CONTINUING PROFESSIONAL UPDATE

MI PODIATRIC CPU TOOLS
Driving excellence in foot health

Mr Anthony J Maher
FRCPodS

Mr Timothy E Kilmartin
FRCPodS PhD

Juvenile Hallux Valgus Part 1. Anatomy, Aetiology & Pathophysiology

Preface

Back in 2010 Dr Tim Kilmartin and Anthony Maher wrote an extensive review of juvenile hallux valgus with the intention of publishing it as a chapter in a paediatric podiatry textbook; unfortunately the book never came to fruition and the manuscript sat unpublished for the next 12 years. The original 'chapter' was based partly on a review of the historic literature relating to juvenile hallux valgus but also drew on both authors' clinical experience in podiatric surgery and Dr Kilmartin's PhD work.

Following initial discussions with the editors of the Canonbury Continuing Professional Update series it was agreed to publish the original manuscript split into two parts. In this first article we aim to provide podiatrists with a thorough understanding of the anatomy, aetiology, and pathophysiology of juvenile hallux valgus. In the second article we will provide an evidence-based approach for the podiatrist wishing to systematically manage this common and important foot deformity.

Introduction

If hallux valgus becomes apparent in childhood, severe forefoot deformity is almost inevitable in later life. While in most cases there is a long family history, this is not apparent in all who present with the condition. In such cases, parents will often worry that the problem is entirely a result of poor footwear. Clinical management should begin with comprehensive explanation of the cause, the natural progression and the non-surgical and surgical treatment options that are available. A long-term treatment plan should be agreed with the child and parents which will involve baseline measurement of the deformity, night splints to slow progression, orthotic treatment to relieve symptoms and regular clinical measurement of the deformity. The aim of treatment should be to prevent involvement of the second toe and minimise symptoms without restricting the child's activities and also minimise self-consciousness of the condition. If the hallux valgus deformity begins to underride and sublux the second toe, surgical correction is indicated but preferably when skeletal maturity is confirmed on x-ray assessment.

Key Point
Hallux valgus (HV) is not the most frequently occurring paediatric foot complaint. However, when it occurs, HV can cause significant anxiety for parent and child alike. When adults are questioned regarding the onset of HV, up to 57% report first noticing the deformity during childhood [1]. The deformity may initially be asymptomatic affecting only the first metatarso-phalangeal (MTP) joint, though it will inevitably deteriorate through the patient's life, altering digital alignment and potentially disrupting the normal function of the forefoot [2]. The potential for HV to cause significant morbidity throughout life should not be disregarded.

Normal Anatomy and Function

Before assessing or planning to treat hallux valgus (HV), the clinician must understand the relevant normal anatomy and function of the first ray and the structures which comprise it. HV is primarily a pathological abnormality of the 1st MTP joint and medial column of the foot, though it can progress to encompass the neighbouring 2nd digit and ultimately the whole forefoot.

Anatomy

The first MTP joint contains two synovial articulations. The first articulation is the head of the first metatarsal which articulates with the base of the proximal phalanx. This articulation represents a partial ball and socket arrangement. The second articulation is between the plantar cartilage of the metatarsal head and the dorsal surface of the two sesamoid bones. The articular surface of the proximal phalangeal base and the dorsal surface of the two sesamoids create a cup-like structure through which the metatarsal head can rotate and slide[3 4].

The sesamoid bones located plantar to the metatarsal head are bound to each other by a strong inter-sesamoidal ligament. Ligamentous fibres arising from the sesamoids attach to both the metatarsal head and proximal phalanx. Fibres from the lateral sesamoid also have an attachment to the intermetatarsal ligament which subsequently inserts into the plantar plate (a strong ligament which arises from the metatarsal and insets into the base of the proximal phalanx) of the 2nd MTP joint. The 1st MTP joint is further stabilised by oblique medial and lateral fibres which form collateral ligaments[3 4].

Development

Embryonic limb buds first appear between the 4th and 8th intrauterine (IU) week. By the 10th IU week, the feet will have formed, and ossification of the long bones will have begun in earnest. The first metatarsal is usually identifiable at this point while ossification of the hallucal phalanges begins a week or two later. Growth of the metatarsal bones proceeds rapidly during the first 6 years and ossification of the bones of the first MTP joint will continue through the first two decades of life, with completion between 18-20 years[3 5 6].

