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Knee Angular Deformities (Genu Valgum, Genu Varum, Blount Disease) in Children

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Abstract

The knee undergoes known physiological angular changes during development. At birth, a varus is present, which resolves by 18 to 24 months of age. Thereafter, the varus develops, reaching its maximum extent by 3 to 4 years of age. By age 7, children are expected to have a final alignment, with a near neutral mechanical axis. Variations in shape can occur in different populations. Persistent or more severe deformities can lead to biomechanical changes, affecting gait and aesthetics, and causing pain, functional limitations, and instability. In the long term, especially in bow legs, this deformity is associated with early osteoarthritis. Growth correction is an effective treatment for angular knee deformities; it can be temporary, using a plate or screw, or permanent, without implants. This method has a lower learning curve, less scarring, and fewer complications than other treatments.

Diagnosis of knee deformities requires caution and must combine clinical, imaging, and endocrine genetic testing. In treatment, based on the diagnosis, determining the cause of knee deformities will give good results.

***Keywords**: Genu Varum; Genu Valgum; Knee Deformities; Guided Growth; Lower Limb Deformities*

Introduction

Angular deformities of the lower limbs are common in children. In most cases, they are a change in the normal growth pattern and are completely benign. The appearance of symmetrical and asymptomatic deformities, stiffness, systemic disorders, or syndromes indicates a benign condition with excellent long-term outcomes. In contrast, asymmetric deformities and deformities accompanied by pain, stiffness, systemic disorders, or syndromes may indicate a serious underlying cause and require treatment. This article will look at the main topics of angular deformity in children in a practical way.

Bowed legs are a common orthopedic problem in children. As children grow, the knee undergoes a sequential change in axial development, from varus to valgus. The differences in foot shape and position that parents notice as their child learns to walk often reflect differences in normal physiological development. Because parents are unaware of normal lower limb development and growth and want their child to have normal alignment, they often become concerned and seek medical advice. Many children are referred to orthopedists for treatment of physiological varus and valgus, which is unnecessary and sometimes harmful. To do this, the doctor needs to know when to consider the problem physiological or pathological, by taking a detailed history of the problem, performing a detailed examination to rule

out pathological causes. Causes Most parents bring their young children to the doctor for symptoms of bow legs or bowed knees, which are simply a variation of normal growth and development that will resolve as the child grows. Whether pathological or physiological, it is necessary to differentiate by taking a detailed medical history, carefully examining and measuring several parameters including the intercondylar distance, intercondylar distance and assessing the tibiofemoral angle.

Coronal plane deformity of the knee joint, also known as genu varum or genu valgum, is a common finding in the clinical practice of pediatricians and orthopedists. These deformities can be physiological or pathological. If left untreated, pathological deformities can lead to abnormal joint loading and thus a risk of early osteoarthritis.

Anatomy of the growth plate

Histology

The growth cartilage consists of a fibrous component, a cartilaginous component, and a bony component. The fibrous component surrounds the growth cartilage and is divided into an ossified groove called the groove of Ranvier and a ring surrounding the cartilage called the ring of LaCroi [1]. The function of the groove of Ranvier is to provide chondrocytes for the growth cartilage to increase in diameter and length. The ring of LaCroi lies between the ossified groove and the periosteum of the metaphysis, enveloping the growth cartilage and providing mechanical support to the growth cartilage. The fibrous component protects the growth cartilage from shear forces. The cartilaginous component of the growth cartilage is divided into a reserve zone, a proliferative zone, and a hypertrophic zone (Figure 1). The hypertrophic zone itself is divided into a maturation zone, a degenerative zone, and a zone of temporary calcification. Adjacent to the cartilaginous component is the bony component of the growth cartilage. This is where the chondrocytes are converted into bone.

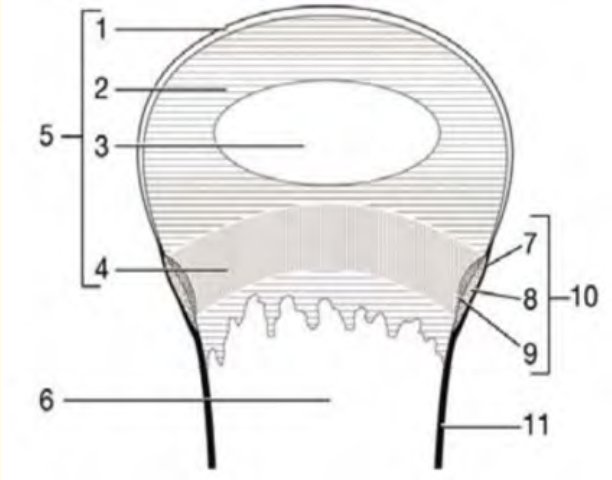


Figure 1: *Components of the epiphysis and metaphysis. (1) articular cartilage; (2) epiphyseal cartilage; (3) secondary center of ossification; (4) epiphyseal plate; (5) epiphysis; (6) metaphysis; (7) fibrous layer of the periosteum; (8) ring of lacroix; (9) groove of ranvier; (10) fibrous components of the epiphyseal plate; (11) cortical bone.*

Vascular supply

Several arteries supply blood to the growth cartilage (Figure 4). The epiphyseal arteries supply the growth cartilage through numerous branches that branch into the growth cartilage, supplying the hypertrophic zone up to 10 cells deep. No blood vessels penetrate the

hypertrophic zone, leaving the hypertrophic zone avascular [1]. The perichondral arteries supply the fibrous structures of the growth cartilage. The feeding arteries supply four-fifths of the blood supply to the metaphysis and do not pass through the open cartilage. Branches of the epiphyseal arteries supply the remainder. The terminal branches of these vessels terminate in small vascular loops or capillary tufts beneath the last row of intact intercellular spaces of the growth cartilage. The chondrocytes are dead at this level, which is important for understanding the development of osteochondral necrosis. a. Venous drainage of the metaphysis occurs via the large central vein of the shaft. In humans and cats, the femoral growth plate may be partially supplied by branches of the epiphyseal artery; However, in dogs, there is no such blood supply.

Physiology of the growth plate

Because of the different blood supply in different regions of the growth plate, cell metabolism is similar. In the proliferative zone and the upper half of the hypertrophic zone, metabolism is anaerobic, whereas in the lower half of the hypertrophic zone, metabolism is anaerobic.

Chondrocytes in the reserve zone are spherical, fewer in number, and separated by more matrix than in other regions. Cells in the reserve zone contain many lipid vacuoles and abundant endoplasmic reticulum, suggesting protein production.[1]. The oxygen tension in this zone is relatively low, consistent with low cell activity (Figure 2). This may indicate that oxygen and nutrients reach this zone only by diffusion, which may be important in the etiology of osteochondrosis and hypertrophic osteodystrophy. The function of this zone may be to supply chondrocytes to the proliferative zone.

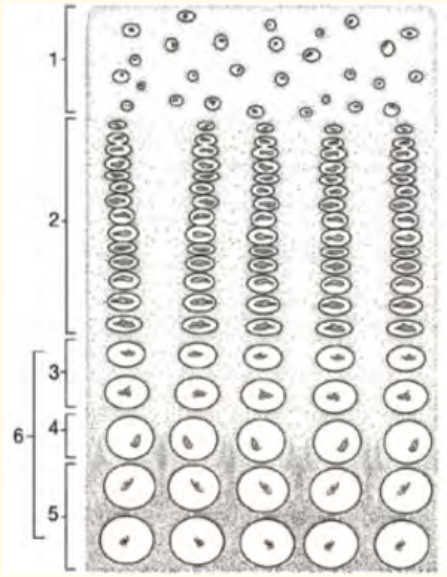


Figure 2: *Zones of the cartilaginous component of the epiphyseal plate. (1) reserve zone; (2) proliferative zone; (3) zone of maturation; (4) zone of degeneration; (5) zone of provisional calcification; (6) hypertrophic zone.*

In the proliferative zone, chondrocytes are flattened and arranged in columns parallel to the longitudinal axis of the bone. Oxygen tension is higher than in other zones, as is cellular metabolism, resulting in high concentrations of cellular metabolites [1]. The primary function of this zone is cell proliferation; other functions include intracellular matrix, proteoglycan, and collagen formation (Figure 3). Collagen has high tensile strength and supports the mechanically weak proteoglycan gel within the cartilage of this zone.

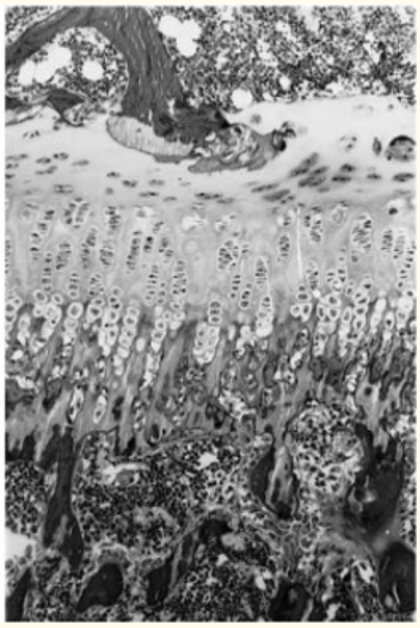


Figure 3: *Histology of the epiphyseal plate.*

The hypertrophic zone is divided into a maturation zone, a degenerative zone, and a zone of temporary calcification [1] (Figure 4). The onset of the maturation zone can be accurately determined based on cell shape. Chondrocytes become spherical and are five times the size of chondrocytes in the proliferative zone at the base of the zone. It has been shown that insulin-like growth factor stimulates hypertrophy of chondrocytes in this zone, thereby promoting longitudinal growth. The cytoplasmic content of chondrocytes in the mature zone, including glycogen, decreases rapidly in cells distal to the proliferative zone. The terminal cells, adjacent to the degenerative zone, show evidence of cell destruction and death. The oxygen tension in this part of the hypertrophic zone is low, indicating reduced metabolic activity. Chondrocytes in this zone lack cytoplasmic glycerol phosphate dehydrogenase, an important substrate for aerobic energy production by the cell. When glycerol phosphate dehydrogenase is lacking, anaerobic metabolism occurs and lactate accumulates. This environment may contribute to the death of chondrocytes in the degenerative zone. The calcium content of the mitochondria and cell walls of chondrocytes decreases as the cells are destroyed. The lost calcium accumulates in the matrix vesicles, starting from the middle of the hypertrophic zone. This matrix calcification is called primary or transient calcification. It occurs mainly in the matrix blood vessels of the longitudinal septa of the cell column. Other structures such as collagen fibers and proteoglycans are also calcified.

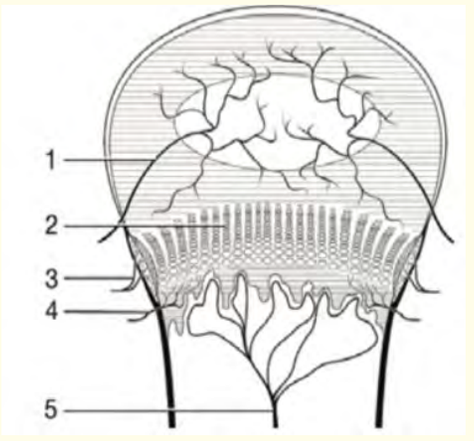


Figure 4: *Blood supply of the epiphyseal plate. (1) Epiphyseal artery; (2) epiphyseal plate; (3) perichondrial artery; (4) metaphyseal artery; (5) nutrient artery.*

Growth plate closure and contribution to overall growth

In dogs, major growth occurs between 3 and 6 months of age. Most dogs reach 90% of their adult size by the end of the 9th month. Most growth plates close between 4 and 12 months of age, depending on the anatomical location and breed. However, our clinical impression is that the growth plates of some large breeds may not close until 15 to 18 months of age. The time frame for growth plate closure in the forelimbs and hindlimbs of an average dog. The growth plates that contribute to the total axial growth of long bones remain open longer than those of smaller bones (e.g. carpals, tarsi). It is generally accepted that epiphyseal closure occurs earlier in smaller animals. Cats have a similar pattern of growth plate closure. Epiphyseal closure begins at 4 months of age and is usually complete by 7 to 9 months of age. However, complete closure of the distal femoral epiphysis can occur in cats as late as 20 months of age. Studies have evaluated the contribution of each epiphysis to overall growth [2].

The distal femoral epiphysis accounts for 70% of the longitudinal growth of the femur and 40% of the overall growth of the lower limb. Bone growth in the distal femoral epiphysis is the most rapid of all epiphyses. The distal femoral epiphysis is a rare injury with many complications. Of all the commonly encountered epiphyses, it is the most common epiphyseal injury in children [3]. It accounts for 5% of all epiphyseal fractures.

Clinical

Data collection

Clinical evaluation and radiographs (full AP length weight bearing) were performed preoperatively, immediately postoperatively, and before and immediately after plate removal. To minimize unnecessary radiation exposure to children, only clinical evaluation was performed at follow-up visits, including measurement of the intercondylar and mid-tibial distance. Cosmetic concerns, knee pain due to subluxation, patellar instability, difficulty running or climbing stairs, and frequent falls were considered symptoms of genu valgum. A detailed history of the onset and progression of the deformity was sought. The tibiofemoral angle and interankle distance were recorded in all patients at baseline and every 3 months as described below. In the standing position with the patella deviated to one side, the anterior superior iliac spine (ASIS), patellar center, and ankle center (the midpoint between the medial and lateral midpoints of the ankle) were marked. The anterior superior iliac spine and patellar center were connected, and the patellar center and ankle center were connected by 2 lines. The tibiofemoral angle was measured using a goniometer based on the above lines.

Laboratory tests included all routine tests, including serum calcium, phosphorus, and alkaline phosphatase (ALP). All patients underwent baseline computed tomography. The underlying cause of genu valgum was sought and treated medically if necessary before surgery [5].

If these values were abnormal or if axis deviation was observed, the patient underwent radiographs. If the patient did not have a lateral plane deformity before surgery, no lateral decubitus radiographs were obtained at follow-up visits, except in the case of decreased knee range of motion.

Genu varum and medial tibial torsion are normal in infants and young children, with maximal varus occurring between 6 and 12 months of age. During normal development, the lower limb gradually straightens to a zero tibiofemoral angle by 18 to 24 months of age (when the child begins to stand and walk). As normal growth continues, the knee gradually deviates into valgus (hollow knee). This valgus deformity reaches its maximum at around 3-4 years of age, with an average lateral tibiofemoral angle of 12 degrees [6]. Finally, by age 7, the genu valgum spontaneously corrects to an adult lower limb alignment of 8 degrees valgus in females and 7 degrees in males (Figure 5). The greater valgus in females may be due to their wider pelvis.

Both extrinsic and intrinsic factors can influence this normal angular alignment of the lower limb.

During childhood development, knee alignment should follow a predictable change from varus to valgus as described by Salenius and Vankka [7]. By the age of 6 years, this transition stabilizes at approximately 5-7º tibiofemoral valgus (Figure 5). By the age of 10 years, a 10º valgus deformity cannot be expected to spontaneously improve [7]. The presence of the deformity leads to abnormal joint loading, which may lead to future osteoarthritis. In addition, the deformity may lead to cosmetic problems, functional limitations, and abnormal gait mechanics.

