

CHAPTER

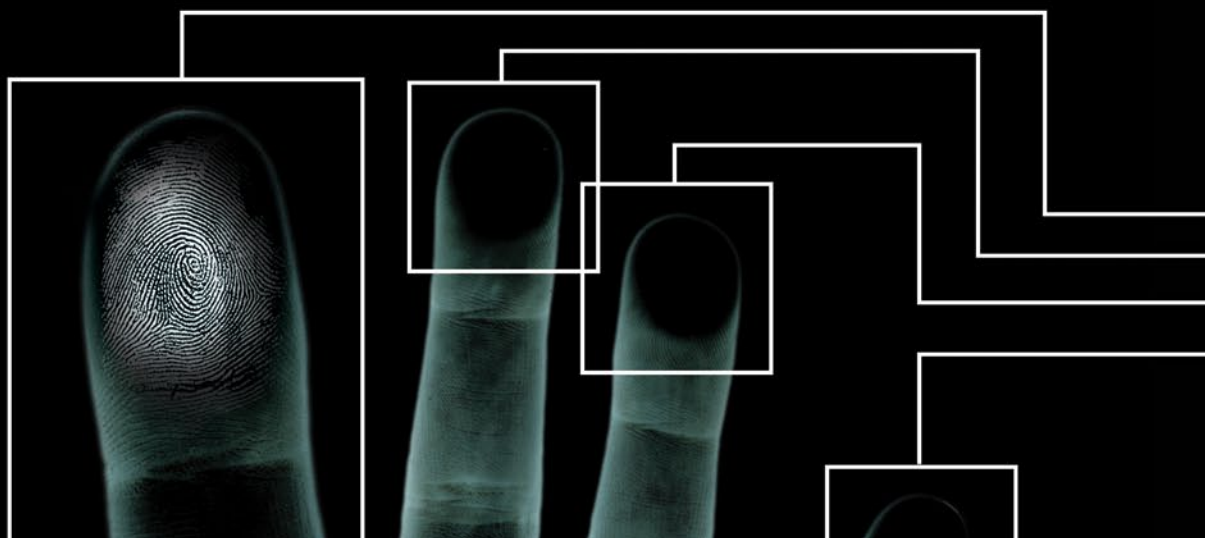


EMBRYOLOGY AND MORPHOLOGY OF FRICTION RIDGE SKIN

KASEY WERTHEIM

CONTENTS

3	3.1 Introduction	12	3.7 Pattern Formation
4	3.2 Embryology: Establishing Uniqueness and Pattern Formation in the Friction Ridge Skin	18	3.8 Genetics
5	3.3 Limb Development	21	3.9 Uniqueness: Developmental Noise
7	3.4 Differentiation of the Friction Ridge Skin	22	3.10 Summary: Keys to Uniqueness and Pattern Formation
8	3.5 Primary Ridge Formation	22	3.11 Reviewers
11	3.6 Secondary Ridge Formation	24	3.12 References





CHAPTER 3

EMBRYOLOGY AND MORPHOLOGY OF FRICTION RIDGE SKIN

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3.1 Introduction

Friction ridge skin has unique features that persist from before birth until decomposition after death. Upon contact with a surface, the unique features of friction ridge skin may leave an impression of corresponding unique details. Two impressions can be analyzed, compared, and evaluated, and if sufficient quality and quantity of detail is present (or lacking) in a corresponding area of both impressions, a competent examiner can effect an individualization or exclusion (identify or exclude an individual). The analysis, comparison, evaluation, and verification (ACE-V) methodology, combined with the philosophy of quantitative–qualitative examinations, provide the framework for practical application of the friction ridge examination discipline. But at the heart of the discipline is the fundamental principle that allows for conclusive determinations: the source of the impression, friction ridge skin, is unique and persistent.

Empirical data collected in the medical and forensic communities continues to validate the premises of uniqueness and persistence. One hundred years of observations and statistical studies have provided critical supporting documentation of these premises. Detailed explanations of the reasons behind uniqueness and persistence are found in specific references that address very small facets of the underlying biology of friction ridge skin. This chapter brings together these references under one umbrella for the latent print examiner to use as a reference in understanding why friction ridge skin is unique and persistent.

The basis of persistence is found in morphology and physiology; the epidermis faithfully reproduces the three-dimensional ridges due to physical attachments and constant regulation of cell proliferation and differentiation. But, the basis of uniqueness lies in embryology; the unique features of the skin are established between approximately 10.5 and 16 weeks estimated gestational age (EGA) due to developmental noise.

