Muscle spindle activity in the affected upper limb after a unilateral stroke

L. R. Wilson, S. C. Gandevia, J. T. Inglis, J.-M. Gracies and David Burke

Department of Clinical Neurophysiology, The Prince Henry and Prince of Wales Hospitals and Prince of Wales Medical Research Institute, University of New South Wales, Sydney, Australia

Summary

Weakness, loss of dexterity and exaggerated reflex responses to proprioceptive and cutaneous stimuli are typical features of hemiparetic stroke. Since the extent to which altered fusimotor drive contributes to these deficits has not been established, this study was designed to assess fusimotor function in stroke patients by comparing three aspects of muscle spindle afferent behaviour (background discharge rate, responses to reflex inputs and responses to voluntary contractions) in 11 subjects affected by recent cerebrovascular lesions, with those in 18 healthy volunteers. The mean background discharge rates of muscle spindle afferents in the radial nerve when subjects attempted to relax the recorded limb completely were 6.6 ± 5.3 Hz (n = 26) in patients and 6.4 ± 6.1 Hz (n = 76) in control subjects. The variability of discharge rate of active afferents was also similar (0.12 ± 0.07 and 0.09 ± 0.10, respectively). Reflex activation of fusimotor neurons was assessed using trains of electrical stimuli to the superficial radial nerve or to the palm of the hand, and using natural skin stimuli. Neither type of cutaneous stimulation affected muscle spindle afferent discharge in the absence of an EMG response. During deliberate voluntary contractions muscle spindle discharge rates were enhanced similarly in both the control and patient groups, indicating that volitional drives could access fusimotor neurons in the patients. Qualitatively, spindle behaviour was similar in patients and control subjects. These findings suggest that fusimotor function is not disturbed any more or less than skeletomotor function in hemiparetic patients and it is concluded that fusimotor dysfunction probably contributes little to their deficit.

Keywords: muscle spindle afferent; fusimotor drive; spasticity; stroke; hemiparesis

Introduction

A stroke commonly results in loss of strength, dexterity and coordination. In addition to impaired movement there are often exaggerated tendon jerks, a velocity-dependent increase in muscle tone (i.e. spasticity) (Lance, 1980) and changes in resting posture. It has been suggested that increased dynamic fusimotor drive contributes to the spasticity associated with stroke (Rushworth, 1960, 1964; Jansen, 1962; Dietrichson, 1971), although the few studies that have addressed this question have provided varying conclusions. Alternatively, it has been suggested that inability to mobilize fusimotor activity appropriately for a task might contribute to the loss of dexterity, and that disinhibition of spinal reflex pathways to fusimotor neurons might disrupt ongoing movements by allowing afferent inputs triggered by movement to activate fusimotor neurons reflexly (Burke, 1988). One study in which muscle spindle afferent activity was recorded in two subjects with hemiparetic spasticity found no evidence for increased dynamic or static responsiveness of spindle endings (Hagbarth et al., 1973, 1975b). In another study, muscle spindle afferent discharge in response to electrical- or reflex-induced muscle twitches was reported to be enhanced during contractions of remote muscle groups (the Jendrassik manoeuvre) in both the control and patient groups (Szumski et al., 1974). It was argued that this reflected activation of dynamic fusimotor neurons by the remote muscle contraction. However, for this conclusion to be valid, inadvertent EMG in the recorded muscle would need to be excluded, and there is now substantial evidence indicating that reflex reinforcement by remote muscle contraction does not occur through fusimotor mechanisms (Hagbarth et al., 1975a; Burke, 1981).

In healthy subjects the levels of background fusimotor drive to voluntarily relaxed muscles appear to be low (Vallbo et al., 1979; Burke, 1981; Gandevia and Burke, 1992), though there is limited evidence that fusimotor neurons can be selectively activated by reflex inputs in otherwise relaxed muscles (Gandevia et al., 1986, 1994). On the other hand, there is considerable evidence that fusimotor neurons can be activated by even the weakest voluntary efforts, with
Table 1  Patient details—type of lesion and physical effect

