Effect of Forces on the Growth, Development, and Maintenance of the Human Body

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The purpose of this paper is to present an overview of growth principles that are influenced by mechanical factors. These general principles are followed by some biomechanical examples of the growth and development of weight-bearing body areas, and examples of growth principles and mechanics related to therapeutic applications.

Key Words: Biomechanics, Child development disorders, Extremities, Fetus, Spinal cord.

The growth, development, and maintenance of the body is affected by several factors including genetics, nutrition, drugs, hormones, and mechanical forces. Each may work separately or in combination to cause normal or abnormal development. Although all of these factors can make important contributions to the formation of the body’s components, we will address the mechanical aspects in this article.

Forces are constantly acting on the body. These forces can affect the size and shape of the body parts, especially those of the musculoskeletal system. Cultural and occupational practices provide several examples of the effects of forces on the body. Several cultures have used cradle boards, which flatten the children’s heads. Others have restricted the size of women’s feet by binding them. The beauty secret of the Padaung tribeswoman is based on the pull of gravity on brass rings around her neck; the gravitational pull gradually pushes the clavicles and ribs downward. Other examples of children’s problems that may occur from their having been in a poor position in the uterus, during sleeping, or while sitting.

Many physicians have become “extraordinarily impressed” with the importance of mechanical influences on the human body as they have noted that many aspects of growth, development, and maintenance are mechanical. Several investigators have traced the effects of forces on the various skeletal structures after a child is born. Others have studied forces related to problems in adults, and others have emphasized the forces that cause deformity in the fetus. Only recently, however, have authors reported on mechanical aspects involved in the development of the embryo.

The following sections of this article will present some general growth principles that are mechanically oriented, examples of mechanics influencing growth and development of selected body areas, and examples of growth principles and mechanics in therapeutic applications. We have termed the study of the effects of forces on the musculoskeletal system during the entire life span developmental biomechanics. By understanding these principles and recognizing these and other examples, physical therapists will be able to prevent and treat musculoskeletal disorders more effectively.

GENERAL GROWTH PRINCIPLES

The general shape of the human body depends on the proper development of skeletal structures. A deficiency in this development leads to gross abnormalities in appearance and function. Most authorities believe that extrinsic factors can greatly modify both the external and internal design of bones. Wolff’s law of bone transformation addressed this phenomenon in the 1800s. The general idea of this law is that every change in form or function of a bone brings about definite changes in the bone’s internal and external architecture in accordance with certain mathematical laws. This law includes the principle that bones will increase or decrease in their mass according to their function. Some authors have made this law more specific in relation to endochondral growth, appositional growth, and external and internal bone form. By understanding the specific reactions of tissues and growth processes to loads and by applying principles of basic biomechanics, clinicians can help direct the normal growth and development of the musculoskeletal system. They can also use this knowledge to prevent deformities or to correct the deformities once they have occurred.

EFFECT OF LOADING ON TISSUE TYPE

The type and duration of loading can influence the type of tissue or articulations being formed. During the process of tissue differentiation, forces are important in determining the type of tissue formation. In many instances, the type of tissue depends on the type of loading that occurs. Almost all tissues are sensitive to the loads that are placed on them. They respond to tension, compression, shearing, torsion, and bending in a manner that contributes to their progressive differentiation.
Generally, chondrogenesis occurs with intermittent loading, and osteogenesis occurs with continuous loading. Storey presented a spectrum of loads from continuous compression through intermittent loading to continuous tension that provided for functional adaptations of connective tissue and joints. With continuous compression in a constant direction, bones become connected by bars of cartilage that act as shock absorbers and as a flexing system. A synchondrosis is an example. With intermittent compression and a range of movement between bones, articular cartilage is formed. A pathological example is cartilage forming in a pseudarthrosis. With a decreasing magnitude of intermittent compression and an increasing amount of tension, symphyses are formed. With intermittent loads of tension and compression of equal magnitudes along with sliding, condylar cartilage develops. With increasing intermittent tension, sutures are formed such as joints in the skull. With continuous tension in one direction, thick collagenous tissue develops into tendons and fascia.

**EFFECTS OF LOADING ON AMOUNT AND DIRECTION OF TISSUE GROWTH**

The magnitude and direction of loading can influence the amount and direction of tissue growth as well as affecting the type of tissues and joints being formed. The following principles show how loads on the musculoskeletal system can exert this influence on tissue growth.

