ORIGINAL ARTICLE

# The use of multidisciplinary assessment and scientific measurement in advanced juvenile idiopathic arthritis can categorise gait deviations to guide treatment

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**Background:** It is difficult to identify the range of gait deviations associated with juvenile idiopathic arthritis (JIA) using simple clinical observations.
**Aims:** To use objective gait analysis to accurately describe biomechanical gait abnormalities in JIA and to search for common patterns, which may subsequently serve as a basis for therapeutic intervention.
**Methods:** Children with persistent polyarticular arthritis and symmetrical joint involvement were referred to the Gait Analysis Laboratory and independently assessed by a multidisciplinary team. Gait analysis was performed using an in-house Visual Vector System and the Novel PEDAR in-shoe plantar pressure measurement system. Clinical groupings were based on the extent of joint restriction: minimal (group A), and moderate–severe (with supinatory foot deformity (group B), or with pronatory foot deformity (group C)). Gait analysis enabled classification of each subject into one of four gait patterns: either near normal (pattern I) or one of three adaptive patterns defined by the predominant abnormality—lower limb pain (pattern II), lower limb deformity (pattern III), or a combination of pain and deformity of the lower limb (pattern IV).
**Results:** Of the 15 subjects assessed as part of this study, seven were placed into clinical group A, six into group B, and two into group C. All the subjects with gait patterns I and II were found in clinical group A. Both subjects from clinical group C exhibited gait pattern III. All subjects from clinical group B and the remainder from group A exhibited a mixture of gait patterns III and IV.
**Conclusion:** Despite the initial clinical observations it was not always possible to predict the resultant gait pattern. Scientific gait analysis allowed a clear distinction to be made between primary and secondary gait deviations, and accurate targeting of physiotherapy and orthotic interventions to suit each individual. Prospective quantitative analysis in a larger sample is under way to support the clinical effectiveness of these findings.

The manifestations of juvenile idiopathic arthritis (JIA) include joint swelling, effusion, tenderness, painful limitation of joint movement, and subsequent disturbance of gait.[1] Clinical assessment of the gait is difficult in children, because of the complexity and rapidity of movement, and the variability induced by the child's developmental stage, mood, and social awareness. In children with JIA, the gait pattern is further complicated by subtle compensatory gait alterations in response to joint pain and limb deformity. Severe disease in several joints may lead to complex gait abnormalities, which are often extremely difficult to evaluate clinically.

There is little published information on formal gait analysis of JIA patients. In early papers, it was suggested that gait abnormalities were a reflection of the overall functional ability of the child with chronic arthritis,[2] and that most patients tended to walk on the lateral foot border with limited loading of the medial metatarsal heads, hallux, and lesser toes.[3] The most affected phases of the gait cycle were thought to be initial and final contact.[4] The first report of controlled quantitative gait assessment in JIA showed significant alterations from normal in recorded kinematic and temporal data,[5] and a later study reported both kinematic and kinetic deviations.[6] Predominant abnormalities included increased hip flexion, reduced terminal stance knee extension, and reduced ankle plantar flexion, but further analysis of the range of gait patterns was not reported.

The treatment of JIA encompasses a team approach, including physiotherapy to prevent secondary deformities and preserve muscle strength, while maintaining a natural gait pattern.[1] This is of particular importance in growing children who may not have a fully mature and established gait pattern at the onset of the disease. Advances in gait analysis can lead to improved understanding of the biomechanics of gait abnormalities and may enable physiotherapists and orthotists to adopt a targeted approach to effectively treat and monitor each abnormality. Improved instrumentation and accurate measurement may differentiate between primary and compensatory gait deviations. The objective of this study was to accurately describe biomechanical gait abnormalities in JIA and search for common patterns, which may subsequently serve as the basis for therapeutic intervention.

## SUBJECTS, MATERIALS, AND METHODS

This study was a prospective, open, descriptive analysis of 23 children with gait abnormalities and arthritis, referred for gait assessment over a three year period (August 1996 to September 1999). The local research ethics committee approved the study, and all subjects or their parents gave informed consent.

