Developmental Flatfoot—Part 1

Here's what you need to know about this common condition.

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Goals and Objectives

To instill a knowledge and appreciation of the developmental flatfoot

To be familiar with its incidence and attendant pathomechanics

To dispel common myths and misconceptions surrounding this entity

To learn its specific etiology with special reference to phylogenic and ontogenic influences as well as the role of osseous immaturity and osseous malalignment in its production

To understand and recognize its clinical significance

To be able to differentiate the developmental flatfoot from other pediatric flatfoot conditions

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*Following this article, an answer sheet and full set of instructions are provided (pg. 164).—**Editor***

There is no better way to begin a discussion about flatfoot in children than is illustrated in a quote from Herman R. Tax, DPM which encapsulates the issue with the prevalence and the importance of identification and proper conservative management of the excessively pronated pediatric flatfoot.

Figure 1: The feet of most infants present with a discernible medial longitudinal arch; however, due to an increased plantar fat pad, the untrained observer is incorrectly led to believe that it is absent.

"There is a serious misconception on the part of the public as amongst a great number of health professionals to equate the problem of 'flatfoot' with excessively pronated feet in children. This is a matter of grave concern since flatness of the longitudinal arch can be a normal or abnormal finding in foot posture, whereas the excessively pronated foot is flattened as part of a pathologic structural malposition. This inherent biomechanical defect is present in the arch of a great majority of children and is the basic reason for most postural pathology of the lower extremity."[1]

Excessive pronation of the feet should in no way be considered a "normal" con-

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dition to be automatically outgrown. In fact, as far back as 1917, an eminent New York orthopedist Royal Whitman wrote in his *A Treatise on Orthopedic Surgery* text that pronation of the feet commonly seen in children is more likely to worsen than improve over the years.[2] According to Dr. Tax, the "father of podopediatrics", only an insignificant number of the millions of children receive the simple available care necessary to minimize the problem.[1]

Introduction

The fastest growing segment of the population today is over 65 years of age. The population triangle has become a rectangle with as many people over 65

Many of the lower extremity adult musculoskeletal parameters are achieved by age six.

years old as there are under 21 years old. When these baby boomers were born 65 years ago, it was anticipated most would only live to be 65. However, due to advances in healthcare, medication, and education, the 65-year-old can today expect another 20 years of life. The 75 year old can expect another 10 years. Those in the 70 year age group are now referred to as the "young" elderly. These individuals are characteristically independent, active, and many are still voluntarily employed.

The life expectancy of a child born today is approximately 78 years, but when that child reaches 75 years of age, advances and discoveries in healthcare will not only give them the extra 10 years as enjoyed today, but likely

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TABLE 1:

Pediatric Flatfoot Selected Conditions

- Calcaneovalgus
- Convex pes valgus
- Accessory navicular
- Tarsal coalitions
- Neurological disorders
- Syndrome Component
- Iatrogenic
- Mechanical
- Developmental

Figure 2: Abnormal pronation with marked forefoot abduction, severe calcaneal eversion, medial displacement of body weight, and super-structural manifestations.

Figures 3: 17 year old female with untreated asymptomatic flexible flatfoot whose parents were told she would grow out of it. Now she's off to college and her foot pain is preventing her from participating in sports.

Figure 4 a, b: The developmental flatfoot has the appearance of a normal arch off-weight-bearing and an absence upon weight-bearing.

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20+ years! Furthermore, and most importantly, what will determine the quality of life for this child at 80, 90 or even 100 years of age is the ability to walk without pain.

Life is movement and mobility is of paramount importance in maintaining health. At the foundation of this mobility system and its only link to the ground stands the foot. Therefore, anything that can be done to improve alignment and function during the developmental years will pay great dividends in terms of improved mobility later on in life, in essence preparing the child's foot for a century of walking.

The correct procedure for the identification of the developmental flatfoot is to distinguish it from similar disorders.

