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Features of motor stereotype kinematics and kinetics in children with achondroplasia: a comparative cross-sectional study

T.I. Dolganova✉, L.V. Smolkova, D.V. Dolganov, A.M. Aranovich, N.S. Gvozdev, D.A. Popkov

Ilizarov National Medical Research Centre for Traumatology and Orthopedics, Kurgan, Russian Federation

Corresponding author: Tamara I. Dolganova, rjik532007@rambler.ru

Abstract

Introduction The study of the kinematic and kinetic parameters of gait in children with achondroplasia would allow a more detailed understanding of the features of their locomotion and determine the strategy of planned treatment.

Purpose To evaluate features of locomotor kinematics and kinetics in children with achondroplasia and compare with peers without orthopedic pathology.

Materials and methods The locomotor profile was assessed by video gait analysis. Kinematic data were recorded by Qualisys7+ optical cameras (8 cameras) with passive marker video capture technology synchronized with six dynamic platforms KISTLER (Switzerland). The analysis of kinematics and kinetics was carried out in the QTM (Qualisys) and Visual3D (C-Motion) programs with automated calculation of the values of indicators of the total peak power in the joints. Three groups were formed for gait analysis: 1) children 6–7 years old, achondroplasia (6 subjectsle, $n = 12$ limbs); 2) their peers, children without orthopedic pathology, 6–7 years old (8 subjects, $n = 16$ limbs); 3) children without orthopedic pathology 3–4 years old similar in height (8 subjects, $n = 16$ limbs).

Results In children with achondroplasia, statistically significant disorder in locomotor kinetics and kinematics were found. The former are associated with a longitudinal deficiency of limb segments and decreased walking speed. The latter are not associated with a longitudinal deficit, but manifested in all planes, namely: an increase in the maximum forward inclination of the pelvis, a flexion position in the hip and knee joints, and dorsal flexion of the ankle joint; increased maximum angle of hip abduction and varus deformity of the knee joint; increased rotational range of motion of the pelvis.

Discussion Since the characteristic features of the main gait profile begin to appear in children by the age of 4–5 years, and is associated with the formation of the activity of central and spinal generators that induce the self-organization of motor stereotypes, we believe that the deviations detected in the locomotor kinematics are secondary pathogenetic manifestations of the kinetics due to the longitudinal deficit in limb segments.

Conclusion Features of locomotor kinetics in children with achondroplasia are due to the longitudinal deficit of the limb length and are associated with low walking speed. Significant deviations of the locomotor kinematics were not associated with the longitudinal deficit of the segments, but were detected in all planes and are related to the entire biomechanical chain.

Keywords: achondroplasia, healthy children, gait analysis, locomotor kinetics and kinematics, limb lengthening

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INTRODUCTION

Achondroplasia is a skeletal dysplasia, a genetic disease with an incidence of 3.7–4.6 per 100 thousand newborns [1, 2]. Disproportional body constitution in achodroplasia is characterized, among other things, by a violation of the coefficient of proportionality between the height and body weight: a significant selective lag behind healthy peers in long bone growth of the lower extremities and to a less extent in the growth of the contractile part of the muscles, but complete preservation of their contractile properties [3, 4, 5].

It should be considered that in children under 5 years of age, the age when the maximum muscle strength parameters are recorded, the anatomical and functionally optimal lengths of the limbs do not coincide. Longitudinal muscle growth in young children may runs independently of bone growth, while in adolescents it is largely influenced by bone traction [6].

Other gait features include excessive abduction of the femurs combined with a flexion position in the hip joints, hypermobility of the knee joints in the frontal plane with the formation of varus deformity of the knee joint due to the tibia [7] and its recurvatum [8, 9].

The above features in combination with a wide pelvis, varus and torsion deformities of the lower legs, thoracolumbar kyphosis, lumbar lordosis, and reduced muscle strength together lead to delayed motor development and impaired walking function [2, 9, 10, 11, 12]. The consequence of biomechanical disorders is an inevitable increase in energy consumption by walking: increased oxygen consumption and intensified metabolism during motor tests [13]. In the literature, kinetic analysis of gait in children with achondroplasia, including joints, have been reported without comparison with the control group [10, 14].

The number of works that studied the kinematic and kinetic features of gait in children with achondroplasia is limited and insufficient [8, 15]. Uncertainty adds to the observation of a weak correlation between radiological and kinematic parameters describing angular deformities. The standard for gait assessment is three-dimensional computer-assisted gait analysis (3DGA). A combination of kinetic and kinematic parameters of the general gait pattern is recommended as an expert level for objective documentation of detected changes [16, 17].

The study of kinematic and kinetic parameters of gait in children with achondroplasia would allow to understand details of locomotion in such a disproportional condition, especially in comparison with peers (by age) and children without skeletal dysplasia but with the closest definitive dimensions of the lower limb segments, and to monitor changes in the parameters during growth of the children that undergo pathogenetic pharmacological treatment [10, 18, 13].