**Effects of Loading on Endochondral Growth of Bones**

Once endochondral growth begins, functional loads appear to have considerable influence on the final form of the bone. The epiphyseal plate may react in four ways in response to loads: 1) growth can increase longitudinally; 2) growth can decrease longitudinally; 3) growth can be deflected by shearing; or 4) torsional growth can occur from continual or intermittent twisting.

**Perpendicular loading.** A load applied to an epiphyseal plate that is not parallel to the direction of growth (perpendicular to the epiphyseal plate) will deflect the growth along the line of the deforming force. As long as the load is maintained, new growth will be deflected in the direction of the load resulting in a lateral displacement of the epiphysis. The chondral growth gradually yields to the shearing component of the load, which effectively realigns the direction of growth. Depending on the magnitude and direction of the load, tilting of the epiphyseal plate may or may not occur.

**Torsional loading.** Because the epiphyseal plate is least resistant to torsion, a torsional load will lead to a rotational deflection of the growth columns around the circumference of the epiphyseal plate. Newly formed bone will grow away from the epiphysis in a spiral pattern, which gives a torsional change. Examples of this effect include normal alignment of tibial rotation, a torsional deformity of the vertebrae occurring in scoliosis, and rotational change resulting from the physicians' and therapists' correction of tibial torsional problems.

Gravity may be sufficient to affect epiphyseal growth. Muscle forces, however, are often much greater than those of gravity. Joint alignment tends to be more influenced by the load caused by muscular forces than by the pull of gravity. An imbalance in muscle forces or lack of muscle forces secondary to non-weight bearing may lead to skeletal deformation, especially if these abnormal forces are applied over a long period of time.

**Effect of Loading on Appositional Growth**

**Compression and tension.** Compression stimulates appositional growth, and lack of compression leads to a reduction of bone tissue. An increase or decrease in tension may produce results in fibrous tissues such as tendon and ligaments that are similar to those produced in bone. Increased weight bearing results in an increase in the thickness and density in the tibial shaft. Lack of weight bearing, however, resulting from factors such as bed rest, immobilization, neurological disorders, or space flight, is followed by bone atrophy. Unused bones are usually smaller in size and abnormal in shape.

**Bending.** When a bone is bent under a load, it modifies its structure by remodeling. The process, however, is relative to the normal shape of bone. When the surface of a bone becomes less concave or more convex, net loss of bone occurs as a result of osteoclastic activity. The more concave or less convex surface has a net increase of bone as...
a result of osteoblastic activity. Frost termed this process \textit{flexure-drift}. The process appears to be related to electrical potentials induced by a piezoelectric effect when bone is deformed. Intermittent current seems to be better for proper bone formation than a continuous current. Hence, intermittent loading of a bone produces better development.

**Effects of Loading on Trabecular Bone**

The internal structure of bones is developed to resist the loads they bear. A change in the magnitude or direction of the loads that produce stress within the bone produces a demonstrable change in the internal architecture of the bone. The trabeculae are organized to provide maximum strength with a minimum of material. Compression appears to be the major load to develop trabeculae; however, tension loads can also produce trabeculae. Bony trabecular hypertrophy in response to increased load, but new trabeculae cannot appear without a preexisting framework to build on. The trabecular system is related to forces of both weight bearing and muscle pull. Without these loads, the trabeculae become thinner and may even disappear. This reduction of bone mass leads to mechanical incompetence. During about the first four or five decades of a normal individual's life, the internal replacement and adjustment of bone mass is maintained according to the mechanical demands made upon it. Because mechanical loads provide a continual stimulus, the remodeling process continues. As an individual ages, however, the process proceeds at a slower rate. The bone attempts to maintain maximum density in areas of maximum loading. Some authors, however, have stated that this adaptive remodeling loses its effectiveness and results in an increased bone resorption and decreased bone formation in most people by the age of 35 to 40 years. Bone loss can be about 25 to 30 percent over 20 years or between 0.5 to 1.5 percent each year.