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (years)</th>
<th>Gender</th>
<th>Lesion (site)</th>
<th>Time since stroke (months)</th>
<th>Wrist strength* (% contralateral side)</th>
<th>Upper limb spasticity (Ashworth score)</th>
<th>Overt sensory loss</th>
<th>Spindle afferents (no.)</th>
<th>Multiunit sites (no.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>57</td>
<td>M</td>
<td>Right brainstem</td>
<td>4</td>
<td>0</td>
<td>2</td>
<td>Yes</td>
<td>1</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>58</td>
<td>M</td>
<td>Right brainstem</td>
<td>2, 6, 17‡</td>
<td>68</td>
<td>75</td>
<td>Yes</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>39</td>
<td>M</td>
<td>Left temporoparietal†</td>
<td>1</td>
<td>50</td>
<td>58</td>
<td>Yes</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>66</td>
<td>M</td>
<td>Right internal capsule</td>
<td>1</td>
<td>59</td>
<td>60</td>
<td>No</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>59</td>
<td>M</td>
<td>Left temporoparietal</td>
<td>3</td>
<td>0</td>
<td>0</td>
<td>No</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>6</td>
<td>75</td>
<td>F</td>
<td>Right frontoparietal</td>
<td>1, 2‡</td>
<td>82</td>
<td>94</td>
<td>Yes</td>
<td>5</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>24</td>
<td>M</td>
<td>Right internal capsule</td>
<td>4, 6, 15, 19‡</td>
<td>5</td>
<td>9</td>
<td>No</td>
<td>7</td>
<td>3</td>
</tr>
<tr>
<td>8</td>
<td>47</td>
<td>M</td>
<td>Left internal capsule†</td>
<td>4</td>
<td>22</td>
<td>44</td>
<td>Yes</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>9</td>
<td>55</td>
<td>M</td>
<td>Right internal capsule</td>
<td>2, 4‡</td>
<td>0</td>
<td>5</td>
<td>Yes</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>10</td>
<td>59</td>
<td>M</td>
<td>Left internal capsule</td>
<td>1</td>
<td>80</td>
<td>98</td>
<td>No</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>11</td>
<td>57</td>
<td>M</td>
<td>Right frontoparietal</td>
<td>1</td>
<td>0</td>
<td>0</td>
<td>Yes</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

M = male; F = female. *Values are averaged for patients studied on more than one occasion; † haemorrhagic stroke; ‡ multiple studies.

recruitment of most muscle spindle afferents in contractions of <5% maximal strength (Valbbo, 1974; Edin and Valbbo, 1990a; Wilson et al., 1997a). Even if fusimotor dysfunction did not contribute to reflex abnormalities in stroke patients at rest, such dysfunction might become manifest during voluntary tasks. So far there have been no assessments of fusimotor activity during deliberate voluntary contractions in stroke patients.

In the present study, muscle spindle afferent discharge was recorded during periods of attempted relaxation, during a range of stimuli which are known to produce exaggerated reflex effects on α-motor neurons in spastic patients, and finally during deliberate voluntary contractions. The stimuli were selected with the aim of accessing reflex pathways that could potentially elicit exaggerated fusimotor responses in stroke patients. Cutaneous afferents were stimulated mechanically (by stroking the skin) or electrically. Mechanical skin stimuli were used in addition to electrical stimulation because such stimuli are encountered during daily activities and should be of greater relevance to the clinical deficit in stroke patients. The effects of activating supraspinal pathways were assessed by making an unannounced sudden loud sound to induce a startle response (probably mediated through reticulospinal tracts) (Rothwell et al., 1993), by asking subjects to rotate their heads to either side (tonic neck reflexes), by clenching the contralateral fist (Jendrassik manoeuvre) and finally by deliberate isometric contractions of the parent muscle. A preliminary account of some findings has been published (Wilson et al., 1997b).

Method

Subject selection and assessment

This study was conducted on 11 patients (21 experiments) and 18 healthy adults all of whom gave informed consent in writing to the experimental procedures which had been approved by the local ethics committee (Committee on Experimental Procedures Involving Human Subjects, University of New South Wales). The mean ages of the control and patient groups were 33 years (range 21–52) and 54 years (range 24–75), respectively, a difference that is probably of no consequence given that there is no reason to believe that fusimotor function changes with age in adults. All patients had single unilateral cerebrovascular lesions caused by either an infarct or haemorrhage confirmed by CT scan or MRI. The initial recordings were made 1–6 months after the stroke, and all patients had weakness and exaggerated stretch reflexes in the affected upper limb with no major contracture. Patients were excluded from the study if they had additional confounding neuropathology (e.g. peripheral neuropathy, secondary diabetes), communication difficulty or risk factors for either bleeding or infection at the neural recording site (e.g. coumarin-based anti-coagulant medication). The strengths of the forearm flexors and extensors (wrist and fingers) were measured (Colebatch and Gandevia, 1989), and the strengths of muscle groups in the paretic limb expressed as percentages of those for the contralateral limb.

