

## REVIEW

# Sensory control of normal movement and of movement aided by neural prostheses

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## Abstract

Signals from sensory receptors in muscles and skin enter the central nervous system (CNS), where they contribute to kinaesthesia and the generation of motor commands. Many lines of evidence indicate that sensory input from skin receptors, muscle spindles and Golgi tendon organs play the predominant role in this regard. Yet in spite of over 100 years of research on this topic, some quite fundamental questions remain unresolved. How does the CNS choose to use the ability to control muscle spindle sensitivity during voluntary movements? Do spinal reflexes contribute usefully to load compensation, given that the feedback gain must be quite low to avoid instability? To what extent do signals from skin stretch receptors contribute? This article provides a brief review of various theories, past and present, that address these questions. To what extent has the knowledge gained resulted in clinical applications? Muscles paralyzed as a result of spinal cord injury or stroke can be activated by electrical stimulation delivered by neuroprostheses. In practice, at most two or three sensors can be deployed on the human body, providing only a small fraction of the information supplied by the tens of thousands of sensory receptors in animals. Most of the neuroprostheses developed so far do not provide continuous feedback control. Instead, they switch from one state to another when signals from their one or two sensors meet pre-set thresholds (finite state control). The inherent springiness of electrically activated muscle provides a crucial form of feedback control that helps smooth the resulting movements. In spite of the dissimilarities, parallels can be found between feedback control in neuroprostheses and in animals and this can provide surprising insights in both directions.

**Key words:** feedback control of movement; muscle receptors; muscle spindles; neuroprostheses; sensorimotor control; tendon organs.

## Introduction

Several reviews have described in detail the anatomy and physiology of sensory receptors implicated in the control of bodily movement (Matthews, 1972; Hulliger, 1984), the sense of movement (kinaesthesia) (Proske & Gandevia, 2009, 2012) and the underlying neuronal mechanism [proprioception: sensing the body's own movements (Sherrington, 1907)]. This article reviews the nature of the sensory signals, the ways in which they might be used by the central nervous system (CNS) and the application of this knowledge to neuroprosthetic devices.

The mammalian body contains thousands of sensory receptors. The human upper limb contains about 4000

muscle spindles, 2500 Golgi tendon organs and a few hundred joint receptors (Barker et al. 1962; Voss, 1971; Hulliger, 1984). Sensory receptors in skin are even more numerous and varied. For example, the surface of the human hand alone has about 17 000 myelinated cutaneous afferents (Johansson & Vallbo, 1979).

## Sensory receptors involved in motor control

### Muscle spindles

The mean number of spindles in a muscle is roughly  $38 * (\text{cube root of mass in grams})$  (Banks & Stacey, 1988). Thus a 125-g muscle contains about 190 spindles. Spindles are attached at each end to the surrounding *extrafusal* muscle fibres that produce the forces for bodily movement. Much smaller *intrafusal* muscle fibres within the muscle spindle don't contribute measurably to these forces, their role being to control the stretch-sensitivity and baseline activity (*bias* or *offset*) of the spindle's afferents. They do this by pulling on the central non-contrac-

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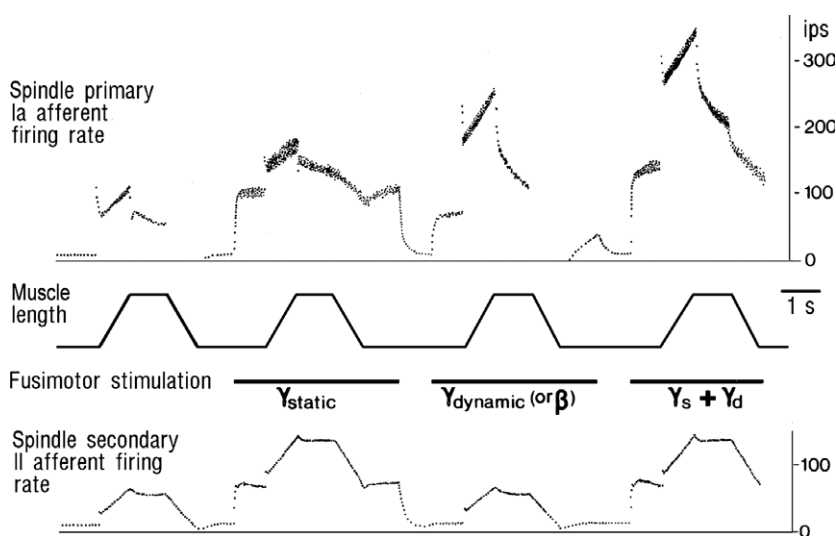
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tile region of the spindle that contains the stretch-sensitive *spindle primary (group Ia) afferents* and *secondary (group II) afferents*. Intrafusal muscle fibres are activated by *fusimotor neurons*, also called  $\gamma$ -*motoneurons* by virtue of their conduction velocity. Some  $\alpha$ -*motoneurons* send branches to intrafusal muscle and these are called  $\beta$ -*motoneurons*. Details of intrafusal and fusimotor subdivisions and their effect on spindle afferent firing are summarized in Fig. 1 and in the associated animation ([angeltear.com/spindle/spindle.html](http://angeltear.com/spindle/spindle.html)). Briefly, *static* fusimotor neurons ( $\gamma_s$ -*motoneurons*) increase the firing rate of both spindle *primary (group Ia)* and *secondary (group II)* afferents in the absence of a length change of the receptor-bearing muscle (biasing action). This can keep Ia and II afferents firing during rapid muscle shortening. The *sensitivity* of Ia and II afferents to muscle length changes is increased by  $\gamma_d$  and  $\gamma_s$  action, respectively.