Both the hallucal phalanges and first metatarsal demonstrate secondary centres of ossification. Unlike the lesser metatarsals, which have a secondary centre of ossification at the head of the bone, the first metatarsal has a secondary centre proximally located at its base. This usually appears between 1 and 2 years of age. The hallucal phalanges, as with all phalanges of the foot, have a secondary centre at the base of the bone which appears between 2 to 8 years of age. The typical radiolucent 'gap' noted on x-rays between the primary and secondary centre of ossification is termed the growth plate or epiphyseal plate. The growth plate is an area of cartilage which usually ossifies by 18-20 years of age. The two sesamoid bones first appear between 7 to 8 years of age as secondary centres of ossification within the cartilaginous plantar plate of the 1st MTP joint[3 5 6].

The medial cuneiform, which articulates distally with the base of the first metatarsal does not appear until 1-2 years of age and is typically followed 6-18 months later by the navicular[5].

Function of the First MTP joint

Several intrinsic and extrinsic muscles control motion of the first MTP joint. Extrinsic motion is primarily achieved through the extensor hallucis longus (EHL) muscle which dorsiflexes and the flexor hallucis longus (FHL) muscle which plantarflexes. These two functions are supported intrinsically by extensor hallucis brevis (EHB) and flexor hallucis brevis (FHB). EHB inserts dorso-medially into the joint capsule and assists with dorsiflexion of the hallux, while FHB inserts into the base of the proximal phalanx plantarly, assisting plantarflexion. Of note here, the FHB tendon divides into two distinct slips prior to its insertion at the base of the proximal phalanx. The tendons then encapsulate the sesamoids leaving only the dorsal surface exposed[3 7 8].

Abduction (away from the midline of the body) of the hallux is achieved by the twin heads of the adductor hallucis muscle which insets into the lateral joint capsule. Adduction is achieved by the abductor hallucis muscle which inserts medially into the joint capsule[4 7].

Figure 1 HAV deformity in a teenager

The primary motion of the normal first MTP joint is dorsiflexion within the sagittal plane. However, the metatarsal head is a curved structure which allows the proximal phalanx to adduct or abduct in addition to dorsiflexion and plantarflexion. Motion in the transverse plane is limited by the collateral ligaments. When compressed against the metatarsal head the sesamoids also resist transverse plane motion. The more curvature there is within the metatarsal head, the greater the degree of transverse plane motion that will be possible. Hence a flatter or ridged metatarsal head will be capable of less transverse plane motion[3 7 8].

During gait, the axis of sagittal plane motion through the first MTP joint is not fixed. During propulsion, the hallux is fixed to the ground and in order to achieve dorsiflexion of the 1st MTP joint, the heel raises, the rearfoot supinates and the metatarsal plantarflexes to rotate around a sliding axis which moves from the centre of the metatarsal head in a proximal and superior direction.

Aetiology of Juvenile Hallux Valgus

Dagnall asserted that there has not been a definitive study of the aetiology of HV, though many theories have been proposed[9]. HV is not a common pathology amongst children. A survey of 6000 school children in the United Kingdom by Kilmartin et al. found an incidence of 2%[10]. That compares with an observed incidence of up to 33% of adults in shoe wearing populations[11]. When questioned, about the onset of hallux valgus, up to 57 percent of adults with HV recall an onset during childhood[1]. Childhood onset may be considered an important factor in patients who during adulthood go on to require treatment for HV. It is however unusual to document hallux valgus in children younger than 9 years of age[12]. Coughlin, in a review of 45 patients, noted a mean onset age of 11.8 years[13]. Though it is clear that not all cases of juvenile HV are symptomatic, Hardy & Clapham suggested that most patients will present to a clinician before the age of 14[14].

We do not know the true incidence of juvenile HV and likewise we do not know how many patients with juvenile hallux valgus will go on to require treatment, either as a child or an adult [15]. Taking account of the published reports of surgical intervention for HV, Coughlin suggested that as many as 84 percent of those presenting for treatment are female[15]. Multiple aetiologies have been proposed for HV, a discussion of these follows below.

