in *A*. In *B*, *C* and *D*, the traces are aligned at the first vertical line to facilitate comparison of the conduction times for this segment. The volley in *A* has the same conduction time as the fastest fibres in the mixed peripheral nerve. It is faster than both cutaneous afferents and motor fibres, and is therefore presumably an antidromic volley in group I muscle afferents. The second peak in the mixed nerve action potential in *B* has the same conduction time as the cutaneous afferents in *C*. In *A–D*, stimuli were of 1 ms duration. Vertical calibration: *A*, 5 μV; *B*, 10 μV; *C*, 5 μV (upper trace) and 2·5 μV (lower trace); *D*, 10 mV.

Cutaneous afferent activity. Afferent volleys evoked by percussion on the Achilles tendon were recorded from six purely cutaneous fascicles of the tibial nerve in the popliteal fossa: two innervating the heel (calcaneal branch of the posterior tibial nerve), three the hallux and medial aspect of the sole (medial plantar nerve) and one the lateral aspect of the sole extending to toe 5 (lateral plantar nerve). In each recording, cutaneous mechanoreceptors responded to percussion when the foot was secured such that the cutaneous innervation zones were not directly stimulated by

contact or pressure (Fig. 6*B*). At the popliteal fossa the cutaneous afferent volleys so evoked had latencies of onset of 10–15 ms and to peak of 25–30 ms. Cutaneous mechanoreceptors will also be activated at the percussion site and in the foot, leg and thigh at fixation and other contact points.

To determine the difference in arrival time of simultaneously initiated muscle afferent and cutaneous volleys, the peripheral nerve conduction velocities for the posterior tibial mixed nerve potential (muscle afferent, see Fig. 7) and the sural sensory potential (cutaneous) were determined in eight subjects for the ankle-to-popliteal fossa segment. The velocities were, for the muscle afferents, mean 56·1 m/s (range 50·6–63·6 m/s) and, for the cutaneous afferents, mean 48·2 m/s

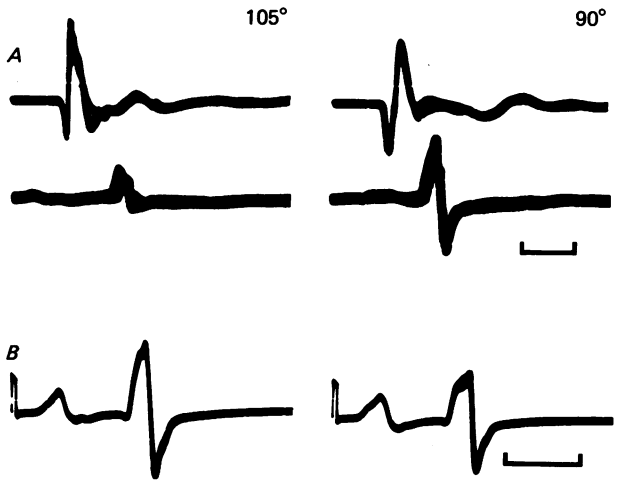

Fig. 8. The effect of dorsiflexion of the ankle from 105° to 90° on the sizes of the Achilles tendon jerk (*A*) and the H reflex (*B*). *A*, the strength of percussion was adjusted to keep the peak-to-peak output of an accelerometer on the Achilles tendon constant (upper trace). Reflex e.m.g. of triceps surae is greater in the more dorsiflexed position (lower traces). *B*, e.m.g. of triceps surae showing stimulus artifact, a small M wave (which remains constant), and the H reflex response (which is smaller in the more dorsiflexed position). Five sweeps superimposed for each trace. Horizontal calibrations: both 20 ms.

(range 45·2–49·1 m/s). Given that the group Ia conduction time from popliteal fossa to cord is approximately 15 ms in man, a cutaneous volley departing the popliteal fossa at the same time would arrive approximately 2·5 ms later (assuming that the two afferent volleys maintain the same relative conduction velocities in the popliteal fossa-to-cord segment as in the ankle-to-popliteal fossa segment).

The effects of ankle rotation. Passive dorsiflexion of the ankle does not alter the electrically induced afferent volley provided that the movement does not disturb the stimulating electrodes. An unaltered direct motor response (M wave) confirms that the conditions of stimulation have not changed and that any change in the reflex response is not due to a different afferent volley. In three subjects, passive dorsiflexion of the ankle from 105° or 100° to 90° was sufficient to inhibit the H reflex (Fig. 8*B*), as has been documented more fully by other authors (Mark, Coquery & Paillard, 1968; Herman, 1969; Delwaide, 1971). This change in joint position had the opposite effect on the ankle jerk (Fig. 8*A*). In these studies, tendon percussion was adjusted so that in the two positions it produced similar outputs from an accelerometer on the Achilles

tendon, thus ensuring that the intensity of percussion remained constant. The output of a second accelerometer on medial gastrocnemius indicated that the spread of the percussion wave to the calf had been altered by stretching triceps surae, thus suggesting that spindle endings in triceps surae probably 'saw' a different stimulus in the two positions. In the more dorsiflexed position, a stronger ankle jerk was recorded. A wider range of joint rotation could not be investigated reliably because larger movements altered the amplitude and/or shape of the M wave.