**Abbreviations:** GRF, ground reaction force; JIA, juvenile idiopathic arthritis; MTH, metatarsal head; MTJ, mid-tarsal joint; MTPJ, metatarso-phalangeal joint; PPP, peak plantar pressure; ROM, range of motion; STJ, subtalar joint; VV, visual vector

**Table 1** Summary of presenting clinical findings following initial assessment, showing subject sex, age, duration of arthritic symptoms, subtype of arthritis, and reported painful joints, with respective gait patterns

| No. | Sex | Age (years) | Duration (years) | Disease subtype (course) | Lower limb pain | Gait pattern |
|---|---|---|---|---|---|---|
| *Clinical group A* | | | | | | |
| 1 | F | 12 | 1.5 | RF– (polyarthritis) | Ankles/feet | II |
| 2 | M | 12 | 2 | RF– (polyarthritis) | Ankles/feet | II |
| 3 | M | 7 | 5 | RF– (polyarthritis) | Ankles/feet | II |
| 4 | F | 14 | 3 | RF– (polyarthritis) | Ankles/feet | II |
| 5 | F | 17 | 11 | JPsA (polyarthritis) | Ankles/feet | IV |
| 6 | F | 4 | 3 | Systemic (polyarthritis) | Ankles/feet | I |
| 7 | F | 8 | 5.5 | RF– (polyarthritis) | Ankles/feet | I |
| *Clinical group B* | | | | | | |
| 8 | F | 16 | 14.5 | RF– (polyarthritis) | Knees | IV |
| 9 | M | 15 | 12 | Systemic (polyarthritis) | Ankles/feet | IV |
| 10 | F | 18 | 14 | JPsA (polyarthritis) | Ankles/feet | IV |
| 11 | M | 8 | 6 | RF– (polyarthritis) | Ankles/feet/knees | IV |
| 12 | M | 12 | 4 | Systemic (polyarthritis) | Knees | III |
| 13 | M | 12 | 5 | RF– (polyarthritis) | Ankles/feet/knees | III |
| *Clinical group C* | | | | | | |
| 14 | M | 10 | 7.5 | Systemic (polyarthritis) | Ankles/feet/knees/hips | III |
| 15 | F | 11 | 5 | JPsA (polyarthritis) | Knees | III |
| Mean (SD) | | 11.73 (3.91) | 6.60 (4.27) | | | |

JPsA, juvenile psoriatic arthritis; RF–, rheumatoid factor negative.

An experienced physician, physiotherapist, and orthotist initially assessed each child. Clinical history, joint pain, and assessment of specific posture type and mobility were recorded, including muscle strength, tone, and range of motion (ROM) tests. Particular attention was paid to foot and ankle function, with assessment of plantar/dorsiflexion, subtalar, mid-tarsal, and metatarso-phalangeal joint movements. All clinical data were entered into an MS Excel spreadsheet and descriptive, non-parametric statistical analysis was performed where appropriate.

Gait analysis was performed using an in-house visual vector (VV) system and the novel PEDAR in-shoe plantar pressure measurement system (Novel GmbH, Munich, Germany). The software generated VV was a real time display of the resultant ground reaction force (GRF) vector, which is the resultant of all forces imparted on the body from the floor during stance phase. This was superimposed on a split video image, viewing the sagittal and coronal planes of the subject loading a Kistler force platform (type 9281B, Kistler Instrumente AG, Switzerland). The VV was refreshed at 50 Hz and was drawn by combining the subject weight, component force values, point of force application, and photogrammetry aspects of the software. The VV was recorded using a standard S-VHS VCR which, when played back in slow motion or using a high quality freeze-frame option, allowed detailed analysis of the individual phases of gait. Established normative data[7–9] was used as controls for comparison.

