Treatment of physeal plate growth arrest: A systematic review of literature

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List of abbreviations

Ant.	Anterior
AP	Anteroposterior
DLS	Dynamic locking screw
DRUJ	Distal radio-ulnar joint
Lat.	Lateral
LLD	Limb length discrepancy
Med.	Medial
PMMA	Polymethylmethacrylate
Post.	Posterior
SCFE	Slipped capital femoral epiphysis

SH	Salter Harris
TSF	Taylor Spatial Frame
LLD	Limb Length Discrepancy

Abstract

Background: There are several ways of physeal injury; the most common cause is traumatic injury. Other causes are: infection, radiation, disuse, tumors, arterial insufficiency, burns, frostbite, chronic stress, and iatrogenic injury which may cause interruption of physeal growth. The most obvious and catastrophic complication of physeal injury is arrest of physeal growth. Complete growth arrest may result in limb length discrepancy, while partial growth arrest may cause progressive angular or rotational deformities.

Purpose: To outline different methods of treatment of partial and complete physeal arrest in a systematic review of literature.

Methdology: Potentially relevant articles describing different techniques of management of epiphyseal plate growth arrest either partial or complete will be identified by online literature search using the PubMed & Cochrane computerized literature database.

Results: In total, 45 papers were retrieved from the initial search strategy. 23 studies met the inclusion criteria and are included in this study. From the 23 studies, 5 were about corrective osteotomy, 6 were for physeal bridge resection, 6 for hemiepiphyseodesis and final 6 for distraction osteogenesis.

Conclusion: For growth plate injuries, the aim of management is to keep the metaphysis; epiphysis and physis separate so that the physeal cartilage is able to grow in between to separate them. When there is a deformity as a result of partial growth arrest; the management is mainly operative through deformity correction by different methods such as: osteotomy, hemiepiphysiodesis or bar excision. When there is a limb length discrepancy as a result of complete physeal arrest; the management can be conservative through use of shoe lift, orthotics and prosthesis if shortening is less than 2 cm. If shortening is between 2cm and 5 cm it is treated by operative correction like chondrodiastasis of ipsilateral limb or epiphysiodesis of the contralateral side. For shortening more than 5 cm, the treatment is by limb lengthening.

Keywords: Physis, Salter Harris, Growth arrest, Osteotomy, Epiphysiodesis, physeal bridge & Limb lengthening

Introduction

Anatomy of the physis:

All bone tissue first forms embryonically as a tissue called primary bone (woven bone). Primary bone is an immature bone that has a distinctive woven appearance under the microscope. Primary bone is formed in the fetus through one of two distinct processes: intramembranous ossification or endochondral ossification. Intramembranous ossification begins directly in mesenchymal tissue. Mesenchymal cells differentiate directly into osteoblasts, which then deposit the bone matrix. Bone tissue formed in this way is known as intramembranous bone. Only a few bones form entirely through this process, such as bones of the developing skull, face, and mandible [(1)].

Endochondral ossification, the more complex of the two processes, involves the development of a cartilaginous model, made of hyaline cartilage, from which immature bone develops. Embryonic mesenchymal cells differentiate into chondroblasts, which deposit a cartilaginous matrix, forming a preliminary structural model of the bone. The primary bone tissue of most developing human bones including most bones of the developing axial and appendicular skeletons is formed through endochondral ossification. These bones are called endochondral bones [(1)].

Nearly all the primary bone formed through these ossification processes is later replaced, through a process of bone remodeling, by secondary bone (lamellar bone). Secondary bone is mature bone. Its matrix is more mineralized than primary bone, and it has a lamellar (layered) rather than woven structure [(1)].

Physis has 3 components: cartilaginous (main), bony and fibrous:

-Cartilaginous component: divided into histologic zones.

-Bony component: metaphysis.

-Fibrous component (periphery): groove of Ranvier and the perichondral ring of LaCroix [1].

Histology of the physis:

The physis is a unique cartilaginous structure that varies in thickness depending on age and location. It is weaker than bone in torsion, shear and bending predisposing the child to injury through this delicate area. The physis is traditionally divided into four zones: reserve, proliferative, hypertrophic, and provisional calcification [2]. **(Fig 1)**

Zone of Reserve Cartilage. This zone consists of the bed of resting hyaline cartilage that is held in reserve and from which proliferating cartilage continuously develops throughout the growth phase of an individual. If no epiphyseal cavity, representing a secondary ossification center, has developed, the zone of reserve cartilage will occupy the core of the epiphysis. If a secondary ossification center has developed, the zone will appear as a band of typical hyaline cartilage situated at some point midway between the primary and secondary ossification centers.

Defect through this zone occurs in Gauchers' disease and dystrophic dysplasia [2].

Zone of Proliferating Cartilage. In this zone, hyaline cartilage, originating from the zone of reserve cartilage, is undergoing intensive interstitial growth. Chondrocytes are proliferating rapidly. As they undergo cell division, the daughter cells line up parallel to the long axis of the tissue, forming long columns of isogenous groups.

Defect through this zone occurs in Achondroplasia and Giagantism [2].

Zone of Hypertrophic Cartilage. In this zone, chondrocytes, which have just undergone a rapid proliferation, have ceased their proliferative activity and begin to hypertrophy. In the process, they compress and resorb the lacunar walls, enlarging the lacunae.

