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CASE REPORT

Kinematic gait analysis of patients with amyotrophic lateral sclerosis: presentation of three cases

Análise cinemática da marcha em pacientes com esclerose lateral amiotrófica: apresentação de três casos

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GENERAL INFORMATION

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ABSTRACT

Amyotrophic Lateral Sclerosis is a degenerative disorder of the central nervous system involving motor neurons, which directly affect the gait of a person. The aim of this study was to analyze the gait kinematics of patients affected by this pathology in different stages. A descriptive cross-sectional research was designed with three subjects who were classified in stages I, II and III proposed by Sinaki & Mulder. Regarding the spatial-temporal variables, it was observed that the more the stage of the pathology progresses, the further the values get from normality. In the angular kinematic parameters, there was a greater hip flexion to achieve ambulation, together with a decrease in the extension in the three joints studied. The results showed that the data from the subjects presenting amyotrophic lateral sclerosis differ from normal at all stages and the losses caused by the disease have direct influence on gait kinematics mainly with the progress of the disease when the overall symptoms become more incapacitating.

Palavras-Chave:
Esclerose lateral amiotrófica
Marcha
Cinemática

RESUMO

A Esclerose Lateral Amiotrófica é um distúrbio degenerativo do sistema nervoso central envolvendo neurônios motores que afetam diretamente a marcha. O objetivo deste estudo foi analisar a cinemática da marcha de indivíduos afetados por esta patologia, em diferentes estágios. Trata-se de uma pesquisa descritiva transversal em três sujeitos classificados entre os estágios I, II e III propostos por Sinaki e Mulder. Em relação às variáveis espaço-temporais, observa-se que conforme o estágio da patologia avança, mais os valores se distanciam da normalidade. Nos parâmetros cinemáticos angulares, observa-se maior flexão de quadril para conseguir deambular, somada a uma diminuição na extensão nas três articulações estudadas. Os resultados demostram que os dados destes indivíduos diferem da normalidade em todos os estágios e as perdas causadas pela doença têm influência direta na cinemática da marcha, principalmente com o progresso da doença, quando os sintomas gerais se tornam mais incapacitantes.

Introduction

Amyotrophic Lateral Sclerosis (ALS) is a degenerative disorder of the central nervous system, which affects motor neurons of the cerebral cortex, brainstem and spinal cord. It is considered a fatal disease characterized by paralysis of muscles that control voluntary movements.[1]

Studies show discrepancies related to the incidence in several regions of the world, the largest one being on the island of Guam (3.9/100.000 inhabitants) and the lowest in China (0.3/100.000 inhabitants).[2] The average age of symptoms onset is lower in Brazil compared to other countries (about 52 and 59-62, respectively).[2,3]

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ALS can be classified into six stages of functional dependency according to Sinaki & Mulder.[4] In the early stage of the disease (stage I), subjects are independent in relation to mobility and daily living activities (DLA). A specific group of muscles is mildly weak, which may appear as limitations in performance or endurance, or both. In the stage II, subjects have a moderate weakness in the affected muscle groups, which can be the upper and lower limbs in both sides of the body.

In stage III, subjects have significant weakness in certain muscle groups, which can lead to clinical signs as foot drop and severe loss of strength of the intrinsic hand muscles. This may lead the subject to need assistance for the DLA. The patient may be unable to stand up from a chair without help. Subjects in stages IV and V show marked weakness of muscle groups and the general mobility is very affected, so the need of assistance in their DLA is essential, as well as the use of assistive devices and wheelchairs. Subjects in the last-stage of the disease (stage VI) are bedridden and need full assistance.[4]

The most common impairment observed is a focal asymmetric weakness in the upper or lower limbs.[5] It is also observed muscular atrophy, cramps, fasciculation, gait deviations and changes in the reflexes and muscle tonus. The initial muscle weakness usually occurs in an isolated manner and affects mainly distal muscles. It is progressive and followed by functional limitations, resulting in difficulties at walking, deconditioning and impairments of balance and at postural control.[6]

As the disease progresses, spasticity can lead to contractures and deformities, as well as modified synergistic patterns of movements, abnormal rhythms of limbs, loss of dexterity and fatigue. These changes can consequently interfere with the motor control and function.[7] At advanced stages of the disease, subjects are unable to walk without using of an auxiliary device or completely lose the ability to perform the gait.[8]

Therefore, due to a) increased incidence of ALS in the world;[9-11] b) the fact that one of the major functional losses of these subjects is the ability to walk; and c) the lack of studies that describe the kinematic changes in the gait of this population, mainly related to disease progression, the general objective of this study was to describe the gait kinematics of three subjects with ALS, at different stages of pathology.