The standard Novel PEDAR in-shoe pressure measurement system consists of an insole pair (with all optional European sizes from 24 to 45), a lightweight data collection unit worn around the waist, a trailing wire for RS232 connection to a PC, and processing software for analysis of plantar pressure data. The system includes an air pressure device for accurate calibration of the insoles, each of which contains 99 individual capacitive transducers. Unlike platform systems, the use of plantar pressure insoles allows the collection and analysis of data from a number of steps, while walking in shoes with or without orthoses. Plantar pressure data are commonly displayed as peak pressures produced during gait—the method adopted by this study.

All gait assessments were scheduled for the afternoon, with the laboratory maintained at a constant ambient temperature of about 25°C to minimise the influence of joint stiffness. VV data were recorded barefoot and in non-adapted footwear for a minimum of 10 lengths of the 18 metre walkway, including a minimum of two clean strikes of each foot on the force platform. Peak plantar pressure (PPP) data were recorded in footwear only for a minimum of 12 consecutive steps along the same 18 m walkway, during a separate trial within the same session. Sufficient rest time was allowed during the assessment as required. The multidisciplinary team and at least one parent were present at each stage of the assessment. Following gait analysis, each individual case was discussed by the team members with reference to both the clinical examination and gait assessment, leading to the formulation of orthotic/physiotherapy treatment recommendations.

## RESULTS

Twenty three children with arthritis were referred for gait analysis, of whom eight were excluded because of other diseases or neurological complications, including sarcoidosis, severe fixed knee flexion deformity, renal osteodystrophy, and cerebral palsy. The remaining 15 children had JIA with predominantly symmetrical polyarticular joint involvement[10] (table 1). Lower limb pain was the most common complaint, particularly in the foot and ankle. Only three subjects complained of isolated knee pain; a further three subjects had combined foot/ankle and knee pain, with one subject having additional hip pain. All subjects were able to walk without aids, and had no neurological symptoms or significant leg length discrepancy. Most subjects were taking oral non-steroidal anti-inflammatory drugs and methotrexate, and only one was taking oral prednisolone. Two subjects had undergone triple arthrodesis.

### Clinical groups

All subjects were initially grouped according to clinical presentation, depending on the ROM at the hip, knee, ankle, subtalar, mid-tarsal, and metatarso-phalangeal joints (tables 2 and 3). Seven subjects were identified in clinical group A with minimal fixed deformities or restrictions in hip, knee, and ankle ROM (table 2). Only one subject was found with a 5° extensor lag in knee ROM. Subtalar (STJ), mid-tarsal (MTJ), and metatarso-phalangeal joint (MTPJ) deviations were also generally minimal, with three cases of mild supinatory/cavum

**Table 2** Summary of hip, knee, and ankle joint range of motion from clinical assessment

| No. | Hip | Knee | Ankle |
|---|---|---|---|
| *Clinical group A* | | | |
| 1 | – | L&R 5° EL, GV | Swelling only |
| 2 | – | – | – |
| 3 | – | – | – |
| 4 | – | Mild GV | – |
| 5 | L&R ↑ IFR | – | – |
| 6 | – | – | – |
| 7 | – | – | – |
| *Clinical group B* | | | |
| 8 | – | – | ↓ L&R DF (0°) |
| 9 | L&R 5° FFD, weak abductors | – | – |
| 10 | L&R 5° FFD | – | ↓ L&R DF (–5°) |
| 11 | – | Effusion, GV and 10° EL | ↓ L&R DF (0°) |
| 12 | – | Inflammation | – |
| 13 | L&R 5° FFD | – | ↓ L&R DF (–10°) |
| *Clinical group C* | | | |
| 14 | L&R 5° FFD and painful EFR | – | ↓ L&R DF (0°) |
| 15 | 15° FFD | Mild GV | – |

Each subject was categorised according to clinical presentation: clinical group A (minimal joint restriction), clinical group B (restricted joint motion, STJ supinatory deformity), clinical group C (restricted joint motion, STJ pronatory deformity).
–, Normal; DF, dorsiflexion; EFR, external femoral rotation; EL, extension lag; FFD, fixed flexion deformity; GV, genu valgum; IFR, internal femoral rotation; PSN, position; ROM, range of motion; STJ, subtalar joint.

deformity, two of phalangeal hyperextension, two of minimally reduced range of STJ motion, and one of arthrodesed STJ and MTJ (table 3).

