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Posture and Gait in Patients with Diabetic Distal Symmetrical Polyneuropathy

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equilibrium point observed in these experiments are generated through the same interneurons that are used during natural movements to obtain the same limb position. A given pattern of motoneuron activation may be achieved through several different pathways, which may or may not utilize common interneurons (a limb flexion, for example, may be produced by stimulation of cutaneous afferents, FRAs [flexor reflex afferents], the spinal locomotor center, or the corticospinal tract). Moreover, a given equilibrium point may result from different sets of forces, obtained, for example, by substituting for one another the various synergists or single-joint with multijoint muscles.

In fact, if a point of equilibrium is reached even after a blind electrical stimulation of the cord, should one not suspect that this depends only on the biomechanical characteristics of muscles and bones?

2. More on group II pathways. To aid in David McCrea's effort to make sense of spinal circuits, I would like to provide some information and add some comments on the systems that process group II information.

Group II connections have a wide distribution in the spinal cord but interneurons that mediate group II effects can be divided into functional subunits characterized by convergence from a limited number of muscles acting at different joints. L4 interneurons are known to receive group II afferents from leg extensors (sartorius and quadriceps) and from pretibial flexors (tibialis anterior and flexor digitorum longus). Other interneurons, localized in L6-S1 segments, show a convergence from ankle extensors (such as gastrocnemius-soleus and plantaris) and from more proximal muscles (such as quadriceps and gracilis). In both cases, however, group II afferents originate from muscles that are not strictly synergists but that can be simultaneously stretched during different motor acts. For example, during a flexion of the knee, L4 interneurons are excited only by quadriceps afferents. When flexion of the knee is accompanied by an extension of the foot (plantar flexion) there will be a summation of the effects from quadriceps and tibialis anterior.

On the other hand, L4 interneurons have multiple axonal projections which connect motoneurons of muscles that are different from those that contribute to the afferent input. The functional significance of this result is still unclear, but it suggests that muscle groups that are not directly engaged in the movement receive information about the length of the muscles that are stretched simultaneously. It is quite apparent that the complex scheme of convergence on and divergence from these interneurons adds to (and completes) the more stereotyped distribution of group I afferents. This would probably subserve and regulate the contraction of multiple muscles during complex movements. Recent results obtained in collaboration with Lars-Gunnar Pettersson (Cavallari & Pettersson 1991) in spinal cats indicate that excitatory group II effects from quadriceps and sartorius to posterior biceps-semitendinosus and gastrocnemius-soleus can be relayed by interneurons located in L7 (the same segment of the target motoneurons) and/or by midlumbar (L4-L5) interneurons, which in turn project to hindlimb motoneurons.

In different animals, each of the "parallel" pathways may contribute in different proportions to the final effect on motoneurons, indicating that the two systems may undergo independent control. Moreover, in the acute low-spinal state a group of interneurons located in the rostral lumbar segments (L2-L3) is tonically active and exerts an inhibitory action on L4-L5 as well as on L7 group II interneurons (Cavallari & Pettersson 1989).

Spinal tonic control acting on both group II and group III reflex pathways could also be of importance in regulating the access to the "alternative pathways" (see McCrea's section 8) since it has been shown that it does not act with the same strength on all interneurons. In the same preparation the transmission of the effects from a certain muscle can be privileged. The modulation of the tonic inhibition therefore seems to be crucial in regulating and distributing reflex discharges toward some of the possible pathways.

Posture and gait in diabetic distal symmetrical polyneuropathy

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[SCG] From a clinical perspective, it is tempting to consider the title of the target article by **Gandevia & Burke** to be somewhat "tongue in cheek." There are very few clinicians who treat patients with disturbances of the peripheral nervous system who would even consider the issue open to doubt. If the question were asked at all, it might be posed as "Under what circumstances are kinesthetic inputs most important in the control of natural movement?" But clinical observation lacks scientific rigor, and despite the minimal role ascribed to proprioceptive feedback by the proponents of "muscle stiffness" (Bizzi et al. 1984), the authors provide us with a comprehensive and compelling argument in favor of a significant role for kinesthetic information in the control of upper limb movements.

