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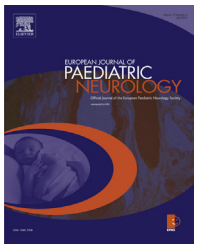

Original article

Whole-body vibration training in children with Duchenne muscular dystrophy and spinal muscular atrophy

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ABSTRACT

Introduction: Whole-body-vibration training is used to improve muscle strength and function and might therefore constitute a potential supportive therapy for neuromuscular diseases.

Objective: To evaluate safety of whole-body vibration training in ambulatory children with Duchenne muscular dystrophy (DMD) and spinal muscular atrophy (SMA).

Methods: 14 children with DMD and 8 with SMA underwent an 8-week vibration training programme on a Galileo MedM® at home (3 × 3 min twice a day, 5 days a week). Primary outcome was safety of the training, assessed clinically and by measuring serum creatine kinase levels. Secondary outcome was efficacy as measured by changes in time function tests, muscle strength and angular degree of dorsiflexion of the ankles.

Results: All children showed good clinical tolerance. In boys with DMD, creatine kinase increased by 56% after the first day of training and returned to baseline after 8 weeks of continuous whole-body vibration training. No changes in laboratory parameters were observed in children with SMA. Secondary outcomes showed mild, but not significant, improvements with the exception of the distance walked in the 6-min walking test in children with SMA, which rose from 371.3 m to 402.8 m ($p < 0.01$).

Interpretation: Whole-body vibration training is clinically well tolerated in children with DMD and SMA. The relevance of the temporary increase in creatine kinase in DMD during the first days of training is unclear, but it is not related to clinical symptoms or deterioration.

1. Introduction

Duchenne muscular dystrophy (DMD) and spinal muscular atrophy (SMA) are among the most common inherited neuromuscular diseases with onset in childhood, both leading to severe physical disability. The incidence of DMD is 1/5000,[1] that of SMA is 1/6000–1/10000.[2] DMD, an X-chromosomal-recessive disorder, is caused by mutations in the dystrophin

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gene that result in the absence of the cytoskeletal protein dystrophin. This leads to increased permeability of the sarcolemma made apparent by elevation of the serum creatine kinase (CK), a marker of muscle damage. However, the exact pathophysiologic mechanism leading to the increased membrane permeability and ultimately severe muscle degeneration in DMD is not fully understood.[3] SMA is caused by an autosomal-recessively inherited deletion on the *SMN1* gene which causes the alpha motor neurons in the spinal cord to degenerate and neurogenic muscular atrophy. CK levels are within the normal range or mildly elevated in patients with SMA. Despite their different aetiologies and pathophysiology, DMD and SMA have many clinical characteristics and treatment approaches in common. Main symptom of both disorders is progressive proximal muscular weakness, leading to impaired motor function with loss of ambulation, development of scoliosis and reduced pulmonary function.[4,5] Complications associated with immobilisation such as contractures and osteoporosis[6,7] contribute significantly to morbidity.

DMD and SMA remain incurable; thus the current treatment approaches, published in Standards of Care for both diseases,[8–10] are mainly symptomatic. Although there are no specific guidelines for physical activity in DMD, regular

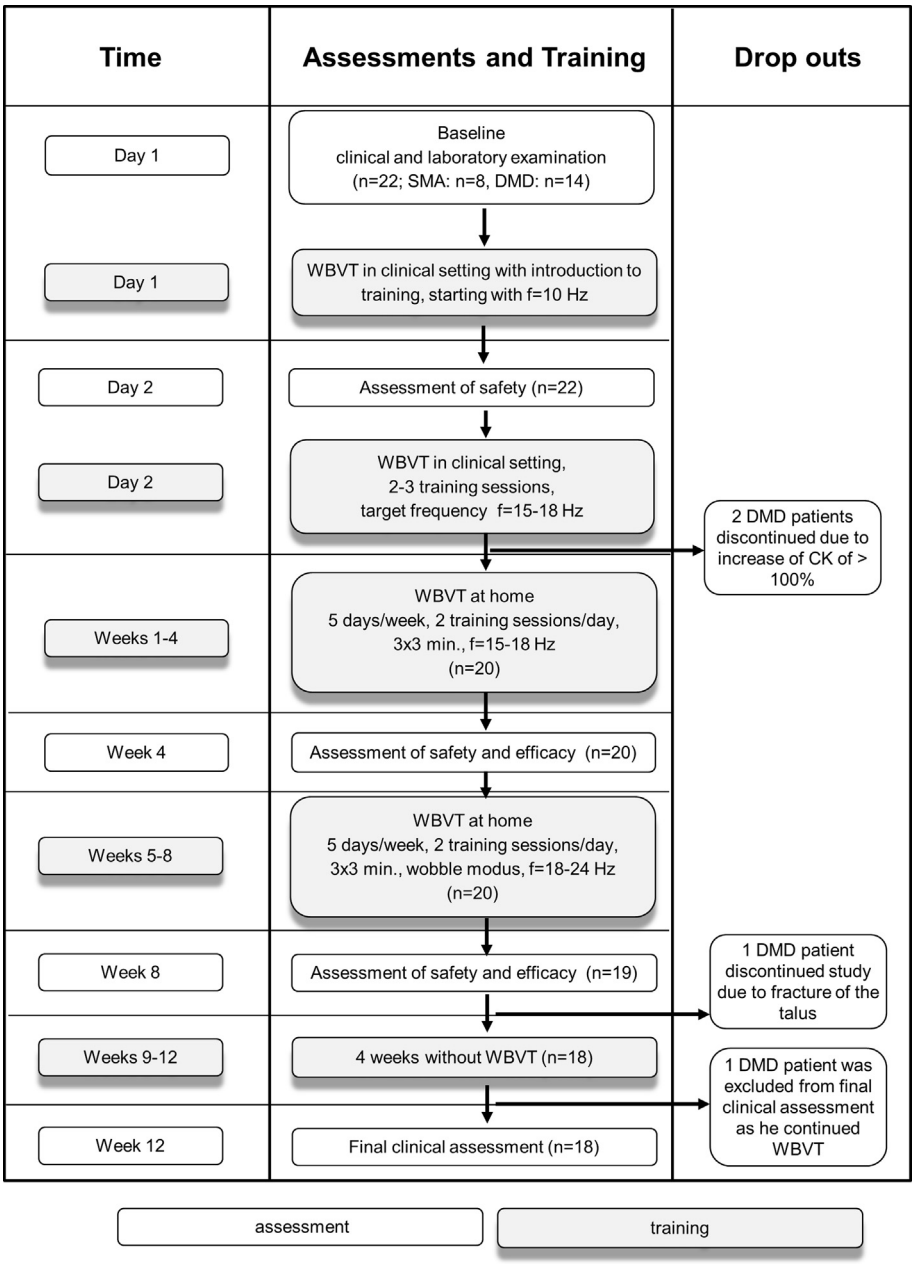

Fig. 1 – Study flow diagram for the complete study period (12 weeks) with the number of patients (*n*) participating. The training programme and the assessments are shown in the middle. The left column specifies the timing and the right column indicates the patients that did not complete the trial. SMA = Spinal muscular atrophy, DMD = Duchenne muscular dystrophy, WBVT = whole-body vibration training, f = vibration frequency, Hz = Hertz.