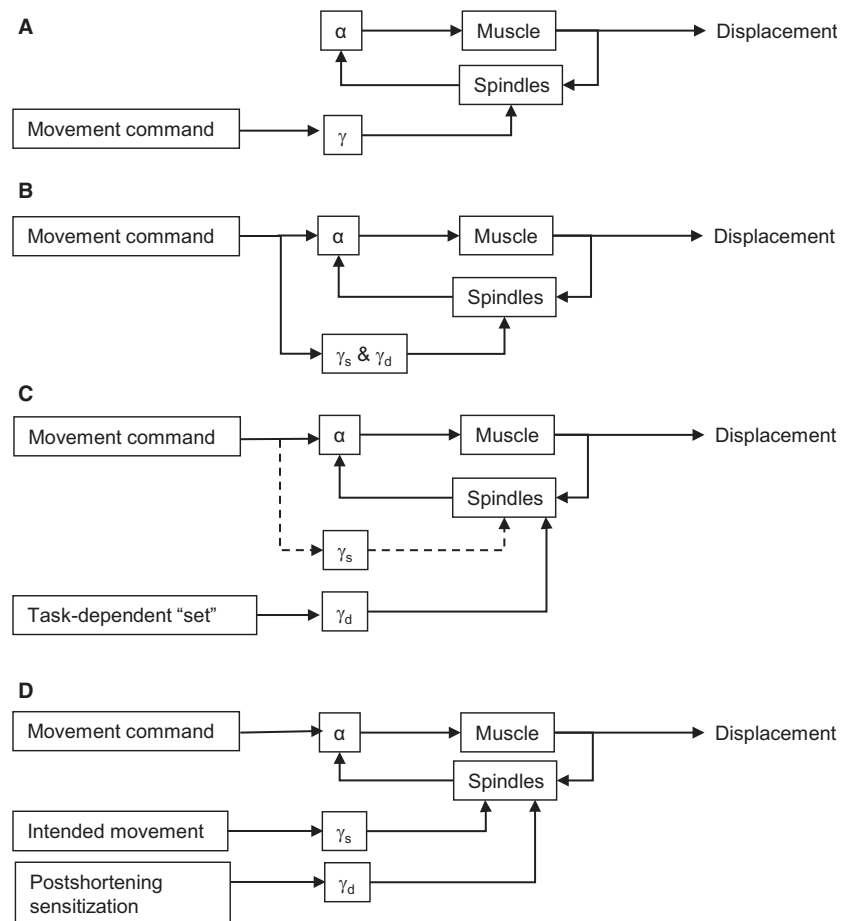
There has been much debate on the nature of fusimotor activity during voluntary movement. The main barrier has been the difficulty in recording from the small axons of  $\gamma$ -motoneurons. This is less of a problem in anaesthetized or decerebrate animals. Four main hypotheses have emerged. The first was the follow-up length servo hypothesis (Rossi, 1927; Merton, 1953), which proposed that in a voluntary muscle contraction,  $\gamma$ -motoneurons are activated first, increasing spindle afferent firing, which in turn reflexly activates  $\alpha$ -motoneurons (Fig. 2A). This theory was replaced by the servo-assistance hypothesis (Matthews, 1970; Fig. 2B), which proposed that  $\gamma$ -motoneurons were co-activated with  $\alpha$ -motoneurons, keeping spindle afferents firing during muscle shortening. It was further suggested that the biasing action of  $\gamma$  drive might exactly compensate for the shortening, except when this was impeded (Phillips, 1969). In this view, spindle afferents were misalignment detectors, sensing a disparity between expected and actual muscle length changes. Early recordings from spindle afferents in awake

humans showed that in isometric contractions, spindle afferents did indeed increase their firing rates, which supported  $\alpha$ - $\gamma$  co-activation and the servo-assistance hypothesis (Vallbo, 1970), though it was arguably at odds with the idea of misalignment detection given that the subjects did not expect the contracting muscle to shorten.