Six subjects comprising clinical group B showed more notable deformities at the hips, knees, and particularly the ankles (tables 2 and 3). Three subjects had 5° fixed flexion deformity (FFD) at the hip, and two had knee joint problems including inflammation or effusion, extension lag, or valgus deformity. Four subjects displayed fixed equinus or reduction in ankle dorsiflexion. Significant deviations were found for the majority of subjects in the subtalar, mid-tarsal, and metatarsophalangeal joints. The group was characterised by a supinatory subtalar joint manifestation with either reduced or no joint ROM, and hyperextended toes.

The remaining two subjects, comprising clinical group C, had fixed hip flexion deformity and subtalar pronatory manifestation. Additionally, one subject presented with reduced ankle ROM and the second with mild genu valgum.

### Gait patterns

Detailed examination of individual VV and PPP data led to the identification of four consistent gait patterns across the entire subject population. Each pattern was classified according to the predominant abnormality. Some of the abnormalities were

**Table 3** Summary of rest position and subtalar, mid-tarsal, and metatarso-phalangeal joint ROM from clinical assessment

| | Subtalar | | | Mid-tarsal | | | |
|---|---|---|---|---|---|---|---|
| No. | PSN | ROM (SPN) | ROM (PRN) | PSN | ROM (ADD) | ROM (ABD) | MTPJ |
| *Clinical group A* | | | | | | | |
| 1 | – | R↓ | R↓ | – | L&R Pain | – | – |
| 2 | L sup | – | – | – | – | – | – |
| 3 | L&R sup (mild) | – | – | – | – | – | Hyperex |
| 4 | Fused | 0 | 0 | Fused | 0 | 0 | |
| 5 | – | – | – | – | – | – | HV and hallux callus |
| 6 | L&R mild cavum | R↓ | L&R↓ | – | – | – | – |
| 7 | – | – | – | – | – | – | Hyperex |
| *Clinical group B* | | | | | | | |
| 8 | L&R sup | ↓ | ↓ | – | ↓ | ↓ | Hyperex |
| 9 | L sup | – | – | – | – | – | 5th MTH callus, and Df hallux |
| 10 | L&R sup | – | 0 | – | ↓ | ↓ | Hyperex |
| 11 | L&R sup | ↓ | ↓ | – | ↓ | ↓ | Hyperex |
| 12 | L&R sup | ↓ | ↑WTB | Add | ↓ | ↓ | – |
| 13 | R sup<br>L prn | ↓ | ↓ | L abd | Mobile | Mobile | Hyperex |
| *Clinical group C* | | | | | | | |
| 14 | L&R prn | ↓ | ↑ | Abd | – | – | – |
| 15 | – | – | ↑ | – | – | – | – |

Each subject was categorised according to clinical presentation: clinical group A (minimal joint restriction), clinical group B (restricted joint motion, STJ supinatory deformity), clinical group C (restricted joint motion, STJ pronatory deformity).
–, Normal; 0, none; ↑/↓, increased/reduced; ABD, abduction; abd, abducted; ADD, adduction; Add, adducted; Df, dorsiflexed; HV, hallux valgus; Hyperex, hyperextended; PRN, pronation; prn, prone; PSN, position; ROM, range of motion; SPN, supination; sup, supine; WTB, on weight bearing.