Inheritance

A familial link has long been considered a likely factor in the aetiology of HV. Johnston in 1954, was perhaps the first to suggest that hallux valgus is inherited and indeed, parents may present their child for treatment for this very reason[16]. Concerned parents will often have first-hand experience of the long term effects of foot deformities and attend for consultations in the hope that preventative remedies are possible (Merriman)[12]. Several papers have considered the importance of inheritance, particularly on the female side with positive female family histories being reported between 72 and 77 percent of the time in patients with symptomatic juvenile HV[13 14]. Positive male family histories are much less common, with a reported incidence of 16 percent[14].

Coughlin further implicated inheritance as a key factor in the development of hallux valgus; of those patients with a positive family history, 94 percent demonstrated maternal transmission[13]. Coughlin also found that the hallux valgus angle tended to be 5 degrees higher in those patients with a positive history[13 15]. Piqué-Vidal et al. undertook a 3 generation study of 350 patients and concluded that 90 percent of patients with hallux valgus had a positive family history consistent with an autosomal dominant trait with incomplete penetrance[17]. Additionally, the authors concluded that inherited hallux valgus deformities are likely to appear before the age of 10, as at this age extrinsic causes such as footwear are unlikely to have been present[17].

Figure 2 An example of fashionable footwear

Footwear

The importance or not of footwear in the development of hallux valgus is a contentious issue with keen advocates on either side of the argument. A number of papers have asserted that fashionable footwear will directly lead to the deterioration of hallux valgus deformities[15]. The relationship between elevated hallux valgus angles and fashionable footwear in European populations has been considered in a classic study by Barnicott and Hardy who compared shod and unshod Nigerians with a mixed group of Europeans[18 19]. The authors found no significant difference between the shod and unshod Nigerians. In both the Nigerians and Europeans there was a significant difference between the sexes, the hallux valgus angle was significantly greater amongst the European females[18 19].

Historically, hallux valgus seems to have been a rarity until populations began wearing shoes[7]. Hallux valgus is 20 times more common amongst Chinese people who wear shoes as opposed to those who are unshod. There has also been a marked increase in the prevalence of HV in Japan since western style shoes have become more popular[7 11].

The argument in favour of footwear as a causative factor is that hallux valgus may develop as a consequence of a relatively wide foot being inserted into a shoe with a relatively narrow toe box. Narrow fitting shoes have been linked with the deterioration of HV in children[15 20 21]. This has however been countered by arguments that juveniles with HV have rarely worn fashionable shoes at the age of onset [13 14]. In a study of American school children, only 24 percent of symptomatic children reported constrictive footwear while 70 percent wore shoes of the most typical American width [13 15].

Footwear is probably best considered to be an aggravating factor in the development and particularly the progression of hallux valgus rather than a cause [21 22]. Even when patients wear sensible shoes, there is evidence that females are more likely to develop HV than males[7 23]. Conversely, modified footwear can be effective in preventing the development of hallux valgus[7]. Craigmille demonstrated that proper shoe fitting and regular replacement has also been demonstrated to minimise the occurrence of HV in school children[24].

Pes Planus

Pes planus has long been associated with hallux valgus deformity, though the basis for this association has not been adequately explained. It has been proposed that pes planus is a predisposing factor for the development of HV[22]. The theory is that any process which creates excessive subtalar joint pronation will leave the mid-tarsal joint unlocked during stance with subsequent medial column and first ray instability. The resultant instability at the first metatarso-cuneiform joint will, in response to ground reaction forces, cause the first metatarsal to adduct and dorsiflex giving rise to an elevated intermetatarsal angle. The pronated foot then fails to resupinate at the end of stance, causing the foot to roll forwards over the medial border of the hallux rather than through active functional plantarflexion and dorsiflexion of the 1st MTP joint [4 8 22].

The combination of an unstable medial column and medial loading of the hallux will, according to the theory, give rise to a hallux valgus deformity. Though pronation has been a popular theory in certain corners, there is little evidence to support the role of pes planus as a causative mechanism in juvenile HV[25]. Kilmartin and Wallace found no association between arch height and HV[25].

