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Identification and classification of toe-walkers based on ankle kinematics, using a data-mining method

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Abstract

A database of 1736 patients and 2511 gait analyses was reviewed to identify for trials where the first rocker was absent. A fuzzy c-means algorithm was used to identify sagittal ankle kinematic patterns and three groups were identified. The first showed a progressive dorsiflexion during the stance phase, while the second had a short-lived dorsiflexion, followed by a progressive plantarflexion. The third group exhibited a double bump pattern, moving successively from a short-lived dorsiflexion to a short-lived plantarflexion and then returning to a further short-lived dorsiflexion before ending with plantarflexion until toe-off. The three patterns were linked to different neurological conditions. Myopathy, neuropathy and arthogryposis essentially revealed group 1 patterns, whereas idiopathic toe-walkers mainly displayed group 2 patterns. Cerebral palsy patients, however, were relatively homogeneously distributed amongst the three groups. Able-bodied subjects walking on their toes showed a high proportion of unclassifiable ankle patterns, due to a variable gait whilst toe walking. Despite the variety of neurological conditions included in this meta-analysis repeatable biomechanical patterns appeared that could influence therapeutic management.

Keywords: Gait analysis; Biomechanics; Data-mining; Toe-walking; Fuzzy c-means

1. Introduction

Toe-walking has been defined as the failure of the heel to make contact with the floor at the onset of stance [1] or as an absence of a heel rocker. In normal gait, the term ''heel rocker'' corresponds to the progression of the limb while the heel is the pivotal area of support [2]. The rear of the foot contacts the floor and rolls into plantarflexion. Without a heel rocker phase, either the mid- or forefoot touches the floor, or the ankle moves toward dorsiflexion.

Toe-walking is a very common gait deviation in children and is considered normal up until three years old [3]. It is also associated with cerebral palsy (CP) [4–10], muscular dystrophy [11–13], neuropathy [14] and in bony deformities such as clubfoot [15]. In the absence of any known cause, idiopathic toe walking (ITW) is the term used to describe the toe–toe gait pattern.

Toe-walking studies usually have one of three major aims. Firstly researchers have tried to distinguish between toe-walker groups, especially between idiopathic toe-walkers and children with mild cerebral palsy [5–8,16]. Fifteen years ago, Hicks et al. [8] identified different causes for toe-walking in CP and ITW: for CP, the cause was excessive knee flexion whereas for ITW, toe-walking was due to plantarflexion. More recently, Kelly et al. [7] pointed out characteristic patterns of knee and ankle motion that differentiated the two pathologies. Kalen et al. [16] found no

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differences between ITW and CP patients in their study using electromyography; however, Rose et al. [5] has since shown that electromyography of functional exercises can help to distinguish between CP and ITW. Secondly researchers have attempted to understand the biomechanical consequences of toe walking. For Kerrigan et al. [17], toe walking may require less ankle plantarflexor, ankle dorsiflexor, and knee extensor strength than normal heel–toe walking, and thus may have compensatory advantages for patients with upper motor neuron injury and weakness in the distal lower extremities. On the contrary, Perry et al. [1] concluded that toe-walking demanded more effort than heel–toe walking, given a higher electromyographic activity in the triceps surae. Thirdly some researchers have sought to distinguish the primary and secondary gait deviations of cerebral palsy [9] by comparing able-bodied subjects walking voluntarily on their toes with CP toe-walkers, although this subject appears to have received less attention in the literature given the paucity of references. All of these studies focus on a particular aspect of toe-walking, either pathological or biomechanical.

Considerable debate concerns treatment choices for toe-walking, especially in children with cerebral palsy [18–21], but also in idiopathic toe-walkers [22] or patients suffering from myopathy [12]. Understanding the causes of toe-walking clearly aids the optimisation of treatment plans; for example, distinguishing between dynamic tightness and fixed contracture [10,23] in cerebral palsy patients has allowed improvement in management [21].

Multidimensional data analysis, particularly cluster analysis, has been used successfully to identify gait patterns in normal walking [24], to classify gait deviations in children with cerebral palsy [25], to distinguish gait patterns in elderly men [26], to classify patients with arthrodesis compared to able-bodied subjects [27], and to monitor progress of gait patterns after a stroke [28]. Unlike the classic clustering methods, the fuzzy c-means clustering method used by Su et al. [27] has the advantage of not assigning a patient strictly to a well-defined group, but rather determines group membership based on all the clusters considered. This method permits the patients most characteristic of each group to be highlighted, the patients in the same group to be compared, and the evolution of a specific patient's gait to be monitored via several post-intervention and/or post treatment evaluations. Thus, the fuzzy c-means clustering method would appear to be appropriate for distinguishing between toe-walking gait patterns. Our purpose was three-fold: firstly to select all toe-walkers from an extensive database and define the proportions of this gait deviation; secondly to research potential patterns in the ankle sagittal kinematics of toe-walkers, and thirdly to link these patterns to differing neurological conditions.

2. Method

2.1. Subjects and database

The entire database of the gait analysis laboratory of the Institut Saint-Pierre was reviewed for toe-walkers. From 1993 to 2003, 1736 patients underwent 2511 gait analysis examinations for various reasons: to evaluate gait before and/or after an intervention, to monitor gait progression or to analyse gait problems. This database seems to be representative of patients examined in a gait lab. From one to eight gait analysis examinations were performed per patient. This study examined 11,950 trials. Fig. 1 summarizes the number of patients per disease. Diseases accounting for less than 10 patients (e.g. poliomyelitis and Marfan's syndrome) or patients displaying symptoms not attributable to a specific disease were classed under 'various'. The patient database included 982 boys and 754 girls, with a mean age of 13.7 ± 8.4 years.

