The Growing Spine

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The Growing Spine

Management of Spinal Disorders in Young Children

Springer

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Dedication

The diverse nature of this textbook, the reasons for its existence, and the important people who made its completion possible warranted this dedication to be rendered in three parts. *First*, we dedicate this book to children with early onset spine deformity (EOS). This constitutes a group of challenging patients with varying natural histories. The text provides the first attempt to define these natural histories as well as patient treatment options and results in a unified approach; such efforts will hopefully lead to the development of more accurate and effective treatment protocols. It is well known that children with EOS have cardiopulmonary and gastrointestinal problems and also suffer from short stature and possibly from pain. One of our main goals in presenting current treatment methods is to enable decisions that will make these patients functional and productive adults with minimal residual deformities. This book will undoubtedly require numerous revisions as our knowledge advances and newer treatment concepts and techniques become available.

Secondly, we dedicate this book to the members of the Growing Spine Study Group (GSSG) and the faculty and participants of the International Congress on Early Onset Scoliosis and Growing Spine (ICEOS). These individuals have devoted a significant portion of their careers to the study of EOS. They have demonstrated a willingness to work cooperatively in furthering our understanding of the various disorders associated with EOS and to share this information with others. Their contributions and generosity of spirit are leading to the development of newer and better techniques for the correction of spinal deformity while simultaneously maintaining maximum growth and development of the spine and chest. Certainly, newer non-fusion approaches must be developed in the future, such as spinal tethers, self-expanding implants, and minimally invasive techniques, all of which aim to decrease the number of surgical procedures that these children must currently endure. This should result in better spinal alignment, fewer surgeries, fewer complications, and more normal growth.

Finally, we dedicate this work to our families. The creation of this textbook has taken a considerable amount of time away from our homes and loved ones. Nevertheless, our families have always recognized our dedication to others and our major goal of improving their lives. Our endeavor would not have been possible without their understanding and support. In particular, *Behrooz Akbarnia* would like to thank his wife, Nasrin Owsia, for her unyielding love and encouragement, his children (Halleh, Ladan, and Ramin), their spouses (Stu and Gil), and his grandchildren (Simia and Kian); as well as his associates Ramin Bagheri and Greg Mundis and support team at San Diego Center for Spinal Disorders, in particular Pat Kostial and Jeff Pawelek for allowing him to take as much time as needed to work on this book. *Muharrem Yazici* would like to thank his parents, Ayyildiz and Zekeriya, for having

taught him that the greatest virtue in life lies in earning by producing; and Ruya, Yildiz Naz, and Mehmed Emir, for silently condoning his borrowing from their time, and for never depriving him of their support. Finally, *George Thompson* would especially like to thank his wife, Janice Thompson, for her support for the past 3–4 years, both during his Presidency of the Scoliosis Research Society and in the preparation of this textbook. There has been little time for family activities.

We hope that this book will be a foundation for future work in this exciting and developing field.

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Foreword I

Spinal disorders, commonly encountered in the growing spine, are particularly prevalent among adolescents, who continue to receive the lion's share of attention and study. However, the spinal disorders present at birth or beginning in the first decade of life are the ones that most threaten a patient's quality and extent of life, and they are the focus of this timely and much-needed book.

While there are many diseases resulting in spinal disorders, the individual incidence of most is small; many have become known as orphan diseases. But, for most of the affected patients, the resulting spinal disorder is a lifetime condition, thus making the cumulative prevalence substantial. These factors conspire to limit the experience of even the largest centers. This and the fact that many of these young children often have comorbidities foster the need for collaboration among subspecialists.

A common denominator for almost all young children with spinal disorders is diminished spine growth. It is now realized that for many of them there is also diminished thoracic growth. The problem is compounded as the diminished growth is usually asymmetrical, resulting in not only a shortened spine and diminished thorax but also angular deformity. To quantify the abnormal, one must know what is normal; this topic is thoroughly documented in *Growing Spine*.

Management of spinal disorders in young children may be as simple as periodic observation or as complex as the outer limits of surgical art and science. The choice hinges on the natural history of the disorder. Long-term observational studies, including critical imaging interpretation, have added greatly to natural history knowledge. Advances in imaging techniques have made it possible to more clearly define the anatomical abnormalities and plan the appropriate corrective surgery.

Management of spinal disorders in young children often requires surgery. Traction, bracing, and/or casting are sometimes utilized to help decrease or at least control spinal deformity until as much spine growth as is reasonably possible has been achieved, thus delaying surgical treatment. As is described in this book, bracing and/or casting are occasionally the successful, definitive treatment for disorders with relatively favorable natural histories.

Surgical goals are to correct as much as possible the existing deformity, optimize normal growth, prevent abnormal growth, maintain maximum mobility, and protect neurological function. The essential tools of surgery are instrumentation and arthrodesis, and generally two approaches have been taken.

In one, posterior distraction instrumentation without arthrodesis was used with the hope that as the straighter spine grew; it would continue to grow straighter. When this did not occur, the process was modified to include planned, periodic re-operation to reapply distraction load. As the distraction rod-spine construct was not intrinsically

stable, supplemental external immobilization with a brace or cast became necessary. Because the distraction hooks often became dislodged, arthrodesis of the upper and lower end two vertebrae was added. After as much lengthening as could be achieved was obtained, the posterior spinal arthrodesis of the spanned vertebrae was performed.

In the other approach, arthrodesis of the convex side of the scoliosis curve was performed, thereby encouraging concave side growth to catch up. The result was not satisfactory. Eventually, concave distraction instrumentation was added to the convex apex convex arthrodesis with somewhat better results.