The purpose of the work was to assess the characteristics of locomotor kinematics and kinetics in children with achondroplasia in comparison with their peers without orthopedic pathology.

MATERIALS AND METHODS

The locomotor profile was assessed using computer gait analysis (CGA) in 6 children (12 limbs) with achondroplasia in an inpatient setting. Inclusion criteria were a confirmed diagnosis of achondroplasia, age 6–7 years, and no previous pathogenetic pharmacological or orthopaedic surgical treatment. The comparison groups included 16 children without orthopaedic pathology. Healthy children were selected for examination according to the criterion of a similar age (6–7 years) and or to the criterion of close “standing height” in a group of children 3–4 years old as far as examination of younger children to study gait parameters is technically difficult. The examined children underwent a computer analysis of walking parameters at the Ilizarov Center Gait Analysis Laboratory. They walked independently, barefoot, on a 7-meter platform at their usual speed.

There were three groups in the study:

Group I, children in the age of 6 to 7 years with achondroplasia (6 subjects, 12 limbs);

Group II, children in the age of 6 to 7 years without achondroplasia (8 subjects, 16 limbs);

Group III, children in the age of 3 to 4 years without achondroplasia who had the closest height with the affected children in standing position (8 subjects, 16 limbs).

Kinematic data were recorded using Qualisys 7+ optical cameras (8 Qualisys cameras) with passive marker video capture technology; synchronized with six KISTLER dynamometer platforms (Switzerland). For setting the markers, the IOR model was used. Analysis of kinematics and kinetics was carried out in the QTM (*Qualisys*) and Visual3D (*C-Motion*) programs with automated calculation of values [19]. Indicators of the total (generation + relaxation) peak power of the joints were calculated [20]; the total general peak power being the sum of the absolute values of generation and relaxation; values of useful peak power being the difference between the absolute values of generation and relaxation on the kinetics graphs [21]. Overall mechanical efficiency was defined as the ratio of positive (useful) peak power to total power [22].

The AtteStat 12.0.5 program was used for statistical data processing [23]. Due to the number of subjects in the groups, nonparametric statistics were used to process the results, accepting a significance level of $p \leqslant 0.05$. Quantitative characteristics of the sample populations are presented in the table as medians with a percentile distribution level of 25÷75% and a number of cases (*n*) equal to the number of limbs. The statistical significance of differences was determined using the unpaired Wilcoxon test.

A permission to conduct the study was obtained from the Ethics Committee of the National Ilizarov Medical Research Center for Traumatology and Orthoapedics (protocol dated October 21, 2021 No. 2(70)). The studies were conducted in accordance with the ethical standards of the Declaration of Helsinki of the World Medical Association "Ethical Principles for Medical Research Involving Human Subjects" as amended in 2000, and the "Rules of Clinical Practice in the Russian Federation" approved by the Order of the Ministry of Health of the Russian Federation dated June 19, 2003 No. 266. The parents of children who participated in the study were present during the tests and gave informed consent for its conduct and publication of research results without personal identification.

RESULTS

Table 1 presents anthropometric data of children of all three groups. The height of patients with achondroplasia was significantly different not only from their peers (by 8.8 σ), but also from children aged 3–4 years (by 2.3 σ). The weight of children and the length of the lower limbs differed significantly only with the group of peer children; there were no significant differences with the group of 3-to-4-year old children.

Table 2 shows the spatiotemporal parameters of gait.

The presented data show that the length of the gait cycle and walking speed in the group of children with achondroplasia were significantly reduced only in comparison with the second group (peers 6–7 years old), which is obviously associated with a significantly shorter length of the lower limbs.

The parameters of movement kinematics are presented in Table 3.

Table 1

Anthropometric data of the examined children

Parameter	Group I (*n* = 6)	Group II (*n* = 8)	Group III (*n* = 8)
Height, cm	97.5 (90÷98,0) *P^2 = 9.19E-6* *P^3 = 0.00169*	120 (117 ÷ 122)	102 (102 ÷ 103)
Weight, kg	17.5 (16.1÷19.0) *P^2 = 0.0347*	23,0 (19,6 ÷ 25,8)	15.4 (14.9÷17.4)
Lower limb length, cm	42.0 (38.0÷44.0) *P^2 = 1.41E-05*	54.5 (52.0÷56.0)	44.0 (42.4÷45.0)

Note: P^2 — level of significance by comparing the parameter of the achondroplasia group with group II; P^3 — level of significance by comparing the parameter of the achondroplasia group with group III