Table 1 summarizes clinical details for the 11 patients.

Data collection

For muscle spindle afferent recordings, subjects reclined in a comfortable chair with the test arm (the affected limb of those with hemiparesis) supported on a padded metal frame. The height of the frame was adjusted so that the shoulder was abducted to 65–70°, the elbow flexed 80–90° and the wrist pronated with the hand resting palm-down, with the fingers and thumb loosely flexed. Because it involved minimal restraint, this posture allowed more complete relaxation and was adopted so that experimental conditions were as ‘natural’ as possible. In some patients, involuntary movements and abnormal muscle tone affected the positioning of the arm during the recording session. Whenever this occurred, passive stretches were applied to aid muscle relaxation and the limb was then repositioned.

EMG activity was recorded from forearm flexors and...
extensors using surface electrodes (1 cm diameter; gain 50 000–100 000; bandwidth 3.2 Hz–3.2 kHz). To help relax the relevant muscles, subjects could watch the EMG recordings on a large oscilloscope.

**Microelectrode recordings**

Insulated tungsten electrodes were inserted percutaneously into the radial nerve 8–12 cm proximal to the lateral epicondyle (Vallbo et al., 1979). Muscle afferent sites were sought during repetitive manual percussion of the relevant muscle or its tendon. Recordings were considered to be from a nerve fascicle innervating muscle when pronounced discharge occurred in response to tendon percussion and voluntary contraction but not in response to superficial skin stimuli. Single afferents from muscle spindles were identified by their responses to maximal muscle twitches produced by intrafascicular stimulation, to stretch applied to the tendon and to voluntary muscle contraction with abrupt relaxation (McKeon and Burke, 1980; Edin and Vallbo, 1990b). No attempt was made to distinguish between primary and secondary muscle spindle afferents. The filtered neural recordings were discriminated through a dual time-amplitude window discriminator (BAK model DDIS 1) and reprocessed off-line using a CED 1401 interface running Spike2 software to reconfirm all unit shapes and to generate instantaneous frequency plots.

**Muscle spindle afferent discharge rates**

The mean resting background discharge rates of muscle spindle afferents were recorded while subjects tried to relax the test limb with the aid of visual feedback of the EMG at high gain. It was not always possible for patients to relax the appropriate muscles completely and in these cases measurements of ‘resting’ background discharge rates were made when muscles were at their most relaxed.

**Stimuli**

**Electrical stimuli**

These were delivered to either the superficial radial nerve where it crosses the abductor pollicis longus tendon (radial) or to the skin on the volar aspects of the proximal phalanges of the fingers (palmar), as in previous studies (Nielsen and Pierrot-Deseilligny, 1991; Gandevia et al., 1994). The stimuli were delivered randomly at 1–3 Hz and consisted of trains of five impulses each of 1 ms duration at 300 Hz, with stimulus intensity set at 1.5–2 times perceptual threshold for a single stimulus. Trains were non-painful.

Skin strokes

These were applied using a light strip of metal (0.4 × 6 cm) fitted with a switch that was activated by the depression of the metal strip. This enabled the onset and completion of applied skin strokes to be detected. Skin strokes were applied manually by transverse sweeps across the exposed extensor surface of the test limb, starting distally at the fingers and progressing proximally to the forearm. Care was taken to avoid direct pressure over the muscle spindle ending under study.

**Joint distraction**

This was applied manually by the experimenter. The proximal part of the limb was fixed while steady traction was applied simultaneously to the distal part. Joint distractions were applied to the thumb, middle finger and wrist. Any trials associated with movement of the limb were discarded. Each stress to the joint lasted about 2–4 s.

**Data recording and analysis**

Background discharge rates were measured over intervals of 30 s when the subject was quiescent and there was minimal or no EMG in the forearm muscles. In these sequences, spindle discharge was steady without visible trends; statistical tests of a stationary state were not used. The coefficient of variation was calculated for actively discharging afferents from these sequences as the standard deviation of the discharge rate divided by the mean rate. Unless indicated, values are given as means ± standard deviation. The χ² test was used for comparing the observed discharge rates with those in other afferent samples. Significance was set at the 5% level.