The next step in the development of the posterior instrumentation approach was propelled by the desire to avoid supplemental brace or cast immobilization and periodic lengthening. Segmental posterior instrumentation utilizing multiple level sublaminar wire anchors and two parallel rods was introduced. This proved unsatisfactory because of auto-arthrodesis and instrumentation breakage.

This was followed by a return to the limited vertebrae anchor approach. Initially, stable end instrumented vertebrae anchor foundations (initially four hooks, transversely connected dual rods, and expandable connectors at the thoracolumbar junction) were used. Periodic lengthening at approximately 6-month intervals is done until further lengthening is no longer possible, at which point definitive arthrodesis is done. Currently, with some modifications, this is probably the most widely used approach. Two related developments are now occurring. The first is experimentation with periodic lengthening utilizing external magnetic power. The second development shifts focus from the end vertebrae to the apex vertebrae. Bilateral apex and end vertebrae pedicle screw anchorage is utilized to provide three-dimensional apex correction, and apex arthrodesis is performed. Spine growth on either side of the apex is then guided by end vertebrae pedicle screw anchors whose connections are free to slide along the rods.

The last several years have brought a number of innovations. The most thoroughly researched and widely used are the techniques and instrumentation utilized with thoracic expansion. Growth modulation through fixation of the convex apex vertebrae continues to be researched as does the possibility of stopping growth of the concave neurocentral synchondrosis.

An important part of the development of this field has been the improved surgical capability of mobilizing the spine. This includes kyphectomy, hemivertebrectomy, vertebral osteotomy, and vertebral resection. The possibility of safely performing these important but invasive procedures has been aided by continued improvement in anesthesia, neurological monitoring, and postoperative care.

Finally, molecular biology has not as yet impacted this field. However, it is not unreasonable to think that the underlying diseases' abnormal metabolic pathways will gradually begin to be understood, raising the possibility of preventative medical therapies.

The compilation of the rapid advances in knowledge about the management of spinal disorders in young children during the past several years has been badly needed. Without it progress is difficult to achieve. The editors and authors of *Growing Spine: Management of Spinal Disorders in Young Children* are at the forefront of this field and are to be congratulated for consolidating the diffuse knowledge base into one easily accessible tome.

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Foreword II

Behrooz Akbarnia, MD, Muharrem Yazici, MD, and George Thompson, MD have all put together a wonderful book regarding aspects of management of the pediatric spine. There are 50 chapters in this textbook. There is a very substantial international flavor as well. To my count, portions of 18 chapters were written by deformity experts outside North America.

The textbook is very complete as it covers aspects of infantile, juvenile, and adolescent deformity and also covers operative and nonoperative management and not only deformity, but also issues of infection, tumor, trauma, and back pain.

Several chapters in this book should be considered classic and probably represent the definitive work by the definitive expert in a particular topic.

Chapter 2, regarding normal growth of the spine and thorax, by Dr. Alan Dimeglio from Montpellier, France, is the most concise and up-to-date manuscript covering this topic, which is perhaps the most important topic to understand if one is to treat complex pediatric spine problems.

Chapter 15, regarding spinal manifestations of the skeletal dysplasias, by Dr. Michael Ain, is certainly the most definitive source of recommendations and suggestions for treatment of complex skeletal dysplasia problems in the pediatric population.

Likewise, Chap. 16, regarding Marfan, Ehlers-Danlos, and other rare syndromes, by Dr. Paul Sponseller, is a very definitive work that is not available in other sources.

Chapter 23, regarding neurofibromatosis, by Dr. Alvin Crawford, is the most complete work regarding treatment of this disorder in the pediatric population that I have seen. Dr. Crawford and Dr. Akbarnia are considered the international experts on neurofibromatosis.

Dr. Johnston and his institution, The Texas Scottish Rite Hospital in Dallas, have the world's experience with utilizing traction for spinal deformities and there is a wealth of extraordinarily useful information included. Also, Dr. Suzuki's career of experience on fusion in the growing child is extremely valuable and apparent in Chap. 29.

The works by Dr. George Thompson and Dr. Behrooz Akbarnia, Chaps. 35 and 36, regarding single growing rod and dual growing rod fusionless techniques, are likewise extremely up-to-date and definitive. Drs. Thompson and Akbarnia, of course, are the international experts on utilizing techniques of "growing rods." There is an extensive amount of useful information and data in those chapters.

Chapter 37, regarding thoracic expansion, by Dr. Robert Campbell, Jr., should be read carefully as well. Dr. Campbell is the world expert on this topic and the information contained in this chapter is unparalleled.

All chapters in this textbook are written by experts in the field, are very informative, and are well referenced. The chapters represent a nice blend of basic factual data, the author's extensive experience, and a bit of innovation. All chapters are excellent. The chapters I mentioned are those that stand above the rest and are, to my mind, the go-to source for any student of complex pediatric spine disease. I look forward to seeing this textbook in print.

The three editors have done a wonderful job. Thank you for allowing me to write this foreword. It is an honor and a pleasure.

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Foreword III

A foreword in a textbook is customarily written as an explanation of why the authors are adding yet another text to the already crowded world of medical publishing. Although the pediatric spine is well represented in both spine and pediatric Orthopaedics textbooks, neither of these can provide the in-depth presentation of a text solely devoted to the subject. It has been 25 years since *The Pediatric Spine* by Bradford and Hensinger, and 15 years since *Disorders of the Pediatric Spine* by Pang. Much has been learned during those many years.

As I looked at the Contents of this book, two things struck me: the thorough and excellent coverage of the topic, and the truly unique and international scope of those chosen to lend their expertise. In all, ten different countries are represented with 47 total authors.