Table 2

Spatiotemporal indices of gait

Parameter	Group I (*n* = 6)	Group II (*n* = 8)	Group III (*n* = 8)
Walking speed, m/sec	0.67 (0.65÷0.68) *P^2 = 0.012*	1.04 (0.97÷1.05)	0.78 (0.72÷0.82)
Length of gait step, m	0.57 (0.53÷0.73) *P^2 = 0.037*	0.97 (0.85÷1.02)	0.74 (0.69÷0.79)
Duration of stance phase, %	62.4 (61.5 ÷ 63.5)	61.9 (61.4 ÷ 62.3)	62.6 (61.7 ÷ 63.8)
Duration of swing phase, %	37.4 (36.3 ÷ 38.3)	38.0 (37.7 ÷ 38.5)	37.4 (36.3 ÷ 38.3)
Duration of double support phase, %	25.3 (22.5 ÷ 28.5)	22.9 (22.5 ÷ 24.6)	25.3 (22.5 ÷ 27.9)
Gait cycles per minute	70.6 (61.7÷72.4)	64.5 (63.3÷65.4)	65.1 (62.1÷66.8)

Note: *P^2* — level of significance by comparing the parameter of the achondroplasia group with group II

Table 3

Kinematic parameters

Parameter	Group I (*n* = 12)	Group II (*n* = 16)	Group III (*n* = 16)
Gait profile score (GPS)	12,3 (11.0÷13.9) *P^2 = 0.00367, P^3 = 0.011*	8,2 (8.0 ÷ 8.4)	9.7 (9.0 ÷ 10.3)
Foot position at initial contact, °	6.6 (3.4÷10.2) *P^2 = 0.00098, P^3 = 0.00011*	−0.3 (−2.6÷1.9)	−2.6 (−3.4÷0.5)
Angle of maximum dorsiflexion of the foot in stance phase, °	16.7 (15.2÷18.2) *P^2 = 0.00015, P^3 = 9.27E-06*	11.6 (10.2÷14.3)	10.6 (8.5÷12.1)
Foot position in swing phase,°	12.5 (7.8 ÷ 14.5) *P^2 = 0.00759, P^3 = 0.00090*	5.7 (2.8 ÷ 6.9)	4.7 (1.8 ÷ 6.6)
Range of plantar flexion, °	20.1 (16.2÷24.1) *P^2 = 0.0009, P^3 = 0.02434*	28.8 (24.6÷32.6) *P^{2-3} = 0.0167*	24.8 (21.8÷25.6)
Supination in stance phase, °	6.1 (1.3÷7.7) *P^2 = 0.0229*	0.7 (−1.8÷2.5)	1.45 (0.45÷4.3)
Angle of foot orientation relative to the movement vector (max value of internal rotation), °	5.8 (1.6÷9.2)	8.2 (4.7 ÷ 13.1)	9.9 (8.0 ÷ 12.6)
Angle of knee flexion at the initial contact of the stance phase;°	6.7 (0,5÷17.2)	3.4 (1.4 ÷ 7.6)	3.7 (0.7 ÷ 5.8)
Angle of peak knee extension in the stance phase,°	10,7 (8,5÷26,7)	5,7 (1,7 ÷ 8,4)	5,3 (2,3 ÷ 9,4)
Angle of maximum flexion in the swing phase , °	72.6 (70.3÷77.7) *P^2 = 0.00187, P^3 = 4.8E-05*	64.6 (61.6÷67.9)	62.8 (60.6÷64.2)
% of gait cycle of maximum knee flexion in swing phase	73.5 (72.0÷76.3)	73.5 (72.7÷74.3)	75.0 (73.0÷75.3)
Varus (+) / valgus (−) of knee joint (max values), °	5.8 (0.0÷11.5) *P^2 = 0.0388, P^3 = 0.00872*	−0.15 (−2.9÷1.2)	−2.0 (−5.0÷2.0)
Angle of hip flexion at the initial contact of stance phase,°	39.9 (33.5÷41.9) *P^2 = 0.0114, P^3 = 0.00173*	29.6 (24.7÷35.0)	27.5 (24.8÷29.6)
Maximum angle of hip extension in stance phase, °	−0.7 (−6.1÷6.6) *P^2 = 0.00236, P^3 = 0.000321*	−11.7 (−15.2÷−7.7)	−13.6 (−16.6÷−7.3)
Angle of maximum hip abduction, °	10.3 (8.9÷ 14.9) *P^2 = 0.00813, P^3 = 0.000763*	4.7 (2.6÷7.8)	6.2 (3.5÷7.3)
Hip joint range of motion, °	40.3 (39.1÷ 47.4)	45.3 (43.4÷48.9)	43.1 (41.3÷44.2)
Femur rotation (max internal rotation), °	8.2 (3.3 ÷ 18.4)	14.6 (7.6 ÷ 16.6)	13.1 (0.1 ÷ 13.9)
Angle of maximum anterior tilt of the pelvis, °	17.1 (14.8÷18.9) *P^2 = 0.000829, P^3 = 5.92E-05*	8.4 (5.3÷12.4)	8.9 (6.4÷10.8)
Range of pelvic rotation by walking, °	27.2 (18.1÷33.3) *P^2 = 0.000529, P^3 = 4.4E-05*	14.2 (12.8÷18.2)	13.1 (12.7÷14.5)

Note: *P^2* — level of significance by comparing the parameter of the achondroplasia group with group II; *P^3* — level of significance by comparing the parameter of the achondroplasia group with group III

In the groups of healthy children, there were significant differences related to age only in the parameter “range of plantar flexion,” which has a correlation with walking speed ($r = 0.412$, $n = 32$, $p < 0.05$). With regard to kinematic parameters in children with achondroplasia (Table 3,

Fig. 1), one can note a significant increase in the integral indicator of the gait profile score (GPS), which is proposed as a quantitative parameter for identifying typical features of the gait nature.

