Correction of Pediatric Angular Deformity by Reversible Hemiepiphyseal Arrest

Essay

submitted for partial fulfillment of the requirements of the master degree in orthopedic surgery

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In the name of ALLAH the most Gracious, the most Merciful

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Contents

Aim of the work

Review of literature on management of axial deformities in children by reversible hemiepiphyseal arrest.

List of abbreviations

- ✓ **1,25(OH)2** 1,25-dihydroxyVitamin D.
- ✓ **25(OH)D** 25-hydroxyVitamin D.
- ✓ **ADTA** Anterior Distal Tibial Angle**.**
- ✓ **aLDFA** anatomic Lateral Distal Femoral Angle.
- ✓ **CORA** Center Of Rotation of Angulation.
- ✓ **CORA** Centre of Rotation of Angulation.
- ✓ **DBP** vitamin D Binding protein.
- ✓ **DHT** Dihydrotestosterone.
- ✓ **ECM** extracellular matrix.
- ✓ **ER** Estrogen Receptor.
- ✓ **FPA** Foot Progression Angle.
- ✓ **GC** Glucocorticoids.
- ✓ **GH** Growth Hormone.
- ✓ **GHRH** Growth Hormone Releasing Hormone.
- ✓ **IGF-1** Insulin-like growth facor-1.
- ✓ **JL** Joint Line.
- ✓ **KAFO** Knee Ankle Foot Orthosis.
- ✓ **LDTA** Lateral Distal Tibial Angle
- ✓ **LPFA** Lateral Proximal Femoral Angle.
- ✓ **MAD** Mechanical Axis Deviation
- ✓ **PETS** Percutaneous epiphysiodesis technique using transphyseal screws

- ✓ **mLDFA** mechanical Lateral Distal Femoral Angle.
- ✓ **MNSA** Medial Neck Shaft Angle.
- ✓ **MPCs** Mesenchymal Precursor Cells.
- ✓ **MPFA** Medial Proximal Femoral Angle.
- ✓ **MPTA** Medial Proximal Tibial Angle.
- ✓ **NWB** Non-Weight Bearing.
- ✓ **PDFA** Posterior Distal Femoral Angle.
- ✓ **PPFA** Posterior Proximal Femoral Angle.
- ✓ **PPTA** Posterior Proximal Tibial Angle.
- ✓ **T3** Triiodothyronine.
- ✓ **T4** Tetraiodothyronine.
- ✓ **TR** Thyroid hormone Receptor.
- ✓ **VEGF** Vascular Endothelial Growth Factor .

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Fig. 10 Mondos hinege.

Fig. 11 Velcro strapping.

Fig. 12 Staples of three thirt-steond-inch -inch 317 M stainless steel. The legs are three-quarters of an inch long; the cross members are five-eighths and seven-eighths of an inch. Modified pliers and set help place the staples close together. The extractor is necessary for removing the staples.

Fig. 13 Oblique incisions are most satisfactory; adequate they afford exposure and cause less scarring.

Fig. 14 The epiphyseal plates undulate, as seen in both projections.

Fig. 15 An instrument set consisting of 8-plates of various sizes, and cannulated screws of 3.5 mm diameter. The plate and screws are made from titanium. Plates are available in various sizes, and screwsare available in 3.5 mm and 4.5 mm diameter and different lengths**.**

Fig. 16 Operative details of hemiepiphysodesis by 8-palate.

Introduction

Angular deformities of the knee, a common problem encountered in pediatric orthopedics, may often be managed expectantly and with benign neglect, requiring only parental reassurance [1]. Most physiological deformities peak between 1 and 3 years (varus) or between 3 and 6 years (valgus) and resolve spontaneously [2].