Myths and Misconceptions

Many health professionals ascribe to the notion that infants under 18 months of age are flatfooted with an absence of the longitudinal arch.[3,4] In some cases this is thought to occur due to increased fat deposits in the sole of the foot.[5] The reality is that most children possess a discernible longitudinal arch. Due to the increased plantar fat pad, the untrained observer is led to believe that the longitudinal arch is absent (Figure 1).

Since the feet of most children appear to be flat and otherwise non-deformed, they are assumed to be normal. The fact is that prevalence or commonality should never be confused with normalcy. Normalcy implies ideal, and an excessively pronated foot is a far cry from the ideal foot. Another myth regarding the foot of the young child is that all children's feet under six years of age are pronated.[6] The reality is that excessive pronation is a poor postural position of the foot and ankle that sets the stage for future dysfunction, deformity, and disability… not only in the foot but in the superstructure, and is abnormal at any age (Figure 2).[7]

Another popular misconception regarding the foot of the young child is that strength and toning exercise programs will improve alignment, function, and osseous development; however, this is not the case. Foot exercises, either intrinsic or extrinsic, cannot be expected to alter the architecture of the foot.[8-11] Finally, the greatest myth of all regarding children's foot problems is: "don't worry, they'll grow out of it." The problem is that these kids don't "grow out of it"; they just grow up and out of the pediatrician's practice (Figures 3). Excessive pronation is more likely to worsen than improve over the years.[2,10,12]

Compensation for inherent structural deficiencies takes place with each step the child takes. The pathologic effects of these compensatory motions are not only limited to the foot and ankle but to the leg, knee, hip, and back as well.[13-16] These repetitive abnormal stresses eventually produce symptomatology with or without attendant deformity. The effects of this dysfunction are not necessarily confined to the foot and ankle but may negatively impact any level of the musculoskeletal locomotor system.

Onset of symptoms may not occur until the "child" begins to participate in sporting activities, thereby placing increased demands on the foot and leg while accelerating and exaggerating excessive pronation.[17-19] Factors influencing the age of symptomatology onset include the degree of structural malalignment, obesity, family history, the presence of contributing systemic disease, ligamentous laxity, level of sports or fitness participation, etc. Over

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TABLE 2:
Mechanical Deficiencies Capable of Producing Pediatric Flatfoot

Genu valgum
Equinus
Transverse plane deficiencies
Morton's syndrome
Limb-length discrepancy
Forefoot varus
Flexible forefoot valgus
Accessory navicular
Tarsal coalitions (adolescents)

TABLE 3:
Equinus Deficiencies Capable of Producing Pediatric Flatfoot

Gastrocnemius
Soleus
Gastrocnemius and soleus
Bony block at the ankle
Forefoot
Metatarsal
Hamstring
Iliopsoas

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80% of the population in the United States suffers from foot problems at one time or another in their lives and the vast majority of these conditions begin in childhood.

Pediatric Flatfoot

The etiology for most types of pediatric flatfoot has been well documented in the medical literature (Tables 1, 2, 3). There is a wealth of information regarding conditions such as calcaneovalgus, vertical talus, and the presence of an accessory navicular.[5,20-25] Neurologic conditions such as cerebral palsy, and syndrome disorders such as Down, Ehlers-Danlos, and Marfan's have been given much attention. The literature is replete with information regarding the flatfoot that results from improper serial plaster immobilization of talipes equinovarus or metatarsus adductus and, to a lesser extent, the mechanically induced flatfoot.[26,27]

Out of this entire group, the entity that has gotten the least attention but the one that is responsible for the largest percentage of pediatric flatfoot cases is the developmental flatfoot.[28,29] The sum total of all other flatfoot cases would not equal the number in the developmental flatfoot grouping. Interestingly, many of the major orthopedic and pediatric texts do not ascribe any clinical significance—or even fail to mention—this widespread condition.[5,20,22,24,25,30]

Definition and Occurrence

The developmental flatfoot is an excessively pronated foot in the weight-bearing pediatric population under six years of age.[1,28,29] Other terms that have been used to describe this condition include pes planus, pes valgoplanus, pes planovalgus, idiopathic hypermobile flatfoot, hyperpronated feet, and floppy feet.[31-34] It is the most common condition affecting the musculoskeletal system in a child of this age group.

