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Review

Secondary gait deviations in patients with and without neurological involvement: A systematic review

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ABSTRACT

Pathologies that lead to biomechanical restrictions in human gait interfere with the tightly regulated muscle activation patterns that control the external moments. In order to maintain proper function, secondary mechanisms are required. The aims of this systematic review were (1) to identify secondary mechanisms in pathologic gait that have been described throughout the scientific literature by means of instrumented gait analysis, (2) to distinguish between active compensatory mechanisms and passive physical effects and (3) to identify common compensatory mechanisms that appear to be independent from the underlying disease. A comprehensive literature search revealed 4080 citations for review, whereof 148 studies entered the full-text review. Thirty-six studies were included and the quality of these studies was assessed by two independent reviewers (kappa = 0.83). The quality of the included studies showed large variation and several methodological issues were identified. Five studies were further identified describing only passive physical effects, leaving a total of 31 studies reporting on compensations. The qualitative analysis revealed common compensations that appeared to be independent from the underlying pathology. In clinical practice, distinguishing primary from secondary gait deviations can be considered highly important since unnecessary treatment may be avoided. However, given the introduction of general principles of compensatory mechanisms and the fact that certain presumed "compensations" were identified as simple passive physical effects, secondary gait deviations have to be further investigated. Computer simulation studies are valuable, especially in respect of the distinction between compensations and physical effects. Furthermore, the need for a uniform terminology was highlighted.

1. Introduction

Healthy human gait uses repetitive reciprocal limb motions in order to advance the body while simultaneously maintaining stance stability [1]. This is achieved by tightly regulated patterns of muscle activations and generated joint moments and powers. Part of the muscle work is done for acceleration and promotion, part is used to control external moments resulting from gravity and inertia. The appropriate use of these external moments is a major factor in the efficient management of human gait energy. Pathologies that lead to deformities (e.g. joint contractures or bone deformities), muscle weakness, sensory loss, impaired motor control or pain interfere with these tightly regulated patterns and hence, active compensatory strategies might be required in order to maintain proper function.

In addition, as the human body underlies to the laws of general physics, further passive segmental movements can follow from a primary deviation as a consequent physical effect. These passive physical effects, however, are often mistaken for active compensatory strategies. Only recently, researchers started to distinguish between those two kinds of secondary deviations [2,3]. Brunner et al. [2], for example identified pelvic retraction and hip flexion in children with cerebral palsy (CP) as a passive physical effect of plantarflexor push under load, implying that there was no active compensatory strategy involved as it has been suggested by previous research [4,5]. Physical effects can be considered passive secondary deviations. They result from gravity acting on the body while moving one or more segments, evoked simply by the anatomical coupling of segments. Hence, every biomechanical constraint following from a primary pathology implicates physical effects. In many cases, however, physical effects cannot be

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identified since the subjects might actively modify or hide them by exerting compensatory strategies in order to enable locomotion. Further (tertiary) deviations can occur as effects on secondary ones, but can be classified again like the secondary ones.

For all these reasons, gait deviations can be divided in principle into the following categories: (1) primary deviations that are directly due to the pathology; and (2) secondary deviations which split into (a) *passive* secondary effects that follow as a physical effect to the primary deviation; and (b) *active* secondary deviations (i.e. compensatory mechanisms/compensations) that act in order to actively offset primary deviations and secondary physical effects. A similar distinction was adopted by several authors investigating gait adaptations due to foot deformities [6–9].

Distinguishing secondary from primary gait deviations is critical in clinical practice, e.g. when planning orthopedic interventions or physical therapy treatment. If the causes of gait abnormalities are identified incorrectly, unnecessary and/or ineffective treatment may be carried out [9]. As a consequence, primary abnormalities will be corrected when treated directly whereas secondary problems may resolve spontaneously once the primary issue is addressed [7,9].

There are several different approaches to identify secondary problems. Most commonly, a pathologic gait pattern is compared to a healthy one and interpreted accordingly. However, this method does neither allow a clear distinction between primary and secondary deviations nor further distinctions between physical effects and compensatory mechanisms. It can only be speculated about the origin of the deviations. Other more reliable methods include comparisons to an additional condition. Stebbins et al. [9], for example assessed 12 children with CP prior to, and following surgery to correct foot deformity, along with a sample of healthy controls. This allowed the investigators to discriminate between the deviations that resolved spontaneously (secondary) and the deviations that persisted (further primary deviations). In another study, Romkes et al. [10], let healthy control subjects mimic the gait of given hemiplegic CP patients. Thereby, the investigators were able to distinguish between primary deviations in muscle activity as a direct consequence of the underlying neurological pathology and deviations due to the biomechanics of toe-walking (i.e. secondary deviations, demonstrated by both the patients and the healthy mimicking subjects). Further methods include in vivo simulations of primary deviations, e.g. simulated restriction of joint range of motion [7], simulated shortening of the hamstrings [11] or simulated leg length discrepancy [12] as well as computer simulations [13–15].

