Pediatric Hand Therapy

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Acknowledgments—Pediatric Hand Therapy

This book is dedicated to all healthcare providers who care for the child's upper limb. Specifically, the goal of this book was to provide a resource for the occupational therapist who cares for children's upper limbs. Several of the conditions and injuries are somewhat rare to see for most providers and therefore, we have tried to make a quick, easy to read resource for these providers and people interested in caring for the child's upper extremity. The work performed to bring this book to completion would not have been possible without the collaboration and efforts of all of the authors of the various chapters as well as my coeditors. I must also thank my parents for their continued support and encouragement. Most importantly, I want to thank my wife, Laura, and our three boys, Noah, Benjamin, and Zachary, for always loving and supporting me. Although I know the time necessary to do projects like this takes away some of our time together, you always continue to support me, understand the work that I am doing, and most importantly love me−−and for that I thank you.

Joshua M. Abzug, MD

Hand surgery and hand therapy are synergistic. The results of hand surgery require preoperative and postoperative communication between the surgeon and the therapist. Our therapists provide invaluable preoperative input into the families and their children regarding expected outcomes, cooperation, and compliance. Our therapist provides critical postoperative care. This care requires communication as the therapist must understand the procedure performed and the status of any repaired structures, such as a nerve or tendon. A stout repair can be managed with early mobilization. In contrast, a weaker repair must be treated with gentle mobilization. At Shriners Hospitals for Children-Philadelphia, hand surgeon and hand therapist work in tandem. We work as a team to provide state-of-the-art surgery and optimum therapy to maximize our children's outcome. Complex procedures mandate a skilled and talented therapist. We require our families to travel back to Philadelphia to begin their rehabilitation process. This prerequisite avoids miscommunication and initiates the rehabilitation process with a therapy team accustomed to the surgical procedure. Our therapists are familiar with the potential trials and tribulations in the early therapy period. Addressing these problems can avoid a suboptimal outcome, which is disappointing to the family, therapist, and surgeon. Over the last 25 years at Shriners Hospitals for Children, I have had the privilege to work with many gifted therapists. They have enhanced our patient's outcomes and made me a better surgeon. I want to thank each and every one of them for all their knowledge, for all their expertise, and for all their service to the children at Shriners Hospitals for Children.

Scott H. Kozin, MD

The key to achieving excellent outcomes in upper extremity rehabilitation is relationships. The relationship between hand surgeon and hand therapist creates the foundation on which communication, shared values, trust, and collaboration are built. The relationship between therapists in the subspecialty of pediatric hand provides a network of colleagues and problem solvers who share resources and opportunities. The relationships among healthcare providers, children, caregivers, and families enable our youngest clients to embrace possibility and participation through surgery, therapy, and adaptation. This text was built on invaluable relationships between therapist and surgeons, and we hope it adds an impactful resource to your pediatric hand therapy practice.

Rebecca Neiduski, PhD, OTR/L, CHT

Preface

Caring for the child's upper extremity is challenging due to the rarity of various injuries and/or conditions as well as the child's inability to cooperate and understand instructions. Despite these obstacles, occupational therapy is a critical component of caring for the child's upper limb. Whether the therapist is helping a child with a congenital limb difference learn how to perform activities of daily living or rehabilitating a child following a traumatic injury, the occupational therapist is maximizing the function of the child. Despite this critical role, few resources exist to aid the occupational therapist in caring for the child's upper limb. The purpose of this book is to provide a comprehensive, easy to read and use reference for the healthcare provider who is caring for pediatric and adolescent upper extremities. The book details the formation and functional development of the child's upper limb. Subsequently, the necessary details and key points of the examination are discussed along with details of various outcome measures. In addition, splinting and taping techniques as well as prosthetic use are emphasized. The remainder of the book is organized into various sections to permit the reader easy access to specific diagnoses. Although the book provides a concise, yet thorough discussion regarding these topics, we envision that the treater will keep the book at "arm's length" as a resource for caring for children with an upper extremity condition. Many of the chapters provide protocols to use during rehabilitation as well as specific splints that are necessary to improve function or during the postoperative course. The goal of this book is to provide the reader with the knowledge to perform a thorough examination, establish an accurate diagnosis, refer for timely treatment, and perform specific rehabilitation including therapy and splinting to maximize the child's outcome.

CHAPTER 1

Embryology and Intrauterine Diagnosis

FRANCISCO SOLDADO, MD PHD • SCOTT H. KOZIN, MD

INTRODUCTION

Someone's first-ever sip of coffee is often an unpleasant experience that renders them pondering how they could ever learn to like such a foul-flavored drink. Similarly, many health professionals' first exposure to embryology, and the basic science that is so integral to it, is often a bitter experience. However, over time, dedicated professionals learn how interesting the field is and how essential the knowledge gleamed is pertinent to patient care. This fundamental principle is particularly true for those who choose to enter a field that evaluates and treats newborns with congenital defects.

Congenital anomalies affect somewhere between 1% and 3% of newborns. Among these infants, roughly 1 in 10 has one or more abnormalities that affect their upper extremities.[1,2] In prevalence, upper-extremity anomalies rank second only to congenital heart defects among malformations present at birth.[3] Most limb anomalies manifest spontaneously or are inherited, with congenital anomalies secondary to teratogens decidedly rare.[4,5]

For those clinicians that evaluate newborns with hand anomalies as patients, or counsel parents who have already born such a child, a basic understanding of embryogenesis, limb formation, and genetics is utterly essential. Also crucial is understanding how these anomalies may relate to more systemic conditions, as these healthcare providers often are required to counsel parents about the potential effect on future pregnancies and what intervention can and should be done. Understanding genetic criteria and their associated anomalies affords such healthcare providers the capacity to make appropriate recommendations to families and/or referral to clinical geneticist and/or genetic counseling.