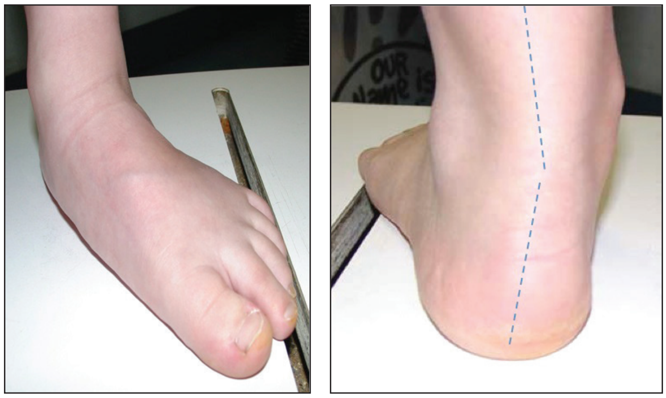

Figure 5a: Developmental flatfoot with medial talar protrusion and medial border convexity. 5b: lateral border concavity, "too many toes" sign, and calcaneal eversion.

Many of the lower extremity adult musculoskeletal parameters are achieved by six years of age. However, a complete musculoskeletal evaluation of any six year old will reveal individual values that deviate significantly from the ideal.[35] Should these asymptomatic, undeformed feet be considered "normal" because they possess commonly occurring, customarily anticipated structural imperfections that cause them to deviate from the ideal but not from the "norm"?

In theory, if the foundation of a building deviated by the same proportional number of degrees as the "average" foot, then the building would neither stand straight nor be capable of supporting heavy loads. In addition, the human "foundation" must be capable of balanced movement; therefore, imperfections in the "average" foot become increasingly significant as the years of use and abuse coupled with the effect of gravity accumulate. The structural morphology of the human foot is dictated by the alignment and quality of the osseous structures, the binding ability of the ligaments, and the reinforcing and secondary stabilizing network of muscles and tendons. The developing foot is flexible, flat, and excessively pronated owing to an immaturity in each of these areas.

In developmental flatfoot the arch is present off weight-bearing and absent on weight-bearing.

Identification

The developmental flatfoot is identified by distinguishing it from similar disorders (Tables 1, 2, 3). Diagnosis is by exclusion or elimination. According to the French orthopedist GF Domisse, "... both knowledge and skill are required in the diagnosis of a condition which is as vital to the individual as wheels to a carriage and wings to a

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Figures 6 a, b: Hubshire maneuver to determine viability of the windlass mechanism as well as ease of medial longitudinal arch restoration.

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bird."[36] The developmental flatfoot has the appearance of a normal arch off-weight-bearing and an absence upon weight-bearing (Figures 4a, b). Off-weight-bearing radiographs appear normal but on weight-bearing, radiographs exhibit increased talocalcaneal, talo horizontal, and talo first metatarsal angles, as well as a decreased calcaneal inclination angle.

There is noted medial talar protrusion accompanied by a medial convexity and lateral concavity (Figures 5a, b). The lateral column of the foot is short relative to the medial column, which is lengthened owing to the medial protrusion of the talar head.[29,37,38] The calcaneus is maximally everted and there is a "too many toes" sign present (Figure 5b).

The Hubshire maneuver and Jack's tests indicate intact functioning of the windlass mechanism, enabling the plantar fascia to stabilize the medial longitudinal arch. Calcaneal inversion is noted upon rising on toes in the Hubshire maneuver, and recreation of the longitudinal arch by dorsiflexing the hallux in Jack's test (Figures 6a, b, c). The ability of these tests to recreate the windlass mechanism rules out the presence of rigid flatfoot conditions such as peroneal spastic flatfoot due to tarsal coalition in the older child, and convex pes valgus in the younger child.