We should focus on the foot in the dorsiflexion position. There is significant supination of the foot in the stance phase, but there are no significant rotational positions of the foot relative to the vector of movements. Varus deformity of the knee joint was detected, associated with deviation of the biomechanical axis of the lower limb, typical of achondroplasia. In healthy children, physiological valgus of the knee joint was found: for 3 to 4-year old children, maximum deviations of the knee joint for valgus of up to 5.0° were recorded, for 5 to 6-year old children it was up to 2.9°.

The flexion position of the femur at the beginning of the stance phase of the gait cycle and a decrease in the angle of femur extension were recorded, which was concordant with the increase in knee joint flexion in the swing phase. There is a significant increase in the anterior tilt of the pelvis and an increase in its rotational movements.

The total gait assessment in the group of patients with achondroplasia also shows a greater deviation from the norm.

In the examined group of patients, knee joint recurvatum which was combined with extension of the femur was detected in only one patient (Fig. 2). In the normal position of the pelvis and a fully preserved range of motion in the ankle joint, the kinetic parameters reflected a pronounced decrease in the strength parameters of the flexors and extensors of the femur and lower leg.

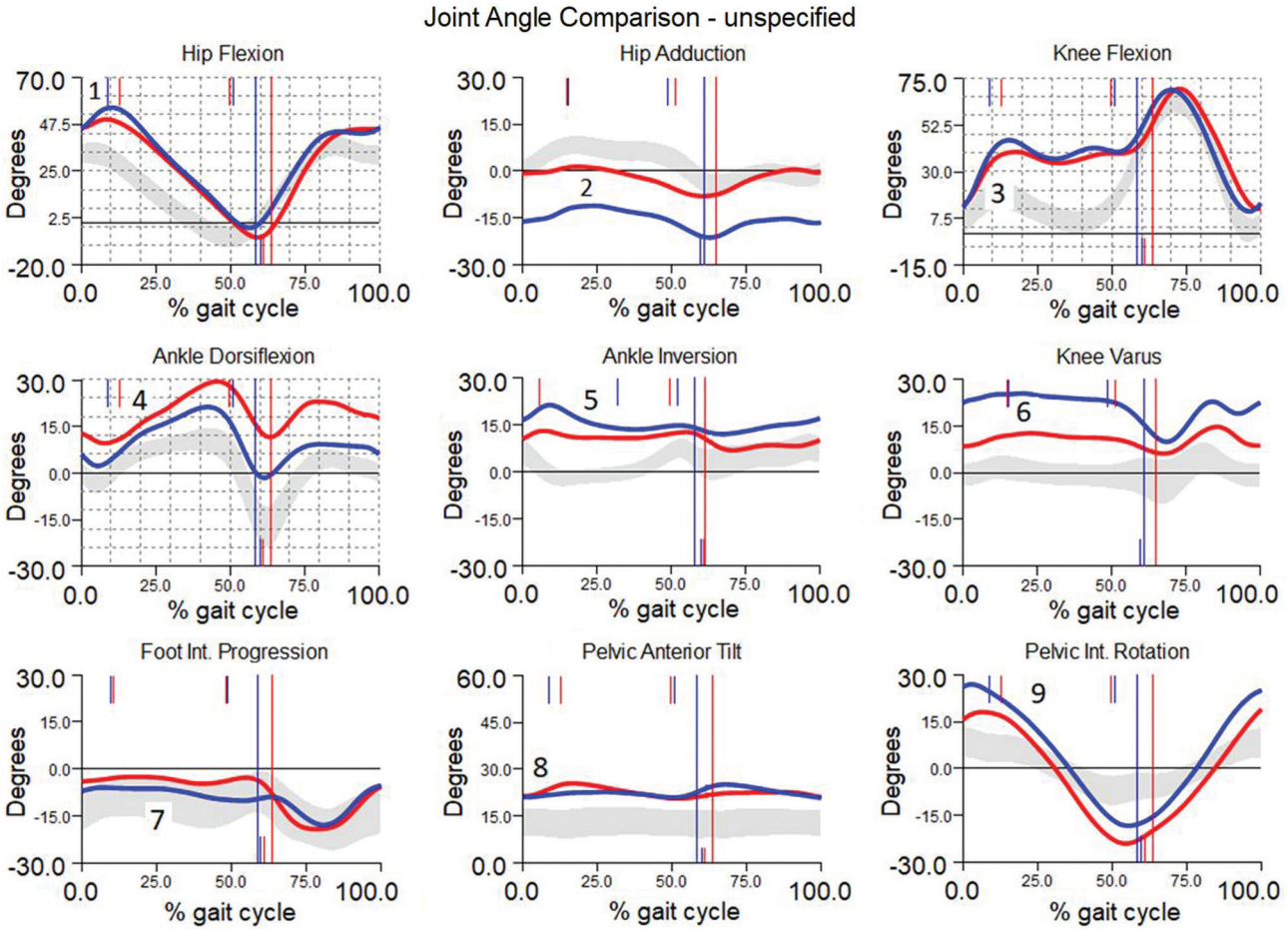

Fig. 1 Example of kinematic graphs showing flexion (1) and abduction of the hip (2), flexion of the knee joint (3), dominant position of the foot in dorsiflexion (4), supination of the foot in the stance phase (5), knee joint varus deformity (6), normal rotational position of the segments relative to the movement vector (7), increased anterior tilt of the pelvis (8) and its rotational movements (9)