The requirements are not the same with all upper-limb congenital anomalies. For example, transverse deficiencies are usually sporadic and carry no appreciable hereditary risk. As such, subsequent pregnancies require no more monitoring than standard care,[1] and there is no need to refer this family to a clinical geneticist. However, concerns about the risks of teratogen exposure elevate when multiple limbs are affected and deficient. This clinical finding suggests some widespread insult to all the developing limb buds and potential teratogen or bleeding abnormality.

Conversely, many other upper-limb anomalies (e.g., radial deficiency) are associated with concomitant, systemic defects (Fig. 1.1).[6] At the same time during embryogenesis when upper-limb anomalies are in their formative stage, other organ systems are developing at the same time. These organ systems can be affected and require evaluation. It is essential that the clinician recognizes those anomalies that typically occur in isolation versus those anomalies that are associated with concomitant anomalies; many of these anomalies may initially be unapparent with dire consequences. This principle is especially crucial when the concomitant anomalies of other organ systems are of greater clinical importance than the limb anomalies. Hand surgeons assessing such patients must focus on the infant's general health before addressing hand malformations.

Some congenital hand anomalies are linked to other musculoskeletal problems, such as ulnar deficiency.[7] Some anomalies can even be associated with more than one musculoskeletal disorder. For example, central deficiency may be linked to the triad of ectrodactyly ectodermal dysplasia and facial clefts (the so-called EEC syndrome) or lower-limb hemimelia (in which

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FIG. 1.1 Nine-month-old boy with an inherited radial deficiency associated with Holt–Oram syndrome. Mother and child's heart anomalies were surgically treated.

either the tibia or fibula is absent or inadequately formed) (Fig. 1.2).

HOW LIMBS DEVELOP IN UTERO

Embryogenesis

After an egg is fertilized, the first stage of growth is called embryogenesis. During this period of time, a sequence of events occurs that will determine the number of limbs, their location, and their orientation.[8] In addition, during this time, between the fourth and eighth week of gestation, most upper-extremity congenital anomalies occur. The sequence of events that determines upper-limb development is as follows:

- Day 26 after fertilization: The limb buds initially become visible. The embryo is only about the size of a single grain of rice, roughly 4 mm in length.[9,10]
- Days 27–47: Over the next 3 weeks, limb buds develop rapidly, but the fingers and toes are not yet identifiable. Even at the end of this period of time, the entire embryo is still only about the size of a lima bean, roughly 20 mm in length.
- Days 48–53: Over the next five or so days, the fingers and toes separate, so that hands and feet become clearly recognizable.
- Day 56: By the end of the eighth week after fertilization, all the essential limb structures are present. Embryogenesis is complete and the next stage of development, the fetal period, has begun.

Fetal Period

Upon the completion of embryogenesis, the fetal period begins. During this stage of development existing structures differentiate, mature, and grow.[3–13] In the limbs, part of the differentiation and maturation process involves the creation of articulations. Joints form as chondrogen condenses into dense plates between limb structures that will ossify to become bones.[14] Joint cavitation develops the articulation further, though each joint's development ultimately requires fetal movement to ensure the joint surface is modeled into its final prenatal form.

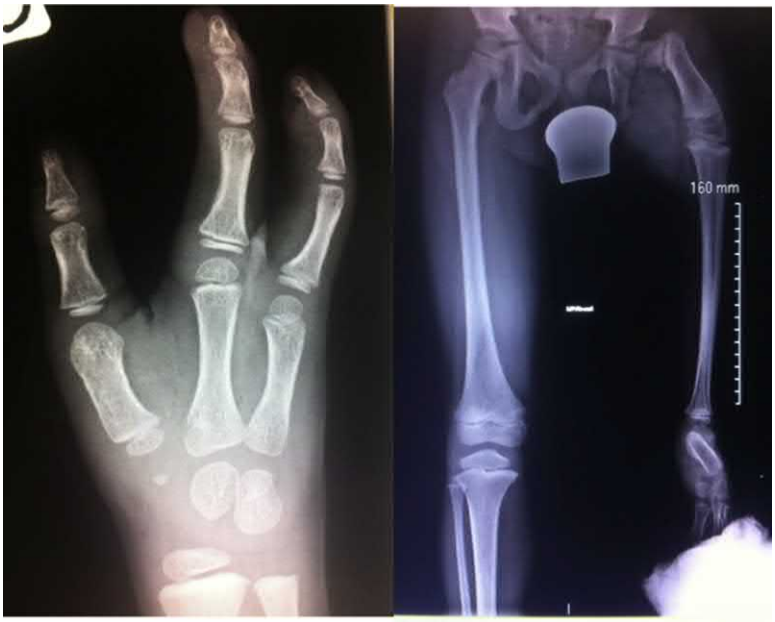

FIG. 1.2 Ulnar longitudinal deficiency associating a proximal femoral focal deficiency.