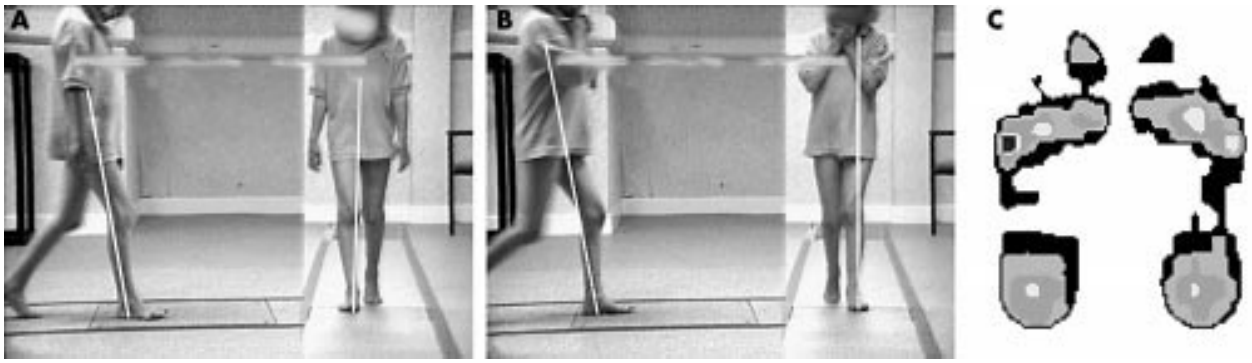


**Figure 1** VV and PPP data for patient 7. The subject number relates to tables 1, 2, and 3. VV data for both L and R limbs at contralateral final contact shows posterior sagittal vector displacement at the knee—increasing knee flexion during loading response. Patient 7 also displayed associated increased R knee external varus moment, and L knee valgus moment. PPP data illustrated increased hallux and 5th MTH pressure.

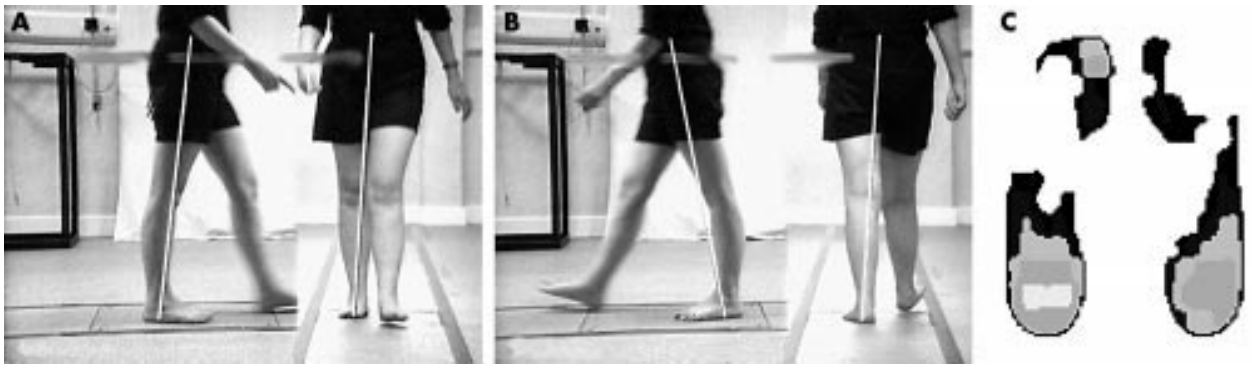


**Figure 2** VV and PPP data for patient 4. The subject number relates to tables 1, 2, and 3. VV data for both L and R limbs at contralateral initial contact showed posterior sagittal vector displacement in the hindfoot, and anterior displacement at the knee. Bilateral knee joint hyperextension and minimal bilateral forefoot pressures are apparent.

predictable from the clinical findings, but more importantly there were unexpected gait abnormalities which were manifest in all three clinical groups.

### Pattern I

These subjects exhibited a near normal gait pattern. We expected the majority of subjects in clinical group A to have this pattern, but were surprised that only two (patients 6 and 7, table 1) in fact did so. The slight increase in loading response knee flexion (fig 1A,B) may have been a result of a marginal increase in stride length in both subjects. Mild external knee valgus and varus moments as illustrated by lateral or medial displacement of the coronal GRF respectively (fig 1A,B) were seen in patient 7, possibly because of a narrow walking base. No significant changes were found between barefoot and shod. The individual PPP distributions showed minimal abnormalities with only occasional isolated mid/lateral forefoot and hallux peak pressures (fig 1C).