The plot thickened when it became possible in the 1970s to record from muscle afferents in freely moving animals. It was quickly noticed that spindle afferents did not fire steadily during unobstructed movements, as they should do if they were misalignment detectors. Rather, their firing rates were deeply modulated. In some cases the firing rates were closely related to the time course of muscle length and velocity (Cody & Taylor, 1973; Cody et al. 1975; Goodwin & Luschei, 1975). In other cases, increased spindle afferent firing during muscle shortening suggested strong  $\alpha$ -linked  $\gamma_s$  activity (Loeb, 1981). In movements that were novel or very difficult, such as walking on a narrow beam, large increases in Ia stretch-sensitivity were observed in cats implanted with dorsal root electrodes. This led to the hypothesis of *behaviour-related fusimotor set*, whereby  $\gamma$ -motoneurons were activated independently of  $\gamma$ -motoneurons (Prochazka et al. 1985; Fig. 2C).  $\alpha$ - $\gamma$  independence was subsequently supported in some studies (Roll & Vedel, 1982; Ribot et al. 1986) and refuted in others (Aniss et al. 1990). Evidence was adduced for specific combinations in flexor and extensor muscles:  $\alpha$ - $\gamma_s$  co-activation with independent  $\gamma_d$  activity (Gottlieb & Taylor, 1983; Taylor et al. 1993) and  $\alpha$ - $\gamma_d$  co-activation with independent  $\gamma_s$  activity (Perret & Buser, 1972; Cabelguen, 1981; Murphy et al. 1984; Bessou et al. 1986; Greer & Stein, 1990; Murphy & Martin, 1993). In an elegant set of studies in walking decerebrate cats Ellaway et al. (2015), presumed  $\gamma_s$  motoneurons fired throughout the step cycle, weakly modulated roughly in time with the phasic bursts of  $\alpha$ -activity in the muscle they innervated, whereas  $\gamma_d$  motoneurons fired at high rates



**Fig. 1** Firing rate responses of group Ia and II spindle afferents to trapezoidal length changes with and without concomitant fusimotor stimulation. The firing rates are typical of displacements of about 10% of muscle rest-length and velocities of 0.05 rest length per second. Horizontal bars indicate periods of fusimotor firing. Note the increase in Ia stretch responses with  $\gamma_d$  stimulation and the biasing effect of  $\gamma_s$  stimulation (ips: impulses per second) (Prochazka, 1996).



**Fig. 2** Four hypotheses of fusimotor control. (A) Follow-up length servo, (B) servo-assistance, (C) fusimotor set, (D)  $\gamma_s$ : 'temporal template' of intended movement,  $\gamma_d$ : sensitizes Ia afferents to stretch following muscle shortening.

during  $\alpha$ -activity and fell silent otherwise. Although this could have been interpreted as  $\alpha$ - $\gamma_d$  co-activation with partial independence of  $\gamma_s$  activity, other considerations, including the behaviour of spindle group II afferents, led to the hypothesis that  $\gamma_s$  firing was a temporal template of the expected movement (Fig. 2D), while  $\gamma_d$  firing sensitized Ia afferents to muscle stretch following shortening (Ellaway et al. 2002; Taylor et al. 2006).

As mentioned, early spindle recordings in humans performing isometric contractions supported  $\alpha$ - $\gamma$  co-activation. Subsequent data collected during unimpeded movement indicated that spindle afferents were affected more by muscle length changes than by presumed  $\alpha$ -linked  $\gamma$ -activity. To add to the confusion, Ia firing recently recorded during hand movements in humans performing keyboard tasks was not only correlated with muscle velocity and acceleration, but also with *future* movement (Dimitriou & Edin, 2008). It was proposed that fusimotor drive reflected internal predictive models (Wolpert & Miall, 1996; Dimitriou & Edin, 2010), an idea similar to that of the 'temporal template of expected movement' (Taylor et al. 2000). The most recent human work has refuted this idea, and instead has not only supported  $\alpha$ - $\gamma$  co-activation and a velocity-signalling role for Ia afferents, but also suggested a concomitant