**Results**

Single- and multiunit recordings of muscle spindle afferent activity were made from the radial nerve of the upper limb in 11 patients (paretic side, 18 experiments) and in 18 healthy subjects. In addition, in three patients, recordings were also made from the non-paretic side in a separate recording session (five single muscle spindle afferents, five multiunit sites). To simplify the discussion these recordings have been categorized separately (stroke ‘non-paretic’ side; see Table 2) and the data have not been included in the group data analysis.

**Background discharge activity of single muscle spindle afferents**

Recordings were made from 102 identified muscle spindle afferents and from 36 multiunit sites dominated by the activity of muscle spindle afferents. Of the single spindle afferents, 26 were from hemiparetic sites and 76 from control limbs: extensor carpi radialis (n = 12 and 32, respectively), extensor digitorum communis (n = 5 and 19), brachioradialis (n = 4 and 5), extensor pollicis longus (n =
Table 2  Muscle spindle afferent discharge rates in relaxed subjects

<table>
<thead>
<tr>
<th></th>
<th>All muscles</th>
<th>Wrist extensors</th>
<th>Digit extensors/abductors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy control*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of afferents</td>
<td>76</td>
<td>35</td>
<td>35</td>
</tr>
<tr>
<td>Rate of all afferents (Hz)</td>
<td>6.4 ± 6.1</td>
<td>7.5 ± 5.7</td>
<td>4.5 ± 5.6</td>
</tr>
<tr>
<td>Active afferents (% total)</td>
<td>63</td>
<td>78</td>
<td>49</td>
</tr>
<tr>
<td>Rate of active afferents (Hz)</td>
<td>10.1 ± 4.6</td>
<td>10.1 ± 4.2</td>
<td>9.3 ± 4.3</td>
</tr>
<tr>
<td>Stroke, paretic side</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of afferents</td>
<td>26</td>
<td>14</td>
<td>8</td>
</tr>
<tr>
<td>Rate of all afferents (Hz)</td>
<td>6.6 ± 5.3</td>
<td>7.1 ± 5.5</td>
<td>5.1 ± 5.7</td>
</tr>
<tr>
<td>Active afferents (% total)</td>
<td>73</td>
<td>79</td>
<td>50</td>
</tr>
<tr>
<td>Rate of active afferents (Hz)</td>
<td>8.8 ± 3.7</td>
<td>9.0 ± 4.4</td>
<td>10.2 ± 2.6</td>
</tr>
<tr>
<td>Stroke, non-paretic side</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of afferents</td>
<td>5</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Rate of all afferents (Hz)</td>
<td>6.5 ± 6.7</td>
<td>10.0 ± 10.0</td>
<td>–</td>
</tr>
<tr>
<td>Active afferents (% total)</td>
<td>80</td>
<td>75</td>
<td>–</td>
</tr>
<tr>
<td>Rate of active afferents (Hz)</td>
<td>7.0 ± 6.0</td>
<td>13.3 ± 9.2</td>
<td>–</td>
</tr>
</tbody>
</table>

Wrist extensors: extensor carpi radialis, extensor carpi ulnaris; digit extensors/abductors: extensor digitorum communis, extensor pollicis longus, extensor pollicis brevis, abductor pollicis longus. *Includes data from a previous publication (Gandevia et al., 1994).

Electrical stimuli
These were delivered to cutaneous afferents in the superficial radial nerve at the wrist or in the palmar aspect of the fingers for 15 identified muscle spindle afferents in the patient group (mean number of stimulus trains per afferent = 36, range 12–45). Peristimulus time histograms were used to assess the effect of the electrical stimuli on muscle spindle afferent discharge. Figure 1A shows an example of a peristimulus time histogram recorded for a muscle spindle afferent innervating extensor pollicis longus during superficial radial stimulation. Electrical stimulation (46 trains) produced no effect on the discharge of this afferent, nor on the discharge of any other afferent. Although there was no change in muscle spindle afferent activity, the stimuli sometimes produced a reflex discharge of α-motor neurons as revealed in the average rectified EMG (see multiunit recording in Fig. 1B). Reflex changes in spindle activity would have been expected at a latency of >100 ms (Gandevia et al., 1994).