At a cellular level, limb buds are an outgrowth of mesoderm into overlying ectoderm. Cells from two mesodermal sources—lateral plate mesoderm and somatic mesoderm. These cell lines migrate from their origins into the limb bud.[3,14] The lateral plate cells eventually become bone, cartilage, and tendon. The somatic cells form muscles, nerves, and vascular elements. Blastemas are clusters of cells that all are destined to differentiate into the same type of tissue. In the fetus's developing limbs, muscular and chondrogenic blastema, derived from lateral plate mesoderm differentiate into muscles and bones, respectively.[15] The level of oxygen tension appears to play a part in this differentiation process. Chondrogenic blastema is located more centrally within the limb bud where oxygen tension is relatively low. Muscular blastema is more peripheral in location where oxygen tension is greater. Both muscles and the cartilaginous structures that ultimately will ossify to become bones develop sequentially, starting proximal and progressing in a distal direction.

Joints form between the ends of adjacent blastemas, a joint capsule surrounding the interzone and the intervening blastemas cavitating within the interzone's center to create the articular space. Joint fluid is produced within this space, while cartilage caps the two ends of each bone. Joints ossify and fuse, resulting in synostosis, when the process mentioned earlier fails. Two joints commonly effected by are the proximal radioulnar and ulnohumeral joints (Fig. 1.3). Another component of fetal development that is required for the formation of a functional mobile joint is movement. When fetuses fail to move adequately, as in arthrogryposis, joint spaces become infiltrated by fibrous tissue resulting in contracted and immobile joints (Fig. 1.4).

Signaling Centers

Three growth signaling centers—the apical ectodermal ridge (AER), the zone of polarizing activity (ZPA) and the Wnt (Wingless type)—central to limb patterning align the three spatial axes of limb development. The axes are labeled proximodistal, anteroposterior, and dorsoventral, respectively (Table 1.1).[14–17] As demonstrated later, our understanding of embryogenesis has been advanced by ingenious experiments performed by embryologists. In these experiments, animal models with limb patterning have been manipulated to permit the dissection and alteration of crucial signaling centers that effect limb development and orientation.[12,13,18]

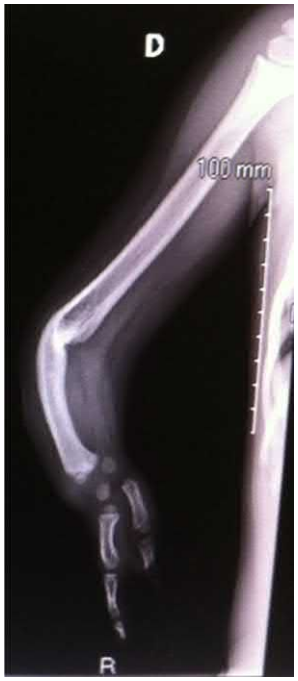

FIG. 1.3 Ulnohumeral fusion or synostosis associated with ulnar longitudinal deficiency.

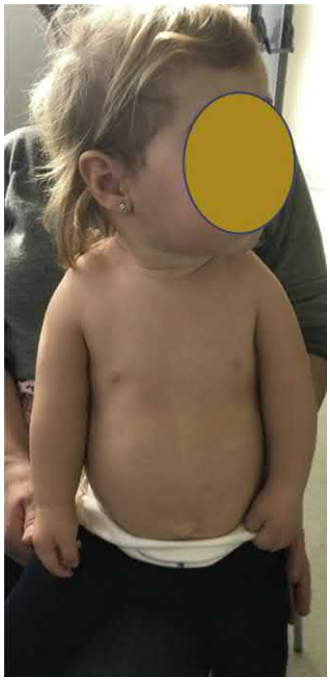

FIG. 1.4 Eleven-month-old girl with arthrogryposis involving predominantly the shoulder girdles. Absence of elbow creases revealing intrauterine poor motion.

TABLE 1.1
Spatial Axes of Limb Development, their Signaling Centers and Malformation Associated.

Signaling Center	Signaling molecule	Limb Axis	Malformation
Apical ectodermal ridge	Fibroblast growth factors	Proximal to distal	Transverse deficiency
Zone of polarizing activity	Sonic hedgehog protein	Radioulnar	Mirror hand
Wnt pathway	Transcription factor, Lmx-1	Ventral and dorsal	Abnormal nail and pulp arrangement Nail-patella syndrome

Proximodistal limb development

Limbs develop in a proximal to distal direction, from shoulder → arm → forearm → hand. The proximodistal signaling center, called the apical ectodermal ridge (AER), is a thickened layer of ectoderm that condenses over each limb bud[14] and secretes proteins that create this effect.[19,20] Experimental models have been developed to mimic proximodistal limb development. They include removing the AER, which results in limb truncation. Conversely, ectopic implantation of the AER induces the formation of additional limbs.[10,12,14] Interestingly, however, removing the AER can be overridden by administering certain fibroblast growth factors that are released by the AER. Moreover, mice deficient in these fibroblast growth factors exhibit complete transverse limb defects.[21,22]

Given these results, transverse deficiencies are now attributed to deficits in the AER or certain signaling molecules, such as fibroblast growth factors, that it produces (Fig. 1.5).

FIG. 1.5 Adactylous form of symbrachydactyly a manifestation of transverse deficiencies. "Nubbins" are the vestiges of digits and are the hallmark of symbrachydactyly. The nubbins are comprised of the remaining ectodermal structures of the distal finger (the pulp, nail fold, and nail).