**Effects of Loading on Cartilage**

Cartilage grows within a wide range of forces. Constant compression on cartilage leads to its thinning, but it may regain its thickness if the compression is slowly released. When intermittent compression is applied, it becomes thicker. Excessive compression causes degeneration of cartilage, and absence of compression is often followed by atrophy. Secondary to either excess or lack of compression, congenital deformities can cause early appearance of degenerative joint disease. The main mechanical factor causing cartilage degeneration appears to be repetitive impulse loading, especially from muscle joint forces. Radin stated that repetitive impulse loading for as little as 20 minutes each day over a period of several months can aggravate the cartilage degeneration. A situation, however, that produces chronically increased stress on the joint can either be the primary cause of degeneration or provide secondary changes in the joint. Joint degeneration can follow trauma, joint dysplasias, subluxations, dislocations, slipped epiphyses, or segmental torsions. Once the initial lesion occurs, physical activity is necessary for the disorder to progress.

**Effects of Loading on Fibrous Tissue**

Soft tissues such as tendons and ligaments adapt in tension. They are self-aligning and their shape does not need to be adapted. Their tissue properties, however, are affected by increased or decreased loading. Intermittent tension causes collagen tissue to increase in thickness and strength. This reaction is evident in the ligaments of exercised animals. Frost termed this response the \textit{stretch-hypertrophy} rule. Lack of loading on ligaments after immobilization, however, decreases their strength and ability to absorb energy.

Frost also expounded on the \textit{stretch-creep} rule. This rule relates to the creep (elongation over time) that occurs within the tissue as a tension load is applied. Many treatment procedures use this concept. The use of casting or night splints to lengthen tendons in the foot is one example. The use of creep can be a valuable treatment technique. This process, however, takes a prolonged period of time. Kite stated that casting, using the principle of creep, may be preferred to surgery. He also warned that forced manipulation of the body part may cause tearing and resulting adhesions in the area. Therefore, knowledge of the advantages and disadvantages of each treatment technique is essential.

**Effects of Loading Related to Rate of Growth**

The effect of load on growth is directly proportional to the speed of growth. Any load applied even for a short time during the period of rapid growth may result in permanent deformity of a bone. Early fetal growth is extremely rapid and reaches a peak around the fifth month. Later in the fetal period, the fetus is exposed to increased extrinsic forces such as increased size, decreased amniotic fluid, and decreased movement. At this time, however, with a slowing growth rate and diminished plasticity, the fetus becomes increasingly able to resist deformation. In general, the modification of tissues is achieved more easily when they are pliable in periods of rapid growth. Rapid growth in small bones provides for production or correction of angular deformities very rapidly. Early correction yields the best corrective results.

**General Comments**

Without the growth process, correction of a deformity is almost impossible. Incorrect application of loads for correction during this time, however, may lead to other deformities. With appropriate management of the deforming forces, the adaptive changes may be reversed or completely avoided. The problems of deformities and their sequelae can be greatly reduced if the clinician uses knowledge of biomechanical and growth principles. The remainder of this article provides examples of growth, development, and maintenance for weight-bearing areas of the body.

**RESULTS OF GROWTH PRINCIPLES IN WEIGHT-BEARING AREAS**

**EXAMPLES OF WEIGHT-BEARING AREAS**

**Spine**

The spine is composed of several structures that provide both static and dynamic stability. The 24 vertebrae consist of an anterior body that is the primary weight-bearing area and a posterior arch formed by the pedicles, laminae, transverse processes, spinous process, and articular facets. The size and mass of each vertebra increases from the cervical to lumbar area in adaptation to
the increasing load from the superincumbent weight. The sacrum is the solid bony base of the spinal column. The top of these five fused vertebrae forms a 45-degree angle with the horizontal plane; this angle equalizes the compressive and shear forces between the last lumbar and first sacral vertebrae. An increase in this angle predisposes this area to accentuation of shearing forces, and a diminution creates elevated compression.59

Spinal longitudinal growth occurs primarily in the vertebral body. Length from cervical to lumbar areas is approximately 20 cm at birth and doubles in the first year. Growth continues at a slow linear rate until the final adult length of 60 to 75 cm. The proportionate size of each area of the spine also alters with growth. The cervical vertebrae become relatively smaller, and the thoracic vertebrae enlarge in percentage of total length. The lumbar area undergoes little alteration in relative proportions.60

Loads on the spine create both primary and secondary trabecular systems in the vertebrae. The primary system, a vertically oriented arrangement through the spinal column, develops to sustain body weight and is the most resistant to atrophy. The secondary systems orient in the oblique direction to counteract torsion, bending, and shear forces and in the horizontal direction to counteract tensile muscular pull. These secondary systems are the most susceptible to atrophic change seen in osteoporosis.61