This flexible deformity must still be distinguished from calcaneovalgus, neurological and syndrome disorders, mechanical causes and iatrogenic flatfoot. This can be achieved through thorough history taking, and clinical and radiographic evaluation. For example, calcaneovalgus is easily recognized by its "up and out" position off-weight-bearing, and the presence of an accessory navicular identified clinically and then confirmed radiographically as the child achieves osseous maturity.

Specialist referral may be appropriate if an underlying neurologic impairment is not apparent or cannot be ascertained. Congenital gastroc equinus will present and be easily identified by an inability to dorsiflex to a right angle with the subtalar joint in neutral to mildly supinatory position. Other forms of equinus as well as forefoot varus and Morton's syndrome may all be assessed clinically.

The developmental flatfoot deformity is unique in that it encompasses several major pedal articulations with triplanar axes of motion. These joint complexes are responsible for connection of the foot to the leg and the foot to the ground, as well as for translating motions from the superstructure and vice versa. As a result of these factors, there is the potential for musculoskeletal dysfunction and derangement in any segment and at any level.

Figure 6c: Jack's test recreating medial longitudinal arch.

Etiology

The developmental flatfoot is caused by phylogenic as well as ontogenic influences, osseous malalignment, osseous immaturity, neuromotor immaturity, and ligamentous laxity.

Phylogeny

The primitive foot was a more hand-like appendage, better suited for climbing, grasping, and conforming to the irregular surfaces of the terrain in which it existed. This three-dimensional unit functioned well in performing the tasks that were essential to its survival.

At one time, it was more important for the human organism to be able to quickly climb a tree than to walk long distances. Since there was no need for shock absorption, the foot remained flexible and flat. Development of the longitudinal arch proceeded as this need increased. The low-arched or flexible flatfoot seen in many children is an atavistic trait or reversion. Dudley J. Morton in his 1935 text, Evolution of The Human Foot ascribed most mechanically-induced foot pathology to a retention of atavistic characteristics in the foot and leg.[37] At least some components of the original Morton's syndrome consisting of short first metatarsal, second metatarsal cortical hypertrophy, posterior displaced sesamoids, and metatarsal cuneiform split have been validated and held to be significant by modern practitioners.[39,40]

Today, the human infant is born with many characteristics inherited from this tree-dwelling ancestry (Table 4).[42] These "evolutionary scars" present at birth and if not outgrown will serve to create or contribute to musculoskeletal pathology later on in life.[35,41-43]

Ontogeny

According to Haeckle's 1899 law of recapitulation, during embryological development, an organism passes through stages that resemble the structural form of several ancestral types of the species as it evolved.[44,45] "Ontogeny recapitulates phylogeny" is a well-known med school pneu-

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monic to remember and understand Haeckle's law. Although in its strict sense, the Haeckelian form of recapitulation is no longer accepted, it is agreed that embryos do reflect the course of evolution and undergo a period where their phylogenetic position rather than selective pressures shapes their morphology.[46,47]

Some pediatric deformities can be directly attributable to arrest of development such as that seen in talipes equinovarus. Premature births carry with them an increased risk of retention of in-utero deficiencies. Additionally the human infant has relatively the largest head and longest legs, which must then be crowded into a snug uterine environment (Figure 7). The comparatively long legs of the fetus cause it to crouch during the later in-utero period. Experts believe that even the full term infant is born in an underdeveloped state necessitating a "premature" birth.