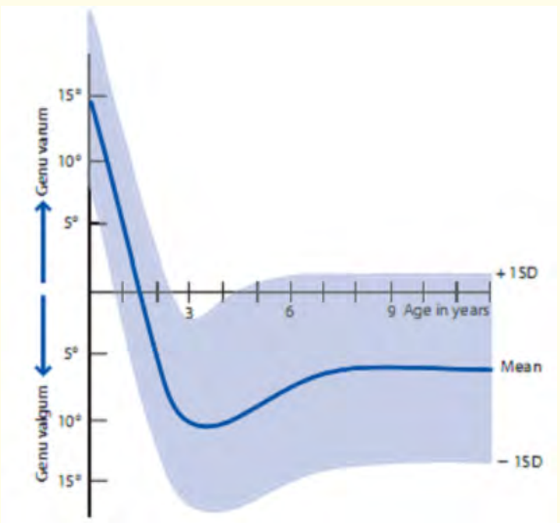


Figure 5: *The tibiofemoral angle changes as the child grows; the alignment of the knee passes from genu varum in early childhood to some degree of genu valgum before assuming the normal adult alignment around six years of age.*

Radiographic parameters

Paley and Tetsworth [8], Paley., *et al.* [9] standardized the radiographic assessment of lower limb deformities and summarized and published this method as a stepwise method called the “axis deviation test” to help determine the source of axis deviation (Figure 6). Long-term radiographs (anteroposterior and lateral) should be obtained, including the hip, knee, and ankle. Correct posture is important: the knee should be straight and the patella should be directed forward.

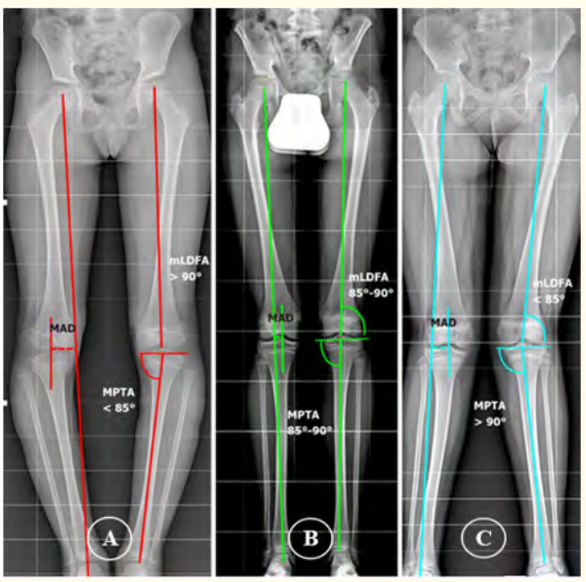


Figure 6: *The mechanical lateral distal femur angle. Medial proximal tibial angle are evaluated. A: Varus; B: Normal; C: Valgus.*

The malalignment test

The mechanical axis (MA) is drawn from the center of the femoral head to the center of the ankle. The angle between the tibia and the shaft (MAD) is measured in millimeters (the dotted lines on the figure are drawn from the center of the knee and the MA). If the MAD is outside the normal range, the cause of the deformity should be investigated. The mechanical distal lateral femoral angle. The proximal medial tibial angle is assessed. A: Varus; B: Normal; C: Valgus. MAD: Angle between the tibia and the shaft; MPTA: Proximal medial tibial angle; mLDFA: Mechanical distal lateral femoral angle.

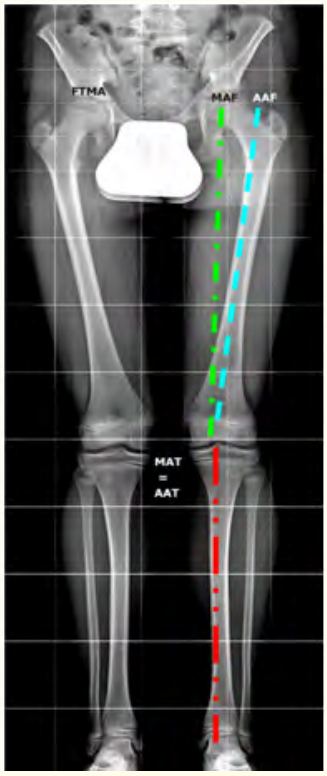


Figure 7: *Lower limb axes evaluated in long standing X-ray.*

Lower limb axes evaluated in long standing X-ray

FTMA: Femoral tibial mechanical axis; MAF: Femoral mechanical axis; AAF: Femoral anatomical axis; MAT: Tibia mechanical axis; AAT: Tibia anatomical axis.

Intercondylar and intercondylar distances

In the bow-legged position, we measured the intercondylar distance, which represents the level of the genu varum and is the distance between the femoral condyles while the lower limbs were positioned so that the medial ankles were touching each other (Figure 6).

In the knee-flexed position, we measured the intercondylar distance, which represents the level of the genu valgum and is the distance between the medial ankles and the medial femoral condyles touching each other (Figure 7).

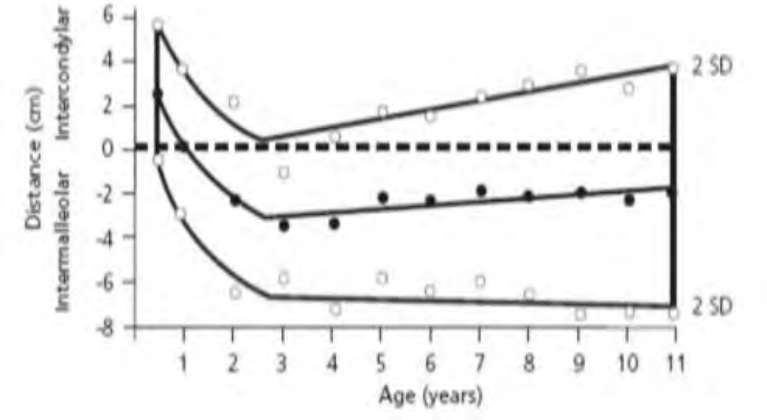
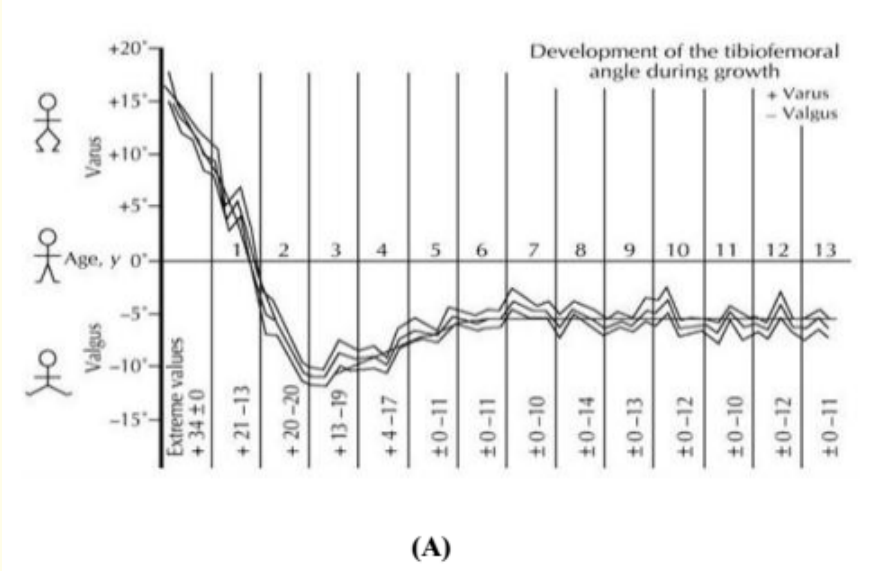


Figure 8: *Standard values of intercondylar and intermalleolar distances in a study of 196 white children. Standard values are solid dots; circles are two standard deviations.*

Tibio femoral angle

The tibiofemoral angle (TFA) or knee angle is the angle formed by the mechanical axis of the femur intersecting the mechanical axis of the tibia. When this angle is narrow, it results in genu varum and when it is too wide, it results in genu valgum. The physiologic progression of TFA from infantile varus to childhood varus is well known (Figure 8 and 9).



(A)

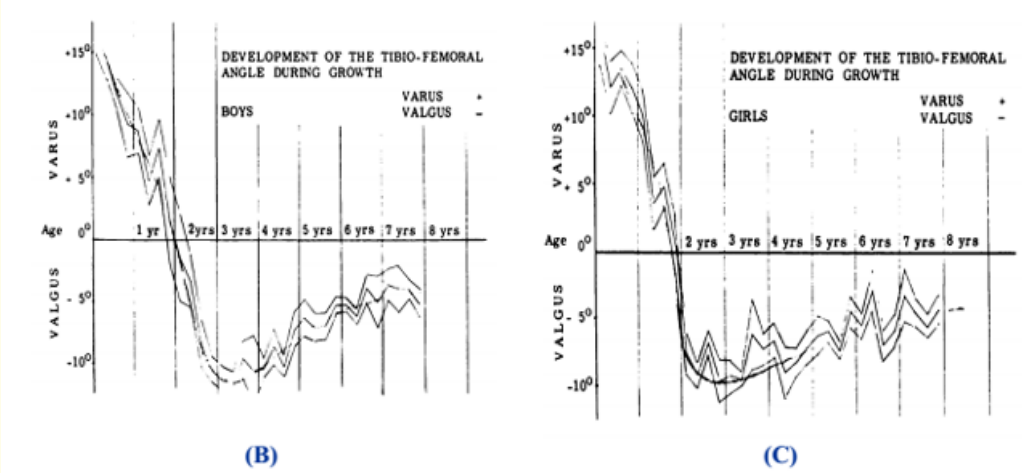


Figure 9A-9C: *Development of the tibiofemoral angle during growth (0-13 years) [1146]. A: The development of the tibiofemoral angle during growth. B: The development of the tibiofemoral angle in boys. C: The development of the tibiofemoral angle in girls.*

Natural history

Bow legs are the result of normal intrauterine posture. In utero, the lower limbs are positioned with the hips flexed, externally rotated, and abducted, the knees flexed, and the legs internally rotated. The combination of external rotation of the hips and internal rotation of the legs results in a varus-shaped lower leg. This varus position is maximal in infancy (12 to 15°) and becomes neutral by 1.5 to 2 years of age.

The knees undergo sequential physiologic changes in alignment from varus to valgus. Bow legs are common up to 2 years of age and gradually progress to knock knees from 2 years of age onwards. By 3 to 4 years of age, the valgus is maximal, and some knock knees persist throughout life.

Numerous studies in the literature have shown that physiological bow legs may appear as early as 1.5 to 2 years of age. They studied the age group from 0 to 18 years using clinical, imaging and radiological methods in different ethnic groups (Figure 10 and 11).

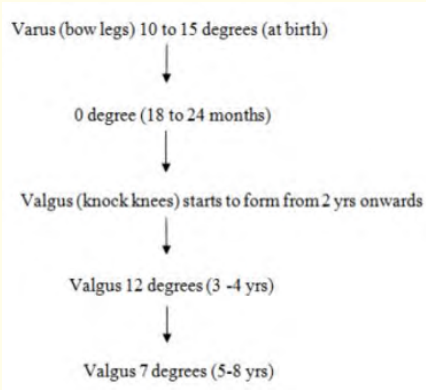


Figure 10: *Flow chart of sequential changes.*

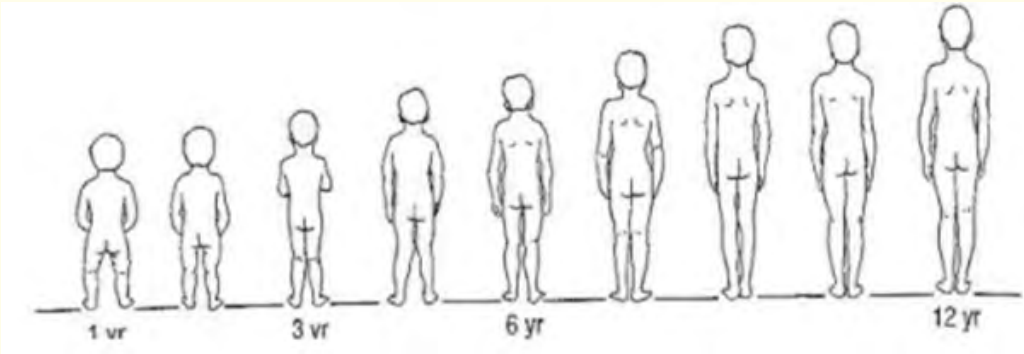


Figure 11: *Progression of TFA [1155].*

History

A detailed medical history of the parents will be taken, including when the problem was identified, when it started, and when it began. Prenatal, intrapartum, and postnatal medical histories are also taken, along with a detailed developmental history. Information about family history similarities, history of trauma, any pain, limping, tripping, falling, and even the child's sitting habits such as "W" sitting are also asked.

Examination

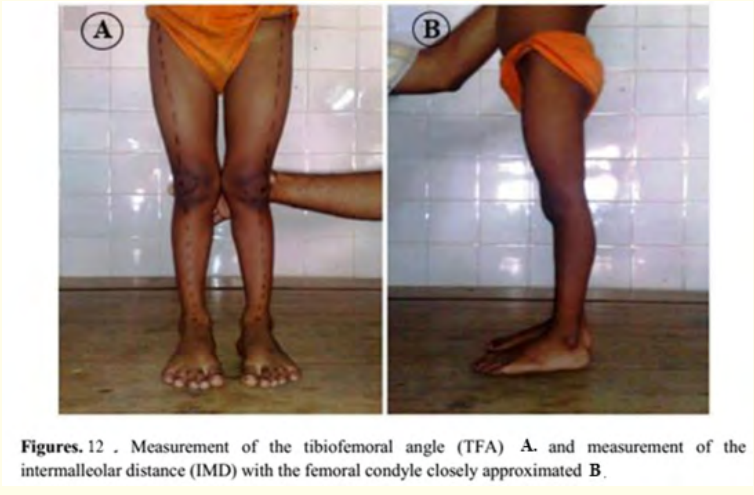
Anthropometric measurements such as height and weight should be measured and recorded on a growth chart. If normal for age, pathological conditions such as hypophosphatemia and rickets should be ruled out. The back and spine should be examined for dimples, bunions, scoliosis, or sinus cavities. A detailed neurological examination should be performed to diagnose any neuromuscular disorders (Table 1).