Pes planus does however, seem to be more prevalent in juvenile HV with studies reporting as much as a 24 times greater incidence of pes planus over the normal population[26]. A second study by Scranton and Zuckerman reported a 51 percent incidence of pes planus in patients with juvenile HV[27]. Others have found a much lower incidence with Coughlin reporting only a 16 percent incidence of pes planus in juveniles undergoing surgery for HV[13]. In reviewing adults with HV, Mann and Coughlin have argued that pes planus is not particularly common in patients with HV unless there is an underlying neuromuscular disorder[28]. The conflicting evidence for the relationship between hallux valgus and pes planus led Easley and Trnka to conclude that there is insufficient evidence either way to prove or disprove a relationship[29].

Hypermobility

Hypermobility of the foot has been proposed by Carl et al. as a possible cause of some hallux valgus deformities though Harris and Beeson note that very few studies have actually examined the link between hypermobility and hallux valgus[30 31 32]. Hypermobility may occur on its own or as part of a connective tissue disorder. Kalen and Brecher found a high incidence of HV in patients with Marfan's syndrome[26]. A hypermobile (and therefore pronated) foot arguably alters the axis of the 1st MTP joint and subsequently places a deforming force on the medial joint capsule[15]. This may lead to the development of hallux valgus as described above. It has been suggested that such a foot may be prone to recurrent deformity following surgical intervention, if the pronatory force is not controlled[22].

Hallux valgus may be a considered a characteristic trait of patients with hypermobility or ligament laxity syndromes but the presence of the two conditions does not necessarily signify a cause and effect relationship. Harris and Beeson undertook a review of 56 juveniles with hallux valgus and concluded that although there appears to be an association between hypermobility and HV, it is not a predisposing factor[33].

Local to the foot, first metatarso-cuneiform joint hypermobility has long been argued as a potential cause of hallux valgus[30 34]. Instability at the first metatarso-cuneiform joint prevents the metatarsal from stabilising during propulsion, resulting in dorsiflexion and adduction of the metatarsal, giving rise to an increase of the intermetatarsal angle by 1-2 degrees[4].

Although hypermobility at this location is a well-defined theory, some authors have argued against the importance of 1st metatarso-cuneiform joint hypermobility. Coughlin and Jones found no association between the severity of HV and the presence of hypermobility. Myerson, Harrison and Beath all found it difficult to assess and define hypermobility of the first ray[35 36 37]. Smith and Coughlin observed that the assessment of first ray hypermobility is usually made with the patient non-weight-bearing, whereas the joint usually functions when weight-bearing and with the support and assistance of the intrinsic muscles, extrinsic tendons, ligaments and plantar fascia[38]. These factors work together as a unit to resist ground reaction forces. The non-weight-bearing assessment of first ray hypermobility could then be considered arbitrary at best. Certainly the reliability of first ray mobility assessments has been questioned[15]. In order to accurately assess first ray mobility, specially designed and calibrated devices have been created and are used for research purposes but such devices are not practical in the clinical setting[38].

Anatomic Factors

Various anatomical anomalies have been described in association with hallux valgus. When detected, these anomalies are sometimes considered causative. As with all the aetiological factors considered so far, caution should be exercised when attempting to label the 'cause' of juvenile hallux valgus.

Metatarsus Adductus: has an incidence of about 1 in every 1000 live births[2]. As with hallux valgus, the condition seems to be more common in females[2]. Metatarsus adductus as a feature of juvenile HV has been disputed for some time. Work by Kilmartin et al. and McCluney and Tinley suggested that metatarsus adductus is not a statistically significant finding in juvenile HV[39 40]. Metatarsus adductus is usually detected early in a child's life and is initially treated by strapping, reverse footwear or, in more serious cases, serial casting may be applied. However children (and indeed adults) may present with an uncorrected or partially corrected deformity which has subsequently given rise to a hallux valgus deformity[41]. Uncorrected, the metatarsus adductus will increase the angle of the adductor hallucis muscle giving it a mechanical advantage and overpowering the opposing abductor and increasing the hallux valgus angle[42]. Secondly, the metatarsus adductus will through adduction of the fore foot, place the hallux against the toe box of the shoe. In this scenario, the shoe itself acts as a deforming force[42]. Banks et al. found a linear correlation between an increasing metatarsus adductus angle and an increasing hallux valgus angle[43]. Metatarsus adductus in its milder forms may be clinically silent but irrespective of whether it may have an aetiological role in the development of HV, the two deformities seem to appear in association.