In vivo simulations allow researchers to better distinguish between primary and secondary deviations, since the primary pathology is artificially induced. However, in vivo simulations do not involve subjects with real pathologies and therefore, data should be interpreted cautiously. The dynamic models used in computer simulation studies, on the other side, might be "fed" with real patient data and allow the researchers to make distinctions between primary and secondary deviations as well as between compensatory mechanisms and physical effects. The disadvantage with this method is the input bias, i.e. the models are "fed" with the data that is thought to be required, but necessary parameters such as rules of adaptations at a longer term are not known. In addition, computer simulations are dependent on the quantitative characteristics of human abnormal walking that have not yet been collected enough to be described as dynamic models [16]. Nevertheless, the possibility of modifying one single parameter such as plantarflexion activity [2] could help decode the complexity of secondary deviations. Simulation studies should therefore be used in order to support the interpretations that are based on the studies involving real patient groups and a control group.

In the literature, deviations in proximal joints following from or compensating for constraints in distal joints and vice versa can be found. Davids et al. [6] and Stebbins et al. [9] for example identified abnormal pelvic transverse plane motion (pelvic rotation) and diminished hip extension during stance as secondary deviations of toe-walking in children with CP. Brunner and Romkes [17] and Matjacic et al. [18] reported plantarflexor hyperactivity during stance phase, which compensated for weak knee extensors in order to provide stance stability in patients with several different orthopedic pathologies.

When investigating gait compensations, it appears that the term "compensation" is usually linked to a specific pathological condition. However, in clinical practice very similar movement patterns can be seen in a variety of underlying disorders, questioning the principle that abnormal muscle activity is the direct result of a neurological disorder [17]. By investigating a group of orthopedic patients suffering from different orthopedic conditions, Brunner and Romkes [17] found two distinctive patterns of compensatory muscle activity, which were independent from the affected joint level, respectively the underlying pathology. They further concluded that these mechanisms corresponded to certain deviations observed in central nervous system (CNS) disorders and that CNS-patients probably do not compensate differently but may be using the same adaptations for muscle weakness as orthopedic patients or any human.

In the literature, the terms "compensation/to compensate" are widely overused and confusion can occur on whether the gait deviation is primary or secondary, respectively passive or active. Thereby, only a sparse amount of studies are concerned with the distinction between physical effects and compensatory mechanisms. Further, it is assumed that there might be general principles of compensation, i.e. compensations that are not directly related to a specific pathology. For treatment planning, a better and more comprehensive knowledge on secondary gait deviations is crucial and therefore, the purpose of the current systematic review was threefold: (1) to identify secondary gait deviations that have been described throughout the literature over the past three decades by means of marker-based three-dimensional gait analysis and involving a control group; (2) to distinguish between physical effects and compensatory mechanisms according to the currently available literature; and (3) to identify common secondary gait deviations that occur across different pathologies and therefore appear to be independent from the underlying disease.

2. Methods

2.1. Electronic database search

In order to provide a comprehensive overview on gait compensations, an electronic literature search was conducted within the databases MEDLINE, CINAHL, EMBASE, BIOSIS Previews, INSPEC and Journal Citation Reports using the search services Ovid, EbscoHost, EMBASE and ISI Web of Knowledge for the time period of January 1980–October 2011.

The search strategy targeted the categories title, abstract and keywords and included the following search terms: *gait, walking, locomotion, compensation, adaptation, deviation, variation, alteration, changes, characteristics, strategy, mechanism, effect, pattern, function, movement, kinematics, motion analysis, gait analysis, motion capture, simulation, model, lower limb, lower extremity, lower body, leg, foot, feet, ankle, knee, hip, pelvis, upper limb, arm, thorax, upper body, upper extremity, joint, human, adult, adolescent, child, elder, patient, subject, woman, man, kid, girl, boy*. Wildcard symbols were used to retrieve all possible suffix variations of the root words. The search was not restricted to specific languages.

2.2. Inclusion/exclusion criteria and screening

Title and abstract of each study were screened and full texts were retrieved subsequently and evaluated for definitive inclusion if they met the inclusion criteria (Table 1). Based on the advantages and disadvantages of the methods commonly applied for the identification of secondary gait deviations and in order to ensure the comparability of the retrieved studies, they were divided in two categories: (1) studies involving a group of subjects with a pathologic gait pattern as well as a