Force-feedback during human walking

To continue to the correspondence of Duysens[1] to my recent letter to the editor[2], I should like to reply further. Duysens[1] ended his letter by stating '…other human studies are controversial', by which he means work other than that on force-feedback. This is true and good for the progress of science. However, the situation with force-feedback during human walking is different. In this case there are no controversies, all the studies that have tested the hypothesis find the same thing: little, if anything. This work, carried out by several capable physiologists using a variety of approaches, is summarized in my letter[2]. In addition, my original letter stated that even in the intact cat the effect (i.e. Ib-afferent input becoming excitatory during the stance phase of walking) is much weaker and often absent. Duysens retrenches by stating that it is not that the phenomenon does not exist, but instead that it is difficult to demonstrate, a position similar to that of supporters of cold-fusion.

The argument based on physiology made by Duysens[1] in his reply is that supramaximal stimulation of a mixed nerve (i.e. the tibial nerve) would be '…much more than selective activation of Ib afferents'[1]. Here we are forced to believe that whatever other afferents were activated, their reflex effects exactly counteracted the alleged Ib effect. If indeed 'much more' than Ib afferents were stimulated, we should have obtained some effect. The most obvious of which would have been a flexion reflex and a consequent shortening of the stance phase. However, a flexion reflex was never observed, nor was the duration of the stance phase or the onset of the swing phase changed. In addition to these arguments support is also provided by the following experimental observation. If, instead of stimulating the tibial nerve supramaximally, it is stimulated at group I strength, the stance phase is not prolonged nor is the swing phase delayed. In the example shown in Fig. 1 the tibial nerve was stimulated at an intensity that produces a substantial H-reflex with no corresponding M-wave. The stimulus frequency was 12.5 Hz, delivered starting at mid-stance and continued until the end of stance. Each stimulus produces a large H-reflex, which would in turn produce a large twitch force. At a stimulus frequency of 12.5 Hz these twitches would sum to a nearly fused tetanus and thus result in a substantial increase of force in the ankle extensors. The key point is that neither the duration of the stance phase is prolonged, nor is the onset of the swing phase delayed. There are two conclusions to be drawn from this experimental result. First, the threshold of the force-feedback pathway, if it exists, must be high. Second, it is clear that during normal human walking the transition from stance to swing is not governed as stated by the force-feedback hypothesis. Humans are not subject to strong and rigid reflex controls. On the contrary, reflexes are modulated by the CNS in a task-dependent manner (see, for example, Ref. 4). Finally, my purpose in writing the original letter[2] was not to 'construct an integrated view' as Duysens[1] claims, but simply, as I stated[2], to complement what Clarac *et al.*[3] had presented in their review article.

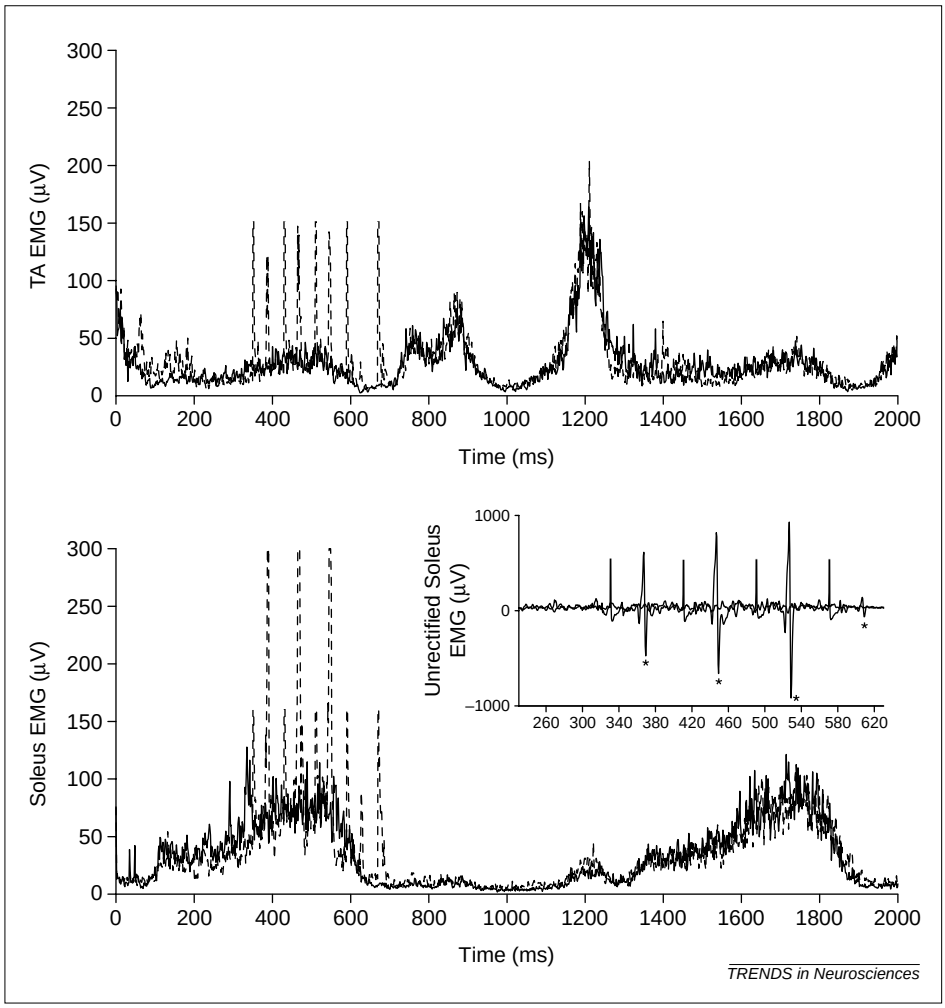

Fig. 1. Example of the effects of a stimulus train (12.5 Hz) delivered at group I strength during the stance phase of human walking. The electromyographic (EMG) activity of the soleus and tibialis anterior (TA) during human walking are shown during a little less than two step cycles. The duration of a single step cycle is approximately 1200 ms. The solid traces represent the control-step cycle (average of 16 step cycles). The broken traces represent trials in which the stimulus train was delivered (average of 16 trials). Each stimulus elicits a large H-reflex that can be seen more clearly in the inset graph (marked by asteriks) at the bottom of the figure. Note that control and stimulated trials superimpose almost exactly, demonstrating that the stimulus train does not change the timing of the step cycle.

Charles Capaday
Dept of Anatomy and Physiology, Faculty of Medicine, Université Laval, Québec City, Québec, Canada.

References

1 Duysens, J. (2000) Reply. *Trends Neurosci.* 23, 529–530
2 Capaday, C. (2000) Control of a 'simple' stretch reflex in humans. *Trends Neurosci.* 23, 528–529
3 Clarac, F. *et al.* (2000) Central control components of a 'simple' stretch reflex. *Trends Neurosci.* 23, 199–208
4 Schneider, C. *et al.* (2000) On the origin of the soleus H-reflex modulation pattern during human walking and its task-dependent differences. *J. Neurophysiol.* 83, 2881–2890

  PII: S0166-2236(00)01709-4