Causes	Genu valgum	Genu varus
Congenital	Fibular hemimelia	Tibial hemimelia
Developmental	Knock knees	Bow legs
Trauma	Over growth, partial physeal arrest	Over growth, partial physeal arrest
Dysplasia	Osteochondrodysplasia	Osteochondrodysplasia
Infection	Growth plate injury	Growth plate injury
Metabolic	Rickets	Rickets
Osteopenic	Osteogenesis imperfecta	-
Arthritis	Rheumatoid arthritis	-

Table 1: *Differential diagnosis of genu valgus and genu varus.*

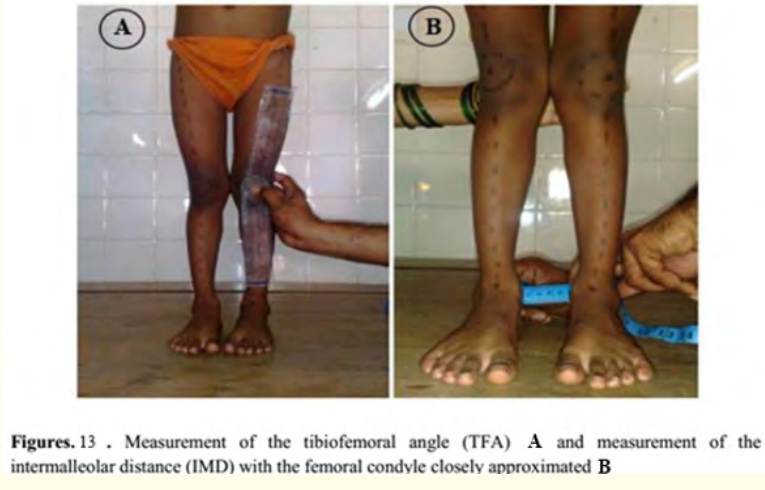
The Trendelenburg sign in the lower limbs and leg length discrepancy should be checked to rule out hip dysplasia. The range of motion of the hips, knees, and ankles should be checked. Evaluation of bow legs and knock knees is done by measuring the intercondylar distance and the intersacroiliac distance, as well as measuring the tibiofemoral angle using invasive or non-invasive techniques. Non-invasive techniques include clinical examination as well as imaging analysis. Invasive techniques mainly include X-rays [10]:

- The intercondylar distance should be measured with the legs together and distances up to 6 cm are considered normal, exceeding this distance requires orthopedic evaluation [10].
- The intersacroiliac distance should be measured with the child standing and estimating the knee. Distances up to 8 cm are considered physiological, exceeding this distance is considered pathological and requires orthopedic evaluation [10].
- TFA is measured as follows, in which the child is asked to stand with the hips and knees fully extended and neutrally rotated, with the knees or ankles touching each other. The anterior superior iliac spine (ASIS) is identified and marked with a skin marker. The center of the patella is palpated and identified with concentric circles of increasing diameter and then marked with a pen. The midpoint between the medial and lateral ankles is marked as the center of the ankle with a standard caliper. Then, using a hinged protractor placed at the center of the patella, each axis of the protractor should be adjusted so that the proximal end of the limb touches the ASIS and the distal end of the limb touches the midpoint of the ankle. TFA is measured with the protractor to the closest degree. This angle corresponds to the suspension angle of the anatomical axis of the femur with the anatomical axis of the tibia. A positive value of TFA indicates valgus, while a varus TFA is given a negative value (Figure 12 and 13) [11].



Figures. 12 . Measurement of the tibiofemoral angle (TFA) **A.** and measurement of the intermalleolar distance (IMD) with the femoral condyle closely approximated **B**.

Figure 12



Figures. 13 . Measurement of the tibiofemoral angle (TFA) **A** and measurement of the intermalleolar distance (IMD) with the femoral condyle closely approximated **B**

Figure 13

After warning signs, the physician assesses the possibility of pathological deformities [12]:

- Unilateral deformities
- Progressive deformities: For example, bow legs that become more severe after two years of age.
- ICD/IMD standard deviation > 2 for age.
- Child's height < 25th percentile for age.

Preoperative examination includes measurement of limb length and clinical deformities (both angular and rotational) and clinical assessment of gait; all patients are able to walk. For valgus, the intercondylar distance is recorded, and for valgus, the interzygomatic distance is recorded (Figure 13). Patellar impressions, long bone torsion, and ligament laxity are also noted. Radiographic evaluation includes AP weight-bearing whole-leg views with the patella facing forward and lateral views of each limb. Patellofemoral views are also performed when necessary. If there is any doubt as to whether the deformity is pathological or physiological, surgery is deferred and the patient is scheduled for a 6-month follow-up [13]. For older adolescents, hand radiographs are performed to assess the expected remaining growth time; if less than 6 months is predicted, this procedure is not recommended. Informed consent is obtained and patients are offered the options of continued observation, osteotomy, or guided growth.

The anatomic tibiofemoral angle (TFA) is measured on radiographs at the time of implantation and plate removal. TFA is used to calculate deformities because radiographic quality does not always allow a clear view of the hip; the use of TFA to calculate deformities after similar treatments has been reported in the literature. All measurements were taken by the same orthopaedic surgeon to avoid interobserver error, and were subsequently reviewed by a second senior surgeon. Valgus deformities exceeding 10° can cause anterior knee pain, circular gait, and sometimes patellar instability [14]. Scoliosis deformities can result in lateral deviation, ligamentous laxity, and a waddling gait.

Genu valgum, genu varum, and Blount disease in children

Genu valgum

Genu valgum, or bowed knee, is a common condition affecting the lower extremities in children and adolescents. Physiologic genu valgum is the most common form, but pathologic genu valgum disorders also occur and may require treatment. The most common pathologic causes of genu valgum are posttraumatic and renal osteodystrophy (Figure 14). Evaluation of children with genu valgum is similar to evaluation of genu varum and includes a careful history and physical examination. In the majority of children with genu valgum, the femoral-tibial angle is within the physiologic range, that is, two standard deviations above or below the mean. Only children with an angle greater than two standard deviations from the mean are considered to have the deformity. Fat thighs, lax ligaments, and flat feet are often the result of a splayed big toe, and this can accentuate the bowed knee shape, making the physiologic genu valgum appear more severe. Measurement of the femorotibial angle (with a goniometer) and the inter-tibia distance are methods of assessing and monitoring genu valgum [15,16].

Growth plate closure and contribution to overall growth

In dogs, major growth occurs between 3 and 6 months of age. Most dogs reach 90% of their adult size by the end of the 9th month. Most growth plates close between 4 and 12 months of age, depending on anatomical location and breed. However, our clinical impression is that the growth plates of some large breeds may not close until 15 to 18 months of age. The time frame for growth plate closure in the

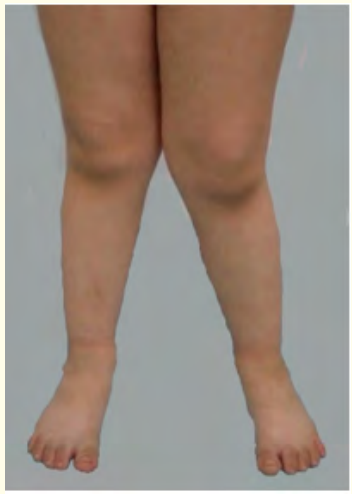


Figure 14: *Girl patient, 9 years older with genu valgum.*

forelimbs and hindlimbs of an average dog. The growth plates that contribute to the overall axial growth of long bones remain open longer than those of smaller bones (e.g. carpals, tarsi). It is generally accepted that epiphyseal closure occurs earlier in smaller animals. Cats have a similar pattern of growth plate closure. Epiphyseal closure begins at 4 months of age and is usually complete by 7 to 9 months of age. However, complete closure of the distal femoral head can occur in cats as late as 20 months of age. Studies have evaluated the contribution of each growth plate to total growth. The distal femoral head accounts for 70% of the longitudinal growth of the femur and 40% of the overall growth of the lower limb. Bone growth in the distal femoral head is the most rapid of all epiphyses. The distal femoral head is a rare injury with many complications. Of all femoral heads, it is the most common growth plate injury in children. The distal femoral head accounts for 5% of all growth plate fractures. The distal femoral head is present at birth and forms both femoral condyles. It fuses with the metaphysis in girls between 14 and 16 years of age and in boys between 16 and 18 years of age. Several studies have shown that the rate of growth disturbance after distal femoral head fracture is very high, often resulting in leg length discrepancy, angular deformity, or both. When the function of the growth plate is severely impaired, anatomical malformations may develop. Direct trauma, diet, hormonal causes, and genetics play an important role in causing growth malformations. At birth, children have a physiological valgus curvature of 10-15 degrees, an internal tibial torsion of 5 degrees, and an external rotation contracture of the hip joint. This curvature reaches its maximum at about 9-12 months of age. This curvature is usually corrected to neutral by 18-24 months of age, after which the limb develops a valgus angle, reaching a maximum of about 12 degrees by 3-4 years of age. This physiological curvature is usually corrected to the adult value of 7 degrees by 8 years of age. The physiological curvature is bilateral and symmetrical; less than 15 degrees and the distance between the two ankles is not more than 8 cm.

Clinical TFA is measured with a goniometer, while ICD and IMD are measured with a tape measure. The lower limbs are carefully positioned during the assessment. The child I is asked to stand upright, ensuring that the hips and knees are fully extended and neutrally rotated, with the knees or ankles touching (Figure 15). Distance between the two coccyx's (Figure 16).

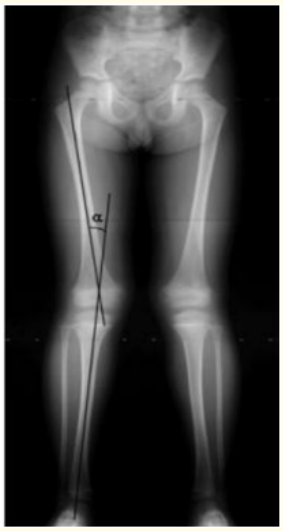
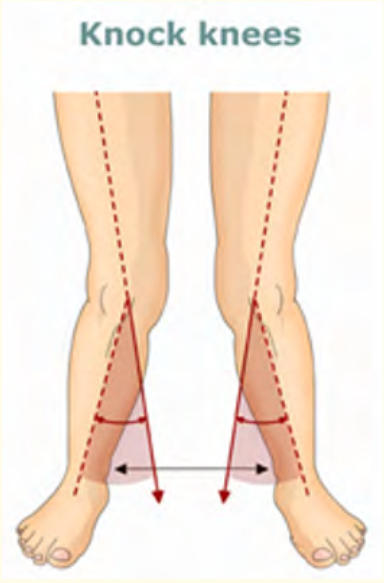


Figure 15: *The teleradiograph of the lower extremity. The radiographs were obtained in standing position, if the subject is compliant, including hip, knee, and ankle joints in a single exposure. The anatomical tibiofemoral angle (aTFA) was defined as the angle (α) between the anatomical axes of femur and tibia [17].*



FFigure 16: *Tibiofemoral angle and intermalleolar distance were measured.*

Radiographic analyses

Standard standing (patella forward) whole-leg radiographs were typically obtained every 3 to 6 months. Radiographic analysis included measurements of the distal lateral femoral angle (mLDFA) and proximal medial tibial angle (MPTA) as described by Paley and Tetsworth.

21 Measurements were performed immediately before surgery and after completion of treatment (defined as hardware removal). All radiographic measurements were performed by 2 orthopedic residents not involved in the study. Residents independently performed measurements twice for each radiograph with at least 1 month between measurements. We converted measured mLDFA and MPTA to valgus angles (angles of deviation from normal) for analysis, assuming normal values of 88 degrees for mLDFA and 87 degrees for MPTA [18]. The following variables were also calculated: correction amount, correction time, and correction rate. Correction time refers to the time interval between surgery and hardware removal. Correction rate is defined as the amount of angle correction divided by the time (in months) from surgery to hardware removal.

Pairwise comparisons were performed to see if there were any significant differences in correction rates between the two groups. Several demographic and surgical variables were examined as possible factors associated with correction rates. These factors included (1) age, (2) sex, (3) BMI, (4) surgical site, (5) implant type, and (6) deviation angle. Correlation analysis was performed to determine if the correction rate was significantly correlated with all of the possible factors mentioned above.

Various causes of genu valgum

1. Developmental - Physiologic, no intrinsic bone disease or congenital anomaly.
2. Congenital - Due to longitudinal deficiency of the fibula.
3. Iliotibial band contracture.
4. Trauma:
    a. Malunion of fracture.
    b. Growth stimulation by greenstick fracture of the proximal tibial metaphysic.
    c. Asymmetric growth arrest due to fracture-separation involving the lateral segment of the upper tibial physis or distal femoral physis.
5. Infection - Causing asymmetric growth disturbance.
6. Arthritis of knee - Rheumatoid, hemophilia.
7. Bone dysplasia - Morquio's syndrome, Ellis-Van Creveld syndrome, Ollier's disease (multiple enchondromatosis), multiple hereditary exostosis, metaphyseal dysplasia, multiple epiphyseal dysplasia.
8. Osteogenesis imperfecta.
9. Metabolic bone disease, particularly.

Pathologic genu valgum deformities

Genu valgum after fractures of the proximal tibial metaphysis

Proximal tibial fractures are relatively common and tend to occur most frequently in children aged 3 to 6 years (range 1-12 years). Boys are affected three times more often than girls, which is typical of all tibial fractures. Skak., *et al.* [19] reported an incidence of 5.6 fractures per 100,000 children per year.

Fractures usually result from direct trauma to the lateral aspect of the extended knee. The main injury patterns are compression (e.g. torus fracture), incomplete compression (e.g. greenstick fracture), or complete fracture. The most common sequelae of proximal tibial metaphyseal fractures are valgus deformity and tibial overgrowth. In 1953, Cozen reported four patients with valgus deformity after nondisplaced or minimally angulated proximal tibial metaphyseal fractures. Several other reports of this complication have been published [19].

Similar valgus deformities have been observed after other injuries to the immature proximal tibiotalar joint, such as osteomyelitis, bone grafting, osteochondroma resection, and osteotomy [20]. The incidence of genu valgum deformity after proximal tibiotalar joint fractures is highly variable. It appears to occur in approximately 50% of cases. Gurnett CA., *et al.* [21] reported on 21 patients with proximal tibiotalar joint fractures, observing the development of a valgus deformity ranging from 11° to 22° in 13 (62%) of them. Robert., *et al.* [22] reported the development of a genu valgum deformity in 12 (48%) of 25 patients. However, Macnab [23] reviewed 40 consecutive patients and found progression of deformity in only 4 (10%). Boyer., *et al.* [24] reported no varus deformity in seven 2- to 5-year-old children with trampoline fractures with one child or adult having a more severe fracture. Varus deformities occurred primarily in association with greenstick or complete fractures and were uncommon after torus fractures.

Other pathological varus deformities

Metabolic disorders

Metabolic causes of pathological varus deformities include vitamin D-resistant rickets, nutritional rickets, and renal osteodystrophy. These disorders are more likely to cause varus than valgus. This is because they have a later onset, when physiologic knee alignment has been achieved. Renal osteodystrophy is the most common metabolic disorder causing varus deformity [20]. Oppenheim., *et al.* [25] described changes in the distal and proximal tibia in children with renal osteodystrophy that are similar to the changes in the proximal tibia in Blount disease.

Treatment is usually initiated after correction of the underlying metabolic disorder. Treatment before this point has a high rate of relapse. Once the metabolic condition has been controlled, treatment can be performed with osteotomy or fixation of the distal or proximal tibia. The latter is particularly effective, provided that the bone has sufficient growth length.

Trauma

Trauma to and around the distal femoral head or proximal tibial head is a common cause of genu valgum [26].

These deformities are progressive and require surgical treatment if there is an asymmetric bridge or rod. The extent of the rod can be assessed by X-ray, CT, or, preferably, MRI. Treatment options include trochanterectomy and fat or silastic grafting (Langenskiöld procedure [27]), along with corrective osteotomy. If the trochanter is wide, complete closure and corrective osteotomy can be performed, while treatment of the leg length discrepancy may be deferred.

Neuromuscular

Children with neuromuscular disorders, such as cerebral palsy, often have valgus foot and excessive external tibial torsion, which can lead to progressive genu valgum. This is more likely due to torsional misalignment than to true knee valgum deformity. Treatment may include soft tissue release to restore muscle balance and osteotomy to correct torsional and angular deformities.

Infection/osteomyelitis

Osteomyelitis can cause valgus directly by injuring the trochanter or by creating reactive hyperemia and stimulating asymmetric growth. Asymmetric epiphyseal arrest is treated similarly to trauma.

Osteodysplasia

Valgus occurs in children with skeletal dysplasias, including multiepithelial dysplasia, epiphyseal dysplasia, epiphyseal dysplasia, and pseudochondral dysplasia. Treatment is based on the diagnosis. Orthopedic treatment is often ineffective in skeletal dysplasia. Pinning or orthopedic osteotomy is often necessary.