Anteroposterior limb development

In animal models, both transplantation of the anteroposterior (i.e., radioulnar or preaxial–postaxial) signaling center, called the zone of polarizing activity (ZPA), and transplanting the sonic hedgehog protein that the ZPA secretes have been demonstrated to cause mirror duplication of the ulnar aspect of the limb.[23] Mutant mice with sonic hedgehog protein in their anterior limb bud develop polydactyly.[24] Also in models, triphalangeal thumbs (thumbs with three phalanges, instead of the usual two) have been found to arise secondary to point mutations that generate ectopic sonic hedgehog compound at the anterior margin of the limb bud.[25] In humans, therefore, both mirror hand and certain forms of polydactyly are now attributed to deficits in the ZPA or sonic hedgehog protein (Fig. 1.6).[3,18]

Dorsoventral limb development

The mechanism behind the development of the dorsum of the finger with its fingernail and the volar surface with its abundant pulp tissue are differentiated and developed is not well understood.[11] The pathway responsible for this differentiation produces one transcription factor, Lmx-1, that induces the mesoderm to adopt dorsal characteristics.[26] In the ventral ectoderm, the Wnt pathway is blocked by a product of a gene called engrailed-1 (En-1). Mice lacking the anteroposterior Wnt signaling pathway, which resides in dorsal ectoderm and secretes Lmx-1, exhibit ventralization of the dorsal surface of their limbs, such that they manifest palmar pads on both sides of their hand: front and back.[27] Conversely, mice lacking the engrailed-1 protein exhibit dorsalization of their limbs' volar surfaces

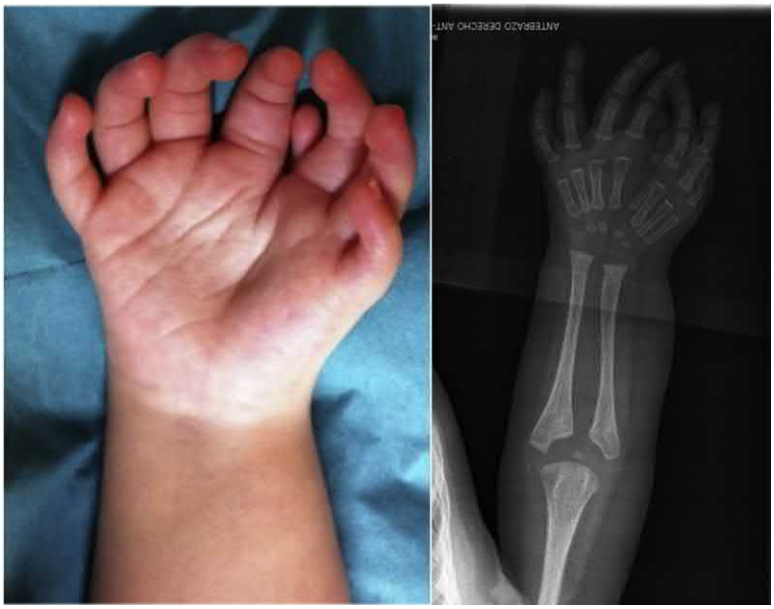

FIG. 1.6 Mirror hand attributed to abnormal anteroposterior limb patterning. The result is duplication of the ulnar field but absence of the radial field.

(so-called bidorsal limbs).[28] Alterations in this latter pathway are relatively rare. Loss of Lmx-1 is associated with a condition called *nail-patella syndrome*, in which affected individuals have small, poorly developed nails and kneecaps. Affected individuals also have musculoskeletal defects in other areas of the body including their elbows, hips, and chest.[29] Other children may present with anomalies that include extraneous nail or abnormal pulp development, both linked to an altered Wnt signaling pathway.[30] In humans, dorsal dimelia with the nails may present on the palmar surface of fingers, is explained by alterations in the Wnt signaling pathway or Lmx-1 (Fig. 1.7).[23]

Programmed Cell Death

Programmed cell death (PCD) is another essential component of proper limb development. PCD is an active process that is genetically controlled. PCD eliminates unwanted cells during embryogenesis.[23] Apoptotic cells undergo a degenerative process, associated with DNA fragmentation, and eventually are engulfed by phagocytes. A clear example of this is the separation of fingers and toes during days 48–53 of gestation. Before day 48, all human digits are webbed. Over the next 5-day period of gestation, interdigital necrosis occurs with extraneous, web-like tissue between fingers and toes undergoing PCD. Failure of interdigital PCD results in syndactyly.[31]

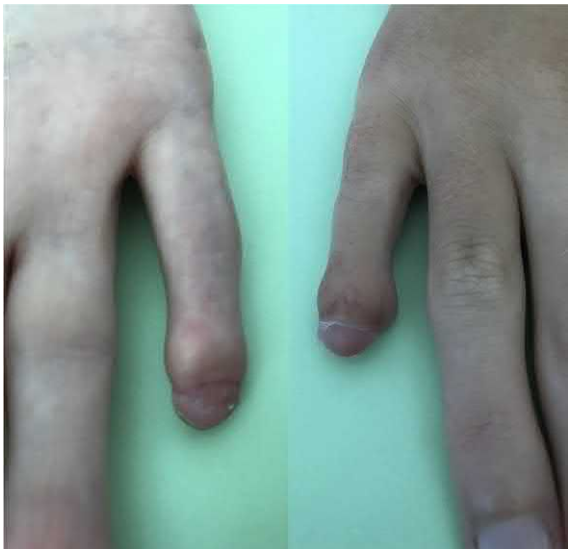

FIG. 1.7 Dorsal dimelia with abnormal dorsoventral limb patterning can cause duplication of the dorsal field resulting in the presence of a nail both in the dorsal and volar sides of the finger.