In reviewing the studies of movement in deafferented humans, **Gandevia & Burke** report chiefly on the work of Sanes et al. (1985), who studied upper extremity movements in seven patients with subacute onset of idiopathic, predominantly sensory, neuropathy. Three other similar patients were studied by Forget and Lamarre (1987), and case studies of similar single individuals are also available (Cole 1986; Cole et al. 1986; Rothwell et al. 1982a). Whereas diabetes mellitus can cause sensory neuropathy in the lower extremity, the study of lower extremity movement in diabetic patients would seem an obvious supplement to the studies described above. Except for the five patients with diabetes mellitus studied during standing by Ojala et al. (1985), we have been unable to locate other studies on patients with partial deafferentation as a result of diabetes.

In fact, the main topics of interest in our own laboratory are the various consequences of diabetic distal symmetrical polyneuropathy (DDSP) – the typical stocking and glove distribution of sensory loss found in many patients with diabetes mellitus (Dyck & Brown 1987). This research began as a response to clinical needs, since we see many patients with foot problems (such as plantar ulceration), most of which are neuropathic in origin (Cavanagh & Ulbrecht 1991). More recently, in working with such patients, we have been struck by their complaints of lower extremity dysfunction during activities of daily life and we have come to believe that the diabetic neuropathic model has been overlooked as far as its potential for providing insight into motor control is concerned. Approximately 20%–50% of individuals who have had diabetes for at least 10 years experience some degree of peripheral neuropathy (Greene et al. 1990; Pirart 1978). This means that there is a pool of between 1 and 2.5 million individuals available for study in the United States, compared to the rather small and unique group of individuals with idiopathic deafferentation used in the studies mentioned above.

The study of patients with DDSP is not immune to the problems mentioned by **Gandevia & Burke**, however. In particular, the disease is variable in its staging and, as its name suggests, it may affect all divisions of the nervous system – sensory, motor, and autonomic (Dyck & Brown 1987; Dyck et al. 1987a; Greene et al. 1990). Yet it is the sensory manifestations that are predominant in most patients, and recent advances in quantitative sensory testing (Dyck et al. 1987b) allow a reasonable quantitative profile of functional deficits to be obtained

(Dyck et al. 1987b), while motor and autonomic deficits can also be determined (Edwards et al. 1984; Low 1984). Thus, a relatively homogenous sample with well-defined sensory deficits and normal strength can be formed by careful selection and screening.

Because the onset of DDSP is generally gradual and the deafferentation incomplete, this model will necessarily underestimate the role of afferent information in motor control – since patients may have learned to adapt to their disability (cf. Wolf et al. 1989) and since there will generally be some degree of residual sensory information. These two factors should not be seen as essentially limiting, however, especially in light of the availability of potential patients.

We have recently presented data on the performance during standing and walking of a carefully screened group of patients with significant DDSP, an age-matched control group of diabetic individuals without significant DDSP, and a further age-matched nondiabetic control group (Cavanagh et al. 1992; Simoneau 1992; Simoneau et al. 1992). Muscular weakness and a variety of medications were excluded and sensory deficit was quantified by vibration perception threshold, monofilament testing, and ankle movement perception threshold. It should be pointed out that these modalities are all principally served by large fiber afferents and we have no information on small fiber status in these patients.

Our findings highlighted the role of somatosensory input during standing, even in the presence of optimal visual and vestibular inputs. The mean sway (measured from a force platform as total movement of the center of pressure) in the neuropathic group with eyes open looking straight ahead was approximately the same as that measured in the nonneuropathic and control groups while standing in the eyes closed, head back position. Thus, during standing, vision and vestibular inputs were apparently not able to compensate for the loss of somatosensory input.