The Newborn

Children are not born with perfect feet. The structure of the newborn foot contains all of the inherited evolutionary imperfections which, if not outgrown, may produce dysfunction and deformity in the adult foot. The foot of the neonate is ill-adapted for weight-bearing. The newborn is not a bipedal organism. Man is one of the few mammals whose young are incapable of standing erect at or shortly after birth. The human infant must undergo considerable developmental "unwinding" outside the womb before the organism possesses all the requisites necessary for bipedal locomotion. This critical developmental "unwinding" takes place on hard, flat, unyielding surfaces in a malleable foot, enclosed in a shoe, and subject to the deforming effects of gravity.

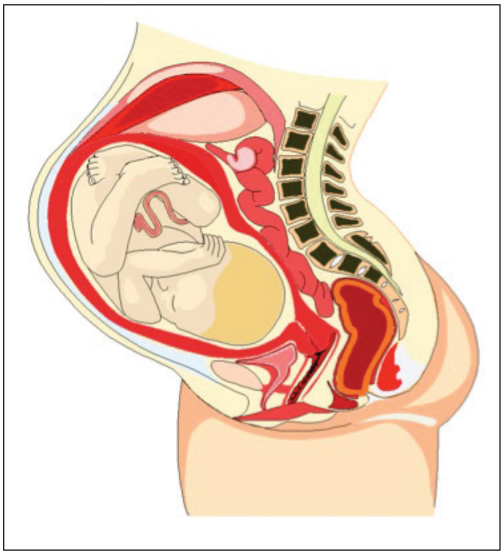

Figure 7: Note the relatively large head and legs crowded into a snug uterine environment during the last trimester.

The navicular is not ossified during the beginning to established walker phase of development.

TABLE 4:

Evolutionary "Scars" Present in the Newborn

- External limb rotations
- Ligamentous laxity
- Coax varum
- Genu varum
- Tibial varum
- Anterior femoral bowing
- Anterior tibial bowing
- Little or no tibial torsion
- Hip flexion
- Knee flexion
- Ankle flexion
- Talar neck adductus
- Metatarsus adductus
- Metatarsal primus adductus
- Subtalar varus
- Forefoot varus

The average infant begins to stand at seven to nine months of age and is walking by one year, with a range of 9-15 months.[48-53] Heel contact during gait occurs at two years of age and a mature gait at three years.[48-54] Up to six years of age, skeletal alignment and development of the foot are dictated by the nature and severity of the deforming forces directed through it, as well as by its ability to resist these forces. When allowed to continue, these compensatory deforming forces retard ideal development while at the same time encouraging the retention of neonatal deficiencies.

According to Wolff's law of bone and Davis' law of soft tissue, adaptation in structure will occur in direct response to function, i.e., form follows function.[55,56] Consequently, musculoskeletal examination of the six-year-old child will usually reveal significant unreduced structural deficiencies that are the basis for pathomechanical foot function. In the developing child, the consequence of poor function is impaired development and retention of in-utero deficiencies.

Osseous Malalignment

The limbs of the newborn are bent and bowed to such a degree that at birth, even the normal newborn possesses significant structural imperfections in all three body planes (Figure 8). On the frontal plane alone, one would note the following: genu varum, 15-20° tibial varum, 8-10° subtalar varus, and 10-15° of forefoot varus.[57] Therefore, the total amount of "normal" varus (the sum of all varus influences into the foot) could reach as high as 45°. All of these deficiencies must be developmentally reduced before ideal foot function can take place. Most skeletal

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deficiencies reduce in magnitude by six years of age, at which point their development is essentially complete. By six years of age, genu varum should have resolved, tibial varum reduced to 0-2°, subtalar varus to 2-4°, and forefoot varus to 0-2°. The total varus component at this point in the ideally developed foot is from 2-8°.