Widely recognized for their role in chondrogenesis and osteogenesis, bone morphogenetic proteins (BMPs) also trigger apoptotic pathways in interdigital mesenchyme to separate the fingers.[23] Antagonists to

BMP are capable of blocking BMP signaling and preventing this process of apoptosis and interdigital necrosis. An obvious animal example of this are bats, mammals whose limbs are webbed, and whose BMP has been shown to be blocked during limb embryogenesis.[32] Similarly, altered signaling of fibroblast growth factors can negate BMP-mediated apoptosis and result in syndactyly, which occurs in individuals with Apert syndrome (Fig. 1.8).

Genes and Molecular Abnormalities and www.omim.org

Research on gene misexpression and altered anatomical and functional development has enhanced general understanding about how limbs develop in utero.[3,18] This research has included intense work focusing on genotype–phenotype correlations that may have substantial clinical implications.

Online Mendelian Inheritance in Man (OMIM, www.omim.org) is a reliable and comprehensive, online compendium of human genes and genetic phenotypes that is updated daily and freely available to help clinicians, investigators, and other interested parties understand the vast evolving field of genetics and countless number of different phenotypes.

Numerous congenital deformities have a known genetic link. However, most possess variable inheritance patterns and breadths of expression. Healthcare

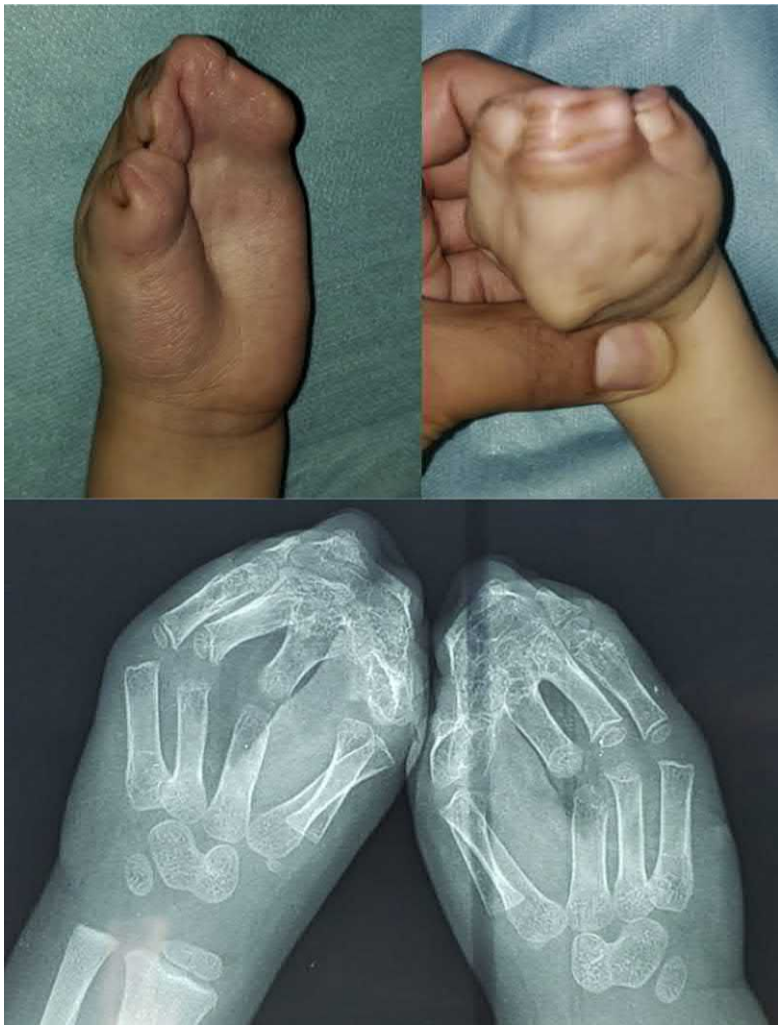

FIG. 1.8 Syndactyly in Apert syndrome is related to altered signaling of fibroblast growth factors resulting in abnormal BMP-mediated apoptosis.

providers who evaluate patients with hand deformities must have basic knowledge about congenital differences that are familial and potentially inherited versus those differences that are not inheritable. This understanding will justifiably recommend to families whether or not they should undergo evaluation by a clinical geneticist. Parents also require appropriate counseling pertaining to the spectrum of phenotypic expressions that can occur with a particular mutation. One misconception that frequently affects parents with a mild phenotype of a congenital disorder is that the extent and severity of their own disorder is a "worst-case scenario" for their child. This misperception can lead such parents to purse with pregnancy and birth, naïve to the possibility that their offspring may have a phenotypically much worse form of their difference. Their severely affected offspring renders those parents devastated, ill prepared, and engulfed with feelings of guilt. Appropriate genetic counseling referral can mitigate the misconception of variable phenotype with similar genotype.

