Published in *Gait & posture, 2019, vol. 68, p. 415-422* which should be cited to refer to this work
DOI : 10.1016/j.gaitpost.2018.12.029

Kinematics can help to discriminate the implication of iliopsoas, hamstring and gastrocnemius contractures to a knee flexion gait pattern.

M. Attias, PhD[1,2] ; A. Bonnefoy-Mazure, PhD[1] ; G. De Coulon, MD[3] ; L. Cheze, PhD[2] ; S. Armand, PhD[1]

Institutions:

[1]Willy Taillard Laboratory of Kinesiology, Geneva University Hospitals and Geneva University, Switzerland

[2] Univ Lyon, Université Lyon 1, IFSTTAR, LBMC UMR_T9406, F69622, Lyon, France

[3]Pediatric Orthopaedic Service, Department of Child and Adolescent, Geneva University Hospitals and Geneva University, Geneva, Switzerland.

Corresponding author:

E-mail: Michael.attias@hcuge.ch

Acknowledgments

This study was founded by the Swiss National Science Foundation (SNF) project 325230_146801.

Abstract

Background: Excessive Knee Flexion Gait Pattern (KFGP) is a common gait deviation in many pathological conditions. The contractures of the muscles that have been identified as being responsible of KFGP are: iliopsoas, hamstring and gastrocnemius.

Research question: How do isolated contractures of the iliopsoas, hamstrings and gastrocnemius impact knee flexion during gait?

Methods: Three levels of contracture (mild, moderate and severe) were simulated bilaterally using an exoskeleton on 10 healthy participants for iliopsoas, hamstring and gastrocnemius muscles. A gait analysis session was performed to evaluate the joint kinematics according to the different simulated contractures. Thirty one parameters were chosen to analyze the kinematics of the thorax, pelvis, hip, knee and ankle. A principal component analysis (PCA) was used to determine the kinematic parameters influenced by contractures.

Results: In addition to a permanent knee flexion observed for the three muscles with contracture: the contracture of the iliopsoas induces a large hip flexion with pronounced anterior pelvis tilt; the contracture of the hamstrings induces an ankle dorsiflexion during the support phase with a posterior pelvis tilt; the contracture of the gastrocnemius induces an absence of first and second rocker of the ankle with a slight flexion of hip and a slight anterior pelvis tilt.

Significance: These results support the identification of the muscles responsible for a KFGP.

A better knowledge of the interactions between contractures and associated joint kinematics of the same and adjacent joints will support the interpretation of gait analyses by more precisely and faster targeting the concerned muscle.

Keywords: Kinematics; Contractures; Gait; Knee flexion; Exoskeleton; Simulation

Introduction

Knee flexion gait pattern (KFGP) is a frequent gait deviation [1, 2] observed in many pathological conditions (e.g. cerebral palsy). It [KFGP] is defined by an excessive knee flexion during the stance phase greater than normally expected [3]. Svehlik et al. defined KFGP more specifically as a value superior to 20° for the minimum knee flexion in single limb support [4]. Other authors defined KFGP by a knee flexion at initial contact superior to15° [5] or 17° [6] corresponding to one standard deviation (SD) above the mean flexion at initial contact for a typically developing children.

KFGP impairs gait performance by decreasing velocity, stride and step length and by increasing walking effort [7, 8]. Physical consequence can be pain at the joint, degenerative arthritis, bony deformities and increased risk for falls due to inadequate foot clearance [9]. To reduce the consequences of a KFGP, treatment of the cause of this pattern is needed. To support the therapeutic choice, a clinical gait analysis (CGA) is generally performed helping to identify and to understand gait deviations. However, interpretation of CGA is complex because gait deviations can be caused by different clinical impairments such as contractures, spasticity, muscle weakness, reduced muscle selectivity or pain [10, 11].

Gait can be altered by the primary causes of gait deviations (impairments) or by secondary compensatory strategies [12, 13]. It is important to distinguish between these aspects because only impairments need to be treated [14]. Secondary, compensatory strategies should disappear once these primary impairments have been treated [15]. Therefore, an original approach to establish an association between primary impairments and gait deviation is to investigate the impact of one isolated impairment on gait deviations [16].

Among impairments that can lead to a KFGP, contractures of *iliopsoas* (PSO), *hamstring* (HAM) and *gastrocnemius* (GAS) muscles are possible causes [1, 8, 16-18].