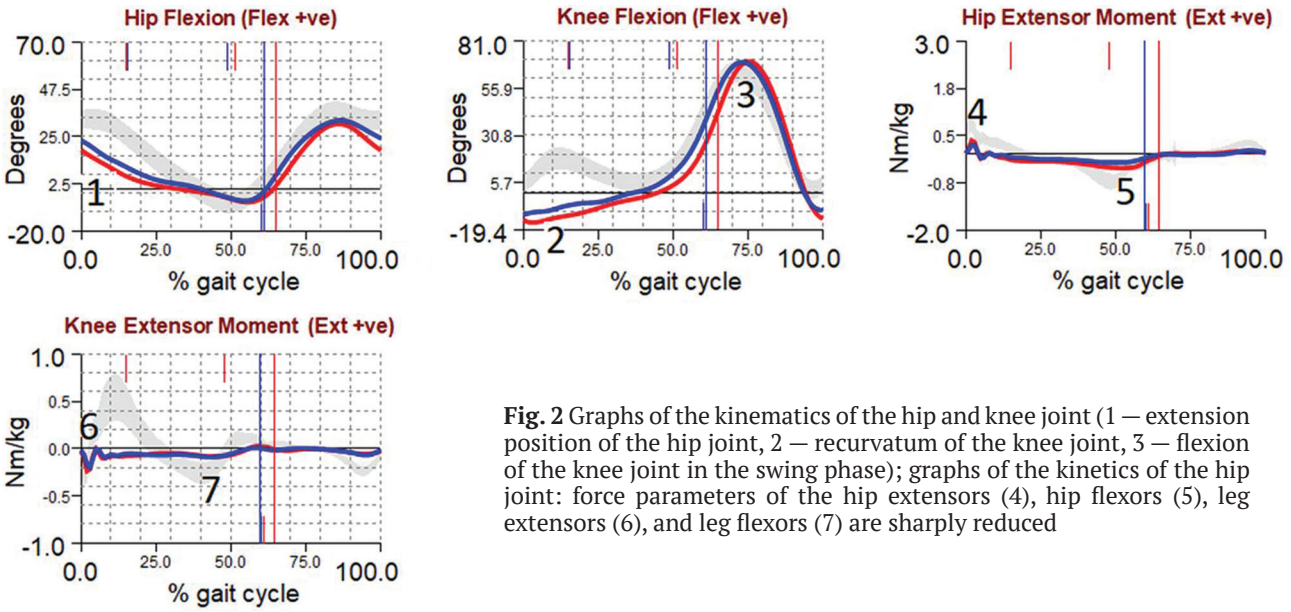

Fig. 2 Graphs of the kinematics of the hip and knee joint (1 — extension position of the hip joint, 2 — recurvatum of the knee joint, 3 — flexion of the knee joint in the swing phase); graphs of the kinetics of the hip joint: force parameters of the hip extensors (4), hip flexors (5), leg extensors (6), and leg flexors (7) are sharply reduced

Tables 4 and 5 present the kinetic parameters of walking in the groups examined.

In the groups of healthy children, a significant age-related increase in the strength parameters of the muscles involved in the motor stereotype was recorded with an increase in walking speed (femur flexors and extensors, femur adductors) and in a heel lift push.

The functional capabilities of the femur extensors and flexors and the lower leg extensors in children with achondroplasia had a significant difference only relative to the peer group (where the mechanical lever is larger). Similar lengths of the segments of the lower extremities (children aged 3–4 years and without orthopedic problems) were not accompanied by significant differences in the strength parameters of these muscle groups. But the indices of the muscle groups responsible for knee flexion and plantar flexion were also significantly reduced in relatively healthy children aged 3–4 years reflecting the influence of the reduced walking speed in such patients. Reduced strength parameters of the adductor muscle group of the femur are a criterion for a decompensated varus deformity of the biomechanical axis of the lower limb.