Viewed from a sagittal perspective, the normal spine is lordotic in the cervical and lumbar areas and kyphotic in the thoracic and sacral areas. These curvatures result from predictable developmental changes. At birth, the spine has two primary posterior convex curves. As the infant lifts his head against gravity, the lordosis in the cervical area increases. Latent tightness of the iliopsoas muscles persisting from fetal flexion, coupled with antigavity work by the infant in a prone position on flexed or extended elbows, in creeping, or in high kneeling encourage increased lumbar lordosis. Weak abdominal muscles provide little anterior pelvic support for the lordotic tendency. Thus, the spine develops increased compensatory curvatures that allow close approximation to the line of gravity and provide inherent stability in all directions.38,59 Abnormal or unusual developmental influences can disrupt the normal symmetry and balance of the spine, predisposing it to deformity.

One of the more prevalent spinal deformities is scoliosis. Although various defects in embryological development, such as failure of vertebral segmentation or undergrowth of chondrification centers, can produce congenital scoliosis,60 the majority of scoliosis is caused after the embryonic period. The problems of muscular imbalance, abnormal muscular tone, persistent abnormal positioning, lack of or abnormal spinal weight bearing noted in the various paralytic or CNS disorders (eg, poliomyelitis, Werdnig-Hoffman disease, muscular dystrophy, myelodysplasia, and cerebral palsy) can create an alteration of spinal dynamics, which predisposes the spine to scoliotic changes.62 The etiology of idiopathic scoliosis, the most frequent nonneuromuscular variant, remains obscure. The possible causes are asymmetrical spinal sensitivity, increase in number of slow twitch fibers on one side of the spine, positional influences, or asymmetrical vertebral and cord growth. Any of these causes could create abnormal forces during spinal development.63,64

Forces of deformation initially affect the viscoelastic structures that are most susceptible to creep. Ligaments and muscles shorten and thicken on the concavity and stretch and eventually relax on the convexity. Cartilage degenerates secondary to heavy compression on the concave side and atrophies secondary to stress-reduction on the convex side. The disk, compressed on the concavity, bulges and demonstrates nuclear migration toward the convexity. Additionally, the disk becomes incapable of maintaining normal vertebral dynamics.62,65

If these changes occur while epiphyses are still unfused, bony deformation results. Compression on the concave side causes growth reduction, and minimally decreased loading on the convex side stimulates overgrowth, which creates vertebral wedging. Rotatory alterations, begun as a sequel of the lateral deformity, cause distortion of the vertebral elements and incongruity of the facet joints. Rib rotation and lumbopelvic dysynchrony often accompany the vertebral distortion.62 The deformation thus creates a self-perpetuating system. Creep with eventual relaxation in combination with the force of gravity causes exacerbation of the problem. The body attempts to remain balanced over the pelvis and frequently develops a compensatory curve.

Similar deformation in an anteroposterior direction can occur with kyphosis. Postural faults and deficiencies of vascular supply have been implicated in addition to the etiologies underlying scoliosis as causative factors of thoracic kyphosis. Lengthening and weakness of posterior viscoelastic elements with shortening of anterior elements results. Fibrous replacement of disk substance secondary to the abnormal pressure leads to loss of vertebral mobility. Excessive anterior pressure interferes with anterior vertebral ossification and creates anterior wedging. The line of gravity assists the deforming forces. The spine compensates by developing an accentuated cervical or lumbar lordosis.

A third spinal condition of relative importance is represented by spondylosis or spondylolisthesis. Both conditions have a defect in the laminar area, specifically the pars interarticularis, but no displacement has occurred in the static spondylosis. Forward slippage of the anterior portion of one vertebra on the vertebra below characterizes spondylolisthesis. The anatomy of the lower lumbar area predisposes this location to the highest frequency of slippage.66-70 Secondary to the normal lumbar lordosis, compressive force is transmitted through the neural arch instead of the vertebral body. Either a single traumatic or continual intermittent force in a hyperextended (hyperlordotic) position can pinch the L5 isthmus between the L4 articular facet and the upward projecting sacral process, fracturing the weakest point, the pars interarticularis. Hence, multiple factors could predispose the area to a hyperlordotic position. In addition to congenital birth defects, these factors include any abnormal or persistent lumbar lordosis secondary to muscular imbalance, abnormal position or muscular tone, or tightness of the iliopsoas or lumbosacral area. Additionally, abnormal or lack of weight bearing could produce reduced calcification in the lumbar area. Subsequent movement superimposed on these conditions of hyperlordosis or decalcification can contribute to fracture and slippage as a sequel of increased shear force.59
The adult hip joint is composed of an acetabulum that faces outward, forward, and downward, and the head of the femur that is inclined at approximately a 125-degree, neck-shaft angle and anteverted 8 to 11 degrees with the shaft. The head of the femur sits deeply in the acetabulum and is secured by the labrum and strong muscular and ligamentous attachments. This mature position is the result of major mechanical influences during development on both portions of the joint.