HAM and GAS cross the knee joint and are both biarticular muscles. Indeed, the HAM also affects the hip joint whereas the GAS affects the ankle joint [3]. Concerning the PSO, it is the main muscle involved in a hip flexion contracture and also lead to a KFGP [1]. Even, if KFGP can be caused by different impairments, we hypothesize that different impairments will lead to different gait deviations at other joints/segments. Therefore, identifying associated gait deviations with possible causes of KFGP will support the interpretation of CGA when a KFGP occurs.

Hence, the aim of this study was to compare the kinematics associated with a KFGP among isolated PSO (referring to a hip flexion contracture), HAM and GAS contractures.

Methods

Participants:

Ten participants were included in this prospective study with no known neurologic or orthopaedic problems. They had the following characteristics [mean (SD)]: 5 females/5 males; age: 25.7 years (3.6); height: 1.71 m (0.1); weight 64.4 kg (11.4).

Ethical approval and participant informed consent were obtained prior the beginning of data collection.

Gait evaluation

To simulate PSO (referring to a hip flexion contracture), HAM and GAS bilateral contractures, participants wore the passive exoskeleton MIkE (Figure 1) and walked along a 10-meter walkway at a spontaneous self-selected speed [9]. The exoskeleton was designed to bilaterally embrace the pelvis, the thigh, and the shank with plastic cuffs and with modified shoes that included attachment points. A particular cut was made on the plastic cuffs to enable reflective markers to be placed directly on the skin as requested for CGA. Each participant walked also with the exoskeleton without simulated contracture for the control condition (CC). The participants were equipped with 34 reflective markers aligned to anatomical and technical landmarks on the head, trunk and pelvis and bilaterally on the arms, thighs, shanks and feet according to the full-body Plug-in-Gait model [19]. Marker trajectories were recorded with a twelve-camera motion analysis system (Oqus 7+, Qualisys, Göteborg, Sweden). Kinematic computation was performed with a minimum of five gait cycles averaged to produce a single angular displacement of the thorax, pelvis and foot segments, and spine, hip, knee and ankle joints.

The level of contracture was selected as mild, moderate and severe contracture according our clinical experience and the literature [16, 20, 21]. Hence, PSO was limited at 0°, -20° and -40° of hip extension angle (knee in extension), HAM was limited at 40°, 70° and 100° of unilateral popliteal angle and GAS was limited at 10°, 20° and 30° of ankle plantarflexion. To set the contractures, the experimenter adjusted the rope length of the exoskeleton in the position used for standard physical examination [22] and controlled it with a manual goniometer (Figure 1).

Thus, nine conditions were performed including three levels of contracture severities for each type of muscle (PSO, HAM and GAS). Before recording the data, each participant walked two to five minutes in each experimental condition. Once the participant was comfortable when walking in the experimental condition, we evaluated his/her gait (two tests of 10 meters each). After each experimental condition, a rest period of two minutes was completed to avoid a possible fatigue effect. The participants were asked to walk at comfortable self-selected speed without other indication. The same day, PSO conditions were performed before HAM conditions with a rest period in between both conditions. GAS conditions were performed another day to avoid fatigue.

Data analysis and statistics

Two steps were performed to analyse the data.

Firstly, in order to evaluate the kinematic parameters influenced by the contracture during walking, a principal component analysis (PCA) was performed on each muscle (PSO, HAM and GAS). The PCA was used to identify the parameters influenced by the degree of contracture for each muscle.

For the thorax, pelvis and spine (defined by the relative movement between thorax and pelvis) the parameters were range of motion (RoM) and mean position during gait cycle for each plane.

For the hip, knee and ankle joints only those parameters in the sagittal plane were analysed. Thus, for the hip joint, the parameters were: RoM, maximum, minimum and mean position during the gait cycle. For the knee joint, the parameters were: angle at initial contact (IC), maximum extension and mean position in stance phase and RoM during gait cycle. For the ankle joint, parameters were: angle at initial contact (IC), maximum plantar flexion and mean position in stance phase and RoM during gait cycle. Finally, for the foot progression angle, only the mean angle during the gait cycle was analysed. These parameters were considered as relevant in a clinical context and reliable according to literature [1, 13, 23, 24]. Indeed, thorax and pelvis obliquity and rotation can influence the gait pattern in many pathologies so that it seems important to consider these specific parameters [25-27]. In addition, hip and knee adduction-abduction and rotation were not selected in the PCA because only the sagittal plane was investigated for these joints. Indeed, the sagittal plane is the plane the most analyzed in clinical gait analysis. Moreover, the adduction-abduction and rotation angles of hip, knee and ankle were not considered in this study due to concerns about the accuracy of this data in our data collection procedures of the hip center and the unreliable identification of knee axis, the poor reliability caused by the soft tissue artifacts [28] and the exoskeleton installation and configuration [9].