Treatment - Surgical

Age at time operation

Boero., *et al.* [28] adopted a different treatment approach depending on the cause; Patients with pathological deformities were treated earlier (2-13 years) than patients with idiopathic deformities (8-14 years). Patients with idiopathic deformities were treated only after it became clear that physiologic correction had failed. Surgical treatment is not recommended before the age of 8 years. After the age of 8 years, angular deformities less than 10° can be considered cosmetic and do not require surgery. However, surgical intervention should be considered for deformities greater than 10° when there is at least 12 months of expected growth remaining. The timing of surgery should take into account that the rate of correction appears to decrease with age and that growth slows as the patient reaches skeletal maturity. In some cases, metal plates are placed in both the femur and tibia to accelerate the correction process before physical closure. Burghartt., *et al.* found that tension band bracing was effective for a variety of deformities around the knee and ankle [29]. They reported results on changes in mechanical deviation and, similar to the study by Ballal., *et al.* found a younger mean age of patients. It appears that multiple factors, including age, diagnosis, growth velocity, and body size, are associated with the observed differences in correction rates. Our study is the first to report correction rates when placing a metal plate in the distal femur in idiopathic varus of the knee. It is important to understand correction rates in order to make informed decisions about the timing of surgery for patients. Theoretically, bone age is preferred in this setting because it provides a more objective criterion for determining the end point of growth. However, no method has been shown to be consistently accurate in predicting the optimal timing of surgery. Age has been identified as another factor that may independently influence the rate of correction when using metal braces. In our study, patients were grouped based on chronological age relative to the time of onset of peak growth velocity. Bone age would be more appropriate. However, not all patients had a bone age recorded at the time of the index procedure. Younger patients in this study tended to have a faster rate of correction than older patients. Unfortunately, there were not enough patients in both groups to be statistically significant. The difference between age groups was not affected by the number of cards, as the allocation rates were similar, one and two cards per group.

Some Procedures [30] (Figure 17)

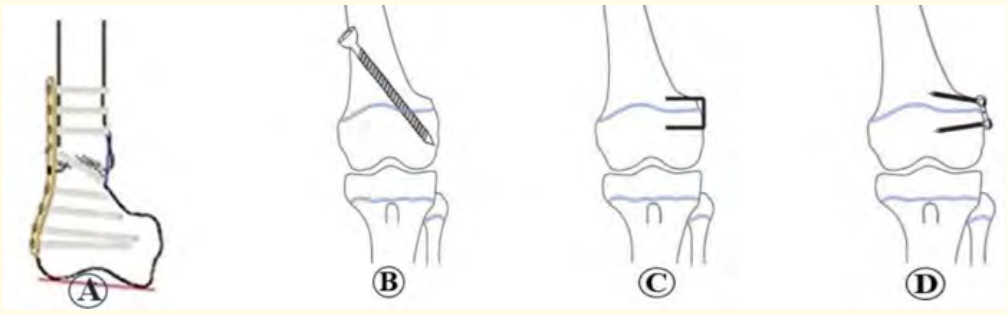


Figure 17A-17D: *A. Distal Femoral Osteotomy; B. Transphyseal cannulated screws; C. Hemiepiphyseal Stapling for Angular Deformity; D. Eight-Plate for angular correction of knee deformities.*

Wedge distal femoral osteotomy (Figure 18)

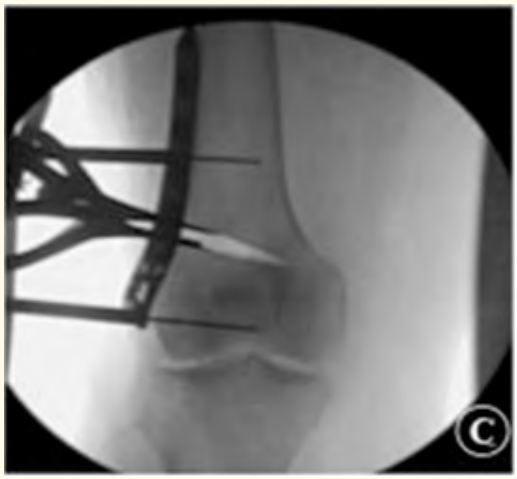


Figure 18: *Intraoperative fluoroscopy images showing a guide pin insertion in the designated trajectory of the osteotomy, the osteotomy performed by microsagittal saw, and laminar spreaders used to create an opening wedge at the osteotomy site based on preoperative planning.*

Hemiepiphyseal stapling for angular deformity (Figure 19)

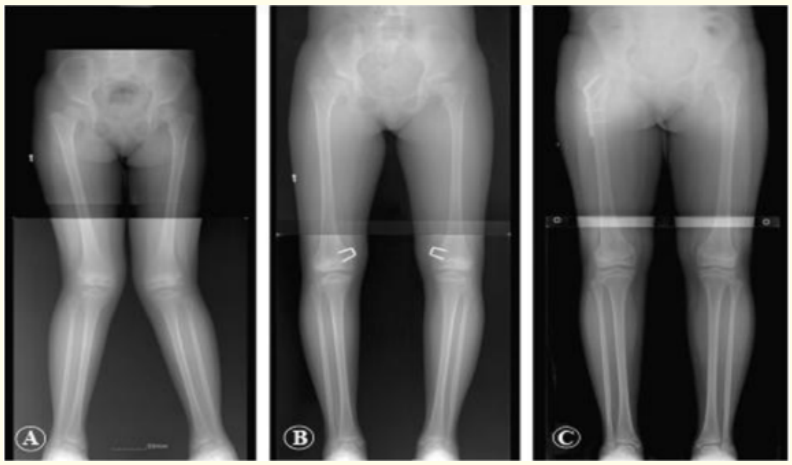


Figure 19A-19C: *An illustrative case. Hemiepiphyseal stapling was performed at age 10.9 ears (A). Staples were removed 17 months later when the deformities were slightly overcorrected (B). Two years after staple removal, the lower extremities remained in physiological alignment with some rebound phenomenon. Right proximal femoral valgization osteotomy also contributed to lateralization of the mechanical axis (C).*

Use of the eight-plate for angular correction of knee deformities (Figure 20)

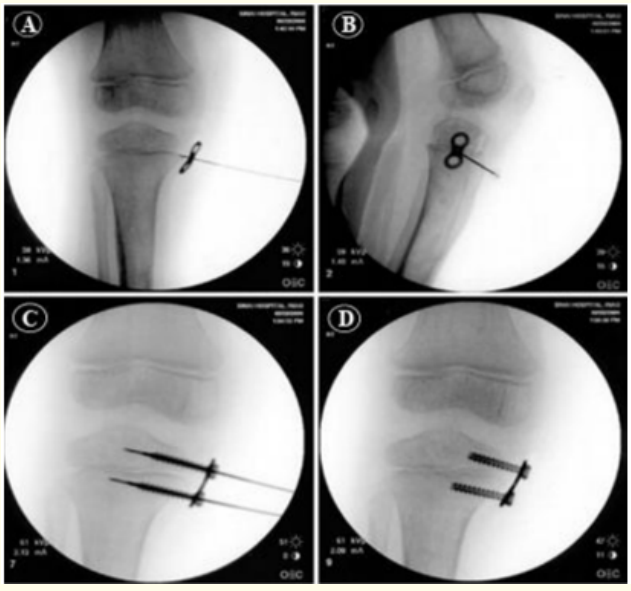


Figure 20A-20D: *Application of the eight plate. A: Anteroposterior view radiograph shows a 1.5-mm Kirschner wire being used to position the eight-Plate so that it straddles the physis. B: Lateral view radiograph shows that the position of the eight-Plate is monitored so that it is not positioned too anterior or posterior in the sagittal plane. C: Anteroposterior view radiograph shows two guide wires with threaded tips inserted in the two holes of the eight plate. Cannulated screws are then inserted over the guide wires. D: Anteroposterior view radiograph shows the final position of the eight-Plate and screws. The longer 32-mm screws depicted here are preferred over the shorter screws, especially when inserted in the metaphysic.*

Percutaneous hemi-epiphysiodesis using transphyseal cannulated screws (Figure 21)

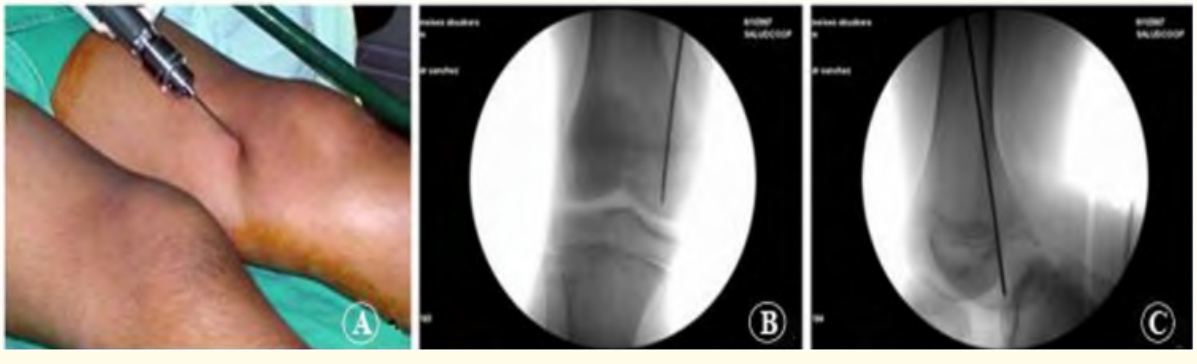


Figure 21A-21C: *Under fluoroscopic control, the elastic nail guide is placed (A) directing the tunnel from cephalic to caudal and towards the lateral part of the condyle (B, C).*

Temporary hemiepiphyseal stapling for correcting genu valgum (Figure 22)

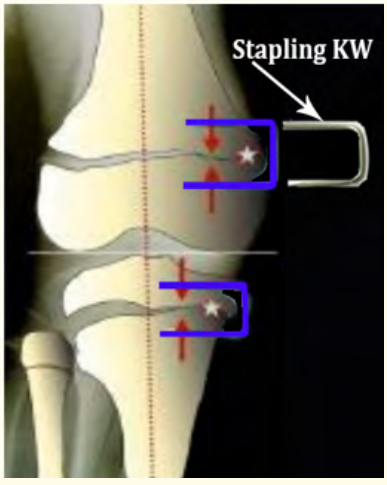


Figure 22: *Operative procedure was Illustrated with Stapling Kirschner wire [31].*

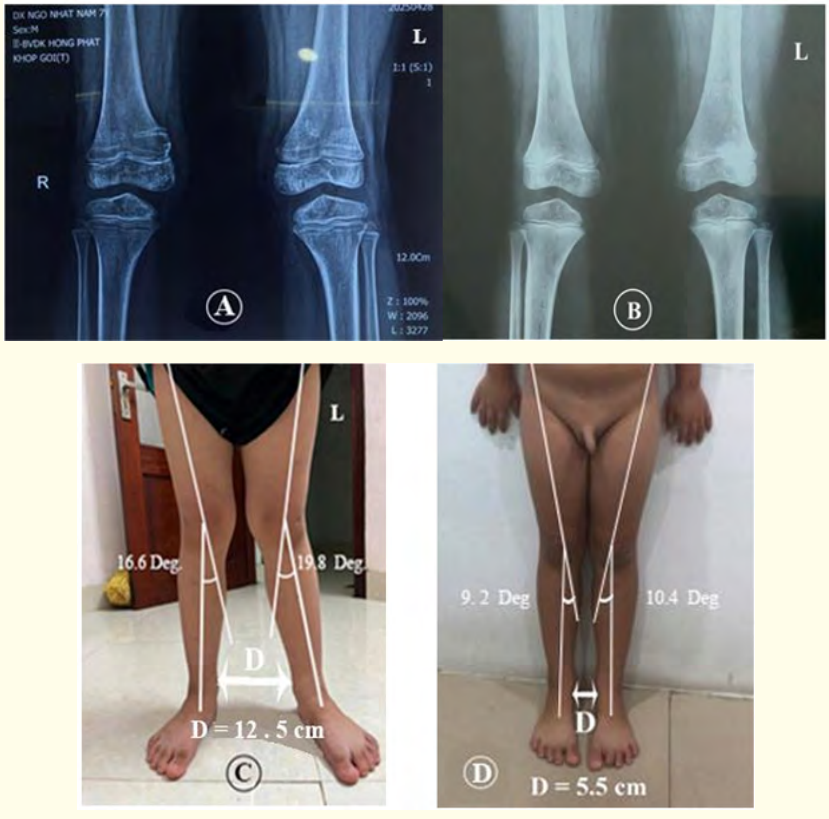


Figure 23A-23C: *7 years 3 months old boy, Genu valgum; Operated according to temporary hemiepiphyseal stapling [31]. A. Pre-operative radiography; B. Before surgery, the distance between the 2 international ankles was 12.6 cm; Tibiofemoral angle on Left 19.8°, Right 16.6°. C. Postoperative 18 months, the distance between the 2 international ankles was 5.5 cm; Tibiofemoral angle on Left 10.4°, Right 9.2°.*

Complications

Other complications in this study included superficial and deep infection, compartment syndrome, and peroneal nerve injury, which are not commonly reported.

The tension wire design is a reasonable option for the treatment of partial disease because it is temporary, easy to place and remove, and maintains an adjustable distal fulcrum.

The combination of heavier loading on the osteochondral plate, which may reduce motion under load due to its abnormal pathology, may place excessive stress on the distal screw, causing it to fail due to fatigue. For these reasons, we believe that the eight-plate system (Orthofix) should not be used in obese patients with Blount disease. Future tension wire designs should include larger, noncannulated screws made of metals with high fatigue strength.

The combination of heavier loads on the body, which may increase motion, along with the load due to its abnormal pathology, may place excessive stress on the distal stapler, causing it to fail due to fatigue. For these reasons, we believe that staplers should not be used in patients who are obese or have Blount's disease.

Conclusion: Kyphoscoliosis is usually diagnosed clinically. Patients with this condition are often asymptomatic but may have pain in the mid-knee and/or ankle. Although the history and physical examination are sufficient to make the diagnosis, there are certain signs that warrant further evaluation of the condition. Although the primary care physician is often the first clinician involved in the care of these patients, it is important to consult a multidisciplinary team including an orthopedist, geneticist, and pediatric endocrinologist depending on the underlying cause. The radiologist also plays an important role in determining the cause. Without a complete medical history, the radiologist may not be sure what to look for or what additional imaging tests to order.

Genu varum

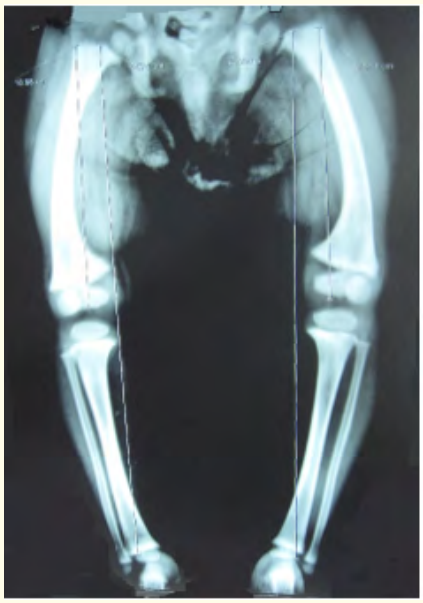


Figure 24: *Imaging genu varum.*

Bow legs, also known as bow legs, are a common childhood deformity and one of the most common causes of parental concern. In most cases, the condition is physiological in origin and resolves spontaneously as the child grows normally. However, there are pathological bow leg disorders that can progress and cause functional impairment (Figure 24).

