Control of a 'simple' stretch reflex in humans

In their recent review article on control of the crayfish 'stretch reflex' during 'walking' Clarac *et al.*[1] concluded that it would be important to understand '...how sensory–motor pathways operate in a real behavioral context'. Indeed this has been done during human standing, walking and running[2–9] and some of this work was reviewed in this journal more than a decade ago[10]. Clarac *et al.*[1] did not mention this body of work in their article. In fact, the authors have done a nice job comparing control of the 'stretch reflex' in several walking invertebrates, as well as a comparison with results in decerebrate cats. A brief comparison with results in human subjects would have provided a nicely integrated view of the subject. My purpose, therefore, is to complete the picture by succinctly summarizing some of the relevant work on modulation of the 'stretch reflex' in human subjects and point to some differences with the crayfish and decerebrate cat.

The amplitude of the H-reflex (analog of the stretch reflex elicited by electrical nerve stimulation) of the ankle extensor, soleus, is strongly modulated during human walking[2,11] and on the spot stepping[4]. Initially, Morin *et al.*[12] showed that the soleus H-reflex is lower during the early part of the stance phase of walking compared to a matched voluntary contraction of the soleus muscle. Later and independently Capaday and Stein[2] investigated the whole of the walking cycle as did Crena and Frigo[4] for on the spot stepping. Both groups showed that the soleus H-reflex increases during the stance phase in a ramp-like fashion in parallel with the soleus EMG, and is strongly suppressed, or completely inhibited, during the swing phase when the ankle flexor tibialis anterior (TA) is active (see details in Ref. 13). The stretch reflex contributes a substantial portion of the motor output during both walking[5] and running[7] (see Refs 14,15 for a detailed examination of the problem). The following neural mechanisms contribute to the modulation of the soleus H-reflex during the normal step cycle: increasing activity of the α-motoneurons during the stance phase[2]; the increase of postsynaptic inhibition of the α-motoneurons during the swing phase[13,16] and, as previously suggested[8,12,17], a tonic increase of presynaptic inhibition of the group Ia afferent terminals projecting to the α-motoneurons. The relation between the amplitude of the soleus H-reflex and the background level of motor activity is not fixed, instead it depends strongly on the motor task[2,8,12,18,19]. For example, the y-intercept of the relation between the H-reflex amplitude and the background level of motor activity is much higher during standing than during walking, whereas the slope is smaller[2]. The H-reflex also decreases during running in comparison with walking[8,9,19]. During the difficult task of walking on a narrow beam, the soleus H-reflex is reduced compared with walking on a normal surface[18]. The task-dependent changes of the input–output properties of this neural circuit have been shown to be of central origin[20,21]. They have been suggested to reflect adaptive control of the stretch reflex parameters in accordance with the biomechanical exigencies of the motor task[10,12,22]. On the basis of computer simulations[17] (see also Ref. 23) and experimental studies in decerebrate cats[24], it was suggested that the task-dependent changes of the input–output properties of this neural circuit were the result of changes of presynaptic inhibition of the Ia-afferents. The direct demonstration of task-dependent changes of presynaptic inhibition during posture and gait has been more difficult[25,26] and has yet to be achieved. In these studies, peripheral nerves such as the femoral nerve, or the common peroneal nerve, were used as inputs to test the state of the presynaptic inhibitory network in the spinal cord. The result is that these conditioning inputs produced little, if any, inhibition during walking compared with standing. There are three possible reasons for this result[25]: (1) the presynaptic inhibitory network is maximally activated during walking; (2) there is occlusion at the interneurons between the afferent volley and the central drive to the interneurons and (3) the conditioning afferent terminals are themselves presynaptically inhibited during walking.