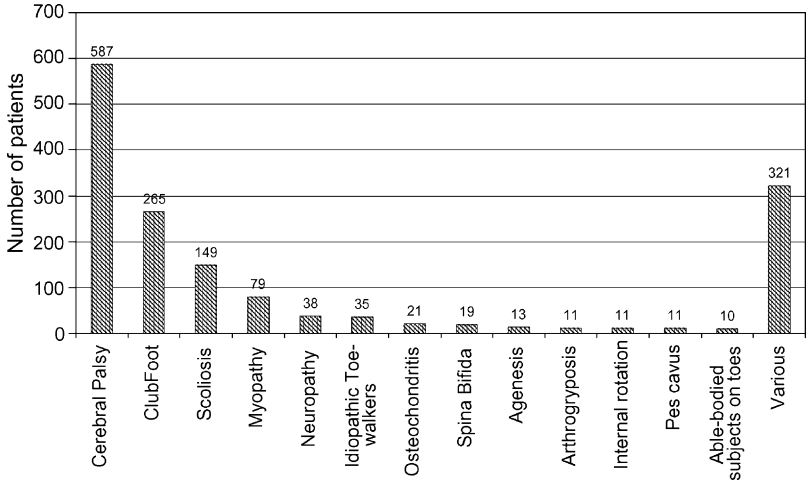

Fig. 1. Number of patients per disease.

2.2. Gait analysis procedure

Gait acquisition comprised a three-dimensional (3D) analysis with a minimum of three trials for each session, including synchronous kinematic, kinetic, and bilateral electromyography (EMG) recordings of the lower limb. A biomechanical engineer oversaw all analyses. A five-camera motion analysis system (Vicon® VX replaced by a Vicon® 512 in 1999, Oxford Metrics, UK) and two force platforms (AMTI®, USA) embedded in the walkway were used. Markers placement was according to Davis's protocol [29], and the subjects walked the length (12 m) of the laboratory barefoot and at their usual speed. Spatio-temporal parameters, joint angle motion, internal joint moments and powers were computed using Vicon Clinical Manager (VCM, Vicon®, Oxford Metrics, UK).

2.3. Data processing

In this study, toe walking was defined by the absence of a heel rocker. Gait trials with flat–foot and fore–foot initial contacts were included; only trials with a heel–foot initial contact were excluded. The slope of the sagittal ankle kinematics during the first nine percent of the gait cycle, corresponding to the first rocker [2,30], was used to select those trials presenting toe-walking. The slope S of the ankle kinematic was calculated during the stance phase: $S = \Delta A/\Delta t$ with A as ankle kinematics and t as time. Without a first rocker, there was no movement towards plantarflexion and the ankle kinematics showed a permanent movement toward dorsiflexion. The entire VCM database was loaded with Access (Microsoft Corporation), which was linked to Matlab (The MathWorks, USA) for all the data processing.

Exclusion criteria were defined on several levels: for patients if all sessions from a patient were incomplete, for sessions if a patient was older than 20 years old to keep a homogeneous population, a session with fewer than three selected trials, for trials if extreme outliers for the selected trials were excluded with the subsequent method. For each angle and for each slope value, the threshold was defined as the first quartile minus three times the interquartile range and the third quartile plus three times the interquartile range [31].

During stance phase, the slope of the ankle kinematics resampled on 30 points was coded as ''1'' if the curve went upwards (towards dorsiflexion, thus a positive slope), and ''0'' if the curve went downwards (towards plantarflexion, thus a negative slope) or if the slope value was nil. To identify patterns in the ankle kinematic data, the fuzzy c-means algorithm (FCM) was used with the following parameters: a fuzzification exponent, $m = 2$; a minimal improvement, $e = 10^{-5}$; and a maximal number of iterations $t = 100$. The input of FCM was the sequences of 0–1 for all the trials selected and the output was the trials membership values to clusters. This clustering method, which allows one piece of data to belong to two or more clusters, was developed by Dunn in 1973 [32] and improved by Bezdek in 1981 [33]. FCM is frequently used in pattern recognition, particularly for the image processing used in MRIs. Su et al. [27] have used FCM to identify gait patterns in arthrodesis patients and describe the method in detail. To determine the number of clusters, the FCM was processed from 2 to 10 clusters and the partition coefficient [33]—which measures the fuzziness of the partition—was used to determine the optimum number of clusters. The optimum was defined when the partition coefficient reached its maximum. The mean of the trial membership values for each pattern was used to analyse the links with the diseases and ankle gait patterns identified. A session was assigned to one of the three groups if the mean membership was higher than 0.5. This criterion was made to avoid classifying an irreproducible session or a different pattern from those identified.

Angles, internal moments and powers in the sagittal plane were reported for each ankle pattern. The mean was weighted by the trials membership values to characterize each ankle pattern. Means were taken over trials that had a membership value superior to 0.5. Partly suggested by Benedetti et al. [34] and Kerrigan et al. [17], the gait parameters found in Table 1 correspond to the local kinematic and kinetic maximum or minimum for curves representing ankle, knee and hip movements in the sagittal plane as well as spatio-temporal parameters. One-way variance analysis (ANOVA) was used to examine the effect of the identified group on gait parameters at an α level of 0.01. All significant ANOVA tests were followed by Tukey's honestly significant difference test for unequal sizes (Spjotvoll-Stoline test) for multiple comparisons in order to determine significant differences at an α level of 0.01. All statistical analysis was done using Statistica for Windows 6.0 (Statsoft, France).