Pathological angular deformities can be either idiopathic or due to congenital syndromes such as skeletal dysplasia. In contrast to physiological deformities, pathological deformities manifest as the underlying disease progresses and acts on skeletal growth, leading to a gradual mechanical axis displacement. Valgus deformities in excess of 10° can cause anterior knee pain, circumduction gait, and occasionally patellofemoral instability [3]. Varus deformities may result in lateral thrust, ligamentous laxity, and a waddling gait. Regardless of whether the etiology is idiopathic, dysplastic, or related to an endocrinopathy, the common goal of surgical treatment is to restore and maintain a neutral mechanical axis [4].

Several methods have been described to attain normal alignment. Although osteotomy has been

considered the gold standard for correcting angular deformities, it is fraught with potential complications [5]. Continued growth may result in recurrent deformities and the need for revision osteotomies [1]. The associated discomfort, immobilization, aggregate hospitalization, recovery time, costs, and risks make osteotomy, the last resort for angular corrections while the physis is still open.

Stapling has waxed and waned in popularity since its introduction by Blount [6], but its use has decreased in recent times because of unpredictability and the fear of permanent iatrogenic physeal arrest [7]. Although stapling can work well, occasional breakage or migration of staples can necessitate the revision of hardware or premature abandonment of this method of treatment [6].

The next advance in reversible hemiepiphyseal arrest was proposed by Me´taizeau [8], who suggested the use of transphyseal screws. However, it is unclear whether this epiphysiodesis truly is reversible [9].

To avoid possible complications, a different hardware construct was adopted, employing a nonlocking extraperiosteal plate and two screws (Eight-Plate); this construct guides the physis growth with few complications and high efficacy [4].

Anatomy and physiology of the epiphyseal plate

- ## Introduction

Mammalian growth plate, also known as epiphyseal plate or physis, is highly specialized mesoderm-derived cartilaginous structure. It develops in the bone bud, secondary to presence of the primary ossification centers and is responsible for bone elongation. The plates are formed by numerous cells that rapidly divide and mature. Post puberty, the epiphyseal cartilage cell division decreases, bone completely replaces cartilage, and the epiphyseal plates fuse together with primary and secondary ossification centers [10].

- ## Cartilage differentiation process

Presently, four major stages of chondrocyte differentiation are known, *i.e.*, mesenchymal precursor cells (MPCs), prechondrocytes, early chondroblasts and terminally differentiated chondrocytes [10].

MPCs initiate chondrogenesis by first migration to presumptive skeletogenic sites from the cranial neural crest, paraxial mesoderm, and lateral plate mesoderm and formation of cell mass condensations. MPCs divide in the center of the condensations to form prechondrocytes that turn off the expression of mesenchymal and condensation markers. Instead of an elongated shape they become rounder with concomitant decrease of intercellular adhesion and intensive endothelial cell proliferation [10].

Simultaneously, most of the primary vessels disappear, and only those stimulated by vascular endothelial growth factor (VEGF) persist. Mesenchymal cells commence synthesis of collagen type I, osteocalcin, osteonectin and osteopontin. Differentiation of prechondrocytes leads to active chondrogenic cells called chondroblasts, which rapidly proliferate and build new bone tissue [10].

Prechondrocyte differentiation leads to the next stage, known as early chondroblasts. Early chondroblasts develop cartilage growth plates by assuming a flattened shape and organizing into longitudinal columns. They proliferate at a high rate until, one layer at a time; they exit the cell cycle and start to increase in size, undergoing prehypertrophy followed by full hypertrophy. They

undergo apoptosis to allow primary ossification centers to expand. Cells located in the middle of the epiphysis of future long bones undergo a similar maturation process that leads to the formation of secondary ossification centers [11].

Chondrocytes are metabolically active cells that synthesize various elements of the extracellular matrix (ECM). They are also a source of so called matrix vesicles, 100 nm in diameter follicles, formed by separation of cellular membrane [11].

- **Growth plate morphology and physiology**

Each growth plate is a sandwich-like, multilayer structure divided into four well defined zones: reserve, proliferative, transformation and degeneration (Fig. 1). The last zone is adjusted to the primary spongiosa zone that finally gives origin for the secondary spongiosa zone [10].

