



Effect of sub-optimal neuromotor control on the hip joint load during level walking

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ABSTRACT

Skeletal forces are fundamental information in predicting the risk of bone fracture. The neuromotor control system can drive muscle forces with various task- and health-dependent strategies but current modelling techniques provide a single optimal solution of the muscle load sharing problem. The aim of the present work was to study the variability of the hip load magnitude due to sub-optimal neuromotor control strategies using a subject-specific musculoskeletal model. The model was generated from computed tomography (CT) and dissection data from a single cadaver. Gait kinematics, ground forces and electromyographic (EMG) signals were recorded on a body-matched volunteer. Model results were validated by comparing the traditional optimisation solution with the published hip load measurements and the recorded EMG signals. The solution space of the instantaneous equilibrium problem during the first hip load peak resulted in 10^5 dynamically equivalent configurations of the neuromotor control. The hip load magnitude was computed and expressed in multiples of the body weight (BW). Sensitivity of the hip load boundaries to the uncertainty on the muscle tetanic stress (TMS) was also addressed. The optimal neuromotor control induced a hip load magnitude of 3.3 BW. Sub-optimal neuromotor controls induced a hip load magnitude up to 8.93 BW. Reducing TMS from the maximum to the minimum the lower boundary of the hip load magnitude varied moderately whereas the upper boundary varied considerably from 4.26 to 8.93 BW. Further studies are necessary to assess how far the neuromotor control can degrade from the optimal activation pattern and to understand which sub-optimal controls are clinically plausible. However we can consider the possibility that sub-optimal activations of the muscular system play a role in spontaneous fractures not associated with falls.

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1. Introduction

Load cells and force platforms make possible accurate measurement of the external forces that act on our body during activities of daily living (Sutherland, 2005). However, the determination of the internal forces that the same physical activity induces on our skeleton through the joints, the ligaments, and the muscle insertions remain difficult to quantify. But this information is of vital importance in a number of research and clinical contexts. For example, the risk of fracture that a given subject faces while performing a given motor task depends not only on the specific bone strength, but also on the internal forces.

The problem is affected by a dramatic indeterminacy. Even if we model the skeleton as a mechanism made of idealised joints, represent each major muscle bundle with a single actuator, and impose all the physiological limits to the force expressed by each actuator, the resulting mathematical problem has more unknowns than equations. The best solution, when the kinematics of each segment has been measured experimentally, is to postulate that the neuromotor control activates the muscle fibres ensuring the instantaneous equilibrium while minimising a cost function (Collins, 1995; Menegaldo et al., 2006; Praagman et al., 2006).

The assumption that in healthy subjects the neuromotor control works in fairly optimal conditions seems reasonable. Indeed, when applied to volunteers this approach predicts muscle activation patterns in good agreement with electromyography (EMG) recordings (Anderson and Pandey, 2001; Erdemir et al., 2007; Heller et al., 2001). Also, the intensity of the hip load predicted is comparable to that recorded with telemetric instrumented prostheses (Heller et al., 2001; Stansfield et al., 2003).

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