3. Results

The research algorithm run on the entire database identified 10426 trials with no first rocker, 5188 for the right side and 5238 for the left side. After eliminating the outliers, the selected trials corresponded to 41% of 2287 sessions with 1570 patients under 20 years old, 54% for both sides, 20% for only the left and 26% for only the right. The percentages of toe-walkers in each disease category are presented in Fig. 2. These percentages are over 50% for four of the diseases and the control group: cerebral palsy (62%), myopathy (51%), idiopathic toe-walking (83%), arthrogryposis (65%) and able-bodied children walking on their toes (100%). The percentages drop under 50% for the other diseases: neuropathy (44%), clubfoot (29%), spina bifida (18%), gait with internal rotation (17%) and pes cavus (20%). Only two diseases presented a proportion of toe-walkers under 10%: scoliosis (6%) and agenesis (3%).

The partition coefficient was used to determine the number of toe-walker clusters; in this case, there was a

Table 1
Mean and standard deviation of angles, moments and power peaks charted in Fig. 3 and spatio-temporal parameters

Code	Variable	Type	Parameter	Phase	Unit	Group 1		Group 2		Group 3		Statistical difference		
						Mean	S.D.	Mean	S.D.	Mean	S.D.	G1 vs. G2	G1 vs. G3	G2 vs. G3
HR1	Pelvis	Angle	Mean	AC	°	14.59	7.64	16.07	7.79	15.98	7.25	*	–	–
H1	Hip	Angle	Value	IC	°	39.15	9.91	42.69	11.44	43.23	9.35	*	*	–
H2	Hip	Angle	Max. Ext.	AC	°	1.14	11.83	0.76	13.13	0.04	9.31	–	–	–
H3	Hip	Angle	Max. Flex.	SwP	°	43.02	9.92	46.06	11.30	46.68	9.60	*	*	–
K1	Knee	Angle	Value	IC	°	22.17	14.52	31.92	17.53	33.16	12.90	*	*	–
K2	Knee	Angle	Max. Flex.	LR	°	27.70	13.68	35.80	15.73	35.53	11.77	*	*	–
K3	Knee	Angle	Max. Ext.	MS	°	15.85	14.98	17.12	18.76	16.22	13.17	–	–	–
K4	Knee	Angle	Max. Flex.	SwP	°	60.14	10.58	62.43	10.28	59.78	8.81	*	–	*
A1	Ankle	Angle	Value	IC	°	−4.68	10.21	−6.40	10.39	−5.23	10.31	*	–	–
A2	Ankle	Angle	Max. Dorsi.	LR	°	5.46	9.16	5.76	9.91	6.69	9.76	–	–	–
A3	Ankle	Angle	Max. Dorsi.	AC	°	14.21	9.82	5.98	9.99	7.49	9.82	*	*	–
A4	Ankle	Angle	Max. Plantar.	SwP	°	−9.72	12.26	−21.82	13.40	−15.62	12.87	*	*	*
A5	Foot progression	Angle	Mean	AC	°	−8.90	14.62	−0.91	15.50	0.62	11.54	*	*	–
MH1	Hip	Moment	Max. Ext.	LR	N m/kg	0.83	0.41	0.87	0.44	1.03	0.51	–	*	*
MH2	Hip	Moment	Max. Flex.	MS	N m/kg	−0.76	0.29	−0.82	0.28	−0.82	0.30	*	*	–
MH3	Hip	Moment	Max. Ext.	SwP	N m/kg	0.39	0.16	0.46	0.18	0.49	0.19	*	*	–
MK1	Knee	Moment	Max. Flex.	LR	N m/kg	−0.27	0.22	−0.22	0.25	−0.18	0.23	*	*	–
MK2	Knee	Moment	Max. Ext.	LR	N m/kg	0.40	0.34	0.46	0.46	0.52	0.43	–	*	–
MK3	Knee	Moment	Max. Flex.	MS	N m/kg	0.09	0.23	−0.04	0.30	−0.21	0.28	*	*	*
KM4	Knee	Moment	Max. Ext.	TS	N m/kg	0.34	0.26	0.43	0.38	0.45	0.31	*	*	–
MA1	Ankle	Moment	Max. Ext.	LR	N m/kg	0.77	0.29	1.25	0.32	1.26	0.37	*	*	–
MA2	Ankle	Moment	Max. Ext.	TS	N m/kg	1.14	0.29	0.99	0.29	1.11	0.30	*	–	*
PH1	Hip	Power	Max. Gen.	LR	W/kg	0.58	0.60	0.81	0.66	1.03	0.74	*	*	*
PH2	Hip	Power	Max. Abs.	MS	W/kg	−0.83	0.45	−0.99	0.45	−0.97	0.50	*	*	–
PH3	Hip	Power	Max. Gen.	SwP	W/kg	0.62	0.44	0.81	0.56	0.87	0.60	*	*	–
PK1	Knee	Power	Max. Gen.	StP	W/kg	0.32	0.44	0.42	0.64	0.75	0.73	–	*	*
PK2	Knee	Power	Max. Abs.	StP	W/kg	−0.58	0.52	−0.64	0.50	−0.67	0.57	–	–	–
PA1	Ankle	Power	Max. Abs.	LR	W/kg	−0.43	0.39	−1.57	0.87	−1.60	0.88	*	*	–
PA2	Ankle	Power	Max. Gen.	MS	W/kg	−0.08	0.19	0.84	0.48	0.80	0.56	*	*	–
PA3	Ankle	Power	Max. Abs.	MS	W/kg	−0.40	0.28	0.20	0.31	−0.48	0.35	*	–	*
PA4	Ankle	Power	Max. Gen.	TS	W/kg	1.45	0.92	1.31	0.91	1.33	0.66	*	*	–
T1	Stance phase				%GC	63.50	5.00	62.22	4.20	63.38	3.79	*	–	*
T2	Swing phase				%GC	36.50	5.00	37.78	4.20	36.62	3.79	*	–	*
T3	Stride length				cm	91.81	20.50	92.04	17.56	93.97	16.65	–	–	–
T4	Stride time				S	1.06	0.18	0.93	0.14	0.95	0.12	*	*	–
T5	Cadence				stride/s	0.96	0.16	1.10	0.16	1.07	0.14	*	*	*
T6	Speed				cm/s	88.99	23.99	100.61	20.08	99.61	17.37	*	*	–