The mechanism of compensation for these congenital structural imperfections in the developing or mature foot is pathological subtalar and midtarsal joint pronation. Of the frontal plane deficiencies present at birth, the most destructive if retained is forefoot varus. Dr. Richard O. Schuster ascribed over 90% of overuse injuries in runners to be influenced by compensation for retained forefoot varus.[58]

Compensation for forefoot varus requires calcaneal eversion beyond the vertical to allow the forefoot to reach the weight-bearing surface. The maximally everted position of the calcaneus causes adaptive contracture of the Achilles tendon, which in turn creates sagittal plane equinus compensation, further compounding the pathology. If left unchecked, this pronatory compensation begets additional frontal plane deformity, viz. forefoot supinatus. This occurs as the late midstance and propulsive foot is driven into an unyielding supportive surface, and with the superstructure passing above, longitudinally torques the unstable forefoot, creating additional frontal plane deformity. Hence, forefoot varus is a major, progressive destructive force into the foot compensated by abnormal subtalar and midtarsal joint pronation, resulting in the development of a flexible flatfoot condition.

On the transverse plane, the range of motion of the hip joint at birth is between 120-150°. External rotation exceeds internal rotation in a 2:1 ratio. The range of hip rotation gradually decreases until approximately four years of age, at which time the range has reduced to 90-120° and is equal internally and externally.

Forefoot abduction with "too many toes" sign is another characteristic seen in developmental flatfoot.

In the child under four years of age, this developmentally externally rotated limb position maintains the foot in an abducted attitude, thereby promoting pedal pronation. In addition, there is a concomitant medial displacement of the line of gravity, which further increases the pronatory forces into the foot, encouraging the development and retention of a flexible flatfoot deformity. The medial displacement of the line of gravity also has negative superstructural implication on the developing lower extremity articulations as well as on posture (Figure 9).

Figure 8: Marked genu varum and tibial varum in the newborn. Note the "C"-shaped appearance of the left foot indicating metatarsus adductus.

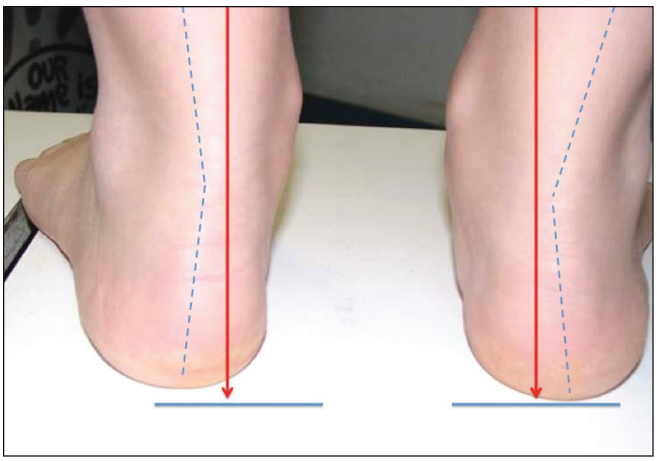

Figure 9: Medial displacement of the line of gravity in an excessively pronated developmental flatfoot. Note the degree of forefoot abduction and lateral concavity.

Osseous Immaturity

Osseous immaturity is another factor which predisposes the developing foot to retain its flexible, excessively pronated, and flattened morphology. Osseous maturity in the lower extremity does not take place until epiphyseal-diaphyseal fusion has taken place. The nature of the osseous pedal segments and its concomitant ability to withstand stress increases directly with age. The osseous framework of a two-year-old is distinctly less developed than that of a six year old.

The ossification sequence in the foot, for the most part, occurs from the rearfoot to the forefoot. The first bones to undergo ossification are the talus and the calcaneus, which are radiographically visible at birth. The last bone to ossify in the foot is the navicular, with an average age of ossification of 33.8 months in males and 23.3 months in females.[59] It is generally agreed that ontogenic development in the foot regarding basic form and position is complete by seven to eight years of age; however,

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overall bone growth continues up to 20 years of age.[60] Complete skeletal maturity occurs at 13 years of age in girls and 15 years in boys.[59,61]

It is interesting to note that at seven months of age when the average child begins to stand unassisted, the ossification process for the navicular as well as for the medial and intermediate cuneiforms has not even begun.[62] These cartilaginous structures are extremely vulnerable to deforming gravitational forces from the limb above. At 12 months of age when the average child is already walking, the navicular and the medial and intermediate cuneiforms are still not ossified. In addition at 12 months, the talus, calcaneus, cuboid, and the lateral cuneiform are all markedly immature and underdeveloped structures.