The evaluation of a child with bow legs includes a thorough medical history and a physical examination. The medical history will usually differentiate physiological from pathological bow legs. Look for the birth history, family history, age at which developmental milestones were achieved, nutritional history, and previous percentiles for height and weight. A family history of short stature or bow legs or the progression of the deformity may indicate a pathological process.

X-rays are usually not necessary in cases of flexed knees. However, if the child is short, has an asymmetric deformity, has a history of progression, or is older than 3 years, an anteroposterior (AP) view of the lower extremities, including the hips, knees, and ankles, should be obtained. The patella should be positioned anteriorly. Measure the femoral-tibial angle, mechanical axis, and angle between the pubic bone and the shaft. Assess the femoral and tibial tubercles, especially those around the knee.

Physiologic genu varum

Pathophysiology

Physiologic genu varum due to intrauterine positioning is a common finding in children from birth to 2 years of age. This condition is often associated with an inward gait due to medial tibial torsion.

The lower extremities appear flexed when the child is standing. However, clinical examination reveals hyperextension of the hips and medial tibial torsion (Figure 25). Posterior capsular contracture is a normal finding in children younger than 1 year. This condition tends to improve over the first 3 years of life, and eventually the internal rotation will slightly exceed the external rotation.

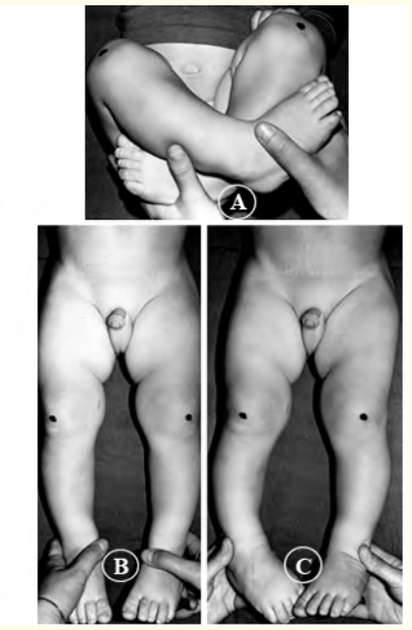


Figure 25A-25C: *A: Physical examination of a 9-month-old boy with physiologic genu varum. The infant may still comfortably assume the in utero position with the hips flexed, abducted, and laterally rotated. The knees are flexed, with the lower legs and feet medially rotated. This position results in a hip flexion contracture, a contracture of the posterior aspect of the hip capsule, knee flexion contracture, and medial tibial torsion. B: When the lower extremities are extended, the posterior hip capsule contracture results in increased lateral rotation (80° to 90°) and limited medial rotation (0° to 10°). When the patellae point laterally, the medial tibial torsion is not readily apparent. C: When the hips are maximally rotated medially and the patellae are directed anteriorly, the medial tibial torsion is more apparent. The medial tibial torsion can be measured by the thigh-foot angle or the transmalleolar axis. The medial tibial torin-toeing during ambulation. This can be assessed by measuring the foot progression angle.*

On radiographs, typical features of physiologic valgus include [32]: Medially tilted transverse planes of the knee and ankle. Slight lateral bowing of the tibia at the junction of the proximal and middle thirds and the femur at the distal third. Thickening and stiffening of the medial cortex of the tibia and femur. The epiphyses, osteophytes, and metaphyses are normal in appearance and there is no evidence of intrinsic bone pathology. The lesions are usually symmetrical.

It can be difficult to distinguish radiographically between physiologic valgus and tibial valgus (Blount disease) in children younger than 3 years. Levine and Drennan [33] developed the diaphyseal angle to aid in distinguishing between these two disorders (Figure 24).

An angle of 11° or less indicates physiologic valgus, and an angle greater than 11° suggests the possibility of progressive tibial valgus (Figure 26). However, a later study by Feldman and Schoenecker showed that angles greater than 16° could predict tibial ulnar deviation, while angles of 9° or less indicated physiological ulnar deviation, and angles of 10° to 15° were indeterminate. The angle between the tibia and ulna has been shown to be well reproducible between and within the same observer. However, it is important to obtain standing radiographs with the knee in a neutral position, as changes in rotation can alter measurements [34].

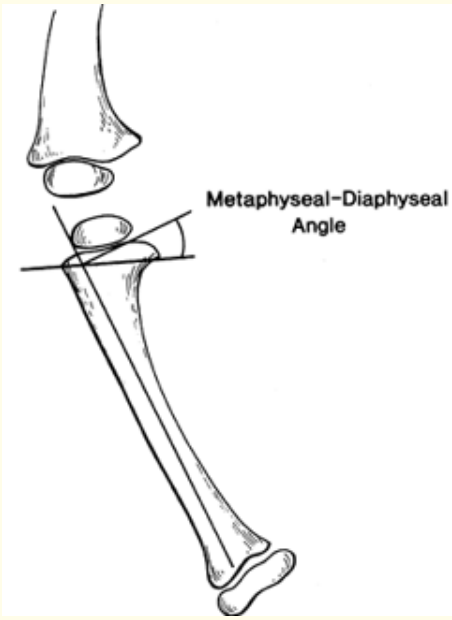


Figure 26: *Metaphyseal-diaphyseal angle. Draw a line between the radiographic corners of the medial and the lateral metaphyses of the proximal tibia, and another line parallel to the longitudinal axis of the tibial diaphysis. Then construct a line perpendicular to the diaphyseal line at the intersection of the metaphyseal and diaphyseal lines, and measure the angle between the right-angle line and the metaphyseal line. (Adapted from Levine AM, Drennan JC. Physiologic Bowing and Tibia Vara: The Metaphyseal-Diaphyseal Angle in the Measurement of Bowleg Deformities).*

If metabolic disorders are suspected, serum calcium, phosphorus, and alkaline phosphatase levels should be measured. Pediatric endocrine evaluation aids in diagnosis and treatment (Figure 27). Physiological scoliosis resolves spontaneously as the child grows and develops normally [35,36].

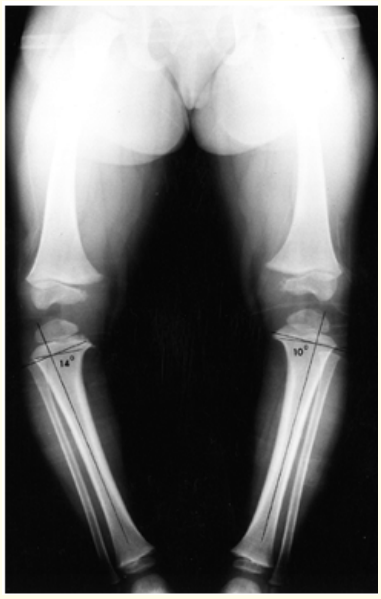


Figure 27: *Standing AP radiograph of an 18-month-old girl with asymmetric bowing of the lower extremities. The metaphyseal-diaphyseal angle is 14° on the right, indicating infantile tibia vara; it is 10° on the left, representing physiologic genu varum. There is already medial metaphyseal irregularity and breaking, as well as mild medial epiphyseal flattening on the right.*

Surgery is rarely indicated. Orthopedic braces or orthopedic shoes are not recommended because there is no evidence that they improve limb alignment. Follow-up of infants and young children with physiologic scoliosis is recommended every 6 months (Figure 28). Accurate clinical measurements reassure parents that their child is progressing well.

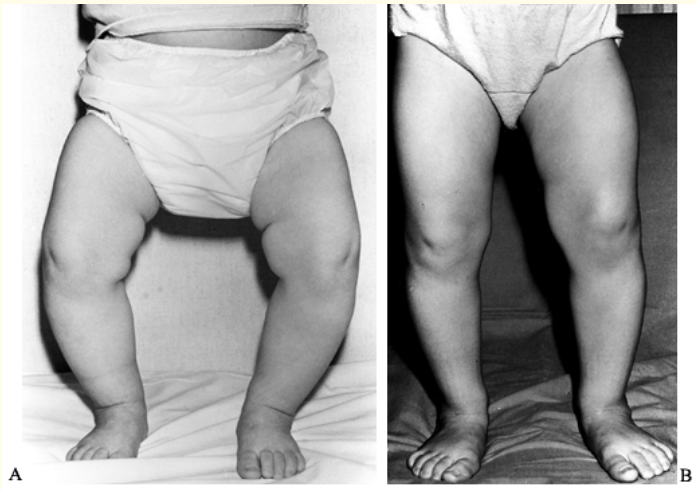


Figure 28A and 28B: *A: 16-month-old girl with physiologic genu varum. Observe the lateral rotation of the thighs and knees and the medial tibial torsion. B: One year later, with no treatment, there has been complete resolution.*

Operative techniques

Techniques for correcting valgus knees are listed in table 2. Common procedures for treating physiologic valgus knees include proximal tibial rotation and fibula osteotomy, proximal tibial hemiarthroplasty, or proximal tibial hemiarthroplasty. The latter two procedures rely on adequate residual growth to achieve complete correction [37]. The nomogram developed by Bowen., *et al.* [38] may be helpful in determining the appropriate timing.

Distal femur
Valgus osteotomy
Closing wedge
Opening wedge
Callotasis
Lateral hemiepiphyseal stapling
Lateral hemiepiphysiodesis
Proximal tibia
Valgus osteotomy (including derotation and diaphyseal fibular osteotomy)
Closing wedge
Opening wedge
Dome
Oblique
Callotasis
Other
Lateral hemiepiphyseal stapling
Lateral hemiepiphysiodesis

Table 2: *Surgical options for genu varum deformities.*

Proximal tibial varus osteotomy and diaphyseal fibula osteotomy

This is the most common procedure for treating persistent physiologic varus because it addresses both the varus and the internal torsion of the tibia. A variety of techniques can be used, including closed, open, oblique, or wedge-shaped. These are essentially the same procedures as surgery for tibial varus or Blount disease.

Proximal tibial varus osteotomy and diaphyseal varus osteotomy are discussed in the section on tibial varus. Internal or external fixation is required to maintain alignment and is often supplemented with a long cast until complete healing occurs.

Proximal tibial hemipinning

Temporary lateral growth retardation of the proximal tibial head with a nail is an effective method for correcting persistent physiologic varus of the knee. If the deformity is severe or residual growth is limited, combined lateral fixation of the distal femoral head and proximal tibial head may be necessary. This procedure will not correct medial tibial torsion at the same time.

Proximal tibial hemi-pinning

More extensive fixation of the proximal tibial head may be effective in the correction of persistent knee dislocations in adolescents. The indications are essentially the same as pin fixation. However, after complete reduction, a second procedure may be required in between to avoid overcorrection. The proximal fibula is usually fixed at the same time. This procedure will not correct any medial tibial torsion.

Rehabilitation and postoperative guidelines

In general, children treated with proximal tibial external rotation and diaphyseal fibula osteotomy are managed similarly to patients undergoing the same procedure for tibial dislocations.

In children treated with suture or proximal tibial head fixation, a knee brace is used postoperatively for approximately 2 weeks. This promotes healing and minimizes discomfort. Active range-of-motion exercises are then initiated and normal activities are allowed, usually 4-6 weeks postoperatively.

Pitfalls and complications

Complications of proximal tibial osteotomy have been well described in the orthopedic literature [39,40]. There is a risk of injury to the peroneal nerve as it passes over the lateral aspect of the proximal fibula, and to the anterior tibial artery as it enters the anterior compartment through the proximal tibiofibular notch. Compartment syndromes have been described, and children should be carefully evaluated in the first 24-48 hours postoperatively. Prophylactic anterior compartment fasciotomy is performed at the time of surgery.

Conclusion: Physiologic varus rarely persists to the point of requiring surgical intervention. There is an association between this disorder and tibial varus (Blount disease). Persistent varus may progress to varus. In my experience, the most common abnormality of physiologic varus is persistent tibial torsion. This is the more common indication for surgical treatment.

Pathological valgus deformity of the knee

Tibial valve disease

Idiopathic valgus deformity of the knee (Blount disease) is the most common pathological valgus deformity of the knee. It is characterized by abnormal development of the medial aspect of the proximal tibial head, resulting in a progressive valgus angle below the knee. The disorder was first described by Erlacher [41] in 1922 and further analyzed by Blount [42] in 1937.

Classification

Tibial valve disease can occur at any age in growing children. Initially, it was classified into two main groups, depending on the age of clinical onset: infantile, with onset between 1 and 3 years of age; and juvenile, with irregular onset, occurring after 6-8 years of age or just before puberty [43]. In 1984, Thompson., *et al.* [44] proposed a three-group classification based on the age of onset: infantile (1-3 years), juvenile (4-10 years), and adolescent (11 years or older). The juvenile and adolescent forms are often combined into a late-onset form of tibial dislocation. However, the rate of recurrence of the deformity after tibial osteotomy for proximal tibial dislocation is much higher in the juvenile group, which justifies the three-group classification. All three groups have relatively similar clinical features, although the radiographic changes in the late-onset groups are less pronounced. Although the exact cause of tibial luxation is unknown, it appears to be secondary to growth inhibition due to increased compressive forces on the medial aspect of the knee. Familial cases have been reported.

The natural history of tibial luxation is a progressive form of luxation. Infantile luxation may cause the greatest degree of deformity due to the longer remaining growth period. In 1952, Langenskiöld [45] described six stages of progressive deformity in infantile luxation. Each stage results in progressive inhibition of epiphyseal growth.

Assessment, indications, relative outcomes

Comparison of the clinical features of infantile luxation and late-onset (juvenile and adolescent) forms of tibial luxation shows significant similarities as well as differences (Table 3). Infantile luxation is the most common form. However, late-onset forms are also common (Figure 27). ACL dysfunction can occur in severe deformities.

Infantile	*Late-onset (juvenile and adolescent)*
African-American race	African-American race
Female predominance	Male predominance
Marked obesity	Marked obesity
Bilateral involvement (80%)	Bilateral involvement (50%)
Medial metaphyseal beak	No medial metaphyseal beak
Medial tibial torsion	Minimal medial tibial torsion
Lower-extremity length inequality	Mild lower-extremity length inequality
	Pain rather than deformity
	Mild medial collateral ligament laxity
	Steadily progressive deformity

Table 3: *Comparison of the clinical characteristics of tibia vara.*

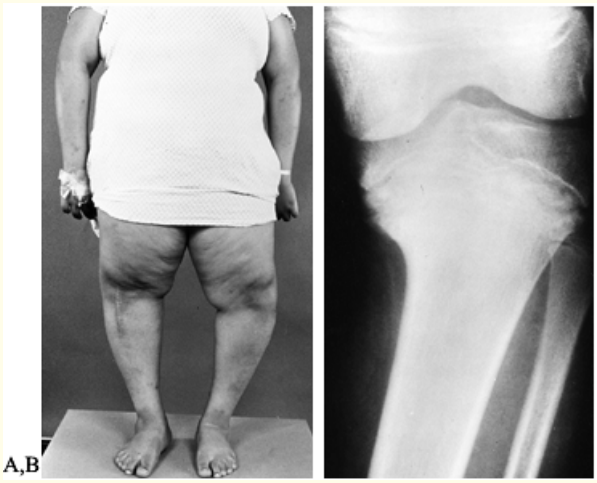


Figure 29: *A: Standing preoperative photograph of a 13-year-old African-American boy with bilateral adolescent or late-onset tibia vara. A previous proximal tibial osteotomy was performed on the right, producing only partial correction. Observe the marked obesity and the untreated left genu varum deformity and its medial tibial torsion. The patient subsequently underwent a laterally based closing-wedge proximal tibial osteotomy, including the physis, and a diaphyseal fibular osteotomy for correction of this deformity. B: On a standing AP radiograph of the left knee, the radiographic changes in adolescent tibia vara are less striking than in the infantile form. There is narrowing of the medial aspect of the proximal tibial epiphysis, physeal irregularity, and increased height of the lateral aspect of the epiphysis.*

On radiographs, the main features of infantile valgus are fragmentation with convexity of the step deformity and the proximal medial tibial process (Figure 29).