Mutations that encode signaling proteins, receptor molecules, and transcription factors can alter normal limb arrangement, resulting in anomalies that range from almost imperceptible to complete limb absence. The number of molecularly identifiable congenital anomalies that practicing hand surgeons are seeing is steadily increasing. Other anomalies, though less well defined at a molecular level, have been mapped to specific chromosomal segments.[3] Table 1.2 lists examples of genes that encode transcription factors and exert some crucial level of control over upper-limb formation (Figs. 1.9 and 1.10).[33–36]

Intrauterine Diagnostics and Treating Upper-Limb Anomalies

Diagnosing congenital anomalies in utero can lead to parental counseling by a geneticist and/or by a surgeon who specializes in the anomaly identified and its treatment. In utero diagnosis may guide parental decisions on difficult personal and ethical questions, such as should gestation be terminated? In addition, in-utero procedures reevolving and may be considered based upon the certainty of diagnosis.[37] In addition, in utero diagnosis permits investigations to screen for associated anomalies, via further imaging studies or other procedures such as amniocentesis and chorionic villus sampling for fetal karyotyping and genetic analysis.[38]

To date, ultrasound remains the primary modality for fetal evaluations and/or monitor fetal development. Ultrasound also guides prenatal care and identifies fetal abnormalities (Fig. 1.11A).[39,40] Ultrasound-based prenatal diagnosis has improved substantially over the last several decades because of technological advances, improved image resolution, increased standardization of prenatal ultrasound protocols, and enhanced training of diagnosticians.[37] Prenatal ultrasound has the potential to identify isolated musculoskeletal abnormalities, including a broad range of hand and upper-limb condition including transverse and longitudinal deficiencies, syndactyly, polydactyly, clinodactyly, and clasped thumbs (Fig. 1.12).[40]

Both the American College of Radiology and American Institute of Ultrasound in Medicine recommend routine second-trimester screening with transabdominal ultrasound, between 18 and 22 weeks of

TABLE 1.2
Consequence of Mutation of Some Genes Encoding Transcription Factors Crucial for Limb Formation.

Gen	Syndrome	Limb anomaly	Inheritance
Hox	Synpolydactyly	Synpolydactyly (Fig. 1.9)	A.D.
	Hand-foot-genital syndrome	Short great toes and hipoplastic thumbs	
	Leri-Weill dyschondrosteosis	Madelung's deformity	
T-Box	Holt-Oram Sd (Tbx-5)	Radial deficiency	A.D.
	Ulnar-mamary Sd (Tbx-3)	Ulnar digits hipoplasia	
Cartilage-derived morphogenetic protein	Grebe chondrodysplasia	Brachidactily	A.R.
	Hunter-Thompson chondrodysplasia	Brachidactily (Fig. 1.10)	

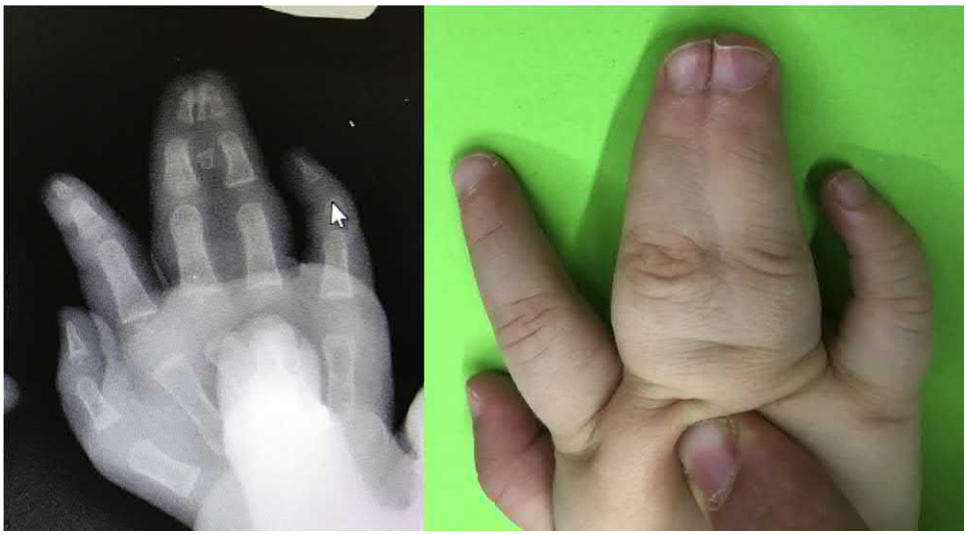

FIG. 1.9 Mutation in Hox genes can result in this autosomal dominant familial synpolydactyly.

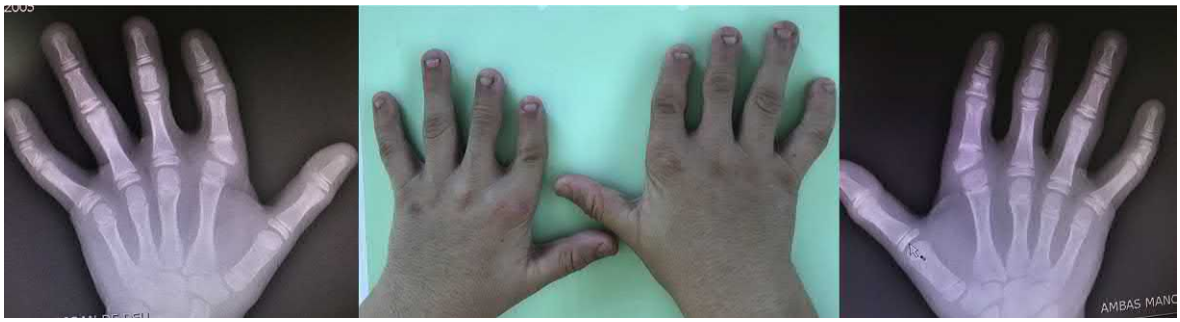

FIG. 1.10 This familial brachydactyly may be explained by an underlying mutation in cartilage-derived morphogenetic protein gen.