inhibition of antagonist  $\gamma$ -motoneurons (Dimitriou, 2014). Figure 3, modified from that study, shows the responses of finger extensor Ia afferents in seven subjects performing voluntary flexion-extension movements of the fingers in the presence or absence of steady, externally imposed torques resisting or assisting extension. Torque-resisting extension elicited a compensatory increase in voluntary extensor EMG activity, particularly during the muscle shortening phases. This was correlated with an increase in bias (offset) and stretch-sensitivity of the extensor Ia responses compared with those in unloaded movements, which was interpreted by Dimitriou as evidence for  $\alpha$ - $\gamma$  linkage. Because the Ia firing rate was well correlated with muscle velocity throughout the cycle, with no obvious increase during the phasic increase in extensor EMG during muscle shortening, this could also have been interpreted as evidence of a tonic increase in fusimotor drive throughout the cycle, related to overall effort. Movements in the presence of imposed extensor torque were associated with elevated flexor EMG activity and this was correlated with a reduction in the extensor Ia responses in the early part of muscle stretch, suggesting reciprocal inhibition of extensor fusimotor drive. Interestingly, if we assume that the CNS decodes movement by taking into account Ia signals from antagonist and ago-

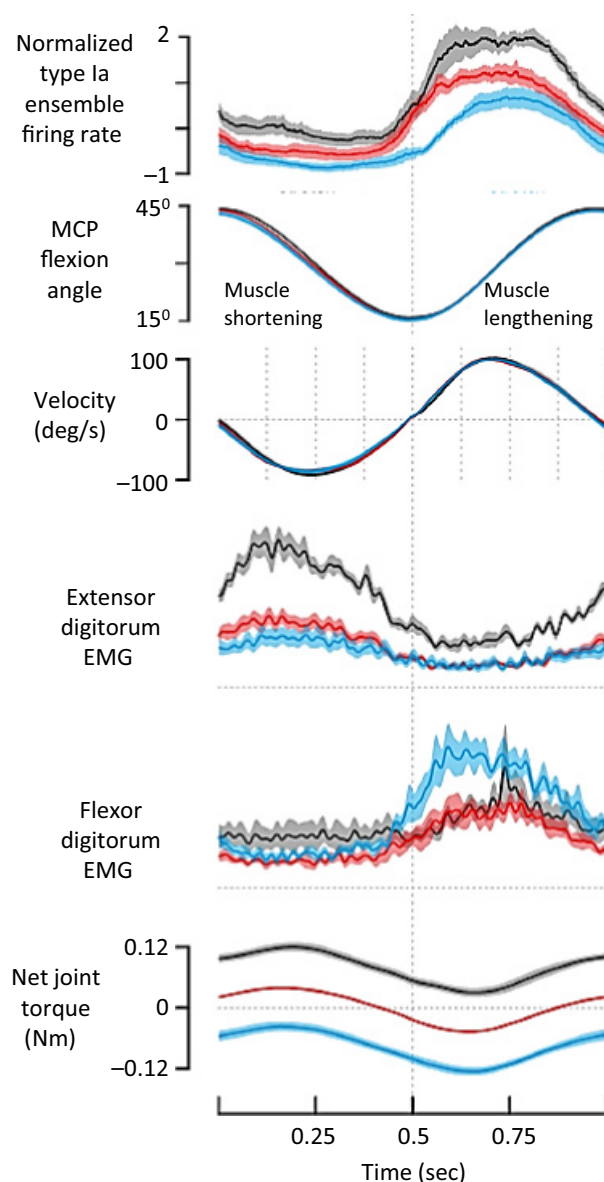
nist muscle pairs, the reciprocal fusimotor effects proposed by Dimitriou would tend to cancel each other out.

So what can we conclude about the role of the fusimotor system? First, there is much evidence that a component of  $\gamma$ -activity is linked in time and strength to the activation of  $\alpha$ -motoneurons (Wilson et al. 1997).  $\beta$ -motoneurons are  $\alpha$ -motoneurons with branches innervating intrafusal muscle fibres, so  $\beta$ -fusimotor drive would also be linked to  $\alpha$ -activity. Secondly, there is a separate component of  $\gamma$ -activity that is *independent* of  $\alpha$ -activity. On this view, some  $\gamma$ -motoneurons fire tonically throughout movement sequences, their firing rate being modulated in relation to behavioural set or to an internal model of the expected movement trajectory.

What is the final effect of the interaction of fusimotor drive and muscle length changes on muscle spindle firing? The simplest view, and the one favoured by the author and several other groups, is that spindle group Ia and II afferents are essentially velocity and length detectors whose sensitivity and bias are adjusted by fusimotor action according to the demands of the motor task. One caveat to this simple view is that tendons of phasically active muscles can absorb a significant portion of the length change occurring between the origin and insertion of the muscle, distorting the relationship between muscle length (or velocity) and spindle afferent firing (Griffiths, 1991; Maganaris & Paul, 1999; Lichtwark & Wilson, 2008; Magnusson et al. 2008). Nonetheless, the time course of limb joint angles during normal locomotion has been inferred reasonably accurately from the firing rates of a handful of afferents recorded with an implanted multi-electrode array. Analysis showed that it was the spindle afferents that provided the most information in this regard (Weber et al. 2006, 2007). From the perspective of mimicking muscle spindle feedback in a neuroprosthesis, the closest practical embodiments are length gauges or Hall effect sensors signalling joint rotation, whose signals are conditioned with variable-gain filters (see below) (Prochazka & Wiles, 1983; Prochazka et al. 1997a; Johnson et al. 1999).