Max.: maximum; Min.: minimum; Flex.: flexion; Ext.: extension; S.D.: standard deviation; AC: all cycles; StP: stance phase; SwP: swing phase; IC: initial contact; LR: loading response; MS: mid-stance; TS: terminal stance; Gen.: generation; Abs.: absorption; GC: gait cycle.
* Significant at $p < 0.01$.

maximum for three groups. Fig. 3c presents the three groups of toe-walkers based on the pattern of the ankle kinematics during the stance phase, as identified by fuzzy c-means clustering. The first group (G1) showed a long and progressive dorsiflexion, followed by plantarflexion until toe-off. The second group (G2) presented a short-lived dorsiflexion, followed by a progressive plantarflexion until toe-off. The third group (G3) exhibited a double bump pattern, moving successively from a short-lived dorsiflexion (as in the second group), to a short-lived plantarflexion, back to a short-lived dorsiflexion and ending with a plantarflexion until toe-off.

Forty-four percent (633) of the sessions (with each side considered independently) belonged to G1, 19% (268) belonged to G2, 16% (232) belonged to G3 and 21% had a mean membership under 0.5. Over 50% of those with clubfoot, scoliosis, myopathy, neuropathy, osteochondritis, spina bifida and arthrogryposis fell into group G1. A high percentage of idiopathic toe-walkers (44%) belonged to G2. Cerebral palsy patients were more evenly spread through the three groups: 36% in G1, 23% in G3 and 19% in G2. A high percentage (45%) of the control sessions—able-bodied persons walking on their toes—presented a membership under 0.5, though 25% of the sessions could be classified as G2, 20% as G3, and 10% as G1 (see Fig. 4 for disease distributions by group).

Spatio-temporal parameters and the sagittal kinematics and kinetics of the ankle, knee and hip indicated significant differences between the groups (Table 1). Spatio-temporal results indicated a lower stance phase (T1) and consequently a higher swing phase (T2) for G2, compared to G1 and G3. Stride time (T4) and speed (T6) were lower for G1. Cadence

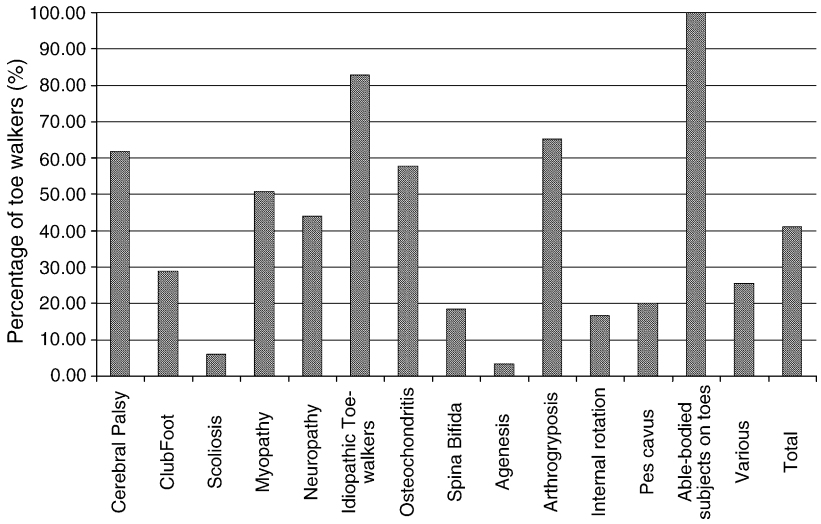

Fig. 2. Prevalence of toe-walking deviation in each disease.

(T5) was significantly different for the three groups. There was no difference in stride length (T3). The mean pelvic tilt (HR1) was slightly lower for G1 than it was for G2.

Plantarflexion at initial contact (A1 in Fig. 3c) was slightly lower for G1, compared to G2. The maximal dorsiflexion in the loading response phase (A2) did not present significant differences. Maximal dorsiflexion (A3) was higher for G1 compared to G2 and G3. Maximum plantarflexion (A4) showed a significant difference in the three groups. The mean angle foot progression (A5) was quasi-neutral for G2 and G3, whereas G1 displayed an external angle. The internal moment at the ankle (MA1 in Fig. 3f) showed a higher peak during loading response for G2 and G3 in comparison with G1. This indicates that the application point of the ground reaction force remained in the forefoot, and certainly in the mid-foot for G1. The flexion moment in the terminal stance (MA2) showed higher values for G1 and G3 than G2. Ankle power (Fig. 3i) was different for the three groups. The energy absorption during loading response (PA1) and the energy generation (PA2) were higher for G2 and G3 compared to G1. In mid-stance, G1 and G3 presented absorption whereas G2 presented energy generation (PA3). Energy generation in terminal stance (PA4) had higher values in G1 compared to G2 and G3.