The changes in the proximal tibial process are less obvious in late-onset forms and are characterized by medial epiphyseal compression, mild posteromedial concavity, cephalad curvature, and mild fragmentation or absence of the proximal medial tibial process. The differences between the three types of valgus appear to be due mainly to the age of onset, the amount of remaining growth, and the intensity of the medial compressive force on the affected side.

An angle between the tibia and the shaft greater than 16° is an early predictor of infantile valgus. After 2-3 years of age, the radiographic features become apparent and the condition can be classified according to the six Langenskiöld grades [46]. The radiographic changes in late-onset forms of tibial eversion are less pronounced but are still of diagnostic value.

Pathophysiology

Histological studies revealed a similar pathological process in all three groups. Only a few proximal medial tibial condyle biopsies were obtained from patients with infantile tibial eversion. Histopathological abnormalities included dense islands of chondrocytes that exhibited greater hypertrophy than expected based on their topographic location, areas of nearly acellular cartilage, and clusters of abnormal capillaries. Langenskiöld [46] in studies of nine and six biopsies, respectively, concluded that the abnormalities were mainly localized to the trochanters and that there was no evidence of avascular necrosis.

These changes were found uniformly throughout the medial and lateral aspects of the growth plate, although they were numerically greater on the medial aspect. They were very similar to those seen in infantile tibial eversion and in slipped femoral epiphyses, suggesting a common etiology [1,2]. Lovejoy and Lovell [49] described two patients with late-onset tibial valgus associated with slipped capital femoral epiphysis. The histopathological abnormalities showed that asymmetric compressive and shear forces acting on the proximal tibial epiphysis resulted in inhibition and distortion of normal endochondral ossification, resulting in tibial valgus. This concept, reflecting the Heuter-Volkmann law, was confirmed experimentally by Arkin and Katz [50]. Golding and McNeil-Smith [48] concluded that children with significant physiologic tibial valgus often walk early and have lax knee ligaments. This leads to asymmetric compression, subsequent inhibition of posteromedial epiphyseal growth, and eventual bridging of the bones, resulting in permanent and progressive valgus. This pathogenesis is consistent with Blount's [51,52] original observation that infantile tibial valgus was first recognized as an increase in physiologic curvature during the first 3 years of life. Langenskiöld [53,54] emphasized that epiphyseal chondronecrosis is the main cause of growth disturbance leading to varus deformity; he attributed the abnormal cartilage to abnormal pressure or shear in overweight children with physiological bow legs. Others agree that abnormal pressure may be the main factor in tibial varus in infants.

In older children or adolescents with minimal residual deformity after physiological varus, rapid growth and repeated weight gain damage the posteromedial portion of the proximal tibial epiphysis, leading to a cycle of varus growth inhibition similar to that described by Golding and McNeil-Smith [48] in the infantile form.

The progression of varus is not due to a bony bridge but rather to inhibition of normal endochondral growth following repeated local trauma. The concept of growth inhibition of the distal tibial epiphysis was biomechanically confirmed in finite element analysis by Cook., *et al*. [55]. As varus increases, the forces acting on the proximal mid-tibial epiphysis also increase. Obesity and mild varus (10°) in older children generate forces sufficient to inhibit growth. However, Henderson and Green [56] reported a case of late-onset tibial malalignment in an adolescent with previously neutral biomechanical axis, suggesting that, at least in some cases, preexisting tibial malalignment is not a prerequisite.

Preoperative management and planning

If the radiograph confirms the diagnosis of tibial malalignment in infants, treatment should be initiated immediately. Orthopedic treatment may be considered for children under 3 years of age with Langenskiöld grade II and possibly grade III malalignment. Approximately 50% to 65% of these cases in children can be corrected with orthotics.

Once the absolute biomechanical axis is achieved, weaning should begin. The child should be followed closely thereafter to ensure that correction is maintained. Currently, a trial of orthopedic treatment for up to 1 year is recommended. If correction is not achieved after 1 year of bracing, corrective osteotomy is indicated. Orthopedic treatment is not indicated after 3 years of age or for severe deformities.

Bracing in children older than 3 years of age carries the risk of delaying corrective osteotomy. Loder and Johnston [57] have shown that delaying orthopedic surgery, even for several months, after the age of 4 years risks not achieving lasting reversal of proximal tibial root compression. This makes the child more likely to require revision surgery to maintain the desired result.

Conservative treatment is contraindicated in late-onset forms of tibial dislocation. The child is too large and the remaining growth is too small to allow for adequate correction. Compliance with orthopedic correction in this age group is difficult.

Overcorrection compensates for the tendency of the knee to return to an adducted position after the patient resumes weight bearing due to posteromedial articular surface pressure and lax collateral ligaments. The goal is to shift the load to the lateral compartment of the knee. Schoenecker., *et al.* [58] have reported that correction within 5° of neutral position usually produces satisfactory results. However, others have recommended overcorrection. Considering the growth plate inhibition phenomenon as suggested by Cook., *et al.* [55], overcorrection of absolute lateral deviation is necessary to reduce excessive medial compression.

Rab [59] described a proximal oblique tibial osteotomy for the treatment of tibial dislocation. This is a uniplane osteotomy that allows simultaneous correction of medial and medial tibial dislocation, while allowing for postoperative cast placement, if required, to improve the position. This ability to correct the osteotomy position postoperatively is important because of the difficulty in achieving satisfactory alignment during surgery.

Procedures that may be used for these children include: Multiple proximal tibial and fibular osteotomies Proximal tibial osteotomy with growth plate resection Intraepithelial osteotomy to elevate the medial tibial articular surface (medial tibial plateau elevation) Excision of the tibial bridge and replacement with interpositional material such as fat or silastic semiepithelial osteotomy of the lateral aspect of the proximal tibial head Oblique proximal tibial osteotomy Ilizarov ring fixation system and callotasis technique In cases of tibial dislocation in children and adolescents, surgery is required to restore the mechanical axis of the knee. The same surgical options as for older children with infantile tibial dislocations are applicable to these groups. The goal is to correct the physiologic genu valgum by careful biometric planning prior to tibial osteotomy. Kline., *et al.* [60] demonstrated that distal femoral malalignment is a component of deformity in late-onset tibial dislocations. Assess this possibility and consider it in the treatment plan. Obtain intraoperative radiographs with the knee extended and gently compress the tibial translation to ensure contact between the medial femoral condyle and the posteromedial articular condyle of the proximal tibia. This technique may help minimize under correction of the deformity. The goal is to achieve at least 5° of tibial translation at the time of osteotomy. Recurrence rates in children are typically approximately 25% and are even higher in boys. Evaluate all pediatric-onset patients with preoperative CT or MRI for evidence of premature or impending closure of the proximal medial tibial epiphysis. If premature closure is not achieved, a simple closed wedge osteotomy or oblique proximal tibial osteotomy with physiologic correction of the translation may be performed. Correction with the Ilizarov ring fixation system and callotasis may be considered, especially if there is a significant lower limb length discrepancy [61].

Internal or external fixation is often required to maintain alignment until complete healing. Reduction of the epiphysis with an external fixator has also been used in Europe [85,87], but this method is not widely used. The method chosen depends on the age of the patient, the amount of remaining growth, and the severity of the deformity. Proximal fibular trochanterectomy is generally recommended for recurrent deformities with early closure of the medial tibial head or for patients 12 years of age or older [62]. Healing is rapid and the correction is permanent. Any discrepancy in the remaining lower limb length is measured by CT scan and treated with adjustment of the contralateral fibular trochanter, when necessary. Henderson., *et al.* reported the results of nine children with late-onset tibial dislocations treated with partial diaphyseal adjustment of the lateral aspect of the proximal tibial head. The average curvature after surgery was 13° (range 3° to 25°).

Operative techniques

Proximal tibial valgus osteotomy and fibular diaphyseal osteotomy

- Make a transverse or transverse skin incision 5 cm below the tibial tuberosity.
- Expose the proximal tibia under the periosteum.
- Release the anterior compartment fascia. The patellar tendon attachments are usually seen proximally in the incision.
- Perform a axial fibular osteotomy through a 3 cm longitudinal incision at the junction of the middle and proximal thirds of the fibula.
- Identify the muscles of the lateral compartment and pull them anteriorly. Split the fibular periosteum longitudinally and rotate it.
- Perform an oblique osteotomy with a small oscillating saw.
- After completing the fibula, perform a proximal tibial osteotomy. This can be a closed, open, or dome osteotomy. This procedure will allow correction of medial tibial torsion and varus deformity. I prefer a closed wedge osteotomy. It is important to correct the medial tibial torsion first, then perform a closed lateral wedge osteotomy. Overcorrection and unnecessary bone resection can occur if the torsion is not corrected first.
- Fix the osteotomy site with a crossed Steinmann pin, compression plate and screws, or an external fixator. I prefer the latter because the pin can be removed in the outpatient clinic without the need for a separate surgery.
- Obtain an intraoperative radiograph with the knee extended to confirm that approximately 5° of lateral deviation has been achieved.
- After closure, immobilize the leg with a long plate with the knee extended and apply gentle external pressure.

Oblique tibial osteotomy

- Make a horizontal incision just below the tibial tuberosity. Make a Y-shaped incision in the periosteum and elevate the periosteum circumferentially.
- Insert a small Steinmann pin at a 45° head angle, 1 cm distal to the tibial tuberosity, and advance under the control of the image intensifier until it passes through the posterior cortex, exiting the proximal tibial head. The angle of insertion determines the degree of correction of the varus and medial tibial torsion. A diagram is provided to assist in determining the appropriate angle of the guide pin before surgery.
- Carefully perform the osteotomy just below the Steinmann pin.
- After completing the tibial osteotomy, perform the fibular osteotomy. This allows free movement of the tibia.
- Drill a hole in an anteroposterior direction, cutting across the osteotomy line and lateral to the tibial tubercle. Insert a 3.5 mm cortical lag screw or cancellous screw, align the osteotomy line, and loosen the screw to allow for later adjustment if necessary.
- After the anterior compartment fasciotomy, close the incision. Place a suction drain at the time of closure.
- Apply a long-leg cast. Rab [59] recommends injecting contrast into the knee to improve the ability to check alignment on radiographs after casting but before applying a long-leg cast.

Excision of the proximal tibial physis

If the deformity recurs in older children with pediatric tibial malalignment, proximal tibial condyle resection may be beneficial.

The procedure is similar to the proximal tibial osteotomy described previously. A similar incision is made, but more proximally. The patellar tendon is mobilized medially and laterally.

A smooth Steinmann pin is placed through the tibial head just below the articular surface. The entire condyle is removed by closed wedge osteotomy. A second pin is placed distally and an external fixator is used.

The fibula is always resected. The advantage of this operation is that it is performed at the site of the deformity, thus allowing for maximum correction and physiologic realignment of the tibia. Healing is usually rapid and there is no risk of recurrence of the deformity. Fixation of the opposite proximal tibial head can be performed at the same time. However, in most cases, the extent of lower limb length discrepancy is monitored with CT scans, and any remaining leg length discrepancy is corrected with surgical fixation of the opposite distal femoral head at an appropriate time.

General and post-operative rehabilitation principles

Post-operative care for children is similar to that following any orthopedic osteotomy of the proximal tibial head. Continue immobilization until complete healing; then engage the child in a home physical therapy program for approximately 2 weeks. Return to normal activities after full rehabilitation. Because of the associated obesity, these children often benefit from dietary counseling.

Complications

Complications during and after proximal tibial osteotomy are common. These include peroneal nerve palsy, anterior tibial artery injury, and compartment syndrome [63]. Compartment syndrome can be minimized by performing an anterior compartment fasciotomy at the time of surgery. If compartment syndrome occurs, the correction is temporarily reduced and a four-compartment fasciotomy is performed. Children with infantile tibial deformity require long-term follow-up to assess the surgical outcome. The deformity may recur, especially in older children and those with advanced Langenskiöld grades. Persistent deformity after skeletal maturity leads to osteoarthritis [64].

Conclusion: We prefer proximal tibial rotation and transtibial fibula osteotomy for correction of varus in young children. This approach allows simultaneous correction of both components of the deformity. It is important to slightly overcorrect the deformity so that the mechanical axis enters the ankle joint. Assessing the degree of correction during surgery can be difficult due to the inability to obtain long-band radiographs. A useful clue is the visualization of the iliac crest on the affected side. Fluoroscopy can be used to measure the mechanical axis on the fluoroscope. The wire can be stretched between the anterior superior iliac spine and the middle of the patella. The distal length of the wire relative to the ankle joint can then be assessed. Under correction is a common problem, preventing proper alignment. Intra-articular contrast at the time of surgery may also be helpful. Once the osteotomy has been internally stabilized, manually manipulate the knee inward to create a more realistic sense of limb alignment under pressure.

Tibia vara caused by focal fibrocartilaginous dysplasia

Valgus secondary to focal fibrochondritis involving the medial aspect of the proximal tibia was first reported by Bell., *et al.* [65] in 1985. Since then, other cases have been reported. Valgus can also affect other regions of the body. Lincoln and Birch [66] reported the condition involving the upper limb. It is a rare cause of pathological varus but should be distinguished from Blount disease because the natural histories of the two disorders are quite different.

Pathophysiology

Biopsy of lesions at the time of orthopedic surgery or for diagnostic purposes have shown consistent histopathological features. Generally, there is a well-demarcated white cartilaginous lesion deep to the metatarsal attachment.

Histological findings include acellular or paucicellular collagenous tissue, inactive fibrocytes, chondrocyte-like cells in the joint spaces, and dense, poorly demarcated fibrocartilaginous tissue.

No giant cells, osteoid, or bone tissue were found in these lesions. The lesions were suggestive of fibrocartilage centrally and tendon tissue peripherally. They did not involve epiphyseal cartilage or the epiphysis. Bell., *et al.* [65] observed that this tissue resembled that normally found at the site of tendon attachment to cortical bone, as described by Cooper and Misol [67] in 1970. They suggested that these children had abnormal development of fibrocartilage at the site of metatarsal attachment. The exact mechanism of this abnormal development is unknown. The defect may be congenital.

Assessment

All children with focal fibrous dysplasia of the tibia have a unilateral tibial curvature. There is no clear sex or lateral predilection. The condition usually begins before age 1 and the deformity progresses until about age 2 before it begins to resolve spontaneously. As it progresses, the deformity may become quite marked, reaching a median curvature of 20° to 30°. Medial tibial torsion and a mild tibial length discrepancy (0.5-1.0 cm) are common accompanying findings.

The lesions are usually painless to palpation, and there is no medial tibial condyle as is often seen in varus in young children. On radiographs, there is a cortical defect in the medial tibial epiphysis proximal to the surrounding sclerosis (Figure 30). MRI will show dense fibrous connective tissue. CT will show similar findings, with an elliptical cortical defect but no soft tissue mass.