3.2 Embryology: Establishing Uniqueness and Pattern Formation in the Friction Ridge Skin

3.2.1 Introduction to Embryology

The uniqueness of friction ridge skin falls under the larger umbrella of biological uniqueness. No two portions of any living organism are exactly alike. The intrinsic and extrinsic factors that affect the development of any individual organ, such as human skin, are impossible to duplicate, even in very small areas. The uniqueness of skin can be traced back to the late embryological and early fetal development periods.

3.2.2 Early Embryological Development: 0–2 Weeks EGA (Raven and Johnson, 1992, pp 1158–1159)

The process of embryological development begins with fertilization of the egg and continues through a period of rapid cell division called “cleavage.” In mammalian eggs, an inner cell mass is concentrated at one pole, causing patterned alterations during cleavage. Although egg cells contain many different substances that act as genetic signals during early embryological development, these substances are not distributed uniformly. Instead, different substances tend to be clustered at specific sites within the growing embryo. During growth, signal substances are partitioned into different daughter cells, endowing them with distinct developmental instructions. In this manner, the embryo is prepatterned to continue developing with unique cell orientation.

3.2.3 Late Embryological Development: 3–8 Weeks EGA (Raven and Johnson, 1992, pp 1160–1164)

The first visible results of pre patterning can be seen immediately after completion of the cleavage divisions as different genes are activated. Certain groups of cells move inward toward the center of the sphere in a carefully orchestrated migration called “gastrulation.” This process forms the primary tissue distinctions between ectoderm, endoderm, and mesoderm. The ectoderm will go on to form epidermis, including friction ridge skin; the mesoderm will form the connective tissue of the dermis, as well as muscle and elements of the vascular system; and the endoderm goes on to form the organs.

Once specialized, the three primary cell types begin their development into tissue and organs. The process of tissue differentiation begins with neurulation, or the formation of the notochord (the precursor to the spinal cord and brain) as well as the neural crest (the precursor to much of the embryo’s nervous system). Segmented blocks of tissue that become muscles, vertebrae, and connective tissue form on either side of the notochord. The remainder of the mesoderm moves out and around the inner endoderm, forming a hollow chamber that will ultimately become the lining of the stomach and intestines.

During late embryological development, the embryo undergoes “morphogenesis”; or the formation of shape. Limbs rapidly develop from about 4 weeks EGA, and the arms, legs, knees, elbows, fingers, and toes can all be seen in the second month. During this time, the hand changes from a paddlelike form to an adult form, including the formation of the fingers and rotation of the thumb. Also during this time, swellings of mesenchyme called “volar pads” appear on the palms of the hands and soles of the feet. Within the body cavity, the major organs such as the liver, pancreas, and gall bladder become visible. By the end of week 8, the embryo has grown to about 25 millimeters in length and weighs about 1 gram.

3.2.4 Fetal Growth: 9–12 Weeks EGA

During the third month, the embryo’s nervous system and sense organs develop, and the arms and legs begin to move. Primitive reflexes such as sucking are noticed, and early facial expressions can be visualized. Friction ridges begin to form at about 10.5 weeks EGA and continue to mature in depth as the embryo passes into the second trimester. From this point on, the development of the embryo is essentially complete, and further maturation is referred to as fetal growth rather than embryonic development.

3.2.5 Second Trimester

The second trimester is marked by significant growth to 175 millimeters and about 225 grams. Bone growth is very active, and the body becomes covered with fine hair called lanugo, which will be lost later in development. As the placenta reaches full development, it secretes numerous hormones essential to support fetal bone growth and energy. Volar pads regress and friction ridges grow until about 16 weeks EGA, when the minutiae become set.

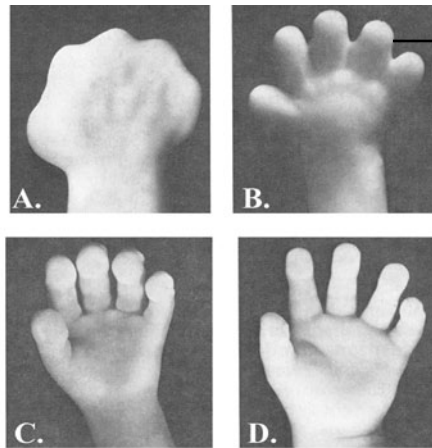


FIGURE 3-1

Growth of the hand progresses from (A) a paddle-like form (magnification = 19.5 X), (B) continues as the fingers separate (magnification = 17.3 X) and (C) the volar pads become prominent (magnification = 7.7 X), and (D) achieves infantlike appearance by 8 weeks EGA (magnification = 4.2 X). (Reprinted with permission from Cummins (1929).)