A minimum of four parameters was selected based on the value of the squared cosines matrix of PCA. If the values was higher than 0.85 for more than four parameters, all the parameters were selected. If the values was lower than 0.85, the four parameters with the highest values were selected. More the value is close to 1, more the parameters explain the variability of gait due to the level of simulated contracture.

Secondly, considering all the conditions, three groups were created based on mean knee position in stance: Group 1: 20-30°; Group 2: 30-40°; Group 3: 40° and more. The differences between the groups and the muscles (PSO, HAM and GAS) on the parameters selected by PCA were analysed by a Kruskal Wallis test with post hoc. Boneferroni's correction was used to limit the problem of multiple comparisons. Walking speed was also compared in the same analysis.

Results

Firstly, the selection of kinematic parameters using PCA was presented in Figure 2. First and second factor of PCA were chosen for all muscles. For PSO with a total inertia (proportion explained by the first and second factor) of 46%; for HAM with a total inertia of 43% and for GAS with a total inertia of 46%. The selected parameters were in the sagittal plane: mean pelvic tilt position,, hip mean flexion position, hip minimum flexion position, hip maximum flexion position and hip flexion at initial contact, knee mean flexion during stance phase, ankle flexion at initial contact, mean ankle flexion position during stance phase and maximal ankle flexion position during stance phase; in the frontal plane, only the range of motion of pelvic obliquity was selected; no parameter was selected on the transverse plane.

Secondly, the selected parameters were compared between the three simulated muscle contractures (PSO, GAS and HAM) for each group of KFGP. The results are presented in Table 1 and illustrated in Figure 3. **At the pelvis level**, PSO and GAS contractures induced an anterior tilt whereas the HAM contracture induced a posterior tilt. PSO induced a more important anterior tilt than GAS. PSO showed more pelvis RoM obliquity than other muscles. **At the hip level**, PSO contracture induced a permanent flexion followed by the GAS with a moderate but permanent flexion whereas the hip flexion was close to that of CC for HAM contracture. **At the knee level**, GAS and HAM contractures induced a more important flexion at IC than PSO that showed only a moderate flexion at IC. **At the ankle level**, HAM contracture induced a permanent dorsiflexion during stance; GAS contracture induced a plantarflexion during the complete gait cycle and PSO contracture showed a kinematics close to the normal.

Discussion

The objective of this study was to compare kinematics associated with a KFGP among isolated PSO, HAM and GAS contractures, simulated using a passive exoskeleton.

Results of this study support that KFGP, can be caused by contracture of different muscles and induce different gait adaptations. The main kinematic associations for each of the three simulated muscles contractures were: 1) PSO contracture induces an important hip flexion with a pronounced anterior pelvis tilt (it should be noted that if the pelvis presents an anterior pelvis tilt, the hips will be naturally in flexion); 2) HAM contracture induces an ankle dorsiflexion during stance phase with a posterior pelvis tilt; 3) GAS contracture induces an ankle plantarflexion (with a moderate hip flexion and a moderate anterior pelvis tilt).

It is important to identify the primary impairment of a KFGP to choose the best treatment strategy. Lengthening of the HAM is often used to treat a KFGP if the popliteal angle is increased. This surgery is often performed in patients with cerebral palsy with a crouch gait pattern in order to improve both knee extension in stance and range of motion [29]. However, it has been demonstrated that CGA is a relevant tool to support clinical decision-making with regards to HAM lengthening. Indeed, in 54% of cases, clinicians change their initial treatment plan based on the CGA results [29]. Thus, the surgery should be indicated when HAM is the cause of joint limitation and the clinician needs to verify and to consider that other impairments may cause a KFGP. Goodman et al. highlighted that limitations in both hip and knee extension of persons with hemiplegia are not necessarily caused by limited length of HAM and/or hip flexors (PSO), but rather as the result of an ankle plantarflexor contracture alone [30]. Rodda et al. showed a reduction of crouch gait in ten patients with cerebral palsy after HAM lengthening at one year and five year post-surgery. An increase of mean anterior pelvic tilt angle was related to HAM lengthening. They explained that they performed too few PSO lengthenings and too many HAM procedures [31]. It can be speculated that a PSO contracture could be the cause of crouch gait in some of these patients and that a PSO lengthening would have also reduced crouch gait with no increase of pelvic tilt angle.