The pelvis is formed from three primary ossification centers: the ischium, pubis, and ilium. These centers converge to form the triradiate cartilage. Endochondral growth in these areas allows the acetabulum to enlarge circumferentially commensurate with spherical growth of the femoral head. The acetabulum, which is the most shallow at birth, provides a large range of motion but also presents the greatest potential for dislocation. The normal angle of the acetabular roof with the horizontal plane is 30 degrees at birth. This angle decreases to 20 degrees by 3 years of age and remains at this level through maturity.

Three growth zones—the longitudinal growth plate, the trochanteric growth plate, and the femoral neck isthmus—contribute to development of the proximal femur. These zones lie on the same line at birth. As the child ages, the longitudinal plate grows at a more rapid rate than the trochanteric plate and shapes the angles of inclination and declination. At birth, the angle of inclination is 150 degrees with approximately 40 degrees of anteversion. If normal compression and tension loads are placed on the proximal end of the femur, both angles decrease with age to adult values.

Unless the femoral head is malpositioned perinatally, the acetabulum and femoral head develop congruently. This congruency is essential for proper development of both portions. External loads guide the development of these areas. The most important are body weight and muscle tension, applied in appropriate magnitude and direction. Any abnormality of compressive load or incongruity of joint structure will lead to bony deformity. For example, a shallow acetabulum or coxa plana may be caused by these abnormal loads.

The trabecular structure of the proximal femur is a consequence of external forces. The two primary systems are the principle compressive lines that develop from a normal weight-bearing load. The secondary systems cross the neck region and greater trochanter, developing as a sequela of muscular tension. All the trabecular systems cross at right angles for greatest resistance to compression and bending stresses.

Abnormal loading or joint incongruity can predispose the hip joint to developmental abnormalities. A frequent deformity is congenital dislocation (CDH) or subluxation of the hip. The most probable causes are hereditary, mechanical, or multifactorial combinations. Joint laxity has been postulated as a genetic problem that can lead to hip deformity. Atypical body position in utero and the resultant mechanical loads may also create hip deformation. Restricted uterine space can trap the fetus in a cross-leg or breech position. The left hip, which is twice as frequently involved as the right, lies against the maternal lumbar spine and may be restrained for prolonged periods in an adducted position.

Forces at or briefly following the birth process may dislocate the flexible hip joint. Because of joint position and force, the breech delivery is often followed by hip dislocation. As many as 30 to 50 percent of all children with CDH were also breech presentations. At least 20 to 30 percent of the children who were breech deliveries have a diagnosed CDH. During birth or immediately after delivery, the hip may be dislocated by passive movement of the relatively flexible, unstable hip joint from fetal flexion into extension. The hip may relocate or remain displaced.

Cultural and environmental factors may lead to hip instability. Swaddling a child tightly in extension with a blanket or carrying a child on a cradleboard can force extension of the hip and cause dislocation. Persistent sleeping or sitting with the hip in the extremes of rotation may provide deforming forces that cause abnormal joint development.

The dislocated or subluxed hip in a preambulatory child may not appear as a developmental problem. If the condition is not corrected within a few months, however, secondary growth alterations and abnormal forces during gait can cause serious permanent sequelae. The magnitude and direction of loads applied to an atypically positioned hip joint will determine the abnormality of growth. A shallow or maldirected acetabulum, coxa vara or valga, or abnormal anteversion or retroversion could result. When the forces of gait are superimposed, early degenerative arthritis can follow.

The child with cerebral palsy commonly develops various types of hip deformity. Abnormal positioning during sleeping and sitting are contributory causes. Muscular imbalance and tone abnormalities, especially in the adductors, iliopsoas, and hamstring muscles, place tremendous potential deforming forces across the developing hip. Delayed weight bearing alters the calcification rate, trabecular development, and angular changes of the hip complex. Weight acceptance in abnormal positions can also create abnormal force development across the acetabulum and proximal femur. Hence, this population is subject to coxa valga, femoral anteversion, hip subluxation or dislocation, and hip dysplasia as adolescents or adults.