Sweat glands mature, and the epidermal–dermal ridge system continues to mature and grow in size. By the end of the second trimester, sweat ducts and pores appear along epidermal ridges, and the fetus begins to undergo even more rapid growth.

3.2.6 Third Trimester

In the third trimester, the fetus doubles in weight several times. Fueled by the mother’s bloodstream, new brain cells and nerve tracts actively form. Neurological growth continues long after birth, but most of the essential development has already taken place in the first and second trimesters. The third trimester is mainly a period for protected growth.

3.3 Limb Development

3.3.1 Hand Development

During the initial phases of formation, the hand undergoes significant changes in topography. Until approximately 5–6 weeks EGA, the hand appears as a flat, paddlelike structure with small protrusions of tissue that will become fingers. From 6 to 7 weeks EGA, these finger protrusions in the hand plate begin to form muscle and cartilage that will become bone at later stages of hand growth (Figure 3-1).

From 7 to 8 weeks EGA, the fingers begin to separate and the bone begins to “ossify” or harden. By 8 weeks EGA, the joints begin to form between the bones of the hand, and the external hand morphology appears similar in proportion to that of an infant.

3.3.2 Volar Pad Development

Volar pads (Figure 3-2) are transient swellings of tissue called mesenchyme under the epidermis on the palmar surface of the hands and soles of the feet of the human fetus (Figure 3-3).

The interdigital pads appear first, around 6 weeks EGA, followed closely in time by the thenar and hypothenar pads. At approximately 7–8 weeks EGA, the volar pads begin to develop on the fingertips, starting with the thumb and progressing toward the little finger in the same radio-ulnar gradient that ridge formation will follow. Also at about 8 weeks EGA, the thenar crease begins to form in the palm, followed by the flexion creases in the fingers at around 9 weeks EGA (Kimura, 1991).

3.3.3 Volar Pad “Regression”

The pads remain well rounded during their rapid growth around 9–10 weeks EGA, after which they begin to demonstrate some individual variation in both shape and position (Babler, 1987; Burdi et al., 1979; Cummins, 1926, 1929). During the period from 8 to 10 weeks EGA, thumb rotation is achieved (Lacroix et al., 1984, p 131). Also at about 10 weeks EGA, the flexion creases of the toes begin formation, followed at about 11 weeks EGA by the distal transverse flexion crease in the palm, and at about 13 weeks EGA by the proximal transverse flexion crease in the palm (Kimura, 1991).

As a result of the volar pads’ slowing growth, their contour becomes progressively less distinct on the more rapidly growing surface (Figure 3-4). This process has been defined as “regression” (Lacroix et al., 1984, pp 131–133),

FIGURE 3-2

A low-power scanning electron microscope view of a fetal hand displaying prominent digital and palmar volar pads. (Reprinted with permission from Carlson (1999), p 152.)



FIGURE 3-3

Normally, 11 volar pads develop and regress on each limb (one on each digit and six on the larger surface of the palm or sole). The hypothenar pad of the palm is divided into distal (Hd) and proximal (Hp) portions. The first (I) interdigital volar pad is also divided into two portions, making a total of 13 potential elevations on each surface. On plantar surfaces, the proximal portions of the hypothenar pad (Hp) and the thenar pad (Thp) are absent, leaving 11 distinct plantar elevations. (Reprinted with permission from Cummins (1929), p 114.)