Cases description

This descriptive observational survey was conducted at the Gait Laboratory of Caxias do Sul University (UCS) in partnership with the Federal University of Health and Sciences of Porto Alegre (UFCSPA), Brazil. The study was approved by the local Ethics Committee (protocol number 362.784).

Three subjects with diagnosis of ALS (according to the criterion "El Escorial" allocated in the definitive ALS category) who presented the functional gait preserved (as classified in stages I, II and III according to the scale proposed by Sinaki & Mulder[4]) took part in this study. Written informed consent was obtained from all participants.

In the city of Caxias do Sul we could find eight ALS subjects, but only three of them were in the stages I, II and III proposed by Sinaki & Mulder.[4]These three patients were selected because they were subjects with a certain degree of muscle weakness, but could still perform functional gait.

All procedures for data collection in the gait laboratory were based on the protocols of Laroche *et al.*[12] For familiarization, participants were first asked to walk 8 meters straight into a self-selected speed in a specific place, so that it would be possible to collect the data. Reflective markers were attached to both sides of the following anatomical references: anterior superior iliac crest; posterior iliac crest; lateral side of the mid-thigh; lateral condyle of the femur; lateral aspect of the tibia; lateral malleolus, calcaneus, and fifth metatarsal head. A model of segments was built to capture the three-dimensional trajectory of the markers during the gait kinematics evaluation, a motion analysis system (VICOM systems MX – Oxford Metrics Group, UK) with seven synchronized cameras that were used at a sampling rate of 100Hz, as shown in ***Figure 1***.

Descriptive statistics of the lower limb kinematic variables were calculated and described by frequency distribution, mean/median and standard deviation.

The characteristics of the participants are reported in ***Table 1***. In summary, the mean age of the total study sample was 52.6 ± 5.4 years. According to the disease phases proposed by Sinaki & Mulder,[4] one subject was classified at the stage I; one subject was classified at stage II, and the third one at stage III. It should be noticed that last subject (the one on stage III) is the only one that needs an auxiliary device to walk outside his house, but for performing the gait analysis the device was removed without presenting risks to the patient, so as not to interfere with the acquisition of the images.

Table 2 describes the results obtained in the analysis of spatial and temporal variables of the gait of the 3 subjects. We can observe that, as the disease progresses within the stages, the functional losses become more severe, interfering directly in the walking patterns and its spatial and temporal variables.

As we can see in ***Table 3***, all the angles presented by the subjects differ from normal, showing that the losses caused by the disease have directly interference in the kinematics of gait patterns. Nevertheless, it could be observed that the subject 1 showed less deviation results compared with the normal gait, even though he has had the disease for a longer period, while subjects 2 and 3 differ significantly from normal, showing greater impairment in ambulation.

Discussion

In the current study, the main objective was to describe the gait kinematics of subjects with ALS who were at different stages of the disease and compare them to the normal values found in the literature. It is remarkable that the ALS appears in many ways, increasing motor control and respiratory symptoms in each distinct subject.[8] However, it is not always that the subject who has had the disease for a longer duration or is at an older age shows the worst progression or impairments.[13]