### Pattern II

Gait pattern II, or antalgic, was characterised by an excessive external knee extension moment with delayed heel rise during terminal stance, illustrated by abnormal anterior displacement of the sagittal GRF vector at the knee and posterior displacement in the foot (fig 2A,B). Furthermore, forced hip extension was visible earlier in stance phase (denoted by anterior sagittal vector displacement at the extended hip), producing predominant heel loading for the duration of single support. In extreme cases the external knee moment was found to produce hyperextension at the joint. The gait pattern was not found to alter significantly between barefoot and non-adapted footwear. Examination of PPP data within this pattern confirmed predominant hindfoot loading, with minimal or absent forefoot pressures (fig 2C). Despite minimal deformities identified during clinical assessment, four clinical group A subjects were found to display the characteristic abnormalities of an antalgic gait (table 1).

### Pattern III

Gait pattern III of lower limb deformity was characterised by anteromedial vector displacement in the forefoot and excessive external knee extensor moment. Extreme cases were associated with premature heel rise at midstance (fig 3A,B). Observed changes in non-adapted footwear were dependent on the severity of calf muscle tightness, STJ stiffness, and overall heel height of the shoe, which increased the effective range of ankle dorsiflexion. The medial forefoot deviation of the GRF was further illustrated by PPP distribution, which showed excessive medial metatarsal head (MTH) loading (fig 3C). The four subjects with pattern III were identified within clinical groups B and C.

### Pattern IV

The predominant feature of pattern IV was excessive supination producing lateral deviation of the coronal GRF in the foot. This was largely a result of cavum deformity and stiff subtalar/mid-tarsal joints. Additionally, bilateral femoral adduction was producing an increased external knee valgus moment, illustrated by lateral displacement of the coronal vector at the knee centre during single support (fig 4A,B). Excessive lateral wear on the subject's own footwear commonly produced further deviation of the coronal vector. Detailed analysis of PPP distribution showed increased lateral forefoot pressures (fig 4C). Four of the six clinical group B

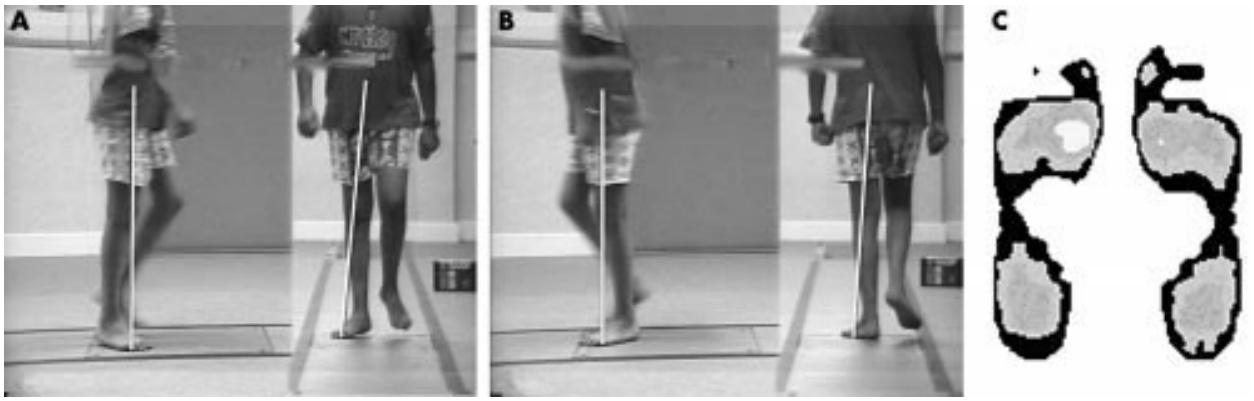


**Figure 3** VV and PPP data for patient 13. The subject number relates to tables 1, 2, and 3. VV data for both L and R limbs at kinematic midstance showed bilateral forefoot abduction, and anterior medial vector displacement in the forefoot. Anterior sagittal vector displacement at the knee produced an increased external extension moment, and premature heel rise was apparent. PPP data showed increased medial forefoot pressure.