Metatarsus Primus Varus: Houghton and Dickson argue that care should be taken when assessing the intermetatarsal angle as opposed to the metatarsus

Figure 3 X-ray of HAV deformity demonstrating an increased angulation at the 1st MTP joint

primus varus angle[44]. The difference being that the metatarsus primus varus angle signifies a medial deviation of the long axis of the metatarsal, as a segment of the first ray. A raised intermetatarsal angle signifies only a raised 'gap' between the first and second metatarsals[44]. Kilmartin and Wallace found no correlation between the intermetatarsal angle and the metatarsus primus varus angle[39]. They concluded that metatarsus primus varus is a consequence of abnormal alignment of the metatarso-cuneiform joint[39].

Obliquity of the Metatarso-Cuneiform Joint: An oblique distal surface to the medial cuneiform is reportedly associated with a hypermobile first ray[32]. Obliquity at this level naturally creates divergence between the first and second metatarsals and a raised intermetatarsal angle. Truslow considered divergence of the first metatarsal and metatarsus primus varus as factors which precede the onset of HV[45]. Others have described an oblique 1st metatarso-cuneiform joint as nothing more than a radiographic artefact[46]. Vyas et al. found that obliquity of the cuneiform distal articular surface was not statistically different between patients with HV and a control group[47].

Metatarsal Length: Technically when metatarsal length is discussed, what is really being considered is the metatarsal protrusion distance or metatarsal parabola[15]. Though previously considered an important factor in the aetiology of HV, there seems to be little agreement that metatarsal length has an impact on the development of hallux valgus. Both long and short first metatarsals have been implicated in the development of hallux valgus[20 48 49]. McCluney and Tinley found that a positive metatarsal protrusion distance (long first metatarsal relative to the second) was a significant predictor of hallux valgus[40].

Figure 4 HAV with underiding first toe and ulcer on 2nd toe

Using a method proposed by Hardy and Chapman, Coughlin found that 28 percent of patients with HV had a short first metatarsal (relative to the second metatarsal), 42 percent had a metatarsal length equal to that of the second and 30 percent had a long metatarsal[13 14]. Perhaps of note, the results were remarkably similar to those of an earlier study by Harris and Beath[15 37].

In the presence of HV, a long first metatarsal tends to demonstrate a greater distal metatarsal articular angle (DMAA) than a short metatarsal. Though not relevant to conservative care, a raised DMAA may influence your choice of surgical intervention[13]. On a similar theme, the long first metatarsal has been associated with more severe deformities and a greater risk of recurrence following surgical intervention[49]. This discussion would probably not be complete without mentioning that most first metatarsal osteotomies used to correct HV will result in a degree of shortening to the first metatarsal and that the shortening, intentional or otherwise, seems to play a role in correction of deformity.

Metatarsal Head Shape: As mentioned above, in order for the hallux to adduct, a curvature of the metatarsal head is required in the transverse plane. Metatarsal head shape has been broadly categorised into flat, ridged and curved[50]. The flat or ridged metatarsal head is considered stable in the transverse plane and less prone to the development of hallux valgus. The more curved the metatarsal, the more able the hallux is to abduct[15 50]. Hence a curved or rounded metatarsal head sets up a favourable environment for the onset of HV (See Figure 3). Interestingly, a study of 100 first metatarsal heads in children by Kilmartin and Wallace found that juvenile hallux valgus could occur in the presence of either a square or curved metatarsal head[51]. Easley reiterates that there is no evidence to support the role of head shape in the development of HV[29]. There also seemed to be no correlation between metatarsal head shape and severity of hallux valgus.

Pathophysiology

The Development Of Hallux Valgus

The question of whether adduction of the first metatarsal (metatarsus primus varus) or abduction of the hallux is the primary event initiating the development of hallux valgus has long been a focus of debate. However, the stages of progression in the deterioration of the hallux valgus deformity have been well documented[15].