The tibial shaft undergoes torsional changes during growth. Torsion at birth is 0 degrees but increases to 23 to 25 degrees of external torsion by maturity. Internal tibial torsion in the adult is, thus, failure of normal tibial external rotation. Because the epiphyseal plate is less resistant to torsion, torsional deformities may readily occur if abnormal forces are present.

The upper end of the tibia undergoes three further developmental alterations in alignment. The pull of the hamstring muscles causes tibial retrotorsion, a natural posterior deflection of the proximal end of the tibia. Retroflexion, or the posterior bend of the shaft, is produced
by the contraction of the gastrocnemiussoleus muscle group. The plateau of the proximal tibia slants posteriorly about 5 degrees in the adult. This value, called tibial retroversion, is a reduction in angulation from 27 degrees in the newborn to 17 degrees at 3 years of age, 7 degrees at 10 years of age, and 5 degrees by 19 years of age.58

Trabecular systems in both distal femur and proximal tibia originate from all sides and align perpendicularly to the articular surface for load resistance. Tra­beculae also develop in areas of muscu­lar attachment in line with fiber direc­tion to resist tensile forces. The patella consists of multidirectional trabecular systems resistant primarily to tension.58

The presence of any abnormal forces during development can create many deformities at the knee. Genu recurva­tion is a frequent knee deformity noted congenitally or postnatally. Malposition in utero with the feet locked in the axilla or under the mandible, as well as a breech position, will cause the knee to develop in extension instead of flexion.17,29,77 Persistence of tibial retrover­sion, quadriceps femoris or hamstring muscle weakness and tightness, or ov­eractivity of the triceps surae muscle can also create a genu recurvatum.78 Genu recurvatum developed in utero can re­sult in contracture of the quadriceps mechanism and anterior capsule; a small, abnormal, or absent patella; a lengthened or absent anterior cruciate ligament; and anterior dislocation of hamstring tendons so that they act as knee extensors. The existence of post­natal genu recurvatum, especially in an ambulatory person, will cause weaken­ing of the posterior joint capsule and stretching and possibly tearing of the anterior cruciate and collateral liga­ments with resultant rotatory and anteroposterior instability.

Many possible etiologies of develop­mental origin have been implicated in genu varum and valgum. Obesity can increase loading and create more abnor­mal mediolateral forces, which alter nor­mal processes of growth. Epiphysial damage or avascularity and metabolic deficiencies can create more deformable bones that will alter under load. Familial relaxation or secondary stretching of collateral ligaments can exacerbate va­rus or valgus positioning. Muscular im­balance or abnormal tone, especially in the thigh, can provide abnormal forces on the developing knee.11

Genu varum can specifically be pro­moted by a persistent prone sleeping position with the hips and knees flexed and the tibiae internally rotated. Both double diapering and straddle carrying can force persistence of normal varus positioning.6 Early weight bearing before structures can tolerate the load has been implicated, as has the absence of normal tibial external rotation. Genu varum produces other lower extremity alterations, including compensatory pronation, talar adduction, and in­toeing. Early onset of osteoarthritis has been noted in some adults secondary to the abnormal loading forces.

The presence of genu valgum has been noted in those patients with exces­sive external tibial torsion or pronated feet. Both of these conditions create forces that exacerbate medial deviation. Prolonged prone positioning with limbs flexed and tibiae externally rotated, as well as "W sitting" with tibiae in exter­nal torsion, can predispose the knees to excessive valgus positioning.10,11 The se­quelea of genu valgum can be medi­al laxity and rotatory instability secondary to ligamentous creep. The iliobibial band will shorten laterally and maintain the deformity. An increased "Q angle" will predispose the patella to lateral tracking and potential chondromalacia. Com­pensatory rotational deformities of the foot may occur to keep the sole flat on the ground.

The most frequent knee disorder is patellofemoral chondromalacia. Several developmental problems have been as­sociated with this disorder. As it forms, the patella conforms to the condyles in utero because the knee develops in flexion. Any embryological defect in artic­ular cartilage might predispose the joint to patellofemoral incongruity. A con­genitally small patella will provide less area for dispersion of force. Any abnor­mality of knee development, either bony or positional, will create patellar mal­alignment and irregular trochlear track­ing.