Oliveira *et al.*,[14] who evaluated the mortality rate

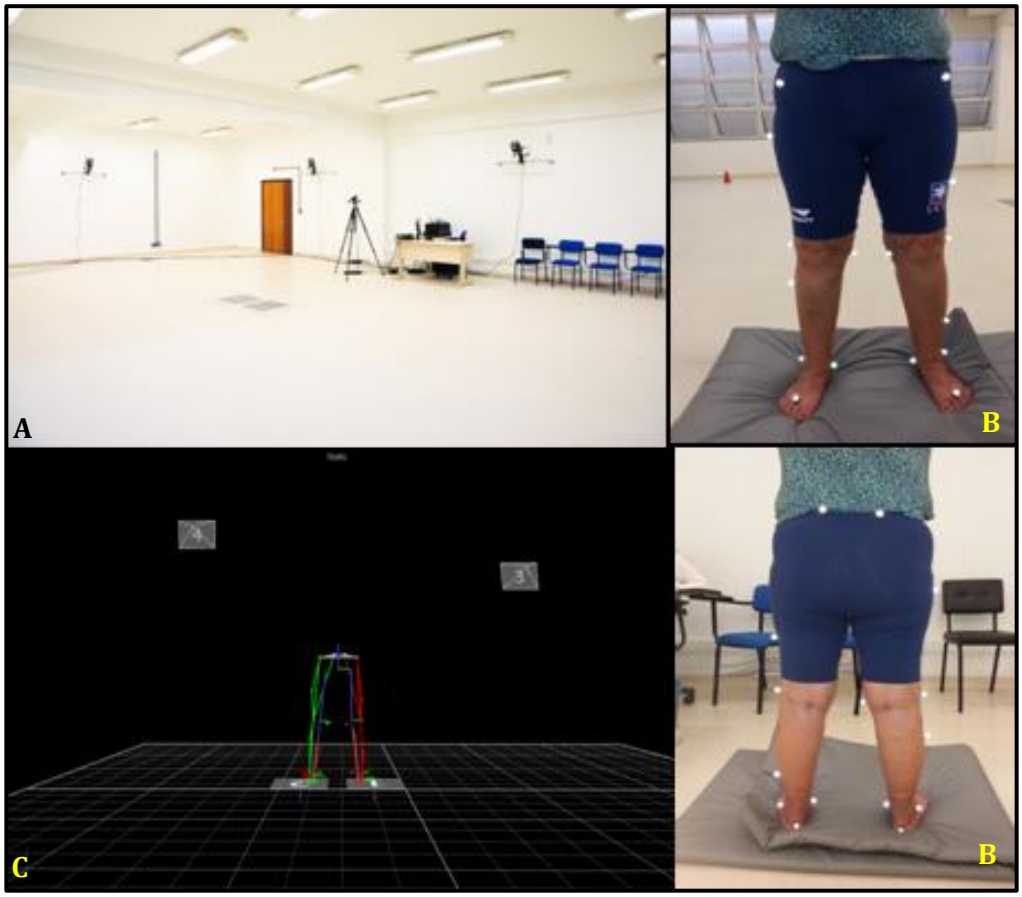

Figure 1. Image of the gait laboratory (A), as well as the markers distributed in the lower limbs of the subject (B) to capture images in the computer (C).

of patients with ALS in relation to time, location and gender, did not find significant differences between the percentage of mortality between men and women, and concluded that there was no significant difference between the age associated with time of progression and death. It was clear from our study that subject 1, who has had the disease for two years, was the one who presented fewer sequels, both concerning space and time variables in relation to angular kinematics.

Considering the spatiotemporal variables, specifically cadence and gait speed, the literature shows that gait speed is directly influenced by the number of steps per minute which the subject can perform (cadence), i.e., the fewer steps per minute, the slower the gait.[14]

Following the progression of the disease that causes great weakness with loss of general mobility, it was expected that the subject would perform fewer steps per minute interfering in the speed in which he was able to walk. Some authors point out that the time-distance measurements, such as speed, cadence, step and stride length have been useful for obtaining information about the overall gait performance and these parameters are essential for professionals who work with these subjects.[15]

Analyzing the length of the stride, there is a considerable difference from the normal for the subjects 2 and 3, who were classified into the more advanced stages of the disease. It is evident that ALS is a type of neurodegenerative disorder which affects the motor neurons that directly or indirectly control the muscle contractions

Table 1. Characterization of the sample considering age, gender, body mass index (BMI), initial form of disease, disease duration, functional stage of the disease and use of assistive devices.

Subject	Age (years)	Gender	Initial form of disease	Disease duration (months)	Functional stage of the disease	Assistive devices	BMI (kg/m^2)
1	60	Male	Spinal	24	I	No	20
2	51	Female	Spinal	8	II	No	28
3	47	Female	Spinal	12	III	Yes	25

Table 2. Spatiotemporal values of the subjects (mean of the right and left sides) and the reference values to cadence, step length, stride length and gait speed.