### Golgi tendon organs

Fortunately, there is much more agreement on the signalling properties of ensembles of Ib afferents from Golgi tendon organs. Unlike spindles, Golgi tendon organs do not have a mechanism to modulate their sensitivity. They respond to force actively generated by the motor units they monitor (Houk & Henneman, 1967; Goslow et al. 1973; Stephens & Stuart, 1974; Stephens et al. 1975). When the firing rates of several Ib afferents recorded during normal movements were summed, the net firing rate was closely related to whole-muscle force (Prochazka & Gorassini, 1998) as predicted in an earlier study (Crago et al. 1982). In a neuroprosthesis, the closest approximation to an ensemble of Golgi



**Fig. 3** Mean normalized firing rates of nine extensor digitorum Ia afferents in seven subjects performing voluntary finger movement without loading (red) or in the presence of steady imposed flexor torque (black) or extensor torque (blue). Imposed flexor torque required a compensatory increase in extensor EMG activity, particularly during muscle shortening. This was correlated with an increase in the offset and stretch-sensitivity of extensor Ia firing compared with unloaded movements, consistent with elevated extensor fusimotor drive. Movements in the presence of imposed extensor torque (blue) required compensatory increases in flexor EMG. The associated reduction in extensor Ia firing in response to muscle stretch suggested reduced extensor fusimotor drive. Ia firing was well correlated with muscle velocity in all cases. Shaded areas represent +1 SEM. Modified with permission from Dimitriou (2014).

tendon organs would be a buckle transducer implanted on a tendon (Walmsley et al. 1978). This would be invasive and of limited lifespan, at least with current technology. Practi-

cal, though less direct, equivalents include force or pressure sensors attached externally to a limb, for example in the heel of a shoe (Burridge et al. 2001) or in the fingertips of a glove (Su et al. 2012).

### Receptors in joints and skin

Early work suggested that joint receptors signalled joint angle over the full range of motion (Boyd & Roberts, 1953). This was challenged in studies suggesting that joint receptors only fired at the extremes of joint angle and not in the mid-range (Burgess & Clark, 1969; Tracey, 1979). Subsequent work showed that at least some joint afferents signalled the full range of motion (Godwin-Austen, 1969; Zalkind, 1971; Carli et al. 1979; Ferrell, 1980; Lund & Matthews, 1981; Ferrell et al. 1987), but it is possible that these afferents were from muscle receptors near the joints (McIntyre et al. 1978; Clark et al. 1985; Gregory et al. 1989). Loading of the joint capsule may be necessary to enable mid-range responsiveness (Grigg & Greenspan, 1977). Most joint afferents have weak polysynaptic connections with  $\alpha$ -motoneurons (Johansson et al. 1991), their role possibly being to inhibit  $\alpha$ -motoneurons when joints are damaged (Iles et al. 1990).

There are several kinds of skin receptors which respond to rapid hair deflection or skin stretch (Willis & Coggeshall, 1991). The skin receptors best suited to signal position are slowly adapting type II receptors which respond to stretching of the skin over and around a joint (Horch et al. 1977; Edin & Johansson, 1995; Edin, 2001, 2004). Type I cutaneous receptors respond more locally, fire less regularly and adapt more rapidly. My colleagues and I have recently been experimenting with artificial skin stretch sensors to control stimulation in a foot-drop neuroprosthesis (see below).

## The role of sensory input to the CNS from muscle and skin receptors

### Kinaesthesia

Muscle and skin afferents both contribute to kinaesthesia (Goodwin et al. 1972; McCloskey et al. 1983; Collins & Prochazka, 1996; Collins et al. 2005). This topic is covered in detail in a recent review (Proske & Gandevia, 2012) and in the article by Proske in this volume of the journal. The following section will focus on the role of muscle and skin sensory feedback in the control of movement.

### Stretch reflexes

Active muscles resist length changes through their intrinsic stiffness, providing the equivalent of negative length feedback (Partridge, 1967). Stretching a muscle causes an increase in firing of its muscle spindle Ia afferents, which monosynaptically excite homonymous  $\alpha$ -motoneurons, activating the muscle to resist the increase in length (Marsden

et al. 1976; Matthews, 1986). This is also equivalent to negative length feedback. Golgi tendon organ Ib afferents respond to the increase in force caused by muscle stretch, particularly when the muscle is active. They reflexly activate interneurons that *inhibit* homonymous  $\alpha$ -motoneurons, thereby resisting changes in force. This is equivalent to negative force feedback (Houk et al. 1970). Concomitant length and force feedback results in spring-like behaviour, the stiffness of which depends on the ratio of length to force feedback gains (Prochazka & Yakovenko, 2002).