At the knee joint (Fig. 3b), flexion at initial contact (K1) and maximum flexion during loading response (K2) was higher for G2 and G3 compared to G1. Minimal flexion (K3) displayed no significant differences in mid-stance. Maximal flexion in the swing phase (K4) was slightly higher for G2 compared to G1 and G3. The internal extensor moment during loading response (MK2) showed higher values for G1 than G3. The flexor moment at initial contact (MK1) and the extensor moment in terminal stance (MK4) displayed lower values for G1 than for G2 and G3. There were significant differences in the extensor moment at mid-stance (MK3) for the three groups. In terms of power, the maximal energy generation (PK1) was higher for G3 than for the others, but there was no difference for the minimal energy generation (PK2).

At the hip joint (Fig. 3a), G1 presented a lower flexion at initial contact (H1) and in the swing phase (H3) than did G2 and G3. The minimal flexion in mid-stance (H2) presented no significant differences. G3 presented a higher extensor moment at initial contact (MH1). The maximal flexion moment in the stance phase (MH2) and the maximal extensor moment in the swing phase (MH3) were lower for G1 than for G2 and G3. In terms of moment, generation at initial contact (PH1) was significantly different for the three groups. The maximal absorption in stance phase (PH2) and the maximal generation at the end of the stance phase (PH3) showed lower values for G1 than for G2 and G3.

4. Discussion

Although fuzzy c-means has already been used in gait analysis [27], to our knowledge, data-mining—defined as extraction of knowledge in large databases—has not yet been used to characterize gait deviations. However, such scans of large databases has often been used in other medical fields, such as oncology and genetics [35,36]. Nonetheless, this data-mining method identified three toe-walking ankle patterns in a variety of diseases, and revealed that toe-walking is a major gait deviation (prevalence $>50\%$) for patients with cerebral palsy, myopathy, arthrogryposis, as well as for idiopathic toe-walkers.

Many classifications exist in cerebral palsy, either based principally on the knee [37,38] or ankle position [4,25,39]. Group 3 of our study resembles the apparent equinus described by Rodda et al. [4], group IV described by Winters et al. [39], and the double bump ankle group of O'Byrne et al. [25]. Rodda et al. [4] thought the cause of toe-walking in apparent equinus was excessive flexion of the knee and