Additionally, malposition or mala­ignment of any portion of the lower kinetic chain can place abnormal force on the patellofemoral articulation.78,79 Both overuse and underuse of the knee have been cited as potential sources of developmental abnormality. Continued heavy compression and increased patel­lofemoral forces in heavy exercise, knee walking, or ambulation in knee flexion will cause articular degeneration. Lack of functional stress in flail, immobile, or nonambulatory limbs can produce atro­phy of articular cartilage.22

Ankle-Foot

The ankle-foot complex undergoes some very complex developmental re­alignment in utero. The primitive foot is initially directed medially and cranially. By the eighth fetal week, the soles face each other like "praying feet." The feet are aligned with the leg as no ankle angulation has yet occurred, so they appear in talipes equinus. Changes in the talus and calcaneus in the seventh fetal month drastically alter foot position. The talus widens as the head begins to torque. The entire bone shifts laterally to align with the axis of the foot. These changes continue postnatally to counterbalance tibial external rotation. The calcaneus grows broader and longer pos­teriorly. The angle of the tuber calcanei with the tibia is reduced from 36 degrees in the third fetal month to 22 degrees at birth, 16 degrees in adolescence, and 3.5 degrees at maturity. Concurrently, lower extremity flexion increases so the feet are positioned in extreme dorsiflexion against the uterine wall and appear in calcaneus position at birth.

The postnatal foot is very elastic with no longitudinal arches. Muscles and lig­aments have not yet developed normal strength and elasticity. Initial stance demonstrates a wide base with external foot position for stability. The feet appear flat and pronated. Gradually, an arch develops as strength increases. By 2 years of age, the arch is apparent in weight bearing with a neutral heel posi­tion and minimal out-toeing of the foot.80,81

Growth of the foot is similar in both sexes until 12 years of age. Half of ma­ture length is achieved in the first year with a linear annual increase thereafter. The male foot grows slightly more rap­idly during adolescent years so total length is slightly greater.82

Trabecular development relates both to compression and soft tissue tension. The calcaneus has primarily a cortical structure with few trabeculae that relate to weight-bearing compression and tensile pull of the triceps surae muscle and longitudinal arch. The pattern in the
talus demonstrates an arch related to compression with vertical lines in the neck area. 83

Because of its distal location, flexibility, and late ossification, the foot is very susceptible to deformities. One of the most common deformities is metatarsus adductovarus. Several prenatal factors can contribute to this deformity. Malposition in utero with the feet wrapped around each other or the body can cause a varus angulation. Increased mechanical pressure from maternal abdominal muscle tone, large fetus, or small uterus and increased hydraulic pressure could make fetal foot repositioning difficult. A neuromuscular disorder causing muscular imbalance or paralysis could also hinder fetal positioning. Postnatal sleeping rotation could create varus deformation. 8,11,17 The functional deformations that might result from metatarsus adductus include widening of the first two toes, in-toeing or heel eversion, and foot supination. Talipes equinovarus is the most frequently diagnosed form of clubfoot. Malposition in utero or failure of lower limb rotation has been implicated along with increased pressure and muscular imbalance. Because of the severity of this deformity, a defect of germ plasma before the seventh fetal week has also been postulated as a causative factor. The major problem is talipes equinus or calcaneus with these bones overlaying each other. The navicular is medially displaced on the talus and the metatarsal shafts adduct. These deformities cause shortening of musculature, ligaments, and skin medially and posteriorly. The remainder of the bones rotate medially and mold into abnormal configurations with deviant articular surfaces. Compressed vascularity causes further delay of appearance of ossification centers. 17,77 Pes planus, diagnosable only at 16 to 28 months of age, can result from primary or secondary developmental abnormality. Shortening of the first metatarsal, or metatarsus primus varus, can cause a primary pes planus, as can muscular imbalance and hypermobility syndromes. Pes planus also occurs as an adaptive sequela of genu valgum or torsional deformities. The flattened, pronated position creates a flexible forefoot that can lead to ligamentous creep. A decreased longitudinal bony arch can result from abnormal compressive loading. Continual medial loading may also create increasing internal rotation of the first metatarsal. The reduction of foot stability will require greater muscular activity for weight acceptance and transfer. The triceps surae muscle is displaced laterally and is shortened. It becomes an evertor and plantar flexor, and the tibialis anterior additionally begins to dorsiflex and evert. Both of these muscles exacerbate the preexisting pronation. 81,84

THERAPEUTIC APPLICATION

The preceding clinical problems are only a few examples of deformities that can be caused by mechanical forces. Early recognition of these causative factors are important for prevention or reduction of the degree of the deformity and disorders through elimination or counteraction of these forces. Intervention strategies must be accurately directed toward wise and judicious use of biomechanical and growth principles to create positive change effectively and efficiently. Intervention can assume any of three possible forms: mechanical, surgical, and therapeutic.