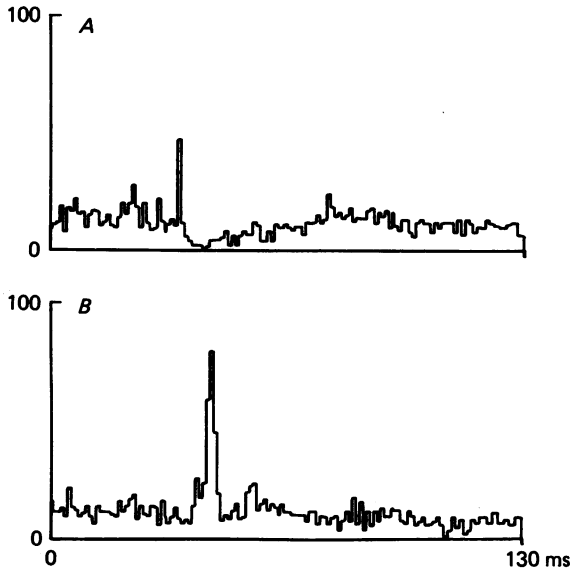

Fig. 9. P.s.t.h.s of the discharge of a voluntarily activated motor unit in soleus in response to weak electrical stimuli to the tibial nerve in the popliteal fossa (*A*) and weak percussion on the Achilles tendon (*B*). The increased probability of discharge in *A* occurs 36 ms after the stimulus and lasts 1 ms. The increased probability in *B* starts 42 ms after percussion and lasts 7 ms. With this motor unit, the discriminator's trigger level was set such that the pulse fed into the computer occurred 5 ms after the actual onset of the e.m.g. potential. Reflex latencies are therefore 31 ms (*A*) and 37 ms (*B*). Bin width 1 ms. The vertical axes are calibrated in absolute counts.

E.p.s.p.s in soleus motoneurones. The rise-times of the e.p.s.p.s produced in soleus motoneurones by electrical stimulation subthreshold for the H reflex and by tendon percussion subthreshold for the ankle jerk were estimated from post-stimulus time histograms (p.s.t.h.s) of the discharge of nine voluntarily activated single motor units in soleus. With seven motoneurones, tendon percussion produced an increased probability of discharge at a mean latency of 36·4 ms (range 34·0–40·0 ms). With eight motoneurones, electrical stimulation produced an increased probability of discharge at 30·6 ms (range 29·1–33·5 ms), the difference with the two stimuli (5·8 ms) being consistent with that required for receptor activation and afferent conduction to the popliteal fossa (see Fig. 1). For the motoneurone of Fig. 9, the latencies were 37 ms and 31 ms, respectively. The duration of the increased probability of discharge reflects the duration of the rise time of the responsible composite e.p.s.p. (see

Methods). As measured from the p.s.t.h.s, the rise times of the e.p.s.p.s would therefore be, for mechanical stimulation, mean 6·6 ms (range 4·0–10·0 ms) and for electrical stimulation, mean 1·9 ms (range 1·0–3·0 ms). With each of six motoneurones for which the effects of both types of stimulation could be determined, the rise time of the mechanically induced e.p.s.p. was longer than that of the electrically induced e.p.s.p., the mean difference in duration being 4·7 ms (range 2·0–8·0 ms). For the motoneurone of Fig. 9, the rise time of the electrically induced e.p.s.p. was 1·0 ms, and the rise time of the mechanically induced e.p.s.p. was 7·0 ms.