Table 4

Kinetic parameters of lower limb joints (relative moment of force normalized by weight; N·m/kg)

Parameter	Group I (*n* = 12)	Group II (*n* = 16)	Group III (*n* = 16)
Hip extension	0.36 (0.33÷0.53) $P^2 = 0.00574$	0.62 (0.55÷0.89) $P^{2-3} = 0.00290$	0.49 (0.38÷0.57)
Hip flexion	−0.25 (−0.34÷ −0.21) $P^2 = 0.00218$	−0.48(−0.56÷−0.31) $P^{2-3} = 0.01668$	−0.32 (−0.38÷−0.28)
Hip adduction	0.32 (0.23÷0.37) $P^2 = 1.94E\text{-}05$, $P^3 = 7.2E\text{-}05$	0.62 (0.57÷0.66) $P^{2-3} = 0.02615$	0.53 (0.45÷0.57)
Knee extension	0.24 (0.09÷0.31) $P^2 = 0.03465$	0.34 (0.25÷0.48)	0.31 (0.21÷0.36)
Knee flexion	−0.11 (−0.13÷−0.04) $P^2 = 0.01789$, $P^3 = 0.005327$	−0.18 (−0.26÷−0.12)	−0.18 (−0.24÷−0.11)
Knee extension at heel lift push	0.06 (0.03÷0.11) $P^2 = 0.041$	0.13 (0.09÷0.14) $P^{2-3} = 0.000111$	0.05 (0.03÷0.07)
Dorsal flexion	−0.09 (−0.10÷−0.08)	−0.14 (−0.15÷−0.09)	−0.11 (−0.13÷−0.09)
Plantar flexion (take-off force)	0.58 (0.53÷0.79) $P^2 = 7.2E\text{-}05$, $P^3 = 0.003993$	1.1 (1.05÷1.21) $P^{2-3} = 2.6E\text{-}06$	0.86 (0.83÷0.91)

Note: P^2 — level of significance by comparing the parameter of the achondroplasia group with group II; P^{2-3} — level of significance by comparing the parameter of group II with group III.

Table 5

Indicators of total (generation + relaxation) peak joint power, normalized by weight (W/kg)

Parameter	Group I	Group II	Group III
Hip joint	1.12 (0.86÷1.62)	1.27 (1.05÷1.46)	1.1 (0.94÷1.22)
Knee joint	1.24 (0.83÷1.81)	1.65 (1.46÷2.15)	1.48 (1.30÷1.69)
Ankle joint (push-off)	2.05 (1.41÷2.17) $P^2 = 0.001253$	3.0 (2.62÷4.0) $P^{2-3} = 0.000205$	2.03 (1.85÷2.27)
Total peak power of all limb joints	4.58 (3.31÷6.25) $P^2 = 0.03075$	6.13 (5.38÷7.06)	4.8 (4.05÷5.09)
Useful peak power of all limb joints	0.63 (0.24÷1.01) $P^2 = 0.000763$	1.66 (1.39÷2.73) $P^{2-3} = 0.000974$	0.88 (0.62÷1.27)
Efficiency of joint function, %	58.7 (53.9 ÷61.5) $P^2 = 0.001476$	63.7 (61.8 ÷67.2)	61.0 (58.0 ÷63.4)

Note: P^2 — level of significance by comparing the parameter of the achondroplasia group with group II; P^{2-3} — level of significance by comparing the parameter of group II with group III.

In patients with achondroplasia, the indicators of normalized weight (W/kg) peak power of the push-off, total and useful peak power of joint work were significantly reduced relative to healthy children of the second group, but did not significantly differ from the parameters of healthy 3–4-year old children, where the length that forms the push was almost of the same lever (segment length).

DISCUSSION

Disorders of enchondral osteogenesis in achondroplasia, mainly in the growth plates, lead to pronounced changes in the skeleton, characterized, first of all, by disproportionate short stature [2, 3, 12, 18]. These growth anomalies are reflected, in particular, in altered gait function, accompanied by lower efficiency and increased energy consumption [13, 15, 24].

Relative to weight, the moments of muscle power of the flexors and extensors of the foot are reduced by 15–30 % in patients with achondroplasia, of flexors and extensors of the lower leg by 40–60 % compared with healthy peers [25]. In this pathology due to a shortened lower limb segment, age-related differentiation of the flexor and extensor muscles is delayed: up to 12 years, the maximum strength of the flexor muscles is 30–50 % greater than the maximum strength of the extensor muscles, i.e. the calculated index of antagonistic muscles corresponds to the values of children aged 4–5 years whose segment lengths are approximately equal to those with achondroplasia [26].