Mechanical stimuli
These were applied to the skin while recording from eight identified muscle spindle afferents in the patient group (mean stimulus count per spindle = 14, range 5–30). EMG activity developed during the natural skin stimulation with seven of the muscle spindle afferents, but the discharge of the afferents did not change (e.g. see Fig. 2). The same was true for the 17 multiunit sites tested, as is illustrated for an extensor carpi radialis site in Fig. 3; there was no evidence of increased neural discharge during repetitive skin strokes over the volar...
and dorsal aspects of the involved limb, even in the presence of a contraction of the extensor muscles.

**Other manoeuvres**

Joint distraction was often difficult to achieve without disturbing the recording site. There was no effect on afferent discharge without an accompanying EMG response for multiunit sites (n = 7) or for a single muscle spindle afferent in the patient group. Similarly, there was EMG activity in response to a sudden loud noise in five out of eight patients tested, but no increased afferent discharge in the absence of EMG. Forceful fist clenching on the contralateral side usually provoked an unintended ipsilateral contraction associated with increased spindle activity. The increased activity always followed the appearance of EMG and did not occur in its absence. The effect of deliberate head rotations could be assessed reliably only twice in the patients because of the tendency for excessive movement and contamination of the neural recordings by EMG. There was no increased muscle spindle afferent activity without EMG in the parent muscle.

**Voluntary contraction**

In all, 20 muscle spindle afferents were studied (patients, n = 10; controls, n = 10) during deliberate isometric contractions of the receptor-bearing muscle. The pattern of muscle spindle afferent discharge during contraction was the same in both the patient and control groups. Increase in EMG activity preceded increases in spindle afferent discharge. The mean discharge rate for all but one of the muscle spindle afferents from the patients increased during deliberate isometric contraction of the affected arm. The mean increase in discharge rate during deliberate contractions was 8.6 ± 7.2 Hz (range 1.4–23.2 Hz) in the patients and 4.1 ± 3.8 Hz (range 0.4–12.4 Hz) in the control subjects (P > 0.27). Although the contractions were not required to be of equivalent effort, the data indicate that the patients could access the fusimotor system voluntarily, perhaps no less effectively than the control subjects.

Figure 4 illustrates the discharge recorded from a muscle spindle afferent (innervating extensor carpi radialis, patient 2 in Table 1) during two consecutive isometric wrist extensions. As is illustrated in Fig. 4, patients often found it difficult to relax the test muscles completely. Inadvertent EMG in the agonist muscle was often accompanied by elevated spindle discharge rates, much as occurred in healthy subjects. This recording shows a large increase in spindle discharge (~35 Hz) during the attempted voluntary contraction. Figure 5 shows data from a muscle spindle afferent while the subject made deliberate ‘grip’ contractions...
with the ipsilateral hand. Muscle spindle afferent activity occurred after the onset of EMG activity in the receptor-bearing muscle, the normal pattern of response in healthy subjects, which has been extensively documented previously (Vallbo, 1971, 1974; Burke, 1981; Edin and Vallbo, 1990a; Wilson et al., 1997).

Recordings from patients’ non-paretic arm
For the five muscle spindle afferents in the non-paretic upper limb of the stroke patients, the mean background discharge rate was the same as for the patient and control groups (6.5 ± 6.7 Hz; see Table 2). There were no fusimotor reflex effects, and the pattern of muscle spindle afferent discharge during voluntary contractions was similar to that recorded for the affected arm and for control subjects.

Discussion
The present study sought evidence for disturbed fusimotor function in hemiparetic patients, all of whom had reflex hyperexcitability typical of ‘spasticity’ (Lance, 1980). No such evidence was found. The discharge rates of muscle spindles in relaxed muscles were the same as in normal subjects, the spindle endings did not have abnormally enhanced reflex responses to peripheral afferent inputs, and there was no difference in their responses to the supraspinal drive associated with reinforcement manoeuvres, head rotation, startle or deliberate voluntary contraction. It must be conceded that, with the exception of the background discharge rates, these findings are qualitative rather than quantitative but, if fusimotor dysfunction had a driving role in the motor abnormalities associated with hemiparetic spasticity, we would have expected to find some evidence of abnormal spindle behaviour. The general impression from the present results is that any fusimotor dysfunction is likely to make a minor contribution to the patients’ disability.