The p.s.t.h. in Fig. 9*A* contains a decrease in the probability of motoneurone discharge occurring immediately after the brief increase at 31 ms. The extent of the decrease is too great to be attributable to refractoriness following the discharge at 31 ms, and presumably results from a combination of active inhibition due to the electrically evoked group I volley and after-hyperpolarization. Such a decrease was not a feature of the p.s.t.h.s to mechanical stimulation (Fig. 9*B*). In human subjects, Pierrot-Deseilligny, Morin, Bergego & Tankov (1981*b*) have shown that electrical stimuli of low intensity activate group Ia and Ib afferents in parallel, and that detectable Ib inhibitory effects occur as early as 1 ms after the onset of Ia excitatory effects. The p.s.t.h. of Fig. 9*A* can therefore be interpreted as the response to a mixed Ia and Ib afferent volley, with a short latency Ia e.p.s.p., the true duration of which is concealed by the development 1 ms later of an Ib inhibitory post-synatpic potential (i.p.s.p.). This interpretation assumes the traditional autogenetic Ia and Ib reflex effects, neglecting recently demonstrated complexities, such as autogenetic Ia inhibition (Fetz, Jankowska, Johannisson & Lipski, 1979). However, if autogenetic Ia inhibition were responsible for the decreased probability of discharge in Fig. 9*A*, it could also be expected with mechanical stimulation (see Fetz *et al.* 1979).

DISCUSSION

Neither the afferent volley responsible for the ankle jerk nor that for the H reflex of soleus is a homogeneous volley in group Ia afferents from primary spindle endings in triceps surae. Both volleys are contaminated by activity in a wide variety of afferents, not only from other mechanoreceptors in triceps surae but also from mechanoreceptors in skin and in other muscles. However, the afferent populations activated by the two stimuli differ, and so too does the pattern of activity in each afferent. The differences in the two afferent volleys are so marked that it is surprising that the resulting reflexes so often behave in parallel.

The conclusion that the two afferent volleys are different is not unexpected given that, in man, the responsible stimuli are not selective. As emphasized by Lance & de Gail (1965), the effective stimulus for the mechanoreceptors responsible for the tendon jerk is not so much stretch but the resulting vibration wave which propagates through bone and muscle stimulating receptors whether the muscle in which they are located is stretched or not. An over-all increase in muscle length need not occur: in hyper-reflexic patients a reflex contraction can often be elicited by percussion which shortens the responding muscle. It is inevitable that the jar produced by percussion will excite sensitive mechanoreceptors in skin and muscle throughout the limb unless widespread denervation can be performed and the femur broken – procedures which have been recommended in animal studies using comparable mechanical stimuli (Morelli, Nicotra, Barnes, Cangiano, Cook & Pompeiano, 1970). Percutaneous electrical stimulation of the tibial nerve in the popliteal fossa, as is normally used in H reflex studies on human subjects, cannot be directed exclusively to the fascicles

innervating triceps surae and, even if it could be, group Ib afferents would be stimulated almost as readily as group Ia afferents.

Man is further disadvantaged by the long conduction distance to the spinal cord and by the slower conduction velocity of human group Ia afferents (maximally 120 m/s for the cat and monkey, but about 65 m/s for man; cf. present study, and also Magladery & McDougal, 1950; Diamantopoulos & Gassel, 1965). If the fastest Ia afferents in an electrically evoked volley take approximately 15 ms to reach the motoneurone pool, the slowest Ia afferents would arrive 6·7–9·4 ms later, assuming that the slowest Ia afferents have a conduction velocity of 40–45 m/s (see Fig. 2*A*). Hence even with synchronized activation of Ia afferents by an electrical stimulus in the popliteal fossa the duration of purely monosynaptic effects on the motoneurone pool could last almost as long as 10 ms. The duration of I a effects on the motoneurone pool will be much longer with tendon percussion because the Ia volley is already significantly desynchronized at popliteal fossa level.

At the motoneurone, the rise time of the composite group Ia e.p.s.p. will be determined not only by the arrival times of the individual component e.p.s.p.s but also by their decay time. It is possible therefore that impulses in the slowest Ia afferents would arrive when the wave of excitation was decaying, and so be unable to play any role in the reflex discharge. However, studies in man suggest that the electrically evoked group I e.p.s.p. in soleus motoneurones has a mean rise time of 3·6 ms (Ashby & Labelle, 1977), and the percussion-evoked group Ia e.p.s.p. in gastrocnemius motoneurones has a mean rise time of 8·2 ms (Noguchi *et al.* 1979). These studies used different muscles and slightly different techniques, but the present study has demonstrated that, in the same motoneurones, the percussion-induced e.p.s.p. is significantly longer than the electrically induced e.p.s.p. (Fig. 9; D. Burke, S. C. Gandevia & B. McKeon, unpublished observation). The long duration of the rise time of the composite e.p.s.p.s has two important consequences: first, *afferents other than Ias from triceps surae may affect the reflex discharge* (see below), and, secondly, *the monosynaptic pathway cannot be the only effective reflex pathway* (D. Burke, S. C. Gandevia & B. McKeon, unpublished observation).