Thus, presynaptic inhibition of afferent terminals might be a fundamental and phylogenetically ancient mechanism for the control of 'stretch reflexes'. However, one important difference between the human and the crayfish 'stretch reflex' stands out. In humans there is no reversal of the stretch reflex, but rather a near complete inhibition during the swing phase, when stretch of the powerful ankle extensors would oppose the intended ankle dorsiflexion. In other words, stretch of the ankle extensors during the swing phase does not assist ankle flexion. The ankle extensor stretch reflex is simply inhibited so as not to oppose dorsiflexion during swing[2,13]. There is also an apparent paradox between what has been shown to occur for the group Ib pathway in the decerebrate cat (i.e. a reversal from inhibition at 'rest' to excitation during locomotion) and the decreased synaptic efficacy between group Ia afferents and the α-motoneurons during human walking. The reader might ask, why would the ankle extensor stretch reflex decrease during the stance phase of human walking, while, at the same time, the group Ib reflex pathway switches from inhibition to excitation? There is no evidence for a reversal of the group Ib effects from autogenic inhibition at 'rest' to excitation during human walking at this time[27–29]. However, there is evidence that loading during the stance phase of infant stepping increases the amplitude and duration of stance[30]. Experiments in our laboratory on adults have shown that when the ankle extensor force during stance is artificially increased by a train of supramaximal nerve stimuli (and consequently, an increased extensor thrust is recorded by the force platform), the duration of the extensor activity is not prolonged and the onset of the TA activity in swing occurs at the expected time (Schneider *et al.* unpublished observation). Either Ib excitation of the α-motoneurons does not occur during human walking, or this mechanism is superceded by other biomechanical requirements of the erect bipedal and plantigrade human gait pattern[31,32]. In fact, in intact conscious cats Ib-excitation during the stance phase of walking is weaker than in the decerebrate[33]. Thus, Clarac *et al.*[1] are right to conclude their article by stating that '...in vertebrates and in mammals in particular, cephalization has resulted in increased control of local circuits by superior structures, and thus probably masks such intrinsic adjustments'. Indeed, the role of the motor cortex during human walking has begun to be studied[31,34,35]. Therefore, it might be possible to understand the function of at least one descending tract (the corticospinal tract) during human walking and its control of reflex circuits.

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Reply

It is true that our recent review[1] of the stretch reflex was mainly devoted to invertebrate models with some references to vertebrate models. We aimed to present the state of our knowledge of the cellular organization of the stretch reflex and its modulations (which are better known in invertebrate preparations). For example, although a lot of work has been done in cat primary afferent (and was mentioned in the review) the presynaptic mechanisms can be studied in 'simple' invertebrate systems, at the level of their ionic components in identified neurons. Similarly, the implication of the motoneurons in the modulation of the stretch reflex has been well characterized in invertebrate preparations. The existence of active electrical membrane properties in motoneurons is largely responsible for the observed changes in the efficacy of the stretch reflex. Such active properties have been demonstrated in motoneurons of insects and crustaceans, but also in some vertebrates, such as the turtle (see review by Kiehn and Eken[2]).

We agree that modulation of the stretch reflex is also present in human (H-Reflex). However, the mechanisms involved are difficult to study. Using nice protocols, such as the one developed by the group of Pierrot-Desseilligny[3], it is possible to estimate the respective roles of presynaptic inhibition and motoneuron excitability in the observed modulation of the stretch reflex. However, the results obtained with such methods remain indirect and do not allow any insight on the involved cellular mechanisms. This was the reason why we did not develop this area in our review. In addition, considering the limited space allowed in the journal, we restricted the presentation of animal models to those in which cellular mechanisms have been shown.

However, the complement made by Capaday[4], is certainly useful to extend the scope of our review to the case of humans.

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Reply

Capaday[4] argues that a comparison with humans would have been provided a 'nicely integrated view'. This is true but not relevant because the scope of this article was not a comprehensive one and in fact we recently published a large comparative review on this topic (Duysens *et al.*[5]). This review makes an extensive comparison with human work, including work on the reversal on the Ib inhibition to excitation. In addition, this review makes a clear link between the original observation on this topic in the cat[6] and the recent work in humans. The view expressed in the letter of Capaday differs in several aspects from our view. This is because of a difference in interpretation of the data. For example, the works mentioned by Capaday in his laboratory as well as those of Schneider *et al.*[7] are taken as evidence against the presence of Ib excitation in the stance phase of humans. These experiments are based on strong electrical stimulation