Basic science is well represented, along with the latest proven and as yet unproven techniques of treatment. Since we have to wait until the end of growth to really know whether our efforts are truly effective, we must be patient and willing to monitor various landmarks of progress over time. These landmarks are coronal curve control, sagittal curve control, torso length, and vital capacity.

Those who read this book will know that they possess the latest knowledge in this unique field.

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Preface

Early onset scoliosis (EOS) is a major topic in pediatric spine deformity today. These challenging deformities occur in almost all differential diagnostic categories. Unfortunately, each diagnosis has a different natural history, making it even more demanding. This is the first textbook on this topic. It is a compilation of the current concepts of evaluation and treatment of the various deformities of the growing spine. We have tried to explore the normal growth of the spine and other associated organs as well as natural history of the various differential diagnostic categories and possible treatment options. It is anticipated that this textbook will need to be updated every two to three years in the future as concepts and treatment guidelines change. Treating the spinal deformity is not the major issue, but controlling the deformity to allow for growth of the spine and the associated organ systems, such as pulmonary, cardiac, and gastrointestinal, is the major goal. Controlling deformity allows for improved spinal growth of the involved child and the controlling associated development of these organ systems. A short trunk has an adverse effect on these organ systems. As a consequence, EOS requires a multidisciplinary care. It involves genetics, pediatrics, pulmonology, cardiology, neurology, neurosurgery, as well as orthopaedic surgery.

Treatment options for very young children are controversial. Bracing, serial Risser casts, and surgery (growth modulation and the use of distraction based or growth-guided techniques such as growing rods) are explored in this textbook.

Preliminary treatment results have demonstrated that growth friendly surgical techniques are effective in controlling or modulating curve progression and allowing for spinal growth. Spinal growth allows for improved capacity of the thoracic and abdominal cavities. Cosmesis is less than ideal as crankshaft remains a significant problem even in the growing rod systems. Surgical treatment complications are high, particularly, infection and implant failure, especially rod breakage. Management of complications is an important aspect of the treatment of EOS. Because of the high complication rate, it is important to make the right decision regarding patient and family selection. They must be cooperative and understanding and be willing to be cooperative during the postoperative period.

Future research is important. The Growing Spine Study Group (GSSG) and other databases will hopefully guide future investigations. Only by defining the results of treatment in a relatively large volume of children over a long period of time can the true effectiveness of each of these techniques be determined. Predicting who will worsen, improving spinal tethers to control progressive deformities and the development of self-expanding or remotely controlled devices that would obviate the need for repeated surgical procedures.

We thank our contributors who are all specialists and experts in a variety of areas involved with early onset scoliosis. We also acknowledge the contribution of the members of Growing Spine Study Group who have continuously provided the information that is the basis for a significant portion of the data presented in this book. Special thanks for assistance in preparing and organizing this textbook are to Sarah Canale and Pooia Salari, without their assistance, the completion of this project would have been very difficult.

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Section I

General

Embryology and Anatomy: Spine/Spinal Cord

1

Shay Bess and Vikas Varma

Key Points

- Development of the spine and spinal cord begins during the third week of gestation.
- Early development includes formation of primitive neural tissue, notochord development, and of the axes of the embryo. The axial skeleton eventually arises from the somites.
- Normal vertebral and neural formation is dependent upon development of these early structures to induce the adjacent cell lines to form the neural arch and distinct vertebral bodies.
- Errors in the formation of these structures lead to induction failure and subsequent spinal dysraphism and congenital scoliosis. Mesodermal vertebrae eventually give way to a cartilaginous anlage, which then ossifies forming the mature vertebral column.
- Neurocentral synchondroses allow continued growth of the spinal canal, and secondary vertebral ossification centers persist until the third decade of life.

1.1 Introduction

Development of the spine and spinal cord begins during the third week of gestation. Early development includes formation of primitive neural tissue, notochord development, and of the axes of the embryo. The axial skeleton eventually arises from the somites, while the central nervous system (CNS) arises from primordial mesoderm. Neurons within the CNS sprout axons form mixed spinal nerves that extend to the appropriate end organs creating the peripheral nervous system (PNS). Mesodermal vertebrae eventually give way to a cartilaginous anlage, which is then progressively ossified, forming the mature vertebrae. Secondary vertebral ossification centers and the neurocentral synchondroses persist until the third decade of life and allow growth of the spinal canal during development. This chapter discusses these key elements of spine and spinal cord development and highlight critical moments during development that can lead to bony and neural malformation.

1.2 Early Development

Initial spine development begins during the third week of gestation. At this stage of development, the embryo exists as a two cell layered structure called the bilaminar germ disc. Approximately on day 15, a groove forms in the midline of the germ disc and progressively elongates. The groove itself is termed the primitive groove. The embryonic cranial and caudal axis is formed as the primitive groove deepens at the cranial end of the embryo, and extends caudally. This central depression is termed the primitive pit, and the mound of cells surrounding the primitive pit is called the

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Fig. 1.1 Photomicrograph of primitive streak in the bilaminar germ disc. The primitive pit, primitive groove, and primitive node form the primitive streak. The head of the embryo will eventually form at the primitive pit and primitive node, and the entire structure (the primitive streak) establishes the embryonic longitudinal axis. (Adapted with permission from ref. [10])

primitive node (Fig. 1.1). The head of the embryo will eventually form at the primitive pit and primitive node. The entire structure (primitive pit, node and groove) is called the primitive streak. The primitive streak establishes the embryonic longitudinal axis, giving rise to left and right sides of the embryo. The cranial/caudal, left/right and ventral/dorsal axes are thus formed in the third week of gestation.

A three layered embryo is subsequently formed by proliferation and migration of epiblast cells through the primitive streak (Fig. 1.2a–c). Epiblast cells invade and replace the hypoblast cell layer, forming the definitive endoderm. Migration of epiblast cells between the epiblast and endoderm layers continues, forming a third cell layer, the mesoderm. Upon establishment of the mesodermal layer, the epiblast is renamed and is now termed the ectoderm or ectodermal layer.

Two midline structures develop in the mesoderm: the prechordal plate and the notochordal process. The notochordal process begins as a hollow mesodermal tube and goes on to become a solid rod structure, called the notochord. The notochord induces formation of the vertebral bodies, and as the early vertebral bodies coalesce around the notochord, the notochord becomes the nucleus pulposus (Fig. 1.3a, b).

Following development of notochord, three distinct structures form in the mesoderm: the paraxial mesoderm, intermediate mesoderm, and lateral plate mesoderm. The paraxial mesoderm lies adjacent to the notochord and gives rise to cell lines that differentiate into the axial skeleton, voluntary musculature and skin

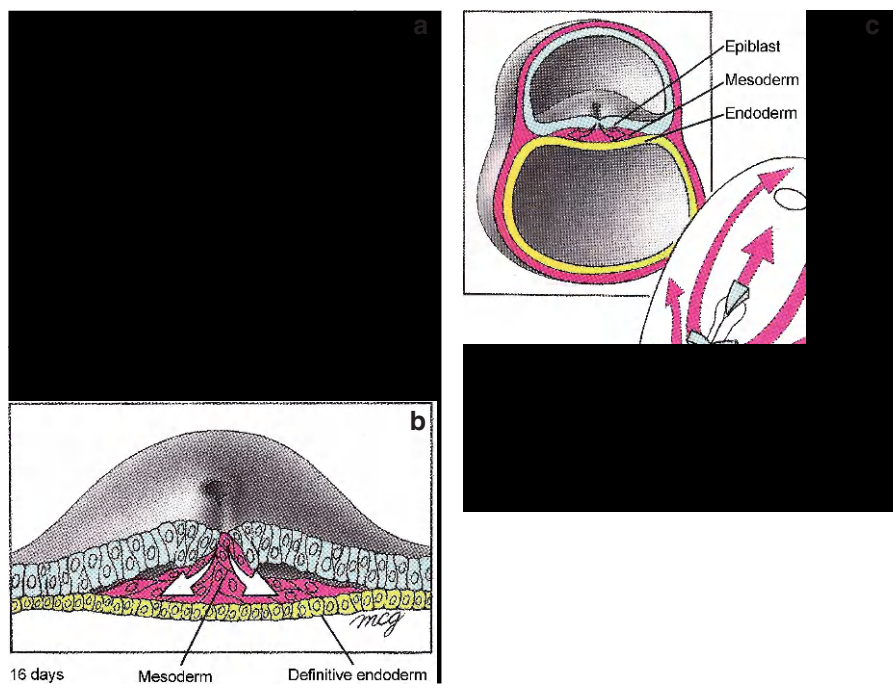

Fig. 1.2 (**a–c**) Proliferation and migration of epiblast cells. Epiblast cells proliferate and migrate through the primitive streak eventually forming the endoderm, mesoderm and ectoderm; the definitive three cell layered embryo. (Adapted with permission from ref. [6])

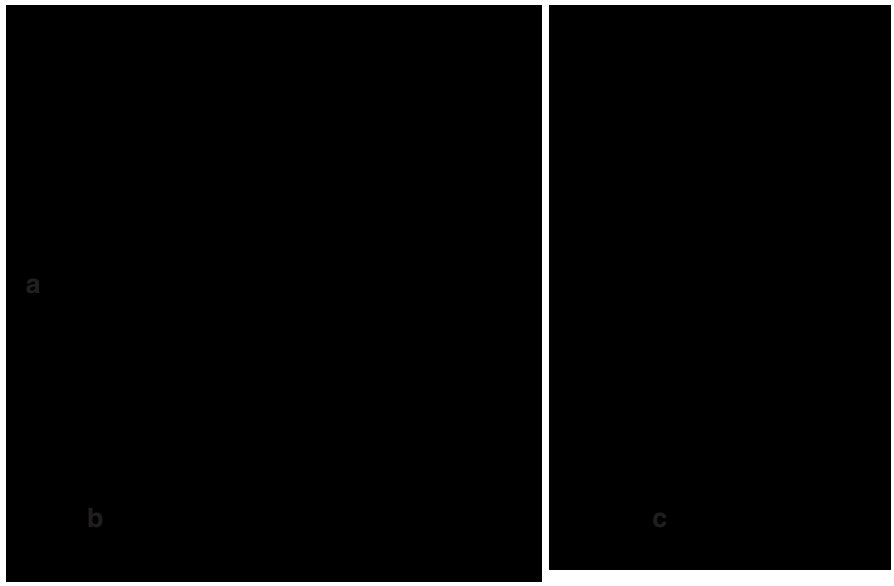

Fig. 1.3 (**a, b**) Formation of Notochordal Process and Notochord. The hollow notochordal process forms within the mesoderm, and goes on to form the solid notochord. The notochord induces vertebral body formation and eventually becomes the nucleus pulposus. (Adapted with permission from ref. [6])

Fig. 1.4 Paraxial mesoderm, intermediate mesoderm, and lateral plate mesoderm formation, location and eventual structures. (Adapted with permission from ref. [4], Figure 16.1 (Section II))

dermis, via formation of somites. The intermediate mesoderm gives rise to the urinary and genital system. The lateral plate mesoderm splits into a ventral and a dorsal layer. The ventral layer forms the mesothelial covering of the visceral organs, and the dorsal layer gives rise to the skin dermis and the parietal lining of the body wall (Fig. 1.4).

1.3 Somite Formation and Differentiation

As indicated above, the axial skeleton, voluntary muscle, and the dermis of the neck and trunk are derived from the somites. The paired somites appear approximately on gestational day 20. The somites arise from the

Fig. 1.5 Somite formation. The paired somites arise from the paraxial mesoderm and form the axial skeleton, voluntary muscle, and the dermis of the neck and trunk. (Adapted with permission from ref. [6])

paraxial mesoderm, and develop in a cranial to caudal fashion at a rate of approximately 3–4 somites per day (Fig. 1.5). Initially, 42–44 somite pairs flank the notochord forming the base of the skull and extending to the embryonic tail. The terminal 5–7 somites eventually regress, forming a final total of 37 somite pairs. Somite pairs 1–4 form the occiput, and bones of the face and inner ear. Somites 5–12 form the cervical spine (there are 8 cervical somites, but only 7 cervical vertebrae, because the first cervical somite participates in occiput formation). Somites 13–24 form the thoracic vertebrae, somites 25–29 form the lumbar vertebrae, and somites 30–34 form the sacral vertebrae. The three caudal, terminal somite pairs persist after regression of the terminal embryonic tail, forming the coccyx. The somite pairs and somite positioning on the embryo form an anatomic template that organizes the spinal column and PNS.

As the embryo develops, the somites separate into subdivisions. Each subdivision gives rise to the ultimate tissue structure that develops from each somite. The first somite subdivisions to appear are the sclerotomes. The sclerotomes ultimately form the bony spinal column. Sclerotomes are formed when a hollow central cavity forms within the somite. This cavity develops in the medial region of the somite, adjacent to the midline notochord and neural tube. The central cavity fills with cells, termed loose core cells, and eventually ruptures, allowing the core cells to migrate towards the midline and envelop the notochord and neural tube (Fig. 1.6).

Fig. 1.6 Sclerotome formation. The central cavity within the somite fills with loose core cells, and eventually ruptures. Core cells migrate toward the midline and envelop the notochord and neural tube forming a sclerotome. The ventral sclerotome forms the vertebral body, and the dorsal sclerotome becomes the vertebral arch. (Adapted with permission from ref. [6])

The cellular structure that eventually surrounds the notochord and neural tube is termed sclerotome. The ventral sclerotome that surrounds the notochord forms the vertebral body, and the dorsal sclerotome that envelops the neural tube eventually becomes the vertebral arch.

Normal vertebral body and vertebral arch development is dependent upon normal sclerotome induction by the underlying notochord and neural tube. Spinal dysraphism, is a spectrum of birth defects originating from failure of neural tube closure (see Chap. 21). This leads to abnormal cell signaling and induction of the overlying sclerotome. Spina bifida is defined as incomplete closure of the neural arch. In the setting of spina bifida, the underlying neural elements are uncovered. Spina bifida occulta indicates that only the neural arch failed to close completely. However, in more severe conditions of spina bifida, the contents of the neural canal can bulge out and become continuous with the overlying skin. The contents of the spina bifida defect are contained by a membranous tissue, a cele. The cele is on the skin surface overlying the spina bifida defect. The contents of the cele may include only the neural meninges (dura and arachnoid) and is termed as meningocele. If the cele contains neural tissue and meninges, it is termed as meningomyelocele.

Once the sclerotomes form and become positioned adjacent to the notochord and neural tube, each sclerotome divides, allowing the spinal nerves to emerge from the neural tube, and exit at their respective level (Fig. 1.7a–d). When the sclerotome division is complete, the caudal half of the suprajacent sclerotome merges with the cranial half of the subjacent sclerotome, forming the vertebra precursor. The division and subsequent refusion explains why there are eight cervical nerves, but only seven cervical vertebrae. The cranial division of the first cervical somite contributes to form the base of the occiput, while the caudal division of the first cervical somite and the cranial division of the second cervical somite form the first cervical vertebra. The first cervical nerve exits above the C1 vertebra, the second cervical nerve exits between C1 and C2, this pattern persists to C7-T1 foramen where the C8 nerve root exits. The sclerotomal cells that remain in the original region of the sclerotome division surround the notochord, and form the fibrous portion of the intervertebral disc, the annulus fibrosis. The enveloped notochord goes on to form the infantile and childhood nucleus pulposis. As the child ages, the original notochord cells of the nucleus pulposis are replaced by fibrocartilageous cells.

1.4 Central Nervous System Development

During early development, two key structures originate in the mesoderm; the notochordal process and the prechordal plate. The prechordal plate induces the overlying epiblast cell layer to form the neural plate. In response to inductive factors produced by the prechordal plate, the neural plate cells differentiate into neurectoderm and proliferate in a cranial to caudal fashion. The cranial portion of the neural plate is broad and gives rise to the brain, while the tapered caudal region of the neural plate forms the spinal cord. The caudal portion of the neural plate overlies the notochord and is bordered by the somite pairs. This positioning allows the caudal neural plate, which will become the spinal cord, to become enveloped by the sclerotomes, forming the spinal canal (Fig. 1.8). The neural plate becomes the neural tube by a process called neurulation, in which the neural plate involutes, until the lateral edges of the folded neural plate and overlying ectoderm meet and fuse in the midline (Fig. 1.9).

Once the neural tube fuses in the midline, it separates from the overlying ectoderm, and differentiates into three distinct layers (Fig. 1.10). The innermost cell layer of the neural tube, the ventricular layer, lays adjacent to the lumen of the neural tube (the neural canal). The ventricular layer contains neuroepithelial cells, which are the precursors to the cells that eventually comprise the CNS. The first generation of cells produced by the neuroepithelial cells is neuroblasts. Neuroblasts eventually become neurons in the CNS. Once formed, neuroblasts migrate away from the ventricular layer to form the mantle layer. The mantle layer eventually becomes the grey matter of the CNS. The neuroblasts in the mantle layer organize into four columns during the fourth week of gestation, forming paired dorsal and ventral columns. The cells of the dorsal column form association neurons that serve to interconnect the motor neurons of the ventral columns with the sensory neurons in the dorsal root

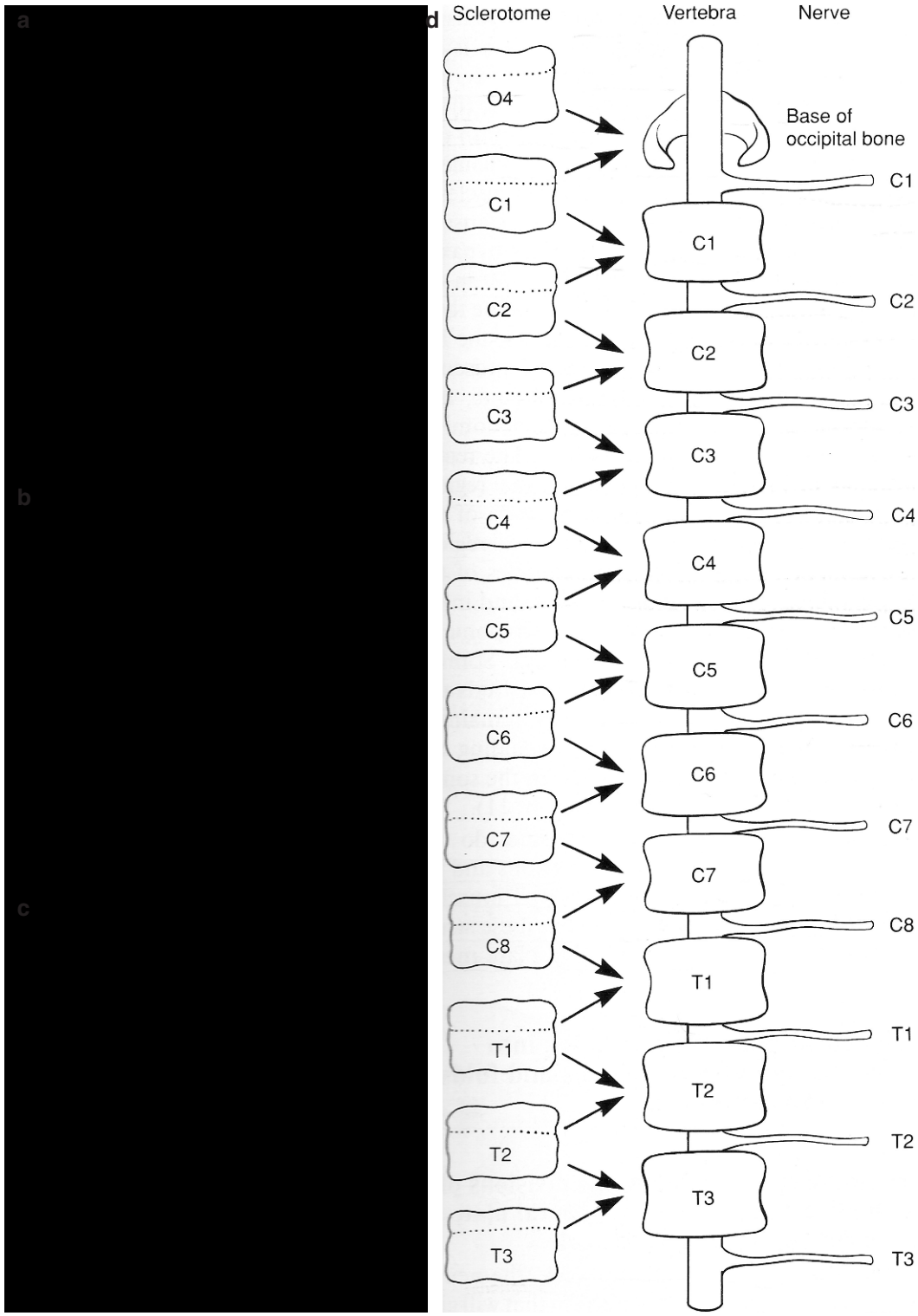

Fig. 1.7 (**a–d**) Sclerotome division and reconvergence. Sclerotome division allows the spinal nerves to emerge from the neural tube, and extend to the periphery. The sclerotomes then reconverge to form the final vertebrae. (Adapted with permission from ref. [6])