Compared with adults, children exhibit lower joint kinetics, speed, and power even after adjusting for age-related dimensional differences due to lower levels of maximum voluntary muscle activation, which is associated with their relative inability to engage or use their “fast twitch” motor fibers, type II [27]. The age-related increase in muscle force parameters in the groups of healthy children is consistent with the data on the kinetics of locomotor stereotypes in healthy children in various speed ranges of movement, where a significant positive correlation was found between walking speed and the total general ($r = 0.907$; $n = 104$) and useful peak power ($r = 0.475$; $n = 104$) of the joint muscles [22].

The temporal parameters of the gait structure determine the walking speed, and the relative duration of the stance phase and the double-support phase decreases with increasing walking speed [28]. Despite the no-lower walking speed in patients with achondroplasia, the temporal parameters of the gait structure (the relative duration of the support, non-support and double-support periods of the gait cycle) do not differ significantly in the groups of patients. The results obtained are consistent with the literature: the predominance of axial deviations with extremely insufficient limb

length determines gait features characterized by shortened stride length, low walking speed [8, 10] and increased rhythm in the absence of differences in the relative walking speed taking into account the length of the limbs [14].

The optimal strategy for improving the condition of children with achondroplasia also requires studying the gait pattern, features of kinematic and kinetic parameters, both from the point of view of treatment planning and for carrying out objective multifactorial monitoring of the effectiveness of therapy [9, 10, 12].

A limited number of studies on the gait of individuals with achondroplasia using instrumental analysis can be found in the literature [8, 10, 24, 29, 30]. Some of the works were carried out to study locomotor function in adults or only after surgical treatment [10, 13, 30, 31]. A number of walking features specific to achondroplasia have been identified by various authors. Kinematic features in the sagittal plane in walking are characterized by a dominant flexion position in the hip, knee and ankle joints (dorsal flexion) [8, 14], which may be a compensatory position due to the anterior tilt of the pelvis. Excessive anterior pelvic tilt may be caused by weakness of hip extensors and abdominal muscles [32]. Structural changes in the lumbar spine (lordosis combined with stenosis) [33, 34, 35] may also contribute to rotation of the pelvis that leads to compensatory adjustments of the underlying segments. Our study also revealed a significant limitation of extension in the hip joint in children with achondroplasia, a decrease in plantar flexion in combination with a significantly greater dorsiflexion in the stance phase of the gait cycle. The newly identified feature is a more pronounced flexion of the knee joint in the swing phase in comparison with the control groups, which can be explained by the flexion position in the hip joint and leads to an increase in passive flexion in the knee joint in the swing phase of the gait cycle.

According to the literature, recurvatum of the knee joint is recorded in patients with achondroplasia [14], which is predisposed by the specific anatomy of the proximal tibia (smaller than normal inclination of the articular surface) [11]. In our study, computer gait analysis in five patients did not reveal recurvatum in the knee joint during the support phase. It is likely that sufficient motor control compensates for this anatomical disorder. In one patient (16% of cases), recurvatum in the knee joint was found, and the kinetics reflected a pronounced decrease in the power parameters of the flexors and extensors of the hip and lower leg (Fig. 2).

A decrease in the magnitude of kinetic parameters (both moments of force and generated power) in the sagittal plane was revealed in patients with achondroplasia in comparison with healthy peers, which is obviously due to the shorter length of the mechanical lever segments. We found that there were no differences when compared with children of similar segment lengths (but younger age). Making comparisons with children aged 3–4 years, it is necessary to consider that at this age the general parameters of the locomotor pattern have not yet been fully formed [36, 37, 38]. The characteristic appearance of the main gait profile begins to appear in children at approximately 4–5 years of age, which may be associated with the formation of the activity of spinal central generators that induce self-organization of motor stereotypes (spinal central pattern generators — CPG). The works of other authors indicate the correspondence of kinetic changes in the sagittal plane to flexion in the joints in patients with achondroplasia [8, 14], but a comparison of the parameters with healthy children with similar segment lengths was not carried out.

It is known that muscle force parameters and peak joint power in patients with achondroplasia are determined by the length of the lever (the length of the limb segment) while their physiological maturity is preserved [39, 40]. A decrease in joint power indicators is also associated with a lower walking speed, because a reliable positive correlation between the power parameters of locomotion and walking speed was found [22].

We believe that the absent differences in the parameters of generated power and moment of force in comparison with younger children with similar lengths of segments of the lower extremities justifies the strategy of lengthening the longitudinal size of long bones (pharmacologically and/or surgically) precisely from the point of view of reducing the gait energy consumption in achondroplasia and, accordingly, an increase in motor capabilities.