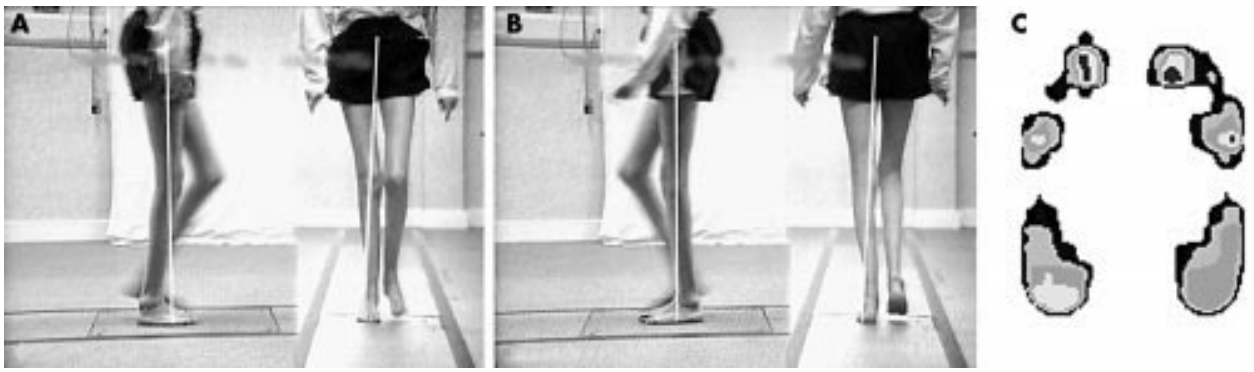


**Figure 4** VV and PPP data for patient 8. The subject number relates to tables 1, 2, and 3. VV data for both L and R limbs at kinematic midstance showed lateral deviation of the coronal vector in the foot because of excessive supination, and at the knee because of femoral adduction. The latter produced a consequent increase in external valgus moment during stance. PPP data illustrated lateral displacement of pressure distribution, particularly in the MTH region.

subjects and a single subject from group A were identified with pattern IV.

## DISCUSSION

This is the first study of gait in children with JIA which highlights a number of different patterns of gait disturbance that may have implications for treatment. We showed four recognisable patterns of gait that could not be reliably predicted from other clinical observations. Of particular importance was the demonstration of abnormal gait patterns in subjects with relatively normal findings from clinical examination (clinical group A). Only two of seven subjects in this group displayed the expected near normal gait characteristics of pattern I. The other five subjects had abnormalities of excessive extension knee moment (pattern II, secondary to lower limb pain) or excessive supination (pattern IV). Other authors have not mentioned these abnormalities in detail, although limited knee extension has been noted in JIA.[6]

The abnormalities found in pattern II subjects were likely to have been the result of a compensatory gait pattern adopted to offload the painful forefoot and reduce discomfort. These findings suggested that temporary orthotic intervention might be helpful to offload painful areas. The changes highlighted by the VV and PEDAR systems also suggested that biofeedback training might have a role in correcting the gait. Gait pattern III was found in two subjects from clinical group B, and two from group C. This pattern contained subjects with limitation of hip, knee, and ankle movements. The two group C subjects, who displayed an increased range of subtalar pronatory motion on clinical examination, appeared to adopt a pronation mechanism to enable tibial progression in the sagittal plane during late stance. The treatment modalities of antipronatory heel cups, heel raises, and midfoot rockers may be of benefit in such subjects.

Significant restriction of ankle dorsiflexion or equinus, and excessive STJ supination with varying degrees of forefoot stiffness were found in subjects from clinical group B. Subtalar supination was invariably associated with increased longitudinal arch curvature, and MTPJ hyperextension. These abnormalities were found in four subjects in gait pattern IV, and were probably caused by a combined result of fixed joint deformity (secondary to long standing arthritis) and adaptation to pain. A single subject from clinical group A proved the exception, in whom additional excessive bilateral internal femoral rotation produced increased hallux pressure and plantar callus.