The reliability and the limitations of the simulated contractures with the exoskeleton were showed in a previous study [9]. The first important limitation concerns the short adaptation time to contracture emulations. Indeed, adaptation time can play an important role in gait strategies adopted by the participant and dependent on the respective type of gait. GAS can act on ankle plantarflexion or on knee flexion/extension, or a combination of both [32]. HAM can influence hip flexion/extension, knee flexion/extension, or both [1]. So there are endless possible combinations of adaptation strategies although most participants choose similar gait adaptations for each condition. The (strategy) choice of the participants (probably also valid for the patients), is certainly guided to optimize their walk by preserving energy [33], to optimize forward progression [20], to assure balance and their stability [34]. The second limitation concerns the walking speed that was not controlled between the different conditions of this study. We know that gait parameters vary due to walking speed [35] and can influence the results of this study. However during the CGA, with patients, we encounter the same problems of different walking speeds according to the impairments. The third limitation is related to the simulation of muscle contracture: the attachment points on the exoskeleton can differ from human morphology, we have considered only the main muscular action line; only a passive limitation of movement was investigated (by imposing an increase of passive constraints after a certain length) without considering the modification of higher passive constraints for all muscle lengths (e.g. length-tension curve) related to spastic muscles and tendons [36] and without considering the complex muscle function modifications that occurs in patients (e.g. cerebral palsy [37]).

In addition, contractures are very often associated with other deficits such as paresis or muscular hyperactivities [10, 11]. These deficits can be combined and create complex gait deviations. However, isolating a clinical impairment (e.g. contracture, weakness, spasticity) is an appropriate and relevant approach to investigate its influence on walking [1, 8, 9, 12, 13, 17, 30, 38-40]. Indeed, it is a good starting point to identify the effects of isolated contractures before understanding the effects of combined clinical impairments.

Clinical contribution

Understanding the mechanisms that affect walking and identifying motor impairments is important to target and to develop treatment strategies. However, the link between motor impairments assessed by clinical examination and the alterations of walking are not obvious and lack scientific evidence [41-44].

The findings of this study prove that KFGP can be caused by either PSO, HAM or GAS contractures. The clinicians should consider all three contracture types during the interpretation of clinical gait analysis and the choice of treatment. In addition, gait deviations associated with a KFGP can support the clinician to better understand the underlying causes of KFGP (e.g. KFGP associated with ankle plantarflexion and anterior pelvis tilt s indicates a gastrocnemius contracture) that should be verified in clinical examinations (e.g. limitation of ankle dorsiflexion knee extended). Therefore, these findings support the understanding and the interpretation of clinical gait analysis by better targeting the altered muscle due to contractures causing KFGP.

Conclusion

This study discriminates kinematic factors associated with a KFGP according to muscle contractures in the perspective of supporting the interpretation of CGA. To resume, the results highlight that for a given level of KFGP 1) PSO contracture induces an important hip flexion with a pronounced anterior pelvis tilt; 2) HAM contracture induces an ankle dorsiflexion during stance phase with a posterior pelvis tilt; 3) GAS contracture induces ankle plantarflexion with a small hip flexion and a small anterior pelvis tilt. This knowledge together with other clinical information (physical examination, imaging) will help to identify the impairment(s) responsible of a knee flexion gait pattern. By improving the understanding of the possible cause of gait deviations, treatment decision can be improved with the final objective to enhance gait quality and, finally, their functional performance in daily life.

Conflict of interest

There are no conflicts of interest associated with this research.

Acknowledgments

This study was funded by the Swiss National Science Foundation (SNF) project 325230_146801.