As the hallux begins to abduct away from the midline of the body, the perhaps confusingly named intrinsic muscle, adductor hallucis gains a mechanical advantage and exerts a passive force on the lateral sesamoid and associated joint capsule. This favours the progression of deformity while the abductor hallucis becomes increasingly ineffective at resisting the deforming forces. The lateral pull on the sesamoid structure pulls the medial sesamoid ever closer to the metatarsal crista. The crista initially resists further progression but, under pressure from the medial sesamoid, is slowly eroded which allows both sesamoids to migrate further laterally[3 4 7 8 15 22].

The hallux is initially pulled towards the second toe where, in many cases, it will rest. This may be a source of inter-digital irritation and subsequent corn formation. The second toe acts as a buttress preventing further migration and stabilising the deformity. With continued lateral force exerted on the 1st MTP joint capsule, the 2nd toe 'buttress' is placed under increasing strain until, eventually the hallux pushes the digit laterally or dorsally by destabilising it at the MTP joint (see Figure 4). Occasionally the hallux will override the 2nd digit[7 8].

As the hallux valgus deformity progresses, the first MTP joint loses its ability to function during toe off, thus increasing the load taken by the 2nd MTP joint. This stresses this much less substantial structure, giving rise to capsulitis or pre dislocation syndrome. This chronic strain weakens the 2nd MTP joint making the digit more likely to abduct or dorsiflex away from the hallux. In cases of severe deformity the 2nd MTP joint may sublux or even dislocate[7 52].

With significant lateral migration of the hallux, the 1st MTP joint will sublux increasing the intermetatarsal angle and creating a more prominent metatarsal head medially. The metatarsal will now be prone to footwear irritation and subsequently an adventitious bursa will develop. Persistent traction from the medial joint capsule and abductor hallucis tendon directed to the medial aspect of the first metatarsal head may be implicated in the formation of an exostosis[15].

Progression of Hallux Valgus

Little has been written of the progression of juvenile hallux valgus into adulthood. Hoshino et al. reported the radiographic progression of hallux valgus over a population life time[53]. The authors concluded that the deformity was unstable during the first 3 decades of life but was also prone to further progression through later years[53]. The deformity does not appear to progress greatly through the 4th and 5th decades though this is followed by a further peak of activity around the 6th decade, after which there seems to be little progression. However it should be noted that hallux valgus will not progress as an isolated pathology[12]. Severe deformity will eventually engulf the whole forefoot with a multitude of associated pathologies. An uncorrected symptomatic hallux valgus which has been allowed to progress over many years is a technically challenging prospect for any clinician. ■

Coming soon!

Juvenile Hallux Valgus. Part 2. Assessment and Management

Having considered the functional anatomy, aetiologies and pathophysiology of juvenile hallux valgus, the second article will move its focus on to the assessment and management of this condition. Topics covered will include clinical and radiographic assessment of HV and both conservative and surgical treatment options.