The identified changes in kinematics in the horizontal plane are not as pronounced as in the sagittal plane. Noteworthy is the significant increase in the range of pelvic rotation. We regard this feature as compensatory, aimed at increasing stride length. This is also noted by other researchers [8, 31]. The works of Kierman et al [8] and Broström et al [14] describe increased external hip rotation of the femur, which was not found in our patients. In the frontal plane, increased hip abduction was observed in children with achondroplasia. Anatomically, the pelvic organs in children with chondroplasia are vharacterized by a wider and more horizontal acetabulum [9]. Hip flexion position may also contribute to increase in hip abduction and knee varus. Increased hip abduction coupled with hip flexion position also influences frontal plane kinematics with a decrease in hip adduction force vector during walking.

Kierman et al [8] opine that the internal torsion of the lower leg compensates for the external rotation of the femur, and the orientation of the foot, as a result, does not deviate significantly from the patient's movement vector. In our study, no significant deviations of the foot were found either. It is obvious that rotational movements and mutually compensating torsion deformities of the femur and lower leg (if they are found) in achondroplasia do not significantly affect the gait and are not primary pathological elements.

Changes in the frontal plane are caused by varus deformity of the lower limb axis and instability of the knee joint. This determines the amplitude of movements in adduction of the tibia, the emergence of a compensatory valgus vector of the moment of force, which can be measured using 3D computer analysis [41], and based on its value, a conclusion can be drawn about compensated or decompensated instability of the knee joint. As children develop and grow, their legs change their shape, starting with an O-shape, passing through an X-shape period, forming physiological valgus of the knee joint, which we observe in groups of healthy children. In this case, the biomechanical axis of the lower limb passes through the middle of the hip and knee joints, close to the outer edge of the talus block, as a result of weight-bearing on the joints is even [42]. Our study fully confirms the importance of varus deviation of the biomechanical axis for the kinematics and kinetics of movements. The deviation is usually bilateral and symmetrical, located mainly at the level of the tibia, and has a complex three-dimensional deformation, which is associated with a relative excessive length of the fibula [43].

Another characteristic feature of achondroplasia is frontal weakness of the knee joint due to the changes in the proximal attachment points of the collateral ligaments.

Moreover, foot supination was found, which was significantly different from the parameters of peers or healthy children with similar limb lengths. We believe that excessive inversion is of a compensatory adaptive nature. We interpret the spatiotemporal characteristics of gait in children with achondroplasia either as a consequence of insufficient length of segments (decreased stride length, increased gait cycles per minute) or as gait instability (increased relative duration of the double-support phase).

It is obvious that the strategy for correcting gait disorders is to increase the length of the segments of the lower extremities to restore the proportions along with elimination of angular deformities. This would result in both an improvement in stride length and a reduction in energy consumption in walking.

Computer (3D-instrumented) gait analysis is a valuable and necessary method of evidence-based medicine in monitoring the effectiveness of surgical and/or pathogenetic pharmacological treatment (vosoritide).

CONCLUSION

Disorders in the kinematic parameters of walking in children with achondroplasia affect the entire biomechanical chain. They are found in three planes: an increase in the maximum anterior tilt of the pelvis, flexion in the hip and knee joints, along with dorsiflexion of the ankle joint in the sagittal plane; an increased maximum hip abduction angle and varus deformity of the knee joint in the frontal plane; an increased rotational range of motion of the pelvis in the horizontal plane.

Deviations of kinetic parameters are determined by a smaller lever (segment length) and a lower walking speed. A similar length of segments of the lower extremities (with children aged 3–4 years without orthopaedic problems) is not accompanied by significant differences in the power parameters of the femur and lower leg extensors. The kinetics of the group of flexor muscles of the lower leg and foot is determined by the speed parameters of walking.

Changes in the horizontal plane are compensatory in nature; if orientation of the axis of the foot relative to the vector of movement is normal they are not pathological.

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Ethical statement *The study was carried out in accordance with the ethical standards of the Declaration of Helsinki (revised in October 2013), approved by the institutional ethics board (protocol No. 2(70) dated October 21, 2021).*

Informed consent *Voluntary informed consent was obtained from all patients for publication of the study results without disclosing their identity.*

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Information about the authors:

Tamara I. Dolganova — Doctor of Medical Sciences, leading researcher, rjik532007@rambler.ru, https://orcid.org/0000-0002-0117-3451;

Lidiia V. Smolkova — postgraduate, slv@odb45.ru, https://orcid.org/0000-0001-9665-0427;

Dmitry V. Dolganov — Candidate of Biological Sciences, senior researcher, paradigma-dv@rambler.ru, https://orcid.org/0000-0002-8708-1303;

Anna M. Aranovich — Doctor of Medical Sciences, Professor, Head of the Department, senior researcher, aranovich_anna@mail.ru, https://orcid.org/0000-0002-7806-7083;

Nikita S. Gvozdev — traumatologist-orthopedist, nikitozgvozdev@mail.ru, https://orcid.org/0000-0003-3428-3742;

Dmitry A. Popkov — Doctor of Medical Sciences, Professor of RAS, Corresponding member of the French Academy of Medical Sciences, Head of the Clinic, dpopkov@mail.ru, https://orcid.org/0000-0002-8996-867X.