Table 1
Crosstalk occurrences in the SEMG signal detected from BR in three electrode configurations.

Task	Electrode diameter and interelectrode distance (IED)																											
	Ø = 18 mm, IED = 50 mm									Ø = 18 mm, IED = 25 mm									Ø = 2.5 mm, IED = 10 mm									
	1[a]	2[a]	3[a]	4[a]	5[a]	6[a]	7[a]	8[a]	TOT	1[a]	2[a]	3[a]	4[a]	5[a]	6[a]	7[a]	8[a]	TOT	1[a]	2[a]	3[a]	4[a]	5[a]	6[a]	7[a]	8[a]	TOT
Static wrist extension	1	1	1	1	1	1	1	–	7/7	1	0	–	1	1	1	1	–	5/8	0	0	0	0	0	0	0	0	0/8
Dynamic wrist extension	1	1	–	1	1	–	1	1	6/6	1	1	1	1	1	1	1	1	8/8	0	1	1	0	0	1	0	–	3/7
Static wrist flexion	0	1	1	0	1	1	0	–	4/7	0	0	0	0	1	1	1	0	3/8	0	0	0	0	0	0	0	0	0/8
Dynamic wrist extension	0	1	–	1	1	1	1	1	6/7	0	1	1	0	1	1	0	1	5/8	0	1	0	0	0	0	0	–	1/7
Dynamic finger extension	0	1	–	1	1	–	1	–	4/5	–	0	–	1	1	1	0	–	3/5	0	0	–	0	1	0	0	–	1/6
Total									27/32 (84%)									24/35(69%)									5/36(14%)

Value '1' represents presence of crosstalk indicated by a surface EMG signal greater than twice its noise level without fine wire signal; '0' represents absence of crosstalk indicated by a surface EMG signal lower than twice its noise level without fine wire signal; '–' represents no result regarding crosstalk due to inability of subject to perform task without activating BR.
[a] Subject #.

Reference

[1] Keenan MA, Fuller DA, Whyte J, Mayer N, Esquenazi A, Fidler-Sheppard R. Deformity. Arch Phys Med Rehabil 2003;84:291–6.

doi: 10.1016/j.gaitpost.2008.10.043

Early signs of pathological pattern evaluated by gait analysis in Duchenne muscular dystrophy patients

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Introduction: Duchenne Muscular Dystrophy (DMD) is an X-linked hereditary disease, caused by a gene mutation that affects dystrophin production. The absence of this protein causes a progressive degeneration of muscular fibres leading to a diffuse progressive hypostenia [1]. An early manifestation of the pathology may occur since 3 years old. In children without symptoms of the disease, clinical functional task assessment tools may not show an early compensatory strategy acted by the child to compensate for the muscular weakness. Our goal was to study the very early sign of the walking alterations by means of gait analysis in a group of young dystrophic child.

Method: A pathologic group, composed by 11 DMD patients aged from 5 to 7 years (mean 6.1+/−0.7 years), was grouped on the basis of the clinical observations: patients were evaluated while performing the following motor-tasks: ability and time to get up from the floor to standing (Gowers test) less than 3 s, capability of running a distance of 10 m, climbing stairs and descending without compensatory strategies. Nobody had a pharmacological treatment at the moment of examination. No alterations of passive range of motion, nor clinical signs of muscular weakness were evident. Patients were then assessed by gait analysis and data was compared with an age-matched control group composed by 9 healthy children, aged from 5 to 9 years. Gait analysis was performed, according with Davis protocol, using a Vicon System and a Kistler force platform embedded on an 8 m walkway. Kinematic (pelvic and lower leg joint angles) and kinetic (joint moments and powers) data of both groups were obtained and then compared for statistical significance differences.

Results: Spatial and temporal parameters showed significant differences between the two groups: cadence increased and step length decreased in a significant way ($p < 0.01$). Instead, the walking velocity was found to be similar in both groups. The pelvis showed significant differences in the range of anterior–posterior tilt, which was increased in the dystrophic patients ($p < 0.01$). The external rotation during terminal stance and internal rotation in mid and terminal swing were increased in patients group ($p < 0.05$). In the frontal plane, during terminal stance and pre-swing phase, there was an increased pelvic obliquity. Range of motion in sagittal plane showed a significant difference at the ankle, with an increased plantarflexion in swing in the dystrophic patients ($-1.86 \pm 5.9^\circ$, control group: $7.5 \pm 2.2^\circ$; $p < 0.01$). Maximum dorsiflexion in terminal stance/pre-swing phase was less in patient group (11.8+/5.3° control group: 18.3+/4.4°; $p < 0.01$). Kinetic analysis showed significant differences in power generation and absorption at the hip joint in mid, and in terminal stance and terminal swing ($p < 0.01$); at the ankle in loading response (bigger absorption), and mid and terminal stance (less production) ($p < 0.01$. At knee there was a less flexor moment in mid-stance ($p < 0.01$). Ankle showed a less dorsiflexor moment in terminal stance-pre-swing and minor range from maximum to minimum ($p < 0.01$).

Discussion: Gait analysis was a useful instrument for evaluating and quantifying the early modifications in the gait pattern of these dystrophic patients, when the clinical evaluation could not clearly evidence specific modification. This analysis can be considered an initial stage during the evaluation and quantification in the progressive weak pattern, and can be useful at evaluating the disease's progression during clinical treatment. Clinical interpretation of early gait pattern, in terms of compensation or weakness, can be useful to define more focused, early rehabilitative program.

Reference

[1] McDonald CM, Abresch RT, Carter GT, et al. Profiles of neuromuscular diseases. Duchenne muscular dystrophy. Am J Phys Med Rehabil 1995;74:S70–92.

doi: 10.1016/j.gaitpost.2008.10.044

Gender differences in the control of head accelerations during level walking

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Introduction: In young individuals, the oscillations of the upper body during level walking are characterised by an attenuation of the linear acceleration going from pelvis to head