gestation, to confirm the fetuses gestational age, evaluate their intrauterine development, and screen for congenital anomalies. Current recommendations only require that the ultrasonographer document that all four extremities are present, although enhanced ultrasound imaging will likely change this basic recommendation. Currently, even with level-2 (''targeted'') ultrasound studies, further detailed assessment of the extremities is ''encouraged,'' but formalized standards are nonexistent.[41] In fact, despite improvements in ultrasound technology and techniques, its sensitivity detecting upper-limb anomalies remains low.[42]

At one tertiary level hospital, the postnatally confirmed sensitivity of prenatal ultrasound detecting upper-extremity anomalies was approximately 40%.[42] This sensitivity was lower when the anomalies were limited to the upper extremities (25% vs. 55%). Sensitivity for upper-limb anomalies was highest for conditions affecting the entire upper extremity (85%) and lowest for those affecting the digits alone (10%). Fetuses with limb-reduction defects, radial longitudinal deficiency, phocomelia, arthrogryposis, abnormal hand positioning, and cleft hand were more likely to be accurately diagnosed, because they were more likely to have an associated anomaly.

Several strategies can be utilized to enhance the sensitivity and accuracy of ultrasound imaging. One such technique is transvaginal ultrasound, which allows better visualization of the fetus and limbs.[40] In some high-risk groups, early risk assessment with ultrasound is performed between 11 and 14 weeks of gestation.

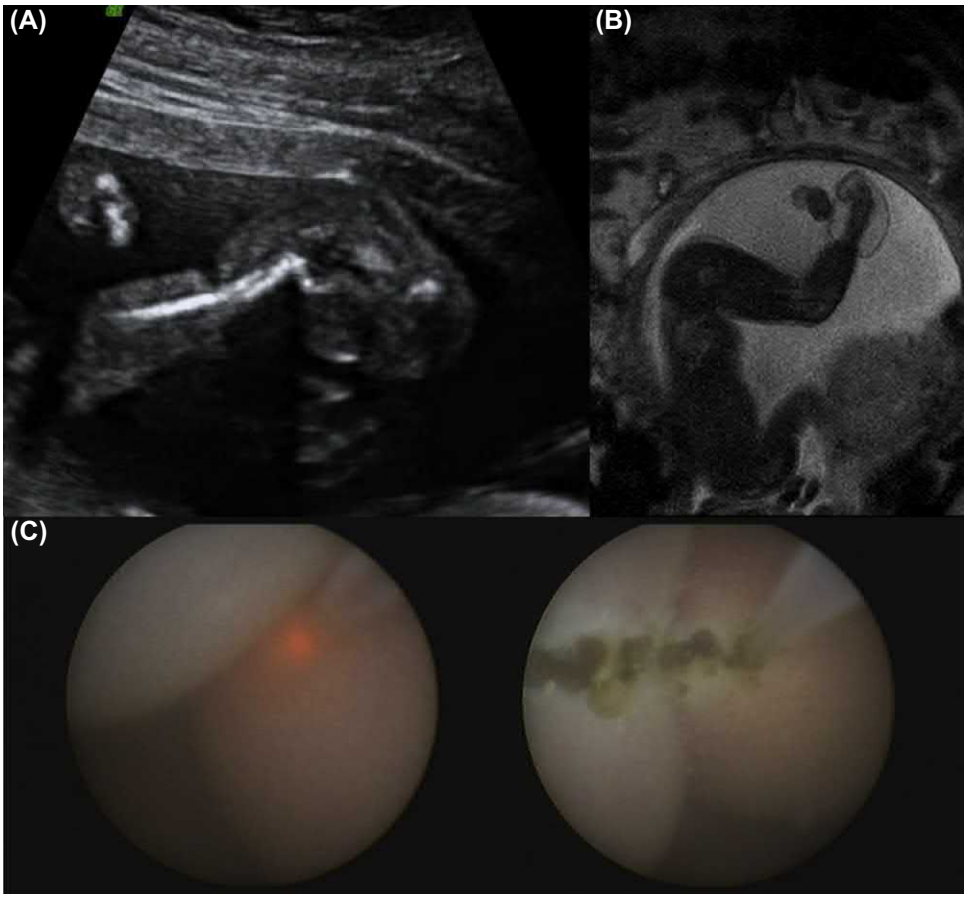

FIG. 1.11 Ultrasound showing severe leg constriction with markedly distal edema and risk of intrauterine amputation **(A)** Prenatal MRI confirming the ultrasound findings **(B)** Fetoscopic image of the leg constriction and longitudinal release with a Yag-Laser fiber **(C)**.

Within this time period and by employing transvaginal techniques, initial limb development can be assessed. Three-dimensional (3-D) ultrasound also improves the modality's diagnostic potential, allowing for the identification and characterization of more-complex anatomical structures. Authorities have advocated adopting 3D ultrasound as the imaging modality of choice for analyzing fetal limbs, particularly their hands (Fig. 1.13).[43]

The hand is best visualized by ultrasound during the late part of the first and early part of the second trimester. At this time, the fingers are large enough to be visualized and characteristically extended and abducted, facilitating the examiner's ability to discern anatomical alterations. Later in gestation, the hands often position in a clasped fist-like appearance obscuring hand anomalies. Additionally, the relative decrease in intrauterine space and amniotic fluid in later gestation limits fetal motion and the likelihood that the fetus will move into a position more suitable for detailed ultrasound assessment.