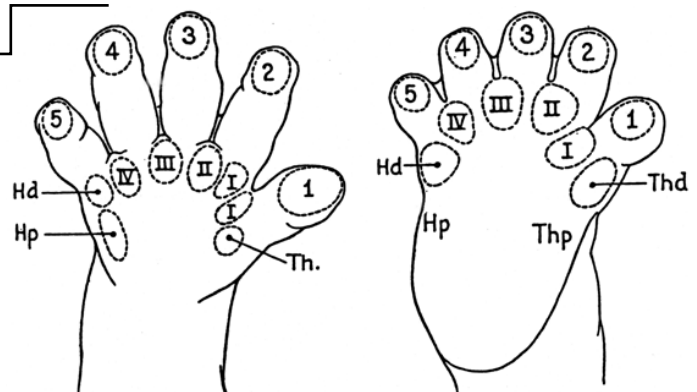
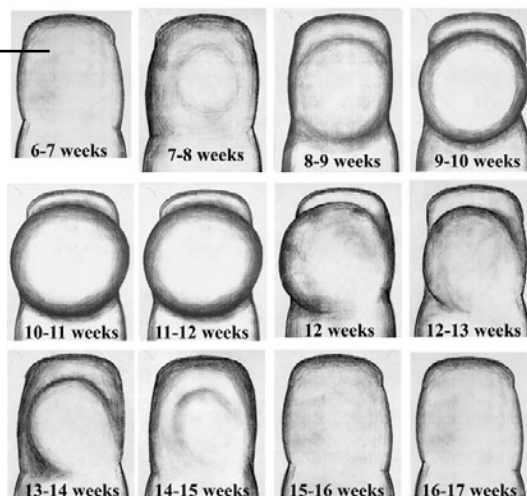


FIGURE 3-4

Drawings that represent a volar pad from initial formation until complete regression, excluding growth of the size of the finger. Actual EGA values are highly variable and are included only as approximations in this figure. (Reprinted with permission from Wertheim and Maceo (2002), p 61.)



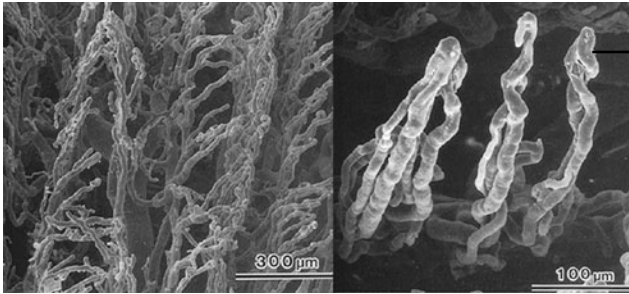


FIGURE 3–5

Scanning electron micrograph of a resin cast of the fine vasculature of the finger of an 85-year-old man shows a complex pattern of capillary loops in dermal ridges. Approximate magnification = 150 X (left) and 700 X (right). (Reprinted with permission from Montagna et al. (1992).)

but it is important to understand that the pad is not actually shrinking; rather, the volar pads are overtaken by the faster growth of the larger surrounding surface. The volar pads of the palm begin to regress as early as 11 weeks EGA, followed closely by the volar pads of the fingers. By 16 weeks EGA, volar pads have completely merged with the contours of the fingers, palms, and soles of the feet (Cummins, 1929, p 117).

3.4 Differentiation of the Friction Ridge Skin

3.4.1 Development of the Epidermis

The primitive epidermis is established at approximately 1 week EGA, when ectoderm and endoderm are separately defined. A second layer of epidermis forms at about 4–5 weeks EGA. The outermost of the three layers is the periderm. The middle layer, which is the actual epidermis, is composed of basal keratinocytes (named because of the keratins these cells manufacture). At about 8 weeks EGA, the basal cells between the epidermis and the dermis begin to consistently divide and give rise to daughter cells that move vertically to form the first of the intermediate cell layers (Holbrook, 1991b, p 64). At this point, the embryonic epidermis is three to four cell layers thick, but it is still smooth on its outer and inner surfaces. Keratinocytes are tightly bound to each other by desmosomes, and the cells of the basal layer are attached to the basement membrane by hemidesmosomes (Holbrook, 1991a, p 5).

3.4.2 Development of the Dermis

The first dermal components to originate from the mesoderm are fibroblasts. These irregular branching cells secrete proteins into the matrix between cells. Fibroblasts synthesize the structural (collagen and elastic)

components that form the connective tissue matrix of the dermis. During the period 4–8 weeks EGA, many of the dermal structures begin formation. Elastic fibers first appear around 5 weeks EGA at the ultrastructural level in small bundles of 20 or fewer fibrils (Holbrook, 1991b, pp 64–101). Nerve development occurs in different stages from 6 weeks EGA onwards. Neurovascular bundles and axons with growth cones are seen in the developing dermis as early as 6 weeks EGA (Moore and Munger, 1989, pp 128–130). In fact, axons can be traced to the superficial levels of the dermis, and in some cