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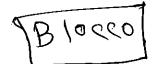


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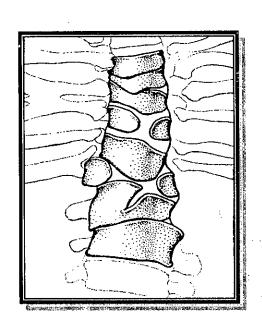
# CONGENITAL SCOLIOSIS

Essay
Submitted for Partial Fulfillment of
M. Sc. Degree in Orthopaedic Surgery

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# TO

MY WIFE,
MY PARENTS
& MY SISTER

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# **CONGENITAL SCOLIOSIS**

# **CONTENTS**

#### **CONTENTS**

Introduction	on	1
Chapter 1	Development and Anatomy of the spine	
•	Development	2
	Anatomy	10
Chapter 2	Biomechanics	
	Biomechanics of normal spine	24
	Biomechanics of congenital scoliosis	31
Chapter 3	Pathology and classification of congenital scoliosis	
	Definition	33
	Etiology,,	33
	Classification	34
	Associated anomalies	38
Chapter 4	Natural History	45
Chapter 5	Diagnosis	
	Clinical evaluation	52
	Radiological examination	55
	Other diagnostic means	56
	Curvature measurements	59
Chapter 6	Treatment	
	General outlines	67
	Non-operative treatment (Bracing & Casting)	68
	Operative treatment	75
	Posterior spinal fusion without instrumentation	76
	Posterior Spine Fusion with instrumentation	79
	Combined Anterior and Posterior Fusion	83
	Combined anterior and posterior Hemi-epiphysiodesis &	
	Hemiarthrodesis	86
	Hemivertbra Excision and Fusion	
	Endoscopic anterior correction of congenital scoliosis	90
	Treatment of congenital kyphoscoliosis	
	Osteotomy in congenital scoliosis	
	Surgical treatment of cervical and cervico-thoracic curves	95
•		
References		101

## **CONGENITAL SCOLIOSIS**

# INTRODUCTION

#### INTRODUCTION

Congenital scoliosis is a lateral curve of the spine that is due to the presence of vertebral anomalies that cause an imbalance in the longitudinal growth of the spine. These vertebral anomalies develop during the first six weeks of intrauterine life, when the anatomical pattern of the spine is formed in mesenchyme. Once the mesenchymal mold is established, the cartilaginous and osseous stages follow that pattern. The vertebral abnormality is present at birth, but the clinical deformity may not become evident until later in childhood when a scoliosis develops and the diagnosis can be made radiographically. Some anomalies cause so little deformity that they remain undetected, so the true incidence of congenital scoliosis in the general population remains unknown. (McMaster and Ohtsuka, 1982)

In this study, a review on normal embryology and anatomy is mentioned briefly as a background in order to understand the pathology of different vertebral anomalies that cause congenital scoliosis. Different types of congenital scoliosis and their natural history are discussed to know when and how we should interfere with individual cases of congenital scoliosis. The first step for treatment of congenital scoliosis properly is by early and precise diagnosis, which makes the responsibility of diagnosis extend to include obstetrician, parents, pediatrician and orthopedic surgeon, as many types of these deformities are known to be rapidly progressive and need very early surgery. Although the advanced imaging methods such as MRI which is of great value in diagnosis of intra-spinal associated anomalies, plain x-ray is still the corner stone in diagnosis of congenital scoliosis. Other investigations should be done in every case to exclude other common associated systemic anomalies.

In the last decades, lines of treatment deviates markedly from conservative methods to the surgical ones. Nowadays conservative means used in the past in the form of bracing and cast become only indicated as an adjunct to surgery. Surgical choices also are developing very rapidly, the ordinary spinal fusion by using Harrington system becomes old fashion and replaced by the new generation of the three dimensional systems (like TSRH and Isola) and the standard open approaches are gradually replaced by endoscopic surgeries. Other operations such as hemivertebral excision, convex hemiepiphysiodesis and wedge osteotomy are very effective in prevention of congenital curve progression specially if it is done early in life.

## **CONGENITAL SCOLIOSIS**

CHAPTER 1

Development and Anatomy of The Spine

## EMBRYOLOGY AND DEVELOPMENT OF THE SPINE

It is important to have a good background about the development of the spine in order to understand the etiology and the pathology of different developmental abnormalities that cause congenital scoliosis.

Development of the fetus is divided into two stages; the embryonic stage and the fetal stage. It is to be noted that all tissues and Systems of the fetus are formed during embryonic stage, while maturation occurs during fetal period.

Thirty six hours after fertilization of the ovum a blastomere is formed, subdivision occurs and morula is formed, fluid accumulates between the cells of morula to form a blastocyst with cell Mass in one side.

#### Formation of embryonic disc:

On the sixth day of fertilization the inner cell mass is differentiated into an outer trophoblast cell layer and an inner embryonic hypoplast layer (endodermal layer).

The ectoderm layer is formed from trophoblast from which it is separated by amniotic cavity, thus by **ninth day** the embryonic disc formed of two layers (bilaminar disc) ectoderm dorsally and endoderm ventrally.

Ectoderm and endoderm become thickened at cephalic end of the embryonic disc to form prechordal plate.

#### ♦ Primitive streak

This is a longitudinal groove formed in ectodermal layer at cephalic end of primitive streak a thickened mass of cell is called *Hensen's node* which surrounding an opening is called *primitive pit*. Cells from primitive streak proliferate and migrate inwardly and cephalically and settle between the ectoderm and endoderm to form mesoderm. Thus by end of **fourteenth day** of fertilization the bilaminar disc become trilaminar embryonic disc. (*French BN*, 1983) (Fig. 1)

On 17th day of fertilization, cells from *Hensen's node* migrate cephalically along the mid line between ectoderm and endoderm and the mesoderm on both sides to form notochord process which extend to the prechordal plate.

Extension of the primitive pit into notochord process to form central canal within it, the ventral part of the notochord process unite with endodermal layer to form neuro-enteric canal which with central canal of notochord joins the amniotic cavity dorsally to the yolk sac ventrally (Fig. 1). The dorsal part of the notochord process reform again to form notochord proper which becomes surrounded from both sides by paraxial mesoblastic cells to form primitive or blastomal vertebral column. (French BN, 1990).

#### Formation of neural tube:

#### (a) Neurulation

The midline ectoderm cephalic to the Hensen's node thickens to form neural plate.

Thickening of the edges occurs to form two paraxial folds leading to formation of neural groove in the mid line along the whole length of neural plate.

Progress of the folds leads to their meeting by **22nd day** in the mid line in the mid dorsal region. Thus with fusing of the folds, begins the transformation of neural plate into a hollow neural tube. This starts in mid dorsal region and extends both caudally and cephalically.

The superficial ectoderm attached at the edges of the neural folds is pulled towards the mid line by fusing the neural folds to fuse dorsal to the neural tube also.

The cells at neuro-ectodermal junction are specialized and become detached from neural tube, which later form the dorsal spinal ganglion cells (Moe et al., 1978).

Temporary delay in the closure of neural tube occurs at cephalic end at the level of lamina terminals (cephalic neuro pore) on 24th day, and caudal end (caudal neuro pore) at the 28th day (French BN,1990). While the neural plate is developing, the mesoderm lies as a complete layer between

ectoderm and endoderm. The paraxial mesoderm thickens on either side of notochord.

Starting from 20th day of gestation, the cells of the paraxial mesoderm undergo segmentation process forming somites, these somites are separated by intersegmental fissures.

The first cranial somites appear in the middle portion of the embryo just caudal to the cranial end of the notochord, which correspond to future occipital area.

This process proceeds in cranio-caudal direction, and at the end of the fifth week, 42somites are formed. On cross section this somites are wedge shaped with cavity in the