Apart from the degree of dispersion of the Ia activity, some of the other differences between the two reflex afferent volleys have predictable consequences:

(i) In man, as in the cat, the electrical threshold and conduction velocity of group Ib afferents are very similar to those of Ia afferents (Pierrot-Deseilligny *et al.* 1981*b*) so that the H reflex afferent volley will be heavily contaminated by Ib afferent activity, presumably much more so that the percussion-evoked volley. In man, detectable Ib (presumably disynaptic) inhibitory effects begin in soleus motoneurones as early as 1 ms after the earliest Ia excitatory effects (Pierrot-Deseilligny *et al.* 1981*b*). Clearly, with a composite group I e.p.s.p. lasting on average 3·6 ms there is adequate time for significant Ib effects on the reflex discharge of most if not all motoneurones. Such an effect is probably present in Fig. 9*A*: the brief duration of the increased probability of motoneurone discharge is best explained by the development of an i.p.s.p. 1 ms after the e.p.s.p. so masking the true duration of the e.p.s.p.

(ii) The electrical stimulus may be but is probably not always delivered in a way that avoids stimulation of cutaneous afferents in the sural and posterior tibial nerves. However, group I afferents from the small muscles of the feet and from calf muscles other than triceps surae will be activated, and their activity would reach triceps surae motoneurones at the same time as the group I activity from triceps surae. Percussion on the Achilles tendon will activate mechanoreceptors in calf muscles other than triceps surae and in the pretibial flexors largely in parallel with those in triceps surae. Allowing for a one-millisecond delay across the Ia inhibitory interneurone reciprocal

inhibitory effects could occur in soleus motoneurones in parallel with the autogenetic and synergistic Ia excitation (cf. however Tanaka, 1980), particularly if transmission at this interneurone had been facilitated by the percussion-induced cutaneous activity (see Hultborn, 1972). The percussion-evoked afferent volley from the intrinsic muscles of the foot would lag behind the volley from the triceps surae, but the fastest afferents would still reach the motoneurone pool during the rise time of the composite e.p.s.p., and so could affect the discharge of high-threshold motoneurones.

(iii) Percussion on the Achilles tendon will produce widespread activation of cutaneous mechanoreceptors, but those in the foot cannot affect the resulting ankle jerk because of their long conduction distance to cord, slower afferent conduction velocity, and polysynaptic intraspinal path. However, cutaneous receptors will be excited at the knee and possibly more proximally, and activity in their afferents would reach the spinal cord within at most 2·5 ms of the triceps surae Ia volley. Low-threshold cutaneous afferents can affect the H reflex and tendon jerk of triceps surae in man (Hugon, 1973; Delwaide, Crenna & Fleron, 1981). These effects could be mediated directly or indirectly by, for example, the 'Ib inhibitory interneurone' (Lundberg, Malmgren & Schomburg, 1977; cf. however, Pierrot-Deseilligny, Bergego, Katz & Morin, 1981*a*).

(iv) The repetitive discharge of Ia afferents in the percussion-evoked volley clearly differs from the single impulse in the electrically evoked volley. Given the long duration of the percussion-evoked e.p.s.p. there is adequate time for second impulses in the fastest Ia afferents to influence the reflex discharge, and this they could do before the arrival of activity in slowly conducting Ia afferents.

The importance of the factors discussed above rests on the duration of the composite e.p.s.p.s in soleus motoneurones. This is not so with a fifth equally important difference between the two afferent volleys: the effect of the degree of background stretch on triceps surae. Of necessity, the mechanically induced volley is dependent on the intensity of the stimulus actually seen by each receptor and on the receptor's ability to respond. Both may be altered by muscle stretch. Within the limited range that could be studied here, dorsiflexion of the ankle has an inhibitory reflex effect on the motoneurone pool of soleus. However, with the tendon jerk, this negative effect is probably masked by the facilitatory effect that stretching triceps surae has on the transmission of the percussion wave and on the responsiveness of spindle receptors. In the spastic patient, the inhibitory reflex effect of dorsiflexion is even more pronounced than in normal man (Herman, 1969; Burke, Andrews & Ashby, 1971), presumably due to release of transmission in 'flexor reflex afferent' pathways. However, dorsiflexion still potentiates the tendon jerk, much as in normal man, so that the seemingly paradoxical behaviour of the two reflexes becomes more pronounced. Given this, it can be confidently predicted that, if the ankle of a spastic patient is sufficiently dorsiflexed that a tendon jerk can be elicited, the degree of accentuation of the tendon jerk will be greater than the degree of accentuation of the H reflex. This difference has, in the past, been interpreted as indicating excessive fusimotor activity in the spastic state (see Dietrichson, 1971). Clearly, any assessment of fusimotor drive that depends on comparing changes in the tendon jerk with changes in the H reflex must be suspect. Few if any of the differences between the reflexes discussed in this paper can be controlled let alone measured in experiments on human subjects.

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