Fusimotor function at rest
Overactivity of the fusimotor system involving particularly dynamic fusimotor neurons, has been postulated to underlie the tendon jerk hyperreflexia and increased muscle tone of spasticity (e.g. Rushworth, 1960, 1964; Jansen 1962; Dietrichson, 1971). The present finding that the background discharge rates of muscle spindle endings in relaxed muscles of spastic patients are essentially the same as in control subjects argues against fusimotor overactivity, at least for static fusimotor neurons. However, qualitatively, the responsiveness of spindle endings to manipulation of the limb was also no greater in the patients, and it is unlikely that excessive dynamic fusimotor drive which was sufficient to produce reflex hyperexcitability would have been missed. The similarity in resting discharge rates for the two groups was somewhat unexpected since the patients tended to be less able to remain relaxed than control subjects.
In a previous study recording from the radial nerve in relaxed healthy subjects (Gandevia et al., 1994), the discharge of two out of 40 muscle spindle afferents increased in response to cutaneous afferent volleys. Identical cutaneous stimulation was used in this study, but it had no effect on the activity of 15 muscle spindle afferents in the affected arm of stroke patients. Similarly, there was no increase in the reflex responses of spindle afferents to any other peripheral input tested. These results argue against increased resting fusimotor drive in stroke patients and are consistent with evidence from many previous studies that have suggested that there is little, if any, fusimotor drive to relaxed human muscles.

The present data are also consistent with data from animal models designed to reproduce the spastic state: chronic spinal hemisection (Melzter, et al., 1963; Fujimori et al., 1966; Aoki et al., 1976), ablation of motor cortex (Gilman et al., 1974), pyramidal tract section (Gilman et al., 1971) and chronic spinal transection (Lieberman et al., 1979), none of which resulted in demonstrable fusimotor over-activity. They are also consistent with quantitative data on the response to stretch of nine spindle endings from the hemiparetic lower limb of two spastic patients (Hagbarth et al., 1973). Together, these data, animal and human, indicate that fusimotor overactivity is not necessary for spasticity to develop.

The question then arises whether fusimotor overactivity ever occurs in spastic patients. Given that in animals, discrete cerebral lesions can result in selective activity of the dynamic fusimotor system and that stimulation within cerebral structures can produce fusimotor activity without skeletomotor activity (e.g. Granit et al., 1955; Appelberg, 1981), it is likely that fusimotor overactivity may occur in patients with appropriately located discrete pathology. However, as argued elsewhere (Burke, 1981), fusimotor overactivity
cannot produce spasticity by itself; the ‘gain’ of the reflex pathway is too low for the hyperreflexia of spasticity to result from an enhanced muscle spindle input to the spinal cord (Vallbo, 1971, 1974) without changes in the way that input is processed by spinal cord circuits (Pierrot-Deseilligny, 1990).

**Reflex activation of fusimotor neurons**

In patients with spinal cord or cerebral lesions, disturbed spinal reflex function is common; in addition to overactive tendon jerks, there may be extensor plantar responses, loss of cremasteric and abdominal reflexes, flexor and extensor spasms, reflex grasping and other manifestations of ‘dibhhibited’ or dysfacilitated spinal pathways. In spinal cats, cutaneous afferents can reflexly activate and inhibit fusimotor neurons (Alnaes et al., 1965; Grillner, 1969; Appelberg et al., 1977). There is some evidence for cutaneofusimotor reflex pathways in intact human subjects, but this has been demonstrable only exceptionally in subjects at rest (Gandevia et al., 1986, 1994; see also Struppler and Velho, 1976) and more commonly when subjects engage in an appropriate motor task (Aniss et al., 1990).

Disinhibition of cutaneofusimotor reflexes would alter the pattern of afferent feedback generated by movement and might, thereby, contribute to loss of dexterity. However, there was no evidence that peripheral afferent inputs could activate fusimotor neurons abnormally in hemiplegic patients at rest. Indeed, peripheral stimuli failed to activate any muscle spindle afferents in the present study unless the stimulus resulted in EMG activity and, accordingly, there was no evidence that cutaneofusimotor reflexes were operative, either in patients or control subjects. However, this does not imply that there is fusimotor hyporeflexia in the patients because the incidence of demonstrable reflex effects is normally quite low in the relaxed state (Gandevia et al., 1994).