Studies have indicated that the loop gain of negative force feedback may in general be quite low (Houk et al. 1970; Hoffer & Andreassen, 1981). Furthermore, during locomotion in the cat, homonymous Ib reflex action has been found to become *excitatory* (Conway et al. 1987; Pearson & Collins, 1993), which is equivalent to *positive* force feedback (Prochazka et al. 1997b; Geyer et al. 2003; Grey et al. 2007). In closed-loop control systems, positive feedback with a loop gain exceeding one is unstable. However, as muscles shorten, the increment in force they generate for a given increment in activation declines. Thus even if the loop gain of positive force feedback initially exceeds unity, as the muscle shortens, loop gain returns to unity and instability is avoided (Prochazka et al. 1997b). This makes positive force feedback mediated by Ib afferents a suitable mechanism to compensate for weight-bearing in the stance phase of locomotion.

Intrinsic muscle stiffness increases with increasing muscle activation, including activation resulting from reflex responses to sensory input. Intrinsic muscle stiffness also depends on biomechanical factors such as muscle length and velocity (Bennett, 1993, 1994; Bennett et al. 1994). In a classical study in the decerebrate cat, the force response to stretch of electrically activated muscles in the absence of stretch reflexes was compared with the response of naturally activated muscles with intact stretch reflexes (Nichols & Houk, 1976). In the electrically activated muscles, force rose rapidly for the first 50 ms but then suddenly declined. In contrast, in the reflexly active muscles, force increased throughout the stretch. It was concluded that reflex action compensated for the yielding that occurred with steady activation alone, thereby linearizing the response and increasing the net stiffness. In later human experiments, electrically activated muscles did not yield nearly as much and so the linearizing effect of stretch reflexes was not as marked (Sinkjaer et al. 1988). At medium activation levels, stretch reflexes increased muscle stiffness by up to 60%, in accordance with the decerebrate experiments, but surprisingly, at both low and high levels of activation the stretch reflex contributions dropped to zero.

It was originally assumed that the short-latency monosynaptic stretch reflex mediated by Ia afferents was important in controlling load-bearing during locomotion (Capaday & Stein, 1986). Sinkjaer and colleagues challenged this,



instead proposing that spindle group II afferents were the main contributors (Sinkjaer et al. 2000; Grey et al. 2001). This has in turn been challenged in experiments which implicated short-latency positive force feedback mediated by Ib afferents (Grey et al. 2007; af Klint et al. 2010). In the cat, large, rapid, perturbations are required to elicit short-latency stretch reflexes during locomotion (Gorassini et al. 1994; Gritsenko et al. 2001). This led to some doubt as to the importance of short-latency stretch reflexes in load-bearing (Prochazka et al. 2002), but other work suggested that longer latency Ib-mediated stretch reflexes may contribute up to 35% of muscle activation during cat locomotion (Stein et al. 2000; Donelan & Pearson, 2004; Donelan et al. 2009).

Neuromechanical modelling has been employed to help clarify these partly contradictory results (Prochazka et al. 2002; Yakovenko et al. 2004; Ekeberg & Pearson, 2005; Prochazka & Yakovenko, 2007a; Stienen et al. 2007; Markin et al. 2010). Mathematical models of muscle afferent responses to muscle length and force variations were used (Prochazka, 2015). In one such model of locomotion, the phasic activation of four hindlimb muscle groups was produced by a locomotor central pattern generator (CPG; Prochazka et al. 2002; Yakovenko et al. 2004). In the absence of stretch reflexes, and with levels of muscle activation adequate to support body weight, the model produced several stable step cycles before becoming unco-ordinated and collapsing. Adding stretch reflexes, including positive force feedback mediated by Ib afferents, resulted in a slight increase in body height during the stance phases. When the CPG-generated muscle activation levels were set slightly too low to support the body, without stretch reflexes the model quickly collapsed. With stretch reflexes, stable steps were restored.

### General conclusions on the role of stretch reflexes

Taking all of the evidence together, stretch reflexes clearly modulate muscle stiffness and this can play a linearizing and stabilizing role during movement. Short-latency, spinally mediated reflex responses to Ia input may play a lesser role than short-latency reflex responses to group II and Ib input. *Supra*-spinally-mediated long-latency responses to Ia, II and Ib input can play a more significant role, but in the words of a recent study: 'Neural activity occurring within the period normally ascribed to the long-latency stretch reflex is highly adaptable to current task demands and possibly should be considered more intelligent than "reflexive"' (Shemmell et al. 2010).