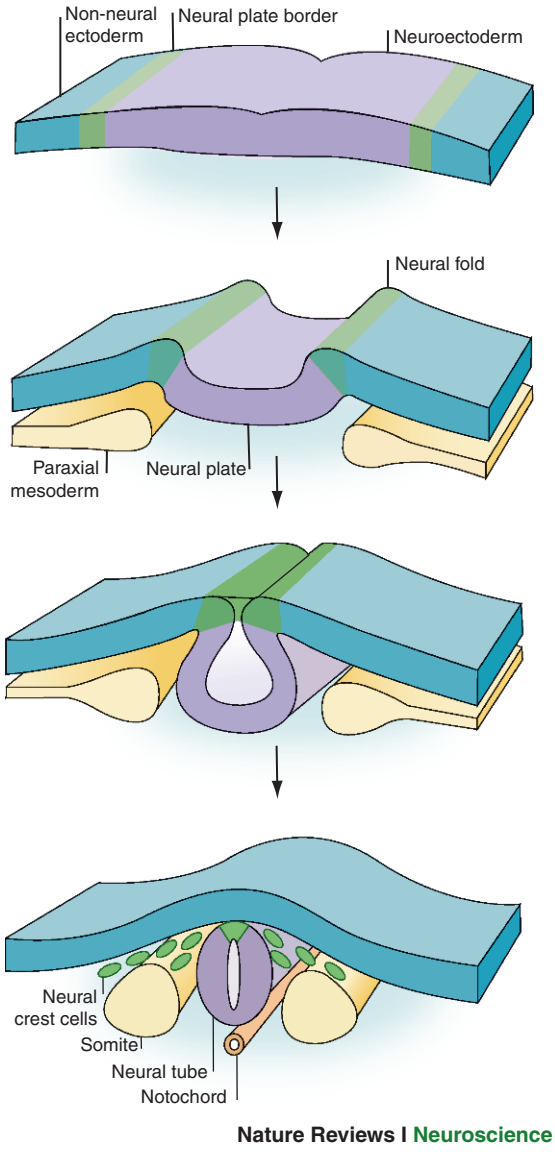

Fig. 1.8 Neural Plate and CNS Formation. The neural plate differentiates from the epiblast, neurectoderm cells and migrate in a cranial and caudal fashion, giving rise to the cranial (brain) and caudal (spinal cord) neural plate. The caudal neural plate is eventually enveloped by the sclerotomes, forming the spinal cord and bony spinal canal, respectively. (Adapted with permission from ref. [3])

ganglia (DRG). Neuronal processes that germinate from the neuroblasts extend peripherally to form the third layer of the neural tube, the marginal layer. The marginal layer becomes the axonal white matter of the CNS.

1.5 Peripheral Extension of the CNS; Formation of the Peripheral Nervous System

Formation of the PNS begins approximately on gestational day 30. Somatic motor neurons in the ventral gray columns extend axon sprouts towards the adjacent sclerotome tissue (Fig. 1.11). The axon sprouts begin at the cervical region, and progressive axonal sprouting extends in a cranial to caudal manner. The ventral axons coalesce as they reach the adjacent sclerotomes, forming distinct segmental nerves and the ventral roots. The somatic system is formed as ventral roots extend past the DRG, inducing the neurons in the DRG to sprout axons. Unlike the somatic neurons in the ventral column, the neurons in the DRG are derived from neural crest cells. The neural crest cells arise from the lateral margins of the neural folds during neurulation. These cells detach from the neural plate, and migrate to different regions of the developing embryo, forming melanocytes, sympathetic and parasympathetic ganglia, and the sensory neurons that reside in the DRG. The axons that extend ventrolaterally from the DRG join the axons in the ventral roots to form mixed spinal nerves. The mixed spinal nerves extend to and penetrate the adjacent sclerotomes and eventually function to innervate the end organs. Other DRG axons grow medially, extending into the dorsal column to synapse with the newly formed association neurons.

1.6 Vertebral Ossification

At approximately the sixth week of gestation, the mesodermal spine precursor transforms into a cartilage model forming the chondrification centers within each vertebra. Two chondrification centers develop in the vertebral body, called the centrum, that go on to fuse in the midline forming a single vertebral body cartilage precursor. If one of the centrum chondrification centers fails to form, a hemivertebra is formed, leading to congenital scoliosis (Figs. 1.12 and 1.13; see Chap. 18). The vertebral arches derive from chondrification centers adjacent to the vertebral body. One chondrification center exists for each neural arch. Chondrification centers for the transverse processes and spinal process

Fig. 1.9 Neurulation. The neural plate becomes the neural tube during neurulation, in which the neural plate involutes and the lateral edges of the folded neural plate fuse in the midline. (Adapted with permission from ref. [6])

Fig. 1.10 Neural tube differentiation. The neural tube differentiates into three distinct layers. The ventricular layer forms the precursor cells that eventually populate the mantle and marginal layers, and comprise the CNS. The mantle layer forms the grey matter of the CNS. The marginal layer becomes the axonal white matter of the CNS. (Adapted with permission from ref. [6])

Fig. 1.11 PNS formation. Axon sprouts emerge from the primordial spinal cord and coalesce as they reach the adjacent sclerotomes, forming segmental nerves and providing end organ innervation. (Adapted with permission from ref. [6])

Fig. 1.12 Vertebral chondrification centers. The mesodermal spine precursor transforms into a cartilage model via the chondrification centers. The chondrification centers are eventually ossified forming the mature vertebrae. (Adapted with permission from ref. [7])

subsequently form, completing the cartilage anlage for the vertebra.