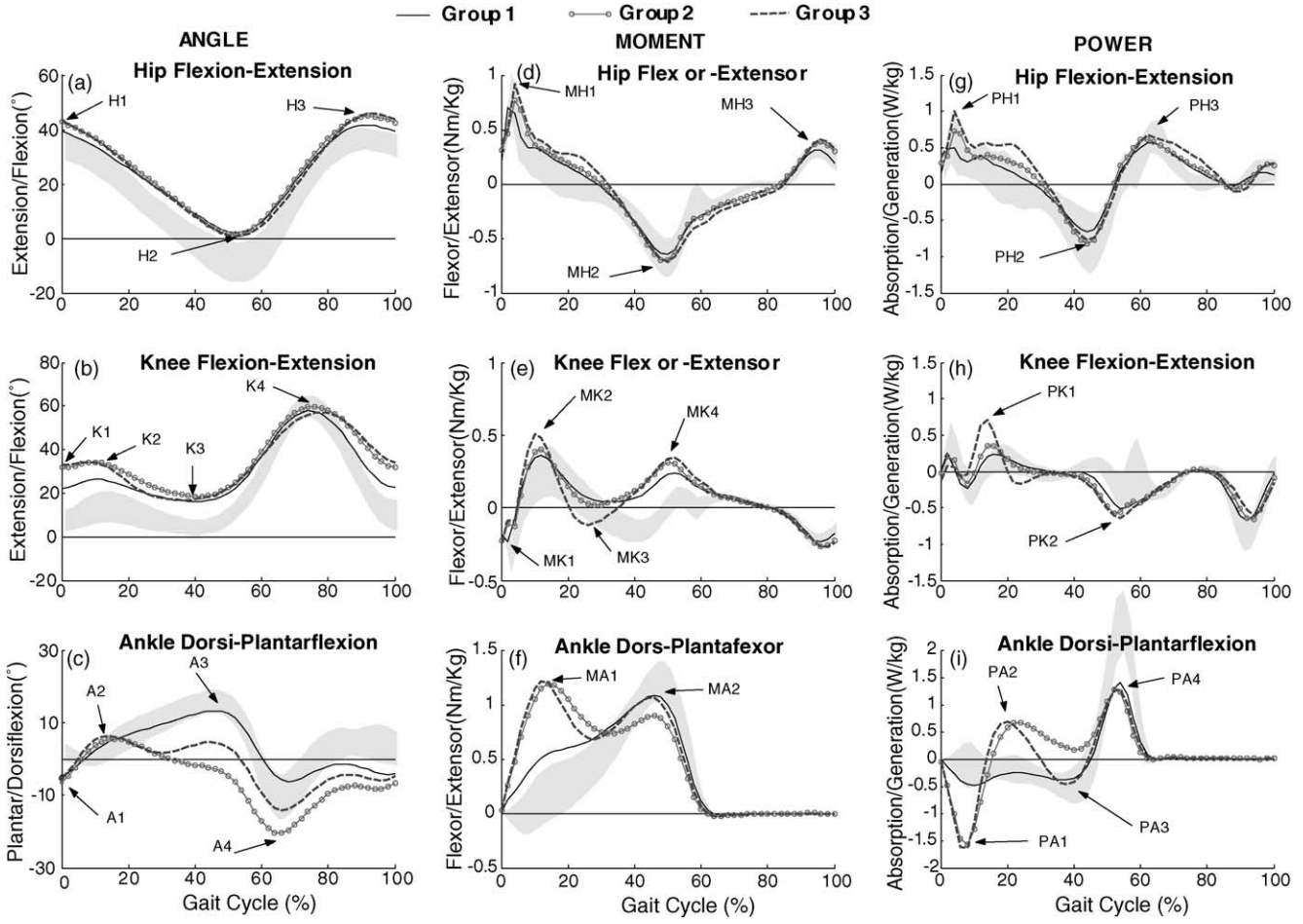

Fig. 3. Toe-walker joint angles (a–c), internal moments (d–f) and powers (g–i) in the sagittal plane for the ankle (c, f, i), for the knee (b, e, h) and for the hip (a, d, g). Mean ± 1 standard deviation forms the normal bandwidth (shadow) for normal subjects. Mean of the three patterns identified with the fuzzy c-mean algorithm are plotted with a black line for group 1, a light grey line marked with circles for group 2 and a dark grey dotted line for group 3. The characteristic curve instances referred to in the results are indicated with arrows.

hip. Our results also show excessive flexion at hip and knee for all three groups, although it was more substantial for groups 2 and 3. Such excessive flexion may be either the cause or the consequence of toe walking, and further investigations will be necessary to establish which it really is. Our group 2 corresponds to the true equinus and the jump gait of Rodda et al. [4] and to Winters' group III. Our group 1 is similar to Winters' group I and group II, and has the same pattern as the crouch gait of Rodda et al., only with more dorsiflexion.

Lin et al. [37] reported moments and powers according to cerebral palsy groups. Ankle plantarflexion moment values in our study gave groups 2 and 3 a double bump pattern, with higher values for group 3 at the end of the stance phase. The only pattern with a double bump in [37] was the jump gait. Our group 3 corresponds to Lin's jump group for power at the ankle, but none of Lin's groups resemble our group 2 with a permanent power generation at 40% of the gait cycle.

Idiopathic toe-walkers (35 subjects) were the most prevalent in our group 2. Hicks et al. [8] have reported an ankle pattern in idiopathic toe-walkers that is similar to our group 2 pattern, with a progressive plantarflexion in mid-stance. In addition, these authors described an ankle pattern in cerebral palsy that is close to our group 1 pattern. The low number of patients in the Hicks et al. study, seven per disease, could explain the fact that they only found a permanent dorsiflexion pattern in cerebral palsy.

There was a high prevalence of myopathic patients (79 subjects) in our group 1. Toe-walking in myopathy has been attributed to compensation for weakness in the quadriceps, fibrosis of the triceps surae and weakness in the tibialis anterior. A number of authors [11–13] have agreed that equinus increases progressively as the disease evolves. The ankle patterns described in these articles correspond to our group 1.

The gait of patients suffering from arthrogryposis has not, to our knowledge, been described in the literature. The arthogryoposis patients (11 subjects) were almost all members of our group 1. More investigations will be necessary to explain their complex gait, and patients often display little range of movement in each joint and a substantial external rotation of the lower limb. Toe walking could be

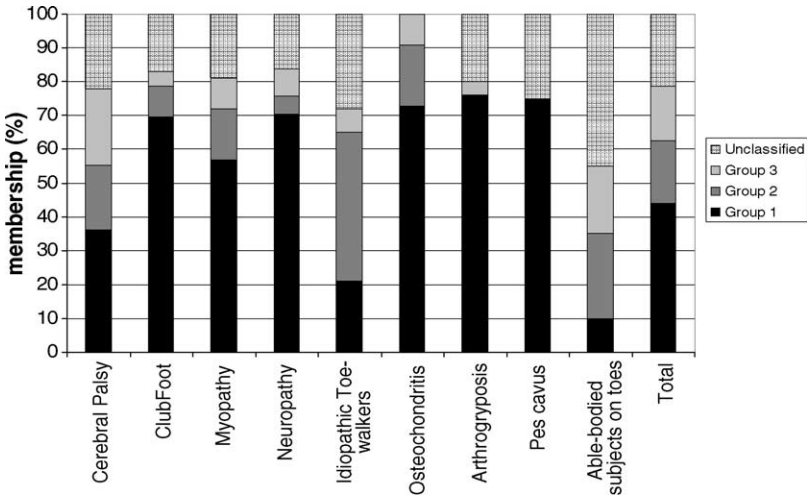

Fig. 4. Group membership per disease. The three groups were identified with a fuzzy c-means algorithm. The unclassified group corresponds to sessions presenting a mean membership inferior to 0.5. Diseases with less than 20% of toe-walkers (Fig. 2) were not reported.

explained by contracture of the triceps surae and fixed equinus associated with weakness in the tibialis anterior.

The gait linked to neuropathy has been described as fore–foot contact associated with premature activity in the triceps surae [14]. The fore–foot contact seems to increase the feeling of safety for those lacking physical self-assurance, such as the elderly [40]. Membership in group 1 was very high for patients suffering from neuropathy (38 subjects).

The proportion of unclassified sessions for able-bodied subjects walking on their toes (10 subjects) could be the consequence of an unreliable gait caused by able-bodied subjects not being used to walking on their toes. In a same session, sometimes a subject displayed pattern 2, and sometimes pattern 3. The mean ankle kinematics for able-bodied subjects on their toes reported by Davids et al. [9] are on the borderline of our groups 2 and 3. It is possible that some of the trials in this study corresponded to group 2 while others corresponded to group 3, with the mean of all trials resulting in the curve presented. However, Perry et al. [1] reported an ankle kinematic curve that corresponds to our group 3.