**Voluntary activation of fusimotor neurons**

Studies in which the corticospinal system was stimulated electrically have shown that fusimotor neurons may be recruited at lower thresholds than skeletomotor neurons (Granit, et al., 1955; Appelberg, 1981). Furthermore, in behaving animals the fusimotor system can be activated independently of the skeletomotor system (Grigg and Preston, 1971; Prochazka, et al., 1976, 1977; Schieber and Thach, 1980). In awake human subjects, cortical stimulation can activate fusimotor neurons (Rothwell et al., 1990) and in voluntary tasks there may be changes in the balance between fusimotor and skeletomotor drives (Burke et al., 1980; Vallbo and Hulliger, 1981). Based on these findings it is conceivable that a corticospinal lesion might disrupt the voluntary control of fusimotor neurons disproportionately. If so, voluntary effort would be associated with an inappropriate feedback from spindle endings in the paretic muscle during attempted voluntary movement and this might contribute to the loss of dexterity. No such alteration in spindle behaviour was apparent. Spindle endings were not recruited voluntarily in the absence of EMG or before the appearance of EMG, and did not achieve higher discharge rates than is usually seen when normal subjects contract the forearm extensors. On the other hand, there was no tendency for spindle activation to be appreciably more difficult to produce. Given that the limbs were paretic but not paralysed, it would be reasonable to conclude that the corticospinal lesion had similar effects on the voluntary drives to α- and γ-motor neurons.

**Supraspinal activation of fusimotor neurons**

Based on changes in the responses of muscle spindles to the relaxation of a twitch contraction, it has been suggested that (i) reinforcement manoeuvres can activate dynamic fusimotor neurons selectively and (ii) dynamic fusimotor neurons may be overactive at rest in spastic patients (Szumski et al., 1974). The first postulate was not supported by subsequent studies that controlled for extrafusal influences on muscle spindle discharge (Hagbarth et al., 1975a; Burke, 1981) and the second is not supported by the present data. Nevertheless, it was appropriate to re-examine whether, in patients, muscle spindle discharge would be affected by the performance of reinforcement and other alerting manoeuvres, and by head rotation, because clinically, muscle tone can be responsive to mental set and changes in body posture. Performance of these manoeuvres commonly provoked unwanted contractions in the hemiparetic limb, as much as is often seen clinically. However, muscle spindles were not activated in the absence of EMG, and spindle activation did not precede the appearance of EMG in the appropriate forearm muscles. Accordingly, there was no evidence for enhanced input to fusimotor neurons from non-volitional (non-corticospinal) descending pathways to compensate for the impaired volitional (corticospinal) drive.

**Alpha–gamma balance in hemiparetic patients**

An important conclusion of the present study is that the corticospinal lesion resulting from a stroke does not significantly disturb fusimotor function, or at least does not do so any more or less than skeletomotor function. This is an important, even if negative conclusion, because it implies that disturbed fusimotor function is not the prime driving force behind the clinical deficits that such patients suffer. This conclusion is consistent with the view that a primary deficit of fusimotor function is not a major factor in disturbances of muscle tone or movement control (Gorassini et al., 1993). It cannot be denied that more quantitative measurements might reveal subtle deficits of fusimotor function, but these would be unlikely to alter the main conclusion of the study. Perhaps the major value of the study lies in a reaffirmation that the motor control deficits of patients with hemiparetic spasticity arise centrally in disturbed central pathways, not peripherally in the muscle spindle. In future studies it might be interesting to look at muscle
spindle afferent activity in flexor muscles in the upper limb because they exhibit spasticity more than extensor muscles. The evidence so far suggests that cerebral lesions producing hemiplegic spasticity do not cause the fusimotor system to become pathologically hyperexcitable at rest, pathologically hyporesponsive to volitional drives or hyperreactive to peripheral afferent inputs.

Acknowledgements
The National Health and Medical Research Council of Australia supported this work. The Canadian Medical Research Council funded J.T.I. and J.-M.G. was supported by a Fellowship from Institut National de la Santé et de la Recherche Médicale, France.

References


Gilman S, Lieberman JS, Marco LA. Spinal mechanisms underlying the effects of unilateral ablation of areas 4 and 6 in monkeys. Brain 1974; 97: 49–64.


Granit R, Holmgren B, Merton PA. The two routes for excitation of muscle and their subservience to the cerebellum. J Physiol (Lond) 1955; 130: 213–24.