References

1. Piggott H. The natural history of hallux valgus in adolescence and early adult life. J Bone Joint Surg Am 1960;42B:749-60
2. Kilmartin TE, Tollafield DR. The child's foot. In: Tollafield DR, Merriman LM, editors. Clinical Skills in Treating the Foot. London: Churchill Livingstone, 1997:305-36.
3. Samojla B. Normal Anatomy of the Forefoot. In: Hetherington V, editor. Hallux Valgus and Forefoot Surgery: Churchill Livingstone, 1994:7-37.
4. Wernick J, Volpe RG. Lower extremity function and normal mechanics. In: Valmassy R, editor. Clinical biomechanics of the lower extremity. St. Louis: Mosby, 1996:1-58.
5. Anderson EG. Appendix 2. Ossification timetable. In: Lorrimer DL, French G, West S, editors. Neale's Common Foot Disorders. London: Churchill Livingstone, 1997:471.
6. Anderson EG. The Growing Foot. In: Lorimer DL, French G, West S, editors. Neale's Common Foot Disorders. 5th ed. London: Churchill Livingstone, 1997:53-64.
7. Phillips D. Biomechanics. In: Hetherington V, editor. Hallux Valgus and Forefoot Surgery: Churchill Livingstone, 1994:39-53.
8. Root M, Weed J, Orien W. Clinical Biomechanics. Normal and abnormal function of the foot. Los Angeles: Clinical Biomechanics corp, 1977.
9. Dagnall C. Introduction. In: Hetherington V, editor. Hallux Valgus and Forefoot Surgery: Churchill Livingstone, 1994:1-4.
10. Kilmartin TE, Barrington RL, Wallace WA. A controlled prospective trial of a foot orthosis for juvenile hallux valgus. J Bone Joint Surg Br 1994;76(2):210-4.
11. Lam SL, Hodgson AR. A comparison of foot forms among the non-shoe and shoe-wearing Chinese population. J Bone Joint Surg Am 1958;40:1058.
12. Kilmartin TE, Tollafield DR, Nesbitt P. The paediatric patient. In: Merriman LM, Tollafield DR, editors. Assessment of the Lower Limb. London: Churchill Livingstone, 1995:319-46.
13. Coughlin MJ. Juvenile hallux valgus: etiology and treatment. Foot Ankle Int 1995;16:682-97
14. Hardy R, Clapham J. Observations on hallux valgus. J Bone Joint Surg Am 1951;33B:276-391.
15. Coughlin MJ. Juvenile Hallux Valgus. In: Coughlin MJ, Mann R, editors. Surgery of the Foot and Ankle. 7th ed. St Louis: Mosby, 1999:270-319.
16. Johnston O. Further studies on the inheritance of hand and foot anomalies. Clin Orthop 1954;8:146-50.
17. Pique-Vidal C, Sole MT, Antich J. Hallux valgus inheritance: pedigree research in 350 patients with bunion deformity. J Foot Ankle Surg 2007;46(3):149-54.
18. Barnicott NA, Hardy RH. The position of the hallux in west Africans. J Anat 1955:355-61.
19. Kilmartin TE, Wallace WA. The aetiology of hallux valgus: a critical review of the literature. The Foot 1993;3(4):157-67.
20. Amarnek D, Jacobs A, Oloff L. Adolescent hallux valgus: its etiology and surgical management. J Foot Surg 1985;24:54-61.
21. Cicchinelli LD, Nakra A. Juvenile Hallux Abducto Valgus Deformity. In: Banks AS, Downey MS, Martin DE, Miller SJ, editors. McGlamry's Comprehensive Textbook of Foot and Ankle Surgery. 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2001:609-22.
22. Valmassy RL. Biomechanical evaluation of the child. In: Valmassy RL, editor. Clinical Biomechanics of the Lower extremeties. St Louis: Mosby, 1996:243-78.
23. Shine IB. Incidence of hallux valgus in a partially shoe wearing community. Br Med J 1965;1:1648
24. Craigmile DA. Incidence, origin and prevention of certain foot defects. Br Med J 1953;2:749.
25. Kilmartin TE, Wallace WA. The significance of pes planus in juvenile hallux valgus. Foot Ankle 1992;13(2):53-6.
26. Kalen V, Brecher A. Relationship between adolescent bunions and flat feet. Foot Ankle 1988;8:331-36.
27. Scranton P, Zuckerman J. Bunion surgery in adolescents: results of surgical treatment. J Pediatr Orthop 1984;4:39-43.
28. Mann R, Coughlin MJ. Hallux valgus: etiology, anatomy, treatment and surgical consideration. Clin Orthop 1981;157:31-41.
29. Easley ME, Trnka HJ. Current concepts review: hallux valgus part 1: pathomechanics, clinical assessment, and nonoperative management. Foot Ankle Int 2007;28(5):654-9.
30. Carl A, Ross S, Evanski P, Waugh T. Hypermobility in hallux valgus. Foot Ankle 1988;8(5):264-70.