Magnetic resonance imaging (MRI) has particular advantages for evaluating neural axis, thoracic, and head or neck abnormalities, relative to the conventional ultrasound, because imaging the fetus with MRI is less dependent upon the presence of normal amniotic fluid volume, fetal position, and maternal body habitus.[44] However, due to artifacts caused by moving extremities, the accuracy of MRI in evaluating the hand and remaining upper limb remains to be determined (Fig. 1.11B).

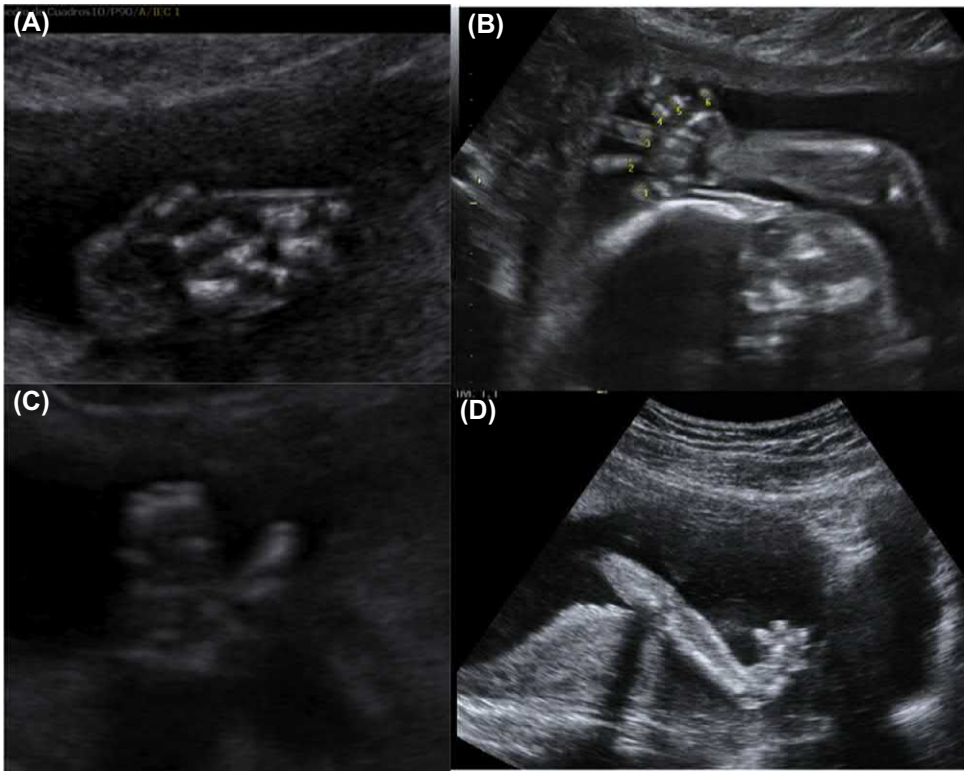

FIG. 1.12 A variety of congenital differences diagnosed by ultrasound at 21 weeks of gestational age: **(A)** complex syndactyly, **(B)** postaxial polydactyly, **(C)** ulnar deficiency with oligosyndactyly, and **(D)** radial clubhand with absent radius.

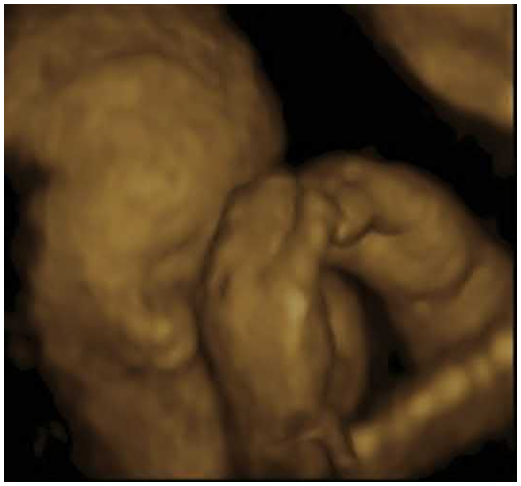

FIG. 1.13 27 weeks 3D ultrasound showing an Apert's hand.

PRENATAL TREATMENT

Fetal surgery is an emerging and established procedure. Intrauterine surgery was initially restricted to the treatment of life-threatening anomalies, given risks to both the mother and fetus (e.g., diaphragmatic hernia, twin–twin transfusion syndrome, giant teratomas, etc.).[45] As the prerequisite of anesthesia (maternal and fetal) and technology (fetal endoscopy) have improved, the risks have been reduced and the indications for intrauterine surgery have been extended to include nonlethal orthopedic conditions, including myelomeningocele and amniotic band syndrome.[46]

Currently, the only upper-limb indication for fetoscopic examination and treatment is the risk of limb amputation by an extremity amniotic band (Fig. 1.11C). The progressive strangulation of a limb by an intrauterine amniotic band leads to gradual worsening of the deformity and ultimate amputation. Prenatal band release arrests the progression of strangulation

and allows the fetal tissue's natural healing capacity to potentially restore the affected limb's normal morphology and function (Fig. 1.11C).[36] Fetal wound repair also occurs without scar formation, which yields the potential application to treating other congenital upper limb deformities (e.g., syndactyly). As future advances in technology and anesthesia decrease maternal–fetal risks further, the indications for prenatal interventions to correct congenital anomalies will likely expand.

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