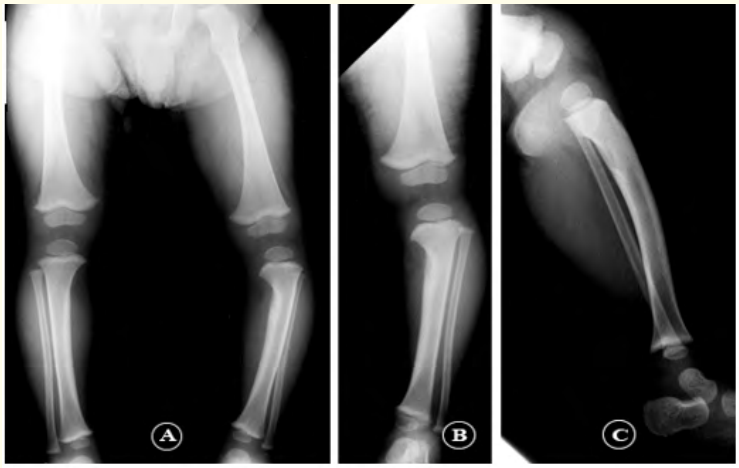


Figure 30: *A: Standing AP radiograph of a 2-year-old Caucasian boy shows asymmetric genu varum involving the left lower extremity. B: Observe the typical radiographic features of focal fibrocartilaginous dysplasia of the proximal tibia. There is a cortical defect involving the medial aspect of the proximal tibial metaphysis. There is associated sclerosis, as well as the mild tibia vara deformity. C: The lateral radiograph.*

Surgical plan

Although data are limited, it appears that only children who have not achieved spontaneous correction by age 4 should undergo corrective osteotomy. This usually involves a proximal tibial osteotomy, distal tibial apical osteotomy, and diaphyseal fibula osteotomy. Because of associated medial tibial torsion, the surgical procedure of choice is an external rotational osteotomy or oblique proximal tibial osteotomy as described by Rab [59].

Surgical technique

The procedures for treating localized fibrochondrodysplasia are similar to those for treating tibial malalignment. There is no intrinsic bone pathology that would affect healing. Immobilization with a long leg cast or a semispica cast is required, depending on the age of the child. A spica cast is generally recommended for young children. After healing, children usually recover quickly and return to normal activities. Long-term follow-up is needed to assess healing and subsequent growth of the tibial head. Regular CT scans are needed to assess limb length.

Complications

Brandish., *et al.* [68] reported a case of persistent peroneal nerve palsy and valgus deformity in a teenager following orthopedic osteotomy. This appeared to be a technical problem. No other complications were reported during surgery for this lesion.

Other pathological genu varum deformities

Vitamin D-resistant rickets and nutritional rickets

Persistent or progressive valgus deformity is common in children with metabolic disorders such as vitamin D-resistant rickets (hypophosphatemic rickets) or nutritional rickets. Vitamin D-resistant rickets is an X-linked dominant disorder of vitamin D resistance that results in defective bone mineralization. Affected children typically have bilateral symmetrical valgus knees; they are relatively short, often in the tenth percentile. Valgus deformity is due to a combination of valgus and affects the distal femur and proximal tibia. Hematologic tests show normal serum calcium and decreased phosphate values. In children with nutritional rickets, the parents have been feeding an abnormal diet (Figure 30).

On radiographs, features include widening of the epiphyses, widening of the epiphyses, and a cup-shaped relationship between the epiphyses and the metaphyses. The curvature is usually symmetrical throughout the femur and tibia. Marked osteopenia and cortical thinning are also common. Serum calcium, phosphorus, and alkaline phosphatase levels, as well as pediatric endocrinology consultation, are necessary to confirm the diagnosis. Medical treatment is important before considering any form of orthopedic intervention [69]. This typically includes oral phosphate supplementation and high-dose vitamin D for children with vitamin D-resistant rickets, and dietary modification for children with nutritional rickets (Figure 31). Surgical correction of lateral knee deformity is usually unsuccessful unless adequate medical control has been achieved prior to surgery. If control is not possible, it is best to wait until the bones are fully grown before attempting to realign the mechanical axes. If metabolic control is possible and the child is young, follow-up is recommended. Children under 5 years of age may improve spontaneously. However, in older children or those who do not improve spontaneously, surgery may be required [69].

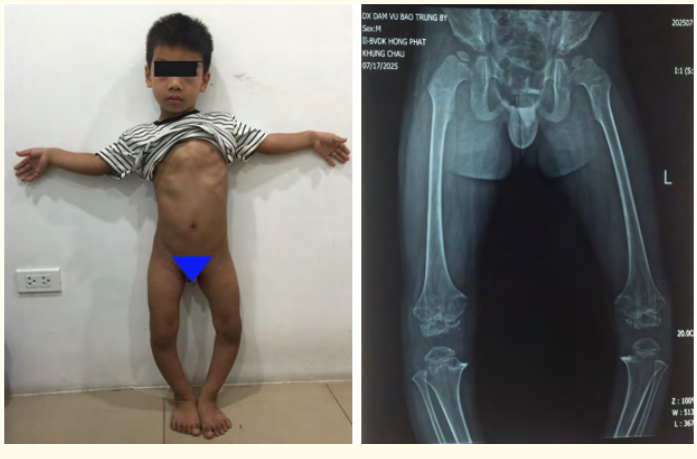


Figure 31: *7-year-old boys have Genu varum with Blood Vitamin D in level is 18 ng/ml (Normal 30-100).*

Surgery may include osteotomy of the distal femur, proximal tibia, or both. If the lesion is extensive, osteotomy of the proximal femur and distal tibia may be required to reposition the lower limb. Postoperative cast immobilization may result in hypercalcemia due to immobilization and may require adjustment of the treatment regimen. Healing time after osteotomy may be twice as long as normal. Delaying major orthopedic surgery until adolescence is often beneficial to minimize the risk of recurrence that is common in young children.

Blount disease

Blount disease, also known as tibiotarsalgia, is an acquired knee deformity of childhood that develops as a result of excessive compressive forces on the proximal tibiotarsal joint, resulting in changes in osteochondral formation. Blount disease can be unilateral or bilateral and presents in two forms-infantile and infantile-which are distinguished by differences in age of onset and clinical presentation (Figure 32).

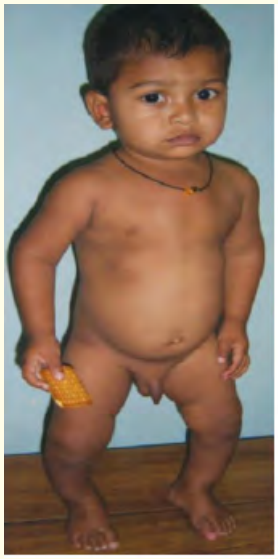


Figure 32: *A child with bilateral bowing of leg.*

Although obesity, delayed walking, and African American heritage are recognized as risk factors for developing Blount disease, the exact pathogenesis of the condition remains unclear. Treatment of Blount disease varies from splinting to surgical intervention and depends on the age and severity of the disease at presentation. Treatment options include knee-ankle-foot arthroplasty, proximal tibial osteotomy with acute or gradual fixation, and hemihumeral fixation. This activity reviews the etiology, pathogenesis, evaluation, and management of Blount disease. It also equips health care professionals with the knowledge and tools necessary to improve the quality of care and outcomes for patients in this unique condition.

Objectives:

- Differentiate Blount disease from other orthopedic conditions with similar presentations to ensure accurate diagnosis.
- Implement appropriate treatment strategies for effective management of Blount disease.
- Apply knowledge of the biomechanical and genetic factors that contribute to Blount disease in clinical practice.
- Collaborate with other health care professionals to provide comprehensive care for patients with Blount disease.

Introduction

Blount disease, also known as tarsal patellar luxation, is an acquired knee deformity of childhood caused by disruption of normal cartilage development in the proximal tibiotarsal joint. The condition develops due to excessive compressive forces on the medial aspect of the proximal tibiotarsal joint, resulting in altered osteochondral formation. Blount disease can be unilateral or bilateral and presents in two forms - infantile and juvenile - distinguished by differences in age of onset and presentation. The infantile or early-onset form is usually bilateral, typically presenting in children between 1 and 5 years of age and tends to worsen after the onset of walking. The juvenile form appears later and may be unilateral or bilateral.

Although obesity, early walking, and African American heritage are recognized as risk factors for developing Blount disease, the exact pathogenesis of the disease remains unclear. Severity varies, ranging from articular cartilage abnormalities to limb length discrepancy [70]. Treatment of Blount disease ranges from splinting to surgical intervention, depending on the age and severity of the disease at presentation. Treatment options include knee-ankle-foot orthosis (KAFO), proximal tibial osteotomy with acute or gradual fixation, and hemitibial fixation.

Radiographic findings are diagnostic, and the Langenskiöld classification system (See figure Langenskiöld Classification System) describes six radiographic stages of Blount disease. Blount disease was first described by Walter Putnam Blount, a pediatric orthopedic surgeon, in 1937.

Causes

Blount disease has a multifactorial etiology, involving both biologic and mechanical factors. Although mechanical overload of the proximal tibia is a significant contributing factor, especially in overweight children who begin walking early, it is not the sole cause of the condition. The infantile form, which also affects normal-weight children, and the higher incidence in African-American patients, suggest a potential genetic component. In addition to mechanical overload, genetic factors have also been implicated in the development of Blount disease.

Epidemiology

The prevalence of Blount disease in the United States is <1%. The infantile form of Blount disease is more common in males than in females. In 80% of cases, infantile Blount disease is bilateral. In contrast, childhood Blount disease, also known as infantile valgus, is usually less severe and more likely to be unilateral. It is most common in children of African and Scandinavian descent.

Pathophysiology

Blount disease results from excessive compressive forces leading to cartilage damage and subsequent delayed ossification. Mechanical overload results in changes in osteochondral morphology. When compressive forces peak at the posteromedial aspect of the epiphyseal cartilage, the patient experiences severe growth inhibition on the medial aspect of the knee, leading to the development of a persistent valgus deformity [71].

History and physical

Valgus is usually considered normal in children under 2 years of age, then progresses to valgus, peaking around 3 years of age. In young and overweight children, persistent valgus is often the initial diagnostic sign [72]. As growth retardation progresses, knee deformities and associated abnormalities gradually worsen, resulting in three-dimensional deformities including varus, arch, internal rotation of the tibia, and limb length discrepancy.

Blount disease in infancy

Blount disease in infancy is usually diagnosed in children between 1 and 3 years of age. The condition occurs bilaterally and is characterized by varus deformity of the tibia and internal torsion of the tibia. Pain is rare, and a palpable "flap" may be evident on the medial aspect of the proximal tibial condyle (Figure 33).

The finding of lateral thrust, which shows the knee deflecting to one side when weight is applied, is an important clinical observation. Irreversible asymmetrical proximal tibial stiffness typically occurs between the ages of 6 and 8 years, rendering conservative treatment ineffective.

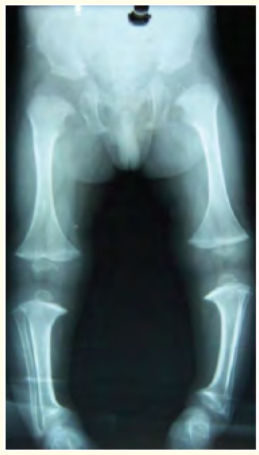


Figure 33: *Radiograph of the lower limb (AP view) showing abrupt medial angulation "beaking" of the medial cortical wall of the proximal tibial metaphysic abnormal stress placed on the posteromedial proximal tibial epiphysis that leads to growth suppression. In about 60% of cases this condition affects both legs. Erlacher reported the first case of tibia vara in 1922 [73].*

Adolescent Blount disease

Juvenile Blount disease usually presents in late childhood or early adolescence and is often accompanied by pain on the medial aspect of the knee. The condition is often associated with overweight or obesity. The disease is usually unilateral and may be associated with abnormalities of the distal femur.

Assessment

History, physical examination, and plain radiographs are sufficient to diagnose Blount disease. In the early stages, clinicians use anteroposterior long-leg radiographs to screen for and measure varus. For accurate measurement, bilateral radiographs from the hip to the ankle are required.

Signs of Blount disease

Signs suggestive of Blount disease include medial epicondyles, wide and uneven medial epiphyses, uneven ossification, and a slope between the epiphysis and metaphysis in varus (Figure 34).

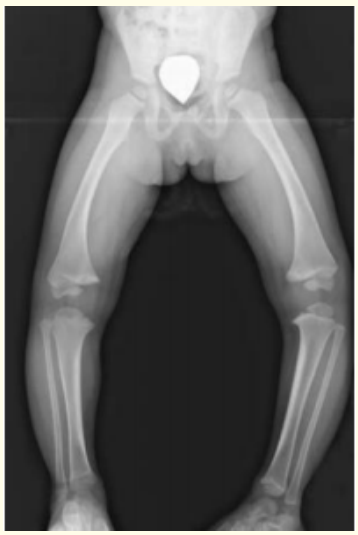


Figure 34: *Standing AP radiograph of the lower extremities demonstrating the typical appearance of early infantile Blount disease, including proximal tibial varus deformity, epiphyseal and physeal irregularity, and medial metaphyseal "beaking".*

Angles for Blount disease detection

Healthcare professionals refer to specific angle measurements to detect Blount disease in children. Various angles, such as the Levine-Drennan angle, are used to assess the relationship between the tibial shaft and its upper growth plate. An angle greater than 11° is often indicative of Blount disease. The angles required to detect Blount disease are shown below.

Average tibial-diaphyseal angle: The average tibial-diaphyseal angle (MDA) can predict the progression of Blount disease.

- A line is drawn from the most distal points on the medial and lateral tibial processes to a line perpendicular to the long axis of the tibial shaft [74].
- Disease progression can be predicted as follows:
- An angle >16° is associated with a 95% risk of deformity progression [74].
- Angles <10° are likely to be physiologic, with a 95% chance of spontaneous recovery [74]. Angles between 11° and 16° should be closely monitored to detect potential progression of tibial varus [74].
- Angular abnormalities include medial and lateral tibial axis deviation, tibial internal rotation, anterior tilt, distal tibial valgus, medial and lateral tibial laxity, and distal femoral deformity [74].

Tibiofemoral angle: The tibial femoral angle measures the severity of the varus deformity.

Mitigated tibial angle: The medial tibial angle (MMBA) is a potential diagnostic screening tool for individuals at risk for Blount disease. When combined with MDA, the MMBA may confirm the diagnosis, leading to earlier diagnosis and improved patient outcomes.

Magnetic resonance imaging (MRI) effectively evaluates cartilage, menisci, ligaments, and osteochondral blood vessels. Additionally, MRI is superior to X-ray in detecting cartilage changes [75]. Therefore, gadolinium-enhanced MRI appears to be beneficial in children with late or missed Blount disease, detected after age 4 years but before the development of achondroplasia on X-ray.

Langenskiöld classification system

Langenskiöld classifies Blount disease into six stages. These stages represent increasing severity and collapse of the medial epiphyseal cartilage, and this classification is particularly applicable to the infantile form of the disease. Epiphyseal bars appear from stage V onwards due to trauma or infection due to disruption of normal cartilage in the growing epiphyseal cartilage. Healing involves bone rather than cartilage. Epiphyseal bars cause angular deformities and limb length discrepancies in skeletally immature children. Although MRI-based classifications, such as the Fort-de-France (FDF), have become more popular recently, the X-ray classification remains the most widely used [76].