Gandevia & Burke predict that "deficits in timing of muscle bursts in highly stereotyped tasks such as . . . locomotion will probably also be revealed" (sect. 2.2, para. 6) and although "sensory ataxia" during gait is widely described in the anecdotal literature (Thomas & Brown 1987), we have not been able to locate any other quantitative studies of gait in individuals with sensory deficits. Our findings during gait (based on automated video analysis of treadmill walking) showed minimal (and statistically insignificant) differences between the same groups used above for the posture experiments. This initial study was confined to an examination of variability in sagittal plane kinematic patterns and we intend to extend it to look at frontal planar movements that have been reported anecdotally to be most affected by neuropathy as well as to perturbations during gait, which might be expected to elicit somewhat blunted responses. Nevertheless, the contrast between the marked decrements observed in neuropathic patients during standing and the minimal changes during gait suggest that somatosensory input is critical in the former task and either less important or redundant (perhaps based on learning) in the second. It is tempting to see a dominance of efference over afference in gait in these results. However, the role played by learning during the many thousands of repetitions of the stride pattern that occurred during conditions of declining sensory information (as the natural history of the disease progressed) needs to be further clarified.

There may be some valid debate over whether or not standing posture should be classified as a natural movement – since the movements that occur are small and not so clearly goal directed as in grasping or walking. Nevertheless, the results have indicated that somatosensory input is much more important in postural control than was previously supposed and that there may be good reason to revisit the patient with diabetic distal symmetrical polyneuropathy in the pursuit of further insight into the motor control of human movement.

How accurately can we perceive the positions of our limbs?

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[SCG] In their target article, **Gandevia & Burke** nicely catalog and describe the many sources of sensory signals that might contribute to kinesthesia and motor control – and there does indeed appear to be ample information available to signal the position and movement of the limbs. With all this kinesthetic information at our disposal, how accurately can we perceive the positions of our limbs? This is the issue I will address in this commentary.

In addressing questions of accuracy, the appropriate measure depends upon the question one is asking. Of the many options available, the commonly used measure of the mean disparity between the perceived position of a limb or joint and the true or target position might seem the best metric. The standard deviation about the mean would, in addition, provide a measure of reliability or precision. In some cases, this mean error might well be the measure of greatest importance. For example, a hunter with shaky hands who shot wildly but whose "average shot" coincided with the target would bag the prize at least once in a while whereas a hunter who shot with great precision but who was consistently off the target might never get the prize. (The latter hunter might do better were he less precise.)

However heuristically useful average measure of error might prove, do real-life motor tasks involve such averages? For the motor control system to program a movement it might do better to know the position of a limb or joint "right now" rather than where it sits "on average." "Where is the joint right now?" and "Where is the joint on the average?" are two different questions and can yield two surprisingly different answers.

Information theory provides a useful way of addressing the question of "where the joint is right now." It provides a measure of the number of bits of information a subject can derive from a unidimensional stimulus array (stimuli that vary along a single dimension such as intensity, frequency, or joint angle). One can also measure a "channel capacity" that can produce a good estimate of the maximum number of stimulus categories, or in our context, the number of different positions over a range that a subject can identify without error (Garner 1962, pp. 74–75). Increasing the number of target positions in the range (stimulus categories) in excess of the maximum indicated by the channel capacity would not increase the number of targets the subject could resolve but would only cause additional errors, with no net increase in information transfer (Hake & Garner 1951).

The measure of information transfer is most closely aligned with variance (Garner & McGill 1956; Miller 1956, p. 81). The noisier the channel, the greater the variability in the responses, the less information that the channel can carry and the fewer the number of stimulus levels that can be resolved without error. Though information theory and the concept of channel capacity existed before the 1950s, they have been used only occasionally in the study of kinesthesia (Durlach et al. 1989; Georgopoulos & Massey 1988; Sakitt et al. 1983; Soechting & Flanders 1989). It is of interest to note that psychophysical studies over the years indicate an upper limit in channel capacity for human sensory systems of seven plus or minus two stimulus categories (for unidimensional stimuli) irrespective of sensory modality, stim-