Forefoot varus is the most destructive frontal plane atavistic deformity seen in the developing foot.

The three year old is already an established mature walker with an adult-like gait pattern. At this stage, the foot is immature and not ideally suited for its static and dynamic functions of support and locomotion. Again, this extremely malleable foot is subject to deforming forces directed through it from the superstructure above.

Since the navicular is the major buttressing segment of the medial longitudinal arch and recipient of stresses from its keystone bone the talus, it is especially valuable in evaluating foot function. Conversely and due to its relative immaturity, it is particularly subject to deformation in the case of dysfunction. It is not until six years of age that the navicular loses its appearance as a "bone island in a sea of lesser density."[63] It is during this same period of navicular immaturity that the child is actively developing and refining a well-coordinated adult, propulsive gait pattern, making this integral segment of the longitudinal arch especially vulnerable to deforming forces. **PM**

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CME EXAMINATION

SEE ANSWER SHEET ON PAGE 165.

1) The most commonly occurring excessively pronated pediatric flatfoot in the weight-bearing pediatric population under six years of age can be ascribed to which one of the following conditions?
- A) calcaneovalgus
- B) tarsal coalitions
- C) gastocnemius/soleus equinus
- D) developmental issues

2) Many of the lower extremity adult musculoskeletal parameters are achieved by what age?
- A) 2 years
- B) 4 years
- C) 6 years
- D) 8 years

3) What is the correct procedure for the identification of the developmental flatfoot?
- A) CT scans
- B) range of motion studies
- C) gait analysis
- D) distinguish it from similar disorders

4) Which one of the following describes the developmental flatfoot?
- A) arch present off weight-bearing and absent on weight-bearing
- B) arch present off and on weight-bearing
- C) arch absent off and on weight-bearing
- D) arch absent off weight-bearing, arch present on weight-bearing

5) Which one of the following indicates intact functioning of the windlass mechanism enabling the plantar fascia to stabilize the medial longitudinal arch?
- A) Hubshire maneuver
- B) Silferskiold test
- C) Homan's sign
- D) Mudler's sign

Continued on page 164

CME EXAMINATION

6) Which one of the following represents an etiologic factor in the developmental flatfoot?
- A) retention of in-utero position
- B) osseous immaturity
- C) retention of atavistic characteristics
- D) all of the above

7) Which important bone in the foot is not ossified during the beginning to established walker phase of development?
- A) talus
- B) calcaneus
- C) navicular
- D) first metatarsal

8) Factors influencing the age of symptomatology onset in the pediatric flatfoot include which one of the following?
- A) ligamentous laxity
- B) degree of structural mal-alignment
- C) obesity
- D) all of the above

9) The weight-bearing developmental flatfoot exhibits a lateral column shortening relative to the medial column, which is lengthened by medial protrusion of the talar head and marked calcaneal eversion. Which one of the following is another characteristic seen in this foot type?
- A) forefoot abduction with "too many toes" sign
- B) hallux flexus
- C) metatarsus adductus
- D) medial concavity

10) The most destructive frontal plane atavistic deformity seen in the developing foot is which one of the following?
- A) forefoot varus
- B) subtalar varus
- C) tibial varum
- D) genu varum

SEE ANSWER SHEET ON PAGE 165.

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EXAM #7/18
Developmental Flatfoot—Part I
(D'Amico)

Circle:

1.	A	B	C	D	6.	A	B	C	D
2.	A	B	C	D	7.	A	B	C	D
3.	A	B	C	D	8.	A	B	C	D
4.	A	B	C	D	9.	A	B	C	D
5.	A	B	C	D	10.	A	B	C	D

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