Three phases occur in the hypertrophic zone

- **Maturation zone:** preparation of matrix for calcification, chondrocyte growth.
- **Degenerative zone:** further preparation of matrix for calcification, further chondrocyte growth in size.
- **Provisional calcification zone:** chondrocyte death allows calcium release, allowing calcification of matrix.

Defects through this zone occur in the following:

- SCFE (not renal)
- Rickets (provisional calcification zone)
- Enchondromas
- Mucopolysarcharide disease
- acromegaly
- Fractures most commonly occur through the zone of provisional calcification, specifically Salter-Harris I fractures [2].

Zone of Calcified Cartilage. The enlarged chondrocytes programmatically begin to synthesize and release alkaline phosphatase into the surrounding matrix, causing the cartilagenous matrix to become calcified. The calcification inhibits oxygen diffusion to the chondrocytes, and the chondrocytes begin to degenerate and die [2].

Zone of Ossification (Resorption). Osteogenic cells from the adjacent marrow cavity differentiate into osteoblasts, which in turn adhere to the surface of the calcified cartilage and begin laying down bone osteoid. The result is the formation of mixed spicules. In addition, osteoclasts can be seen resorbing some of this bone in the process of enlarging the marrow cavity [(2)].

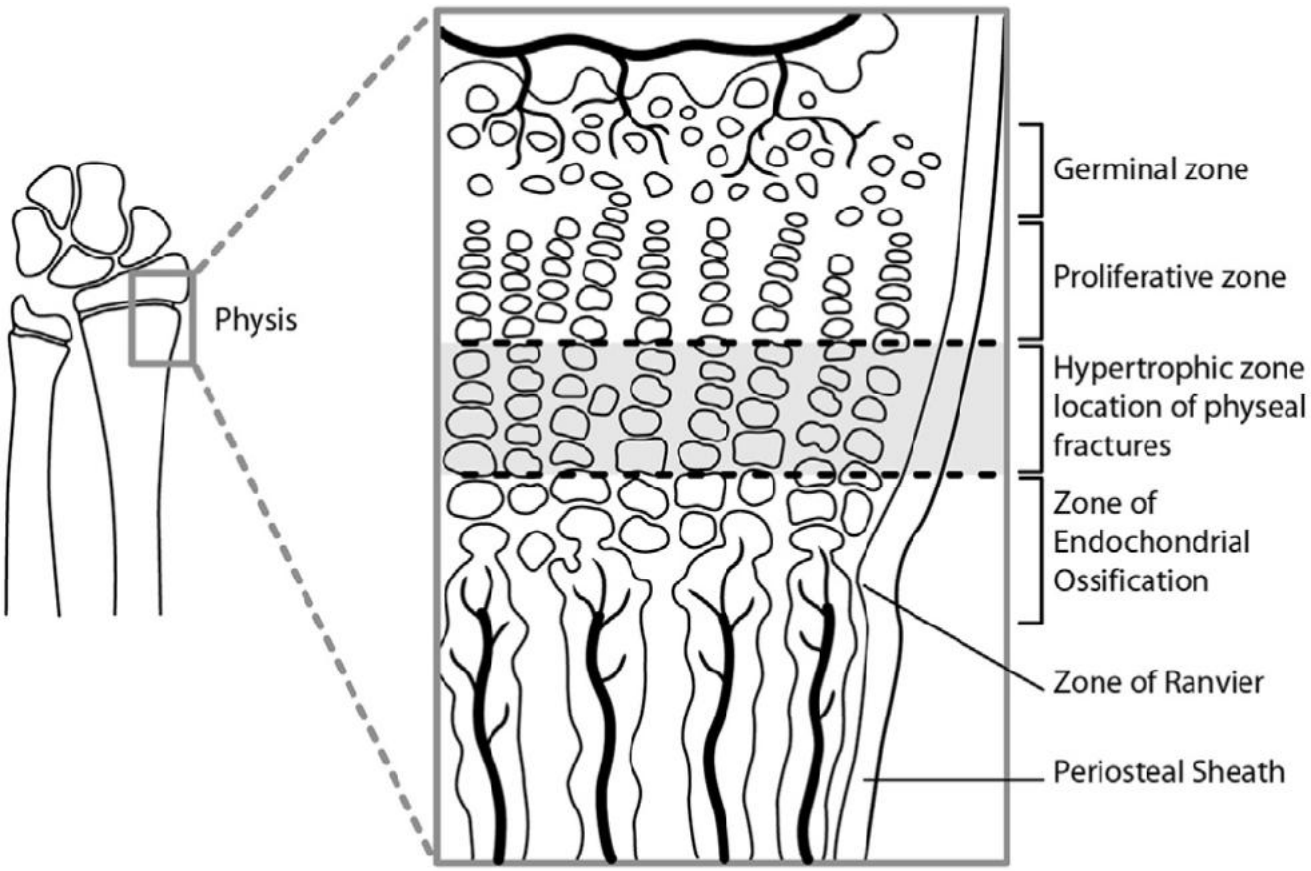

Fig.1 structure of the physis

Ref: ***Mallick A& Prem H (2017); Physeal injuries in children, Surgery (Oxford), Jan 2017: volume 35, issue 1, pages 10-17.***