Each vertebra derives from three primary ossification centers; one for the body (centrum) and two adjacent centers for the vertebral arches (Fig. 1.14). The centra are first ossified in the lower thoracic and upper lumbar regions. Centra ossification progresses more rapidly in the caudal vertebrae, while the vertebral arches are more rapidly ossified in the cervical spine. The cervical lamina are ossified as early as the eighth week, well before the cervical centra ossify. Dorsal, midline fusion of the lamina initially occurs in the lumbar spine, then progresses cranially. Once ossified, the lamina do not fuse to the centrum. Instead, an embryologic joint, the neurocentral synchondroses, persists between the centrum and each lamina. The neurocentral

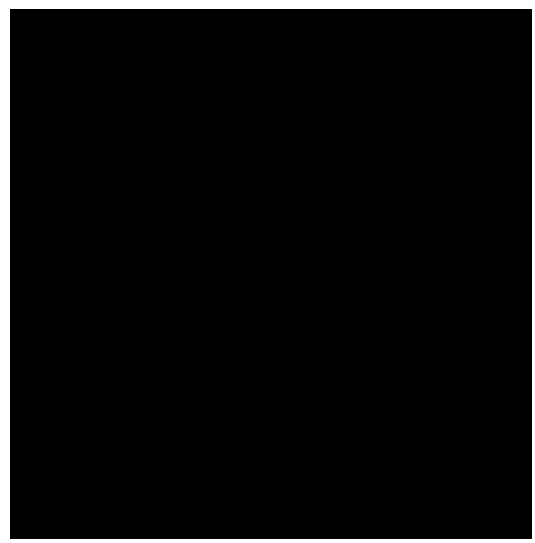

Fig. 1.14 Vertebral ossification centers. The three primary ossification centers of the vertebrae. (Adapted with permission from ref. [5])

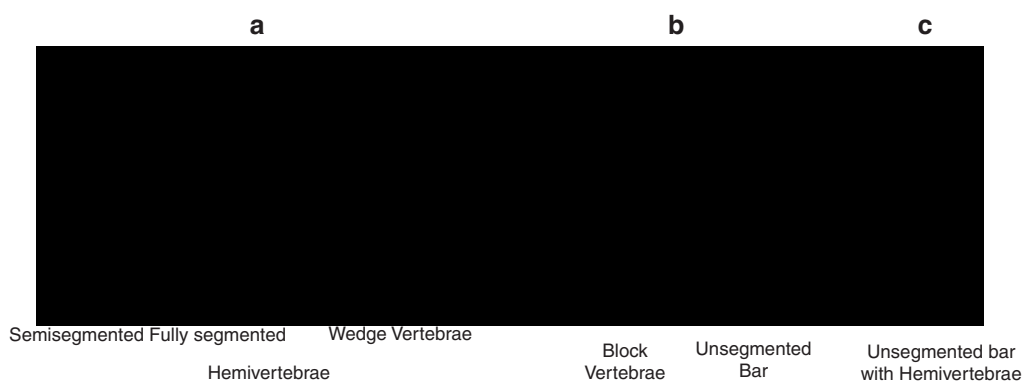

Fig. 1.13 Congenital scoliosis. (Adapted with permission from ref. [2])

synchondroses allow expansion of the spinal canal during growth of the centrum and lamina, and eventually disappear by 6 years of age. Secondary ossification centers at the tips of the transverse processes, spinous process, and ring apophysis develop after birth, and eventually fuse during the third decade (Fig. 1.12).

1.7 Conclusion

Embryological formation of the spine and spinal cord progresses in an organized manner, beginning with formation of the primitive streak, notochord, somites, and sclerotomes. Normal vertebral and neural formation is dependent upon development of these early structures to induce the adjacent cell lines to form the neural arch and distinct vertebral bodies. Errors in formation of these structures lead to induction failure and subsequent spinal dysraphism and congenital scoliosis. These embryological events have been previously described [8–10].

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Normal Growth of the Spine and Thorax

2

Alain Dimeglio, François Bonnel, and Federico Canavese

Key Points

- The growing spine is a mosaic of physes.
- Growth is characterized by changes in rhythm.
- The thorax is part of the spine, it is the "fourth dimension of the spine".
- Lung growth is essentially completed by the age of 8 years with a golden period of maximum growth occurring before 5 years of age.
- Anticipation is the best strategy when dealing with complex spinal deformities.
- The goal of management is to control spinal deformity without impending spinal growth.
- The best strategy is to open the thorax before 5 years of age and keep the spine supple before puberty.
- The immature spine is dominated by the crankshaft phenomenon.
- Early arthrodesis before 10 years of age has a negative effect on the development of the thorax.
- Puberty is a turning point for scoliosis, and skeletal maturation is a precious parameter for follow-up.

2.1 Growth Holds the Basics

It is growth that distinguishes pediatric from adult orthopaedics. It is this ongoing 17-year adventure, punctuated by upheavals that gives this discipline its originality and makes it so interesting. Growth analysis is the evaluation of the effects of time on the growing child. Growth is a complex and well-synchronized phenomenon with a hierarchical pattern that organizes the different types and rates of growth in various tissues, organs, and individuals through time [6, 20, 26].

Growth can be considered as "microgrowth", which is mainly the growth at the cellular level (e.g., in the physes). Although the histologic structure is the same, each physes has its own characteristics and dynamics [20]. The study of height, weight, and body proportions may be considered as the study of "macrogrowth". This study is the culmination of all the effects of microgrowth on the individual: the combined effect of growth of the lower limbs, the trunk, and the upper limbs, increase in weight, and so on [20, 26].

The scope of this process called *growth,* and the changes it brings about, can be better perceived by considering these facts: from birth onward, height will increase by 350% and weight will increase 20 fold, and the spine will double in length [24, 26].

Growth is an essential element in the natural history of any orthopaedic disorder in the growing child [24, 26]. It would be a mistake to assume that only growth in terms of increase in height is important. It is equally important to consider the manner in which the skeletal system develops, that is, the timing of growth in various parts of the body and the changing proportions o f various body segments.

The spine surgeon needs to know the normal values for many parameters and how to measure them. He or she needs to know the significance of these values, for

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