### Phase-switching

In the absence of sensory input, the transitions between the stance and swing phases of the locomotor step cycle would

rely entirely on a pre-set temporal sequence of muscle activations generated by the locomotor CPG. It has been proposed that the timing of the switch between stance and swing phases of locomotion is triggered by finite state rules of the type 'IF in stance phase AND leg is extended AND unloaded AND contralateral leg is loaded THEN switch to swing' (Tomovic & McGhee, 1966; Cruse, 1990; Tomovic et al. 1990; Prochazka, 1993). When such rules were used to fine-tune the timing of phase transitions generated by the CPG, locomotion became much more stable (Yakovenko et al. 2004; Prochazka & Yakovenko, 2007a,b). Similar results were obtained by Rybak and colleagues, who concluded that afferent feedback adjusts CPG operation to the kinematics and dynamics of the limb providing stable locomotion (Markin et al. 2010). In another modelling study, there was no CPG at all; instead, phase-switching was controlled *entirely* by IF-THEN rules (Ekeberg & Pearson, 2005).

## Clinical applications

### Feedback control of stereotyped movements

Because of the practical difficulty of deploying numerous sensors on the human body in assistive devices designed for the rigours of daily use, it has not generally been possible to mimic the biological control mechanisms discussed above in neuroprostheses. It is interesting that in the few cases where analogous mechanisms of control were implemented, these were developed without specific reference to the corresponding biological mechanisms. A good example of this is the control of the stance to swing phase transition of hemiplegic gait with functional electrical stimulation (FES). The control method in most cases is to use under-heel switches or pressure transducers to detect the decline in force that occurs at the end of the stance phase of gait and to use this to trigger stimulation of the common peroneal nerve, which activates the muscles to dorsiflex the foot during the ensuing swing (Liberson et al. 1961). This corresponds to the unloading part of the finite state rule referred to above. In animals, Golgi tendon organs of the foot plantarflexor muscles may provide the sensory input relevant to this rule. Input from the skin of the sole of the foot may also contribute, as suggested by the locomotor deficits experienced by people with peripheral sensory denervation due to diabetes or chemotherapy.

Vodovnik and colleagues, who were among the pioneers of FES in the 1960s, explored the possibility of proportional feedback control of movements about the human elbow joint (Crochetiere et al. 1967; Vodovnik et al. 1967). Although the results were promising, the approach was not tested clinically, possibly because the motor points of biceps and triceps brachii shift under the skin as the elbow flexes and extends. This limits the range of movement over which reliable control can be achieved via surface electrodes. Furthermore, deriving a voluntary command signal was prob-

lematic. The only closed-loop FES devices to have been tested in participants going about their daily lives were a foot-drop stimulator (Prochazka & Wiles, 1983) and a tremor-suppressing system (Javidan et al. 1992; Prochazka et al. 1992). In the former case the signal from a length sensor spanning the front of the ankle was used to control the level of stimulation of the ankle dorsiflexor muscles that lift the foot. The system was modelled on the stretch reflex, the length gauge taking the place of spindles in the ankle dorsiflexor muscles and the stimulator taking the place of  $\alpha$ -motoneurons activating those muscles. This provided proportional negative feedback control of length. The command input to the controller was set to maintain an ankle angle of about 90° during the locomotor step cycle, minimizing the foot-drop that occurs in hemiplegia at the onset of swing and for the brief period at the onset of stance between heel-strike and toe-down. The system worked well in a number of hemiplegic people who went about their daily lives wearing the device. However, the location and attachments of the sensor on the lower leg and the shoe made it vulnerable to incorrect donning, dislodgement and mechanical inputs from clothing. Small shifts in the stimulating electrodes could cause foot inversion and eversion that were not detected by the sensor. For these reasons, under-heel switches were deemed to be more reliable and convenient, and the length feedback system was not pursued further.

In the 1990s a third mode of control was developed, namely the use of an accelerometer to monitor tilting of the lower leg. During locomotion, at the end of the stance phase, the lower leg tilts forward with respect to the vertical as the leg extends. In the Walkaide foot-drop stimulator, when the level of tilt reaches a pre-set threshold value, stimulation is delivered to the common peroneal nerve, activating the ankle dorsiflexor muscles to lift the foot during the ensuing swing phase (Everaert et al. 2013). This corresponds to the extension part of the finite state rule above. In terms of a direct biological equivalent, although there are no sensory receptors that monitor limb tilt, it has been shown that limb joint angles can be derived from signals from muscle and skin receptors and in principle this could be done by neural networks within the CNS (Weber et al. 2007). The Walkaide functions well in many people with hemiplegia, but not all users have enough range and reproducibility of leg tilt for the control method to work reliably (Gunther Gallagher, 2011). An under-heel switch is therefore provided as an alternative.