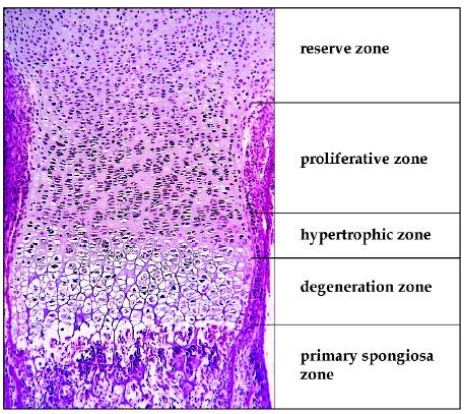

Fig. 1. Longitudinal section of the proximal femoral epiphysis from 21-day-old rat pup (H+E, original magnification ×100) [10].

Resting cartilage cells lying within the **reserve zone** (also known as resting or germinal zone) are formed by small, uniform, compactly located chondrocytes that occur singly or in pairs and are rich in lipid and cytoplasmic vacuoles (Fig. 2). Additionally, in the zone ECM take more place than cells [11].

The direct continuation of the reserve zone is the second layer known as **proliferative zone**. Its chondrocytes are flat and well divided into longitudinal columns (Fig.2) [11].

Below the proliferative zone is another layer known as **hypertrophic zone**. The most characteristic feature of chondrocytes of this level is lack of cellular division and decreased DNA synthesis. Instead of proliferation the cells synthesize high amount of various elements of ECM. Compared to the other zones chondrocytes of hypertophic one are relatively larger. They begin to terminally differentiate and become swollen (Fig. 2). Closer to the primary spongiosa zone the amount of cells with features of **degeneration** increases [11].

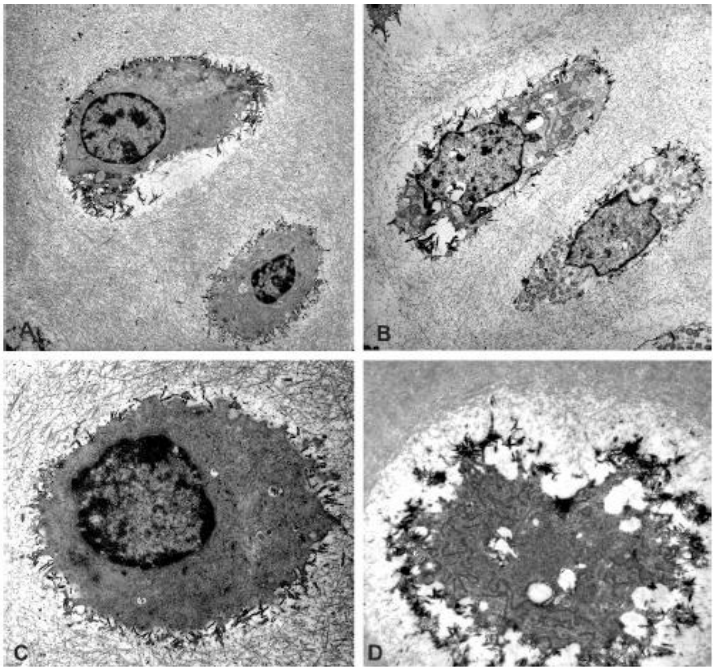

Fig. 2 Chondrocytes of the reserve (**A**), proliferative (**B**), hypertrophic (**C**), and degenerative (**D**) zones of the proximal femoral epiphysis from the 7-day-old rat pup (TEM, original magnification ×3000) [10]

The morphology of the lowest zone – **primary spongiosa** – is similar to the lower level of degeneration zone. However, it is characterized by presence of osteoprogenitor cells. Due to initiation of mineralization process, primary ossified bone lamella and small blood vessels are also visible [11].

The importance of a terminally differentiated chondrocyte seems to be the preparation of calcification by the matrix; since once the cartilage calcifies, apoptosis is required to remove the terminally differentiated chondrocytes. After the cartilage has undergone