MECHANICAL INTERVENTION

Mechanical intervention is primarily the use of orthotics or positional devices for correction and maintenance of alignment. These devices use three-point pressure systems and force couples to create positive deformation. The principle of creep is used to produce slow but permanent change over a long period of time.

The use of orthotic devices and serial casting takes advantage of the plasticity of tissue and the growth potential of bony structures. The method of treatment may use corrective forces similar to those that produced the deformation. 9 Modification of footwear, use of braces such as the Swedish cage or calipers for the hyperextended knee, Pavlik harnesses for dislocation of the hip, or serial casting for clubfoot are all examples of mechanical intervention.

SURGICAL INTERVENTION

Surgical procedures also use biomechanical principles as a basis for correction of a deformity. Selective use of compressive or distractive force can modify a preexisting condition or reverse deforming forces and prevent bony molding. Instrumentation or bony fusion use long-term or permanent stabilizing forces to produce creep and eventual relaxation of deforming structures.

Surgical intervention may be necessary if conservative measures have not been successful. This should be done as soon as the clinical staff establish that conservative measures are not working. 9 Better results from surgery are achieved if it is performed sufficiently early. 85 Early surgical procedures to establish proper bony alignment will allow mechanical forces to provide more normal development. Examples of surgical procedures include epiphysis stapling, which will increase compression across the epiphysis and retard longitudinal growth, and ostotomies, which can provide more normal alignment of the affected body part. Surgical procedures such as joint replacements or arthrodeses may be necessary later as the patient ages.

THERAPEUTIC INTERVENTION

Therapeutic intervention consists of any procedure designed to strengthen, normalize tone and muscular balance, improve posture and coordination, increase joint or muscle range of motion, and normalize function. The principles of growth and biomechanics must be applied to any therapeutic procedure to assess how, when, and where it should be applied. The length of time of application must also be considered, both in terms of specific technique application and positional modification. Only through understanding and application of these principles can therapeutic intervention be effective in reversing deformation.

Mechanical factors have long been considered a part in the destruction process that ultimately leads to joint deterioration. 45-53,86-89 To prevent or delay joint deterioration, trauma should be avoided and deformities should be recognized and treated as early as possible. Early gait analysis may help in recognizing problems before bony destruction takes place. 88

The treatment of osteoporosis is still controversial and poorly understood. 40 Prevention or delay of osteoporosis is the best treatment. 51,44 This can be done in many cases by early strengthening of the bone. Weight bearing and reaction to muscle contractions stimulate osteo-

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blastic activity. Although this bone loss may be universal, recent literature indicates that physical activity early in life may prevent bone loss.20-43 Continued physical activity in adulthood may also prevent or delay the loss of bone mineral.41 In fact, some authors reported that local bone mass may be increased in the elderly with proper physical activity.14,41,90 Osteoporosis seems to be rare in the active individual.44 Weight training can influence increase in bone density.40,43,91,92 Smith and Reddan found that physical activity for the elderly can help in prevention and treatment of osteoporosis.14 Early bone atrophy may be reversed by physical therapy procedures such as providing weight bearing for two to three hours daily.90,91

Fractures of vertebrae and the proximal end of the femur are major complications of osteoporosis.44,93,94 Deformities, especially in the spine, resulting from collapsed vertebrae also occur. Osteoporosis provides little support for internal fixation of fractures and joint replacements.95 Clinicians must be aware of these complications when they treat elderly patients. For example, a hip fracture may occur before a patient’s fall or occur during evaluation or treatment.

SUMMARY

This article provides a broad overview of the growth, development, and maintenance processes that are related to biomechanical principles. It also provides the major mechanical factors related to growth and development. By combining knowledge of these growth factors and the principles of biomechanics, clinicians should be able to evaluate and perform prevention and treatment procedures that are being used today. They should also be able, with some creativity, to devise better procedures for the future.

REFERENCES

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