References

[1] Matjacic Z, Olensek A. Biomechanical characterization and clinical implications of artificially induced crouch walking: Differences between pure iliopsoas, pure hamstrings and combination of iliopsoas and hamstrings contractures. J Biomech. 2007;40:491-501.
[2] Wren TA, Rethlefsen S, Kay RM. Prevalence of specific gait abnormalities in children with cerebral palsy: influence of cerebral palsy subtype, age, and previous surgery. J Pediatr Orthop. 2005;25:79-83.
[3] Perry J, Burnfield J. Gait Analysis: Normal and Pathological Function,. Pomona, CA: Slack; 2010.
[4] Svehlik M, Steinwender G, Kraus T, Saraph V, Lehmann T, Linhart WE, et al. The influence of age at single-event multilevel surgery on outcome in children with cerebral palsy who walk with flexed knee gait. Dev Med Child Neurol. 2011;53:730-5.
[5] Haumont T, Church C, Hager S, Cornes MJ, Poljak D, Lennon N, et al. Flexed-knee gait in children with cerebral palsy: a 10-year follow-up study. J Child Orthop. 2013;7:435-43.
[6] Church C, Ge J, Hager S, Haumont T, Lennon N, Niiler T, et al. Flexed-knee gait in children with cerebral palsy. Bone Joint J. 2018;100-B:549-56.
[7] Cerny K, Perry J, Walker JM. Adaptations during the stance phase of gait for simulated flexion contractures at the knee. Orthopedics. 1994;17:501-13.
[8] Whitehead CL, Hillman SJ, Richardson AM, Hazlewood ME, Robb JE. The effect of simulated hamstring shortening on gait in normal subjects. Gait Posture. 2007;26:90-6.
[9] Attias M, Bonnefoy-Mazure A, De Coulon G, Cheze L, Armand S. Feasibility and reliability of using an exoskeleton to emulate muscle contractures during walking. Gait Posture. 2016;50:239-45.
[10] Gracies JM. Pathophysiology of spastic paresis. II: Emergence of muscle overactivity. Muscle Nerve. 2005;31:552-71.
[11] Gracies JM. Pathophysiology of spastic paresis. I: Paresis and soft tissue changes. Muscle Nerve. 2005;31:535-51.
[12] Romkes J, Brunner R. An electromyographic analysis of obligatory (hemiplegic cerebral palsy) and voluntary (normal) unilateral toe-walking. Gait Posture. 2007;26:577-86.
[13] Houx L, Lempereur M, Remy-Neris O, Brochard S. Threshold of equinus which alters biomechanical gait parameters in children. Gait Posture. 2013;38:582-9.
[14] Davids JR. Quantitative gait analysis in the treatment of children with cerebral palsy. J Pediatr Orthop. 2006;26:557-9.
[15] Schmid S, Schweizer K, Romkes J, Lorenzetti S, Brunner R. Secondary gait deviations in patients with and without neurological involvement: a systematic review. Gait Posture. 2013;37:480-93.
[16] Attias M, Chevalley O, Bonnefoy-Mazure A, De Coulon G, Cheze L, Armand S. Effects of contracture on gait kinematics: A systematic review. Clin Biomech. 2016;33:103-10.
[17] Balzer J, Schelldorfer S, Bauer C, van der Linden ML. Effects of simulated crouch gait on foot kinematics and kinetics in healthy children. Gait Posture. 2013;38:619-24.
[18] Attias M, Bonnefoy-Mazure A, De Coulon G, Cheze L, Armand S. Influence of different degrees of bilateral emulated contractures at the triceps surae on gait kinematics: The difference between gastrocnemius and soleus. Gait Posture. 2017;58:176-82.
[19] Davis RB, Ounpuu S, Tyburski D, Gage JR. A gait data collection and reduction technique. Hum Mov Sci. 1991;10:575-87.
[20] Attias M, Bonnefoy-Mazure A, Lempereur M, Lascombes P, De Coulon G, Armand S. Trunk movements during gait in cerebral palsy. Clin Biomech. 2015;30:28-32.