	Subject 1	**Subject 2**	**Subject 3**	**Normality***
Cadence (steps/minute)	79.30 ± 2.29	69.60 ± 2.39	42.60 ± 6.72	96 to 142
Step lengh (m)	0.55 ± 0.04	0.16 ± 0.03	0.20 ± 0.01	0.80 to 1.20
Striede lengh (m)	1.09 ± 0.07	0.32 ± 0.04	0.39 ± 0.01	1.50
Gait speed (m/s)	0.72 ± 0.06	0.21 ± 0.03	0.21 ± 0.048	1.15 to 1.75

* Normal values[14]

during ambulation.[16] This neuronal deterioration results in neurological dysfunction, in which the impulses initiated by the brain cannot be transmitted to those neurons responsible for the contraction of lower limb muscle fibers, causing generalized weakness.[17]

Therefore, these neurons make certain compensations, for example: patient 3, even being in stage 3 compared to patient 2 (who is in a previous functional stage), presents a step and stride length greater than subject 2. In view of this context, the authors guide a joint observation of all spatial-temporal variables analyzed, suggesting that subject 3, as a way of compensating for her cadence, which is much smaller than subject 2, tends to take larger steps to complete the requested distance, maintaining the same speed.

When it comes to the angular kinematics, we can see from Table 3 that all subjects showed significant changes in the angles involved in the gait. These deviations from normality may be due to disruption of brain-muscle pathway, where the lower limbs did not properly perform voluntary movements, significantly altering the gait patterns.[18] The same applies to individuals affected by neurodegenerative disorders, who showed a change in knee extension during the swing phase while the hip is flexed,[15] possibly due to a neuromuscular deficit of the quadriceps muscle in its concentric contraction phase. The same occurred with the individuals in this study, demonstrating reduced ability to extend the legs while performing the gait, adopting a fully flexed posture. Note that these data are extremely interesting when the professional healthcare outlines the goals during rehabilitation of subjects with ALS.

All values found in the hip, knee and ankle clearly show an asymmetry in the gait of subjects with ALS. Liao

Table 3. Kinematic parameters of the lower limbs expressed in angles (degrees of the right and left side): hip flexion-extension, hip abduction-adduction, hip rotation, knee flexion-extension, ankle plantar-dorsiflexion.

Segments	**Movement**	**Side**	**Subject 1**	**Subject 2**	**Subject 3**	**Normality***
Hip	*Flexion*	R	26.70	28.90	46.50	30
		L	29.90	30.60	34.90	
	Extension	R	-9.06	3.05	14.40	-10
		L	-11.30	7.86	6.54	
	Abduction	R	-3.47	2.97	10.60	15
		L	10.50	4.84	5.63	
	Adduction	R	-12.50	-3.66	-2.07	0
		L	0.32	2.51	-4.16	
Knee	*Flexion*	R	41.80	31.8	54.10	60
		L	47.90	37.60	31.50	
	Extension	R	5.44	-0.19	4.33	0
		L	0.38	11.50	-17.80	
Ankle	*Flexion*	R	15.40	15.80	17.00	10
		L	14.70	23.60	15.20	
	Extension	R	-0.98	12.40	-10.10	20
		L	-0.36	14.50	-18.10	

* Normality values[12] R: right side; L: left side.

et al.[19] analyzed the fluctuations of pace gait in subjects with ALS, Parkinson's disease (PD), and Huntington's disease (HD). The study showed that the pace gait in ALS shows more asymmetry compared to PD and HD. This asymmetry coupled with instability makes walking difficult for these subjects. It has been suggested that there is, probably, an expert system located in the spinal cord, which regulates the alternating movement of the limbs,[20] but the progressive death of motor neurons in this system makes it increasingly less efficient, leading to progressive muscle weakness, fatigue and subsequent muscle paralysis.

This study found some limitations such as a small sample size, due to the difficulty of finding subjects with ALS and who still have a functional gait. This makes it difficult to perform larger comparisons between different subjects within the stages of the disease, increased also by the small number of studies related to the analysis of the kinematic variables.

Conclusion

Through this study, it was possible to conclude that ALS caused alterations in both the spatiotemporal gait and angular variables in the three subjects who were analyzed. The primary findings were that there was a slowdown in spatiotemporal parameters such as cadence and gait velocity, combined with a great difficulty in extending the lower limbs and progressive weakness beginning in the distal muscles. This forces the patient to perform certain moves in to be able to keep walking. In addition, most of the analyzed variables showed a worsening in their values as the severity of the disease increases.

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