In relation to the probable contribution of cutaneous receptors to proprioception, the present author and his colleagues Michel Gauthier and Jacques Bobet recently experimented with the use of a skin stretch sensor to control foot-drop stimulation. The idea was essentially the same as in the system described above involving a length sensor spanning the ankle joint, namely to use continuous proportional feedback control of stimulation to resist increases in

ankle angle exceeding 90°. As discussed earlier, signals from cutaneous stretch receptors around limb joints correlate well with joint displacement, so it was posited that an artificial sensor that mimicked these receptors would provide usable control signals. This was confirmed in experiments in three normal individuals in whom skin stretch was monitored with sensors stuck to the skin over the Achilles tendon, about 15 cm proximal to the sole of the foot. A detailed report of these and related experiments is in preparation. Regarding the feasibility of this approach, sensors in the form of stretchable membranes have recently been developed for long-term monitoring of biological variables, so the requisite technology already exists (Hu et al. 2011).

### Voluntary command signals

Restoring voluntary control of movements of the paralyzed upper limb is an important goal. People with tetraplegia rate this as their primary concern (Anderson, 2004, 2009). As mentioned above, it is a challenge to derive voluntary command signals corresponding to descending drive from the brain, particularly for hand movements. One approach is to monitor the activity of muscles that remain under voluntary control, or the movements they produce. This has been implemented in several upper limb neuroprostheses that have been used in exercise training and activities of daily life. For example, in the implanted Freehand system, signals from a shoulder movement sensor were used to control FES-evoked hand opening and grasp (Smith et al. 1987; Keith et al. 1989). Electromyograms from proximal muscles were used in a second-generation version of this device (Kilgore et al. 2008; Memberg et al. 2014). Wrist movements were used in a surface stimulator (Prochazka et al. 1997a) and a third version of the Freehand system (Peckham et al. 2002). Tooth-clicks monitored by a wireless earpiece were used in a recent surface hand stimulator (Kowalczewski et al. 2011) and an implanted version of this device (Gan et al. 2012). Finally, head movements, also monitored by a wireless earpiece, are used to control hand grasp and release in a surface stimulator currently being developed commercially by the author and colleagues. The shoulder and wrist-controlled versions of the Freehand system enabled users to generate a number of hand movements that allowed simple activities of daily life. The EMG-controlled version allowed proportional control of shoulder, elbow and hand movements with visual feedback (Memberg et al. 2014), though weight-support was required for functional tasks. In the other cases the signals simply provided a trigger to switch from one state to the next in a sequence, a biological function proposed above for input from Ia and Ib afferents. To enable a variety of graded, coordinated hand movements, several degrees of freedom are required of the control signals. Brain machine interfaces could in principle provide such signals, but currently this technology has only been used to control robot arms and

hands (Collinger et al. 2013). Less invasive sensor systems that combine signals derived from 3-D head movements, tooth-clicks and voice commands, are being developed in the author's laboratory.

## Concluding remarks

Sensory input from many thousands of sensory receptors contributes to the control of mammalian movement and to kinaesthesia. The nervous system is able to modulate the sensitivity of the received sensory information at synapses within the CNS, and in the case of muscle spindles, within the receptor itself, by activating intrafusal muscle fibres via fusimotor neurons. In voluntary movements, fusimotor action seems to have at least two components, one proportional to the activity of  $\alpha$ -motoneurons and the other being in some way related to motor task or predicted movement. The details of this second component are still disputed. In the author's view, spindle afferents are predominantly muscle length and velocity detectors whose sensitivity and bias are adjusted by fusimotor action according to the demands of the motor task. A component of fusimotor action linked to muscle contraction helps maintain spindle afferent signalling during muscle shortening and subsequent lengthening. Stretch reflexes mediated by muscle spindles and Golgi tendon organs contribute to load compensation and to higher level control, as exemplified by the switching between phases of a cyclical movement such as locomotion. In this article we have discussed the extent to which these biological mechanisms have been mimicked in neuroprosthetic devices. The main obstacle has been the enormous mismatch between the numbers of sensory receptors in animals compared with the handful of artificial sensors that can be attached to, or implanted in, the body in devices robust and convenient enough to be used by people in activities of daily life. Nonetheless, some analogous feedback control mechanisms do exist. They have provided insights into the way nervous systems have solved the problem of movement control. As technology advances it will be possible to deploy more sensors and this is likely to lead to neuroprostheses that are increasingly biomimetic. A clear understanding of the neurophysiology of motor control, and in particular the sensory receptors Robert Banks has studied in such detail, will then become increasingly important.

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