[21] Drefus LC, Hafer JF, Scher DM. Simulated Ankle Equinus Affects Knee Kinematics During Gait. HSS J. 2016;12:39-43.
[22] Viehweger E, Berard C, Berruyer A, Simeoni MC. [Testing range of motion in cerebral palsy]. Ann Readapt Med Phys. 2007;50:258-65.
[23] Benedetti MG, Catani F, Leardini A, Pignotti E, Giannini S. Data management in gait analysis for clinical applications. Clin Biomech (Bristol, Avon). 1998;13:204-15.
[24] Harato K, Nagura T, Matsumoto H, Otani T, Toyama Y, Suda Y. A gait analysis of simulated knee flexion contracture to elucidate knee-spine syndrome. Gait Posture. 2008;28:687-92.
[25] Bruijn SM, Meijer OG, van Dieen JH, Kingma I, Lamoth CJ. Coordination of leg swing, thorax rotations, and pelvis rotations during gait: the organisation of total body angular momentum. Gait Posture. 2008;27:455-62.
[26] Cappozzo A. The forces and couples in the human trunk during level walking. J Biomech. 1983;16:265-77.
[27] Heyrman L, Feys H, Molenaers G, Jaspers E, Monari D, Nieuwenhuys A, et al. Altered trunk movements during gait in children with spastic diplegia: compensatory or underlying trunk control deficit? Research in developmental disabilities. 2014;35:2044-52.
[28] Leardini A, Chiari L, Della Croce U, Cappozzo A. Human movement analysis using stereophotogrammetry. Part 3. Soft tissue artifact assessment and compensation. Gait Posture. 2005;21:212-25.
[29] MacWilliams BA, Stotts AK, Carroll KL, D'Astous JL. Utilization and efficacy of computational gait analysis for hamstring lengthening surgery. Gait Posture. 2016;49:394-7.
[30] Goodman MJ, Menown JL, West JM, Jr., Barr KM, Vander Linden DW, McMulkin ML. Secondary gait compensations in individuals without neuromuscular involvement following a unilateral imposed equinus constraint. Gait Posture. 2004;20:238-44.
[31] Rodda JM, Graham HK, Nattrass GR, Galea MP, Baker R, Wolfe R. Correction of severe crouch gait in patients with spastic diplegia with use of multilevel orthopaedic surgery. J Bone Joint Surg Am. 2006;88:2653-64.
[32] Matjacic Z, Olensek A, Bajd T. Biomechanical characterization and clinical implications of artificially induced toe-walking: differences between pure soleus, pure gastrocnemius and combination of soleus and gastrocnemius contractures. J Biomech. 2006;39:255-66.
[33] Selinger JC, O'Connor SM, Wong JD, Donelan JM. Humans Can Continuously Optimize Energetic Cost during Walking. Curr Biol. 2015;25:2452-6.
[34] Romkes J, Peeters W, Oosterom AM, Molenaar S, Bakels I, Brunner R. Evaluating upper body movements during gait in healthy children and children with diplegic cerebral palsy. J Pediatr Orthop B. 2007;16:175-80.
[35] Schwartz MH, Rozumalski A, Trost JP. The effect of walking speed on the gait of typically developing children. J Biomech. 2008;41:1639-50.
[36] Dayanidhi S, Lieber RL. Skeletal muscle satellite cells: mediators of muscle growth during development and implications for developmental disorders. Muscle Nerve. 2014;50:723-32.
[37] Mathewson MA, Lieber RL. Pathophysiology of muscle contractures in cerebral palsy. Phys Med Rehabil Clin N Am. 2015;26:57-67.
[38] Matjacic Z, Olensek A, Bajd T. Biomechanical characterization and clinical implications of artificially induced toe-walking: Differences between pure soleus, pure gastrocnemius and combination of soleus and gastrocnemius contractures. J Biomech. 2006;39:255-66.

[39] Houx L, Brochard S, Lempereur M, Remy-Neris O. Simulation of unilateral equinus using an adjustable orthosis in children: design, feasibility and biomechanical effects. Prosthet Orthot Int. 2012;36:131-6.
[40] Harato K, Nagura T, Matsumoto H, Otani T, Toyama Y, Suda Y. Knee flexion contracture will lead to mechanical overload in both limbs: A simulation study using gait analysis. Knee. 2008;15:467-72.
[41] Aktas S, Aiona MD, Orendurff M. Evaluation of rotational gait abnormality in the patients cerebral palsy. J Pediatr Orthop. 2000;20:217-20.
[42] Bonnefoy-Mazure A, Sagawa Y, Jr., Pomero V, Lascombes P, De Coulon G, Armand S. Are clinical parameters sufficient to model gait patterns in patients with cerebral palsy using a multilinear approach? Comput Methods Biomech Biomed Engin. 2016;19:800-6.
[43] Desloovere K, Molenaers G, Feys H, Huenaerts C, Callewaert B, Van de Walle P. Do dynamic and static clinical measurements correlate with gait analysis parameters in children with cerebral palsy? Gait Posture. 2006;24:302-13.
[44] Domagalska M, Szopa A, Syczewska M, Pietraszek S, Kidon Z, Onik G. The relationship between clinical measurements and gait analysis data in children with cerebral palsy. Gait Posture. 2013;38:1038-43.