Interestingly, our group 2 had a pattern similar to the ankle pattern of stair ascent during the stance phase described by Riener et al. [41]. For these authors, forefoot contact during ascent permitted the ankle to be placed in a natural angular range. Idiopathic toe-walkers have been shown to display limited dorsiflexion [5,16], but their available range of passive dorsiflexion appears to exceed that of ambulation [42]. Research by Gordon et al. [43] has demonstrated that muscle tension depends on the degree of overlap between myosin and actin filaments. It is possible that toe walking during stair ascent, or in patients with short triceps surae, such as idiopathic toe-walkers, provides an angle range at the ankle corresponding to maximum force production in the sarcomere length–tension curve. In most cases, idiopathic toe-walkers and persons ascending stairs are able to strike the floor with the heel, but they choose forefoot contact instead. Such a choice could be also due to a more comfortable ankle position, with no relationship to the strength–length curve.

The following hypotheses could be offered to explain the links between our ankle patterns and clinical characteristics. In toe-walking cerebral palsy patients, Tardieu et al. [10] discerned two groups using the ratio between the passive moment and the total internal moment. The first group was characterized by an excessive contraction of the triceps surae muscle and the second group by a contracture of the tendon–muscle complex. Unfortunately, this article did not report ankle kinematics, but Tardieu's groups could correspond to our groups 2 and 3. Group 3 could be linked to excessive muscle activity and spasticity, and group 2 could be linked to contractures that are entirely, or at least partly, responsible for toe walking. Lack of a heel rocker in group 1 could be due to the weakness of dorsiflexor muscles [2]. Other toe-walking causes could be related to attempts to compensate for joint and muscle disorders above the ankle [10] or to the search for a compensatory advantage when walking [17].

Pattern identification could be a useful way to help make therapeutic choices. If group 2 corresponds to triceps surae stiffness caused by a fixed contracture, an intramuscular gastrosoleus lengthening might be appropriate [21]. If group 3 corresponds to triceps surae spasticity or ''dynamic equinus'', botulinum toxin injection or/and serial casts could have a positive effect [20]. If group 1 corresponds to weakness in dorsiflexor muscles or in the muscle above the ankle, it might be interesting to reinforce these muscles to decrease the progression of the weakness, thus allowing the gait to be maintained as long as possible [11], or to increase muscle strength, thus allowing a more comfortable gait.

This study has revealed some consistent biomechanical patterns but further investigations will be necessary to link the three group's gaits to their causes and clinical

examination. Electromyography could potentially be used for this. In the future it would be interesting to examine the relationship between the groups and the results of the different therapeutic treatments for various toe-walking patients.

5. Conclusion

Prevalence of toe walking in a large database confirmed that toe walking is a major gait deviation. Despite examining differing diseases, three major consistent ankle patterns emerged. Identification of clinical causes of these toe-walking patterns could improve gait analysis interpretation and influence therapeutic management. Some hypotheses of causes may explain our findings in part but they need to be tested in further studies.