All previously published studies reported consistent gait deviations in children with persistent arthritis, despite a variety of clinical presentations. Truckenbrodt *et al* stated that gait disturbance and deformity were often complicated by the development of compensatory gait patterns in JIA.[4] Corresponding reductions in hip, knee, and ankle joint moments, and anteroposterior GRF peaks accompanied the kinematic deviations reported by Frigo and colleagues.[6] Lechner *et al* reported similar alterations from normal kinematic and temporal data.[5] The abnormalities were interpreted as either compensation for pain (reducing impact and propulsive forces) or the end result of joint stiffness, excessive muscle tightness, or weakness. It was proposed that monitoring the progressive

deterioration of gait could assist in focusing interventions to reduce secondary complications and protect the unaffected joints.

The limitations of our study were: a small numbers of subjects; and two dimensional measurement of a complex three dimensional movement. The former will be addressed by a larger study with improved statistical power. Three dimensional assessment of forces and moments and PPP measurements, in addition to a complete clinical examination by a multidisciplinary team, will provide comprehensive information about movements in all three dimensions. Without meticulous gait analysis, there is the danger of mismanagement of JIA gait disorders, resulting in serious physical and social consequences on these children during gait maturation. This underlines the importance of using our approach to target the functional biomechanical problem, rather than the presenting complaint, when managing gait in JIA children. If a deviation is found to be an adaptive response to joint pain, treatment should address the pain, rather than the deviation per se. Therefore we suggest that clinical assessment supplemented by VV and dynamic PPP measurements may lead to better outcomes for children with juvenile idiopathic arthritis.

### Conclusion

We have identified subtle differences between adaptive gait patterns in response to pain and those caused by fixed deformity in subjects with juvenile idiopathic arthritis. Despite rigorous clinical examination, it was not always possible to predict the resultant gait deviations accurately—most notably for the subjects in clinical group A and the gait patterns I, II, and III. Early use of gait analysis may help prevent the development of potentially damaging adaptive gait deviations. When fixed deformities are discovered, gait analysis can assist in fine tuning orthoses and optimising gait patterns.

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# ARCHIVIST

## More on antiepileptic drug exposure in utero

About six of every thousand pregnant women has epilepsy and congenital malformations have been reported in up to 14% of children exposed to antiepileptic drugs (AEDs) in utero. A retrospective study in Aberdeen (JCS Dean and colleagues. *Journal of Medical Genetics* 2002;**39**: 251–9) has given more data.

Of a total of 411 women who took AEDs in pregnancy between 1976 and 2000, 258 were contacted and 149 took part in the study. They had 293 children who were assessed from records and questionnaires and by examination. Children exposed to more than one AED in pregnancy (n = 51) were significantly more likely to have major congenital malformations than those (n = 38) born after pregnancies in which the mother did not take an AED. There was no significant increase in rate of malformation, however, when the mother took a single AED in pregnancy. The most frequent malformation was inguinal hernia associated with carbamazepine exposure. Carbamazepine was also associated with hip dislocation, genital abnormalities, congenital heart disease, and submucous cleft palate. Valproate was associated with talipes, other limb abnormalities, and genital abnormalities (hypospadias, hydrocele, undescended testis). Major malformations (those needing treatment in the first year) occurred in 14% of children whose mothers took any AED in pregnancy and minor abnormalities in 42% (rates in nonexposed children 5% and 13%). Developmental delay (speech delay needing referral to speech therapy, or motor delay (not sitting by 10 months or not walking by 18 months, or both) was diagnosed in 24% (exposed) *v* 11% (non exposed) (19% *v* 3% after excluding children with a family history of developmental delay). Facial dysmorphism was found in 52% *v* 25%. Behaviour disorders (autistic spectrum or attention deficit hyperactivity disorder) affected 12% *v* 5% (significant for exposure to carbamazepine or valproate monotherapy or any polytherapy). Neonatal symptoms attributed to drug withdrawal occurred in 20% *v* 3%.

Exposure to antiepileptic drugs in utero, and especially to more than one drug, increases the risks of congenital malformation and of developmental delay in the children of mothers with epilepsy.