Stages of ligament laxity: There are four recognized stages of ligament laxity as follows:

- Stage 0: Normal laxity.
- Stage +: Internal laxity.
- Stage ++: External laxity.
- Stage +++: Multidirectional laxity.

Langenskiöld classification system: The six stages of the Langenskiöld Classification System are as follows (Figure 35) [76,77]:

- Stage I: Abnormal metaphysis.
- Stage II: Curved medial metaphysis.
- Stage III: Development of "ladder" at the metaphyseal beak.
- Stage IV: Osteophytes protrude and occupy a fossa in the medial metaphysis.
- Stage V: Double epiphyseal plate.
- Stage VI: Osteophyte formation.

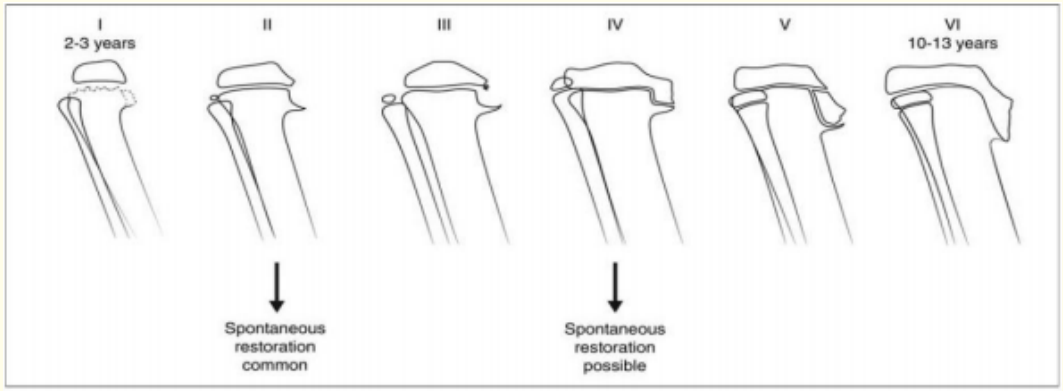


Figure 35: *Illustration of Langenskiöld's radiographic classification of infantile Blount disease demonstrating six progressive stages from mild medial epiphyseal-metaphyseal beaking (stage I) to complete medial proximal tibial physeal arrest (stage VI). Spontaneous correction occurs commonly in stage II lesions and occasionally in stage IV lesions.*

Treatment/management

Treatment of Blount disease depends on the child's age and the severity of the deformity. The discrepancy between chronological age and bone age decreases as chronological age increases. Because of the potential for growth restriction in some patients, preoperative assessment of bone age is important because it can influence the timing and extent of correction. When indicated, surgical intervention aims to restore joint and limb alignment, achieve equal limb length at skeletal maturity, and prevent relapse.

Brace

The KAFO brace is a potential treatment option for children diagnosed before age 4 years and with Langenskiöld stage I or II disease. The brace extends from the upper thigh to the foot and applies valgus pressure to the knee (Figure 36). Orthopedic treatment is most likely to be successful when initiated before age 3 years in non-obese children who wear the brace primarily at night. Treatment lasts for 1 year. In cases where orthopedic treatment is ineffective, osteotomy should be considered before age 4 when indicated. However, the use of braces in hyperactive children aged 3 years or younger poses a significant challenge and limitation to this treatment [78]. Notably, 80% of patients with early-onset, progressive Blount disease who undergo orthopedic surgery before age 4 years recover completely. Weight-bearing deviation is a hallmark of mechanical weakness of the knee.

Figure 36: *Photograph demonstrating a lower extremity brace used in nonsurgical management of infantile Blount disease (particularly in patients with unilateral deformity, presenting at age ≤3 years, Langenskiöld stage III or less). Day, night, and day and-night bracing protocols have been advocated.*

Guided growth

Hemiphysiodesis, also known as guided growth, is a surgical technique designed to gradually correct angular deformities in skeletally immature patients. As an alternative to orthopedic osteotomy, hemiphysiodesis is cost-effective, reduces patient pain, shortens the time required for immobilization, and minimizes surgical risks.

The most common procedure is hemiphysiodesis of the lateral epiphysis using extraperiosteal implants such as staples, pins, or wires. Bone growth on the side of the growth plate where the pin or fixation material is placed is stopped or slowed, allowing the other side of

the growth plate to grow normally and gradually straighten the bone over time. The advantage of this approach is that the entire growth plate can continue to grow after the fixation material is removed.

The child must be growing for at least 4 years for this procedure to be successful. The Hueter-Volkmann principle, which states that compressive forces inhibit longitudinal growth, is the basis of this treatment.

In contrast to varus associated with other disorders, the outcome of guided growth in Blount disease is less predictable, possibly due to the involvement of the proximal epiphyseal cartilage (Figure 37). According to Schroerlucke, patients with Blount disease are at risk of tibial screw fracture after surgery to treat hemiepiphysiodesis (narrowing of the epiphysis) [79]. For patients with moderate to severe Blount disease, parallel plates or solid stainless steel screws without cannulae are recommended [80]. Hemiepiphysiodesis and guided growth systems are currently viable options for those with late-onset Blount disease who present with a varus deformity < 15°, limb shortening < 1 cm, and at least 2 years of bone growth. Surgical intervention is not considered appropriate for children 2 years of age and younger [81].

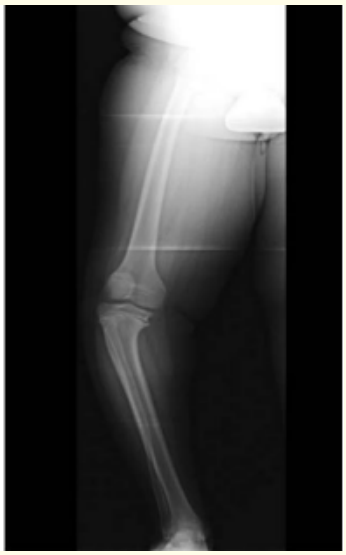


Figure 37: *Standing AP radiograph of the right leg in a patient with adolescent Blount disease. Note the clinical obesity, varus deformities of the distal femur and proximal tibia, medial proximal tibial physeal widening, and distal tibial valgus deformity.*

Osteotomy

Revision osteotomy is typically performed before age 4 years in children with documented and advanced Blount disease or stage I FDF with risk factors [82]. Because of the high recurrence rate in infantile Blount disease, osteotomy is expected to be performed excessively to achieve valgus deviation of 5º to 15°. The goals of osteotomy include lateral translation, external rotation of 10° to 15°, and valgus correction of 5° to 10°.

Various osteotomy techniques have been described for Blount disease (Figure 38), including open and closed wedge osteotomies, open wedge osteotomies, serrated osteotomies, vault osteotomies, and oblique osteotomies. Correction may be performed acutely or gradually with external fixation. Gradual correction results in more precise correction of the mechanical axis and leg length discrepancy.

A systematic review comparing acute and gradual repair for Blount disease found weak evidence in favor of gradual repair, with acute repair resulting in a higher rate of transient common peroneal nerve palsy. There was no difference in reoperation rates between the two procedures [83]. With acute repair, there is a risk of peroneal nerve injury and compartment syndrome regardless of the type of osteotomy and fixation method [84].

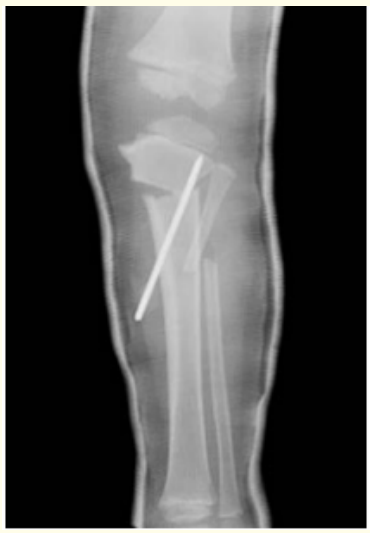


Figure 38: *Postoperative AP radiograph of the lower extremity demonstrating varus deformity overcorrection, with lateral translation of the distal fragment to restore axial alignment following standard high tibial and fibular osteotomy for infantile Blount disease.*

Acute correction

During acute correction, distal varus is stabilized during translation and external rotation to correct the internal rotation deformity. Other necessary surgical procedures, such as greater trochanterectomy and midplane elevation, are performed concurrently with the osteotomy. If the greater trochanter is more than 50% greater trochanteric, a partial epiphyseal osteotomy is appropriate. The osteotomy site should be placed below the tibial tuberosity to prevent patellar depression, which can lead to extensor weakness and knee pain.

Children 3 years of age and older, regardless of stage, or patients with Blount stage III, regardless of age, are considered candidates for osteotomy. Accurate measurement of limb alignment after acute or gradual correction can be challenging. To visualize the alignment of the mechanical axis, some investigators use intraoperative fluoroscopy with an electrocautery filament placed on the skin, over the midline of the hip and ankle [85].

The advantage of acute correction is the ability to immediately correct the deformity. However, this approach increases the risk of compartment syndrome and peroneal nerve injury due to acute extension.

Gradual correction

An osteotomy is performed during gradual correction, and a frame is inserted to allow progressive correction. Commonly used devices include the Taylor Space Frame or the Ilizarov External Fixator. Postoperative treatment typically lasts 12 to 18 weeks. Gradual correction

minimizes the risk of neurovascular injury and compartment syndrome, and allows for correction of the deformity in all planes. However, a potential drawback is pin site infection, due to the length of treatment required (Figure 39).

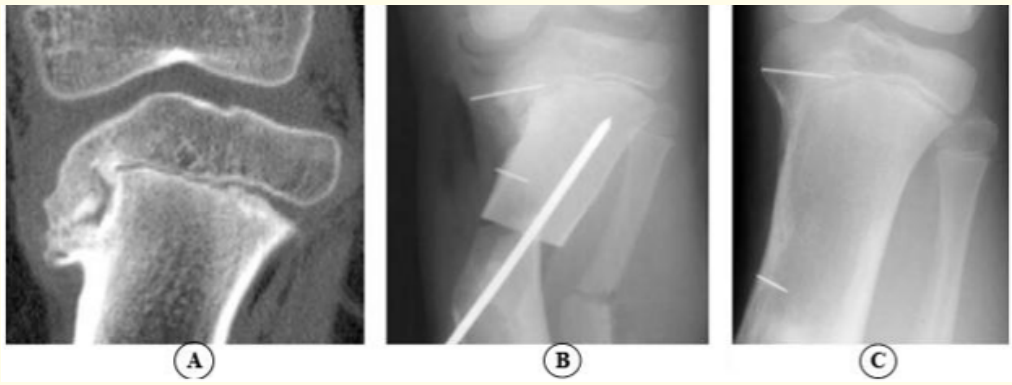


Figure 39A-39C: *A: Preoperative CT scan of the medial proximal tibia demonstrating physeal arrest in a patient with Langenskiöld stage VI infantile Blount disease. B: Postoperative AP radiograph of the tibia after physeal arrest resection and high tibial and fibular osteotomy were performed. Note the metallic markers in the epiphysis and metaphysis, which aid in postoperative monitoring of local physeal growth. C: Postoperative AP radiograph of the tibia demonstrating 30 mm of growth in the same patient at 2 years after surgery.*

Asymmetrical physeal distraction

Asymmetric reduction of the epiphysis is a less commonly used procedure. This procedure involves the insertion of two 6 mm semi-pins into the proximal tibia and two pins into the diaphysis. Once the pins are inserted, reduction is performed at a rate of 1.5 mm/day. A unilateral fixator typically achieves an average angular correction of 13°. Limitations to the popularity of this procedure may be due to the risk of septic arthritis, discomfort during reduction, and the possibility of premature closure of the growth plate [86].

Epistaplasty

Unlike patients with traumatic epiphyseal fractures, patients with Blount disease typically do not have a distinct bony attachment suitable for surgical resection. Epiphysis osteotomy aims to restore normal growth and prevent further deformity. Children who undergo epiphyseal osteotomy before age 7 years, combined with lateral osteotomy at that time, have shown improved outcomes. However, children 7 years of age and older are not considered good candidates for this surgery, and menisci alone are rarely used for these patients [87,88].

Medial tibial plane augmentation

Blount disease may progress to the point where the tibia may deviate laterally, and the medial femoral condyle may enter the posteromedial concavity, resulting in a propulsive gait. Children 6 years of age and older with severe Blount disease, Langenskiöld stage V or VI, and significant posterior concavity in the medial tibial plane are considered good candidates for posteromedial tibial plane augmentation. Surgeons recommend performing an endochondral or transchondral osteotomy, relying on the articular cartilage of the intercondylar notch to preserve the medial tibial plane. At the same time, the focus should be on correcting the posterior concavity of the medial tibial plane by grafting a larger portion of the graft. To prevent recurrence of varus gait, healthcare professionals should perform simultaneous surgical fixation of the proximal tibial and fibular heads [89,90].

Differential diagnosis

Distinguishing infantile varus from physiologic varus of the foot can be difficult. Physiologic varus is characterized by gradual growth of both the tibia and femur, whereas Blount disease varus is acute. MDA >11° suggests Blount disease in affected patients.

Other potential differential diagnoses include rickets, Ollier disease, proximal tibial head damage from trauma, radiation therapy, or infection, osteomyelitis, epiphyseal dysplasia, and radial thrombocytopenia syndrome. Common features of Blount disease include asymmetrical bone bases and acute angular deformity, which are often absent in rickets. The presence of multiple chondromas helps distinguish Ollier disease [91,92].

Prognosis

The prognosis of Blount disease depends on the age and severity of the disease at presentation. Blount disease in infants usually has a favorable prognosis, with a low rate of recurrence of malformations if treated early. Patients in the early stages may recover partially or completely. However, patients in the later stages may progress more severely if not treated early. Patients in the later stages may progress significantly and develop malformations if left untreated [93].

Complications

Pathological complications

Pathologic complications can lead to recurrent deformities and osteoarthritis.

Surgical complications

Surgical complications may include deep vein thrombosis, vascular insufficiency, pathologic fractures, wound infections, dislocations, compartment syndrome, premature closure of growth plates, reactive hypertrophy, and hardware migration.

Improving health care team outcomes

Establishing a comprehensive strategy for the management of Blount disease is critical, including early diagnosis, timely treatment, and seamless coordination among members of the health care team. Health care professionals must collaborate to identify significant risk factors such as obesity and recognize imaging features such as limb asymmetry, which help differentiate Blount disease from metabolic disorders such as rickets. Primary care physicians, physical and occupational therapists, and pediatric orthopedists should collaborate with radiologists to ensure rapid and accurate imaging.

Interdisciplinary communication is essential for early diagnosis and effective care. Nurses and support staff play an important role in facilitating this communication and ensuring that patients receive timely and appropriate care. Collaboration with orthopedists is essential to provide the most effective orthopedic treatment when indicated. Coordinated care is important to prevent the severe deformities, leg length discrepancies, pain, and arthritis associated with Blount disease. By adhering to these principles and promoting effective teamwork, interdisciplinary health care providers can work together to improve outcomes for patients with Blount disease, emphasizing early diagnosis, appropriate treatment, and ultimately better patient-centered care [78].

Conclusion

Lower limb angular deformities are common in young children and often cause great concern for parents. Typically, these deformities are normal variations in growth and development and require no treatment other than observation and reassurance of the parents.

Despite the benign nature of physiologic and extreme valgus, most pathologic causes require appropriate management by an orthopedic surgeon; therefore, careful evaluation of the patient and identification of these pathologic causes is important.

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