31. Harris M-CR, Beeson P. Is there a link between juvenile hallux abducto valgus and generalised hypermobility? A review of the literature. Part 1. The Foot 1998;8(3):125-28.
32. Cantanzariti AR, Blitch IV EL. Proximal Procedures of the First Ray. Part 2. Metatarsal Cuneiform Procedures. In: Banks AS, Downey MS, Martin DE, miller SJ, editors. McGlamry's Comprehensive Textbook of Foot and Ankle Surgery. 3rd ed. Philadelphia: LW&W, 2001:544-55.
33. Harris M-CR, Beeson P. Generalized hypermobility: is it a predisposing factor towards the development of juvenile hallux abducto valgus? Part 2. The Foot 1998;8(4):203-09.
34. Cooper AJ, Clifford PD, Parikh VK, Steinmetz ND, Mizel MS. Instability of the first metatarsal-cuneiform joint: diagnosis and discussion of an independent pain generator in the foot. Foot Ankle Int 2009;30(10):928-32.
35. Coughlin MJ, Jones CP. Hallux valgus: demographics, etiology, and radiographic assessment. Foot Ankle Int 2007;28(7):759-77.
36. Myerson M. Metatarsocuneiform arthrodesis for treatment of hallux valgus and metatarsus primus varus. Orthopedics 1990;13(9):1025-31.
37. Harris R, Beath T. The short first metatarsal: its inclination and clinical significance. J Bone Joint Surg Am 1949;31A:553-65.
38. Smith BW, Coughlin MJ. The first metatarsocuneiform joint, hypermobility, and hallux valgus: what does it all mean? Foot Ankle Surg 2008;14(3):138-41.
39. Kilmartin TE, Barrington RL, Wallace WA. Metatarsus primus varus. A statistical study. J Bone Joint Surg Br 1991;73(6):937-40.
40. McCluney JG, Tinley P. Radiographic measurements of patients with juvenile hallux valgus compared with age-matched controls: a cohort investigation. J Foot Ankle Surg 2006;45(3):161-7.
41. Berenter R, Kosai DK. Serial plaster imobilization of congenital foot deformities. In: Valmassy RL, editor. Clinical Biomechanics of the Lower Extremities. St Louis: Mosby, 1996:405-24.
42. Valmassy R. Common Pediatric Deformities. In: Edwards A, A, Walter JH, Goss LR, editors. Review Text in Podiatric Orthopedics and Primary Podiatric Medicine. Bethesda: American College of Foot and Ankle Orthopedics and Medicine, 2004:607-16.
43. Banks AS, Hsu YS, Mariash S, Zirm R. Juvenile hallux abducto valgus association with metatarsus adductus. J Am Podiatr Med Assoc 1994;84(5):219-24.
44. Houghton GR, Dickson RA. Hallux valgus in the younger patient: the structural abnormality. J Bone Joint Surg Br 1979;61-B(2):176-7.
45. Truslow W. Metatarsus primus varus or hallux valgus. J Bone Joint Surg Am 1925;7:98-108.
46. Brage ME, Holmes JR, Sangeorzan BJ. The influence of x-ray orientation on the first metatarsocuneiform joint angle. Foot Ankle Int 1994;15(9):495-7.
47. Vyas S, Conduah A, Vyas N, Otsuka NY. The role of the first metarsocuneiform joint in juvenile hallux valgus. J Pediatr Orthop B.
48. Munuera PV, Polo J, Rebollo J. Length of the first metatarsal and hallux in hallux valgus in the initial stage. Int Orthop 2008;32(4):489-95.
49. Scranton PE, Jr. Adolescent bunions: diagnosis and management. Pediatr Ann 1982;11(6):518-20.
50. Martin DE, Pontious J. Introduction and evaluation of hallux abducto valgus. In: Banks AS, Downey MS, martin DE, miller SJ, editors. McGlamry's Comprehensive Textbook of Foot and Ankle Surgery. 3rd ed. Philadelphia: LW&W, 2001:481-91.
51. Kilmartin TE, Wallace WA. First metatarsal head shape in juvenile hallux abducto valgus. J Foot Surg 1991;30(5):506-8.
52. McGlamry ED, Jimenez AL, Green D. Lesser Ray Deforomities. In: Banks AS, Downey MS, Martin DE, Miller SJ, editors. McGlamry's Comprehensive Textbook of Foot and Ankle Surgery. 3rd ed. Philapdelphia: LW&W, 2001:253-303.
53. Hoshino T, Inockuchi S, Usami N, Hiraishi E, Hashimoto T. Juvenile hallux valgus in japan: a radiographic comparison with older age groups. The Foot 1998;8(2):93-95.

CPU article number: 007
Editorial Team: **Mike Potter, Ivan Bristow, Anthony Maher & Morwenna Potter**
All articles are selected, commissioned, reviewed and produced independent of Canonbury Products Limited. Canonbury Products Limited kindly provide unrestricted support for the CPU series as a service to the profession.

The editorial team welcomes feedback or suggestions from readers via email: **CPUtools@canonbury.com**