References

[1] Perry J, Burnfield JM, Gronley JK, Mulroy SJ. Toe walking: muscular demands at the ankle and knee. Arch Phys Med Rehabil 2003;84:7–16.
[2] Perry J. Gait analysis normal and pathological function. Thorofare (NJ) Slack 1992.
[3] Papariello SG, Skinner SR. Dynamic electromyography analysis of habitual toe-walkers. J Pediatr Orthop 1985;5:171–5.
[4] Rodda JM, Graham HK, Carson L, Galea MP, Wolfe R. Sagittal gait patterns in spastic diplegia. J Bone Joint Surg Br 2004;86:251–8.
[5] Rose J, Martin JG, Torburn L, Rinsky LA, Gamble JG. Electromyographic differentiation of diplegic cerebral palsy from idiopathic toe walking: involuntary coactivation of the quadriceps and gastrocnemius. J Pediatr Orthop 1999;19:677–82.
[6] Policy JF, Torburn L, Rinsky LA, Rose J. Electromyographic test to differentiate mild diplegic cerebral palsy and idiopathic toe-walking. J Pediatr Orthop 2001;21:784–9.
[7] Kelly IP, Jenkinson A, Stephens M, O'Brien T. The kinematic patterns of toe-walkers. J Pediatr Orthop 1997;17:478–80.
[8] Hicks R, Durinick N, Gage JR. Differentiation of idiopathic toe-walking and cerebral palsy. J Pediatr Orthop 1988;8:160–3.
[9] Davids JR, Foti T, Dabelstein J, Bagley A. Voluntary (normal) versus obligatory (cerebral palsy) toe-walking in children: a kinematic, kinetic, and electromyographic analysis. J Pediatr Orthop 1999;19:461–9.
[10] Tardieu C, Lespargot A, Tabary C, Bret MD. Toe-walking in children with cerebral palsy: contributions of contracture and excessive contraction of triceps surae muscle. Phys Ther 1989;69:656–62.
[11] Armand S, Mercier M, Watelain E, Patte K, Pelissier J, Rivier F. A comparison of gait in spinal muscular atrophy, type II and Duchenne muscular dystrophy. Gait Posture, in press.
[12] Patte K, Pélissier J, Bénaim C, Laassel EM, Guibal C, Echenne B. Gait analysis in Duchenne muscular dystrophy. Ann Readapt Med Phys 2000;43:53–68 [in French].
[13] Sutherland DH, Olshen R, Cooper L, Wyatt M, Leach J, Mubarak S, et al. The pathomechanics of gait in Duchenne muscular dystrophy. Dev Med Child Neurol 1981;23:3–22.
[14] Kwon OY, Minor SD, Maluf KS, Mueller MJ. Comparison of muscle activity during walking in subjects with and without diabetic neuropathy. Gait Posture 2003;18:105–13.
[15] Karol LA, Concha MC, Johnston CE. Gait analysis and muscle strength in children with surgically treated clubfeet. J Pediatr Orthop 1997;17:790–5.
[16] Kalen V, Adler N, Bleck EE. Electromyography of idiopathic toe walking. J Pediatr Orthop 1986;6:31–3.
[17] Kerrigan DC, Riley PO, Rogan S, Burke DT. Compensatory advantages of toe walking. Arch Phys Med Rehabil 2000;81:38–44.
[18] Orendurff MS, Aiona MD, Dorociak RD, Pierce RA. Length and force of the gastrocnemius and soleus during gait following tendo Achilles lengthenings in children with equinus. Gait Posture 2002;15:130–5.
[19] Saraph V, Zwick EB, Steinwender C, Steinwender G, Linhart W. Conservative management of dynamic equinus in diplegic children treated by gait improvement surgery. J Pediatr Orthop B 2001;10:287–92.
[20] Sutherland DH, Kaufman KR, Wyatt MP, Chambers HG, Mubarak SJ. Double-blind study of botulinum A toxin injections into the gastrocnemius muscle in patients with cerebral palsy. Gait Posture 1999;10:1–9.
[21] Steinwender G, Saraph V, Zwick EB, Uitz C, Linhart W. Fixed and dynamic equinus in cerebral palsy: evaluation of ankle function after multilevel surgery. J Pediatr Orthop 2001;21:102–7.
[22] Stricker SJ, Angulo JC. Idiopathic toe walking: a comparison of treatment methods. J Pediatr Orthop 1998;18:289–93.
[23] Zwick EB, Leistritz L, Milleit B, Saraph V, Zwick G, Galicki M, et al. Classification of equinus in ambulatory children with cerebral palsy—discrimination between dynamic tightness and fixed contracture. Gait Posture 2004;20:273–9.
[24] Vardaxis VG, Allard P, Lachance RMD. Classification of able-bodied gait using 3D muscle powers. Human Mov Sci 1998;17:121–36.
[25] O'Byrne JM, Jenkinson A, O'Brien TM. Quantitative analysis and classification of gait patterns in cerebral palsy using a three-dimensional motion analyzer. J Child Neurol 1998;13:101–8.
[26] Watelain E, Barbier F, Allard P, Thevenon A, Angue JC. Gait pattern classification of healthy elderly men based on biomechanical data. Arch Phys Med Rehabil 2000;81:579–86.
[27] Su F, Wu W, Cheng Y, Chou Y. Fuzzy clustering of gait patterns of patients after ankle arthrodesis based on kinematic parameters. Med Eng Phys 2001;23:83–90.
[28] Mulroy S, Gronley J, Weiss W, Newsam C, Perry J. Use of cluster analysis for gait pattern classification of patients in the early and late recovery phases following stroke. Gait Posture 2003;18:114–25.
[29] Davis RB, Ounpuu S, Tybursky D, Gage JR. A gait analysis data collection and reduction technique. Human Mov Sci 1991;10:575–87.
[30] Siegel KL, Kepple TM, Stanhope SJ. Joint moment control of mechanical energy flow during normal gait. Gait Posture 2004;19:69–75.
[31] Hoaglin DC, Mosteller F, Tukey JW. Understanding robust and exploratory data analysis. New York: Wiley, 1983.
[32] Dunn JC. A fuzzy relative of the ISODATA process and its use in detecting compact well-separated clusters. J Cybernet 1973;3:32–57.
[33] Bezdek JC. Pattern recognition with fuzzy objective function algorithms. New York: Plenum Press, 1981.
[34] Benedetti MG, Catani F, Leardini A, Pignotti E, Giannini S. Data management in gait analysis for clinical applications. Clin Biomech 1998;13:204–15.
[35] Shah SC, Kusiak A. Data mining and genetic algorithm based gene/SNP selection. Artif Intell Med 2004;31:183–96.
[36] Li L, Tang H, Wu Z, Gong J, Gruidl M, Zou J, et al. Data mining techniques for cancer detection using serum proteomic profiling. Artif Intell Med 2004;32:71–83.
[37] Lin CJ, Guo LY, Su FC, Chou YL, Cherng RJ. Common abnormal kinetic patterns of the knee in gait in spastic diplegia of cerebral palsy. Gait Posture 2000;11:224–32.
[38] Sutherland DH, Davids JR. Common gait abnormalities of the knee in cerebral palsy. Clin Orthop 1993;139–47.
[39] Winters Jr TF, Gage JR, Hicks R. Gait patterns in spastic hemiplegia in children and young adults. J Bone Joint Surg Am 1987;69:437–41.

[40] Kerrigan DC, Todd MK, Della Croce U, Lipsitz LA, Collins JJ. Biomechanical gait alterations independent of speed in the healthy elderly: evidence for specific limiting impairments. Arch Phys Med Rehabil 1998;79:317–22.
[41] Riener R, Rabuffetti M, Frigo C. Stair ascent and descent at different inclinations. Gait Posture 2002;15:32–44.
[42] Katz MM, Mubarak SJ. Hereditary tendo Achillis contractures. J Pediatr Orthop 1984;4:711–4.
[43] Gordon AM, Huxley AF, Julian FJ. The variation in isometric tension with sarcomere length in vertebrate muscle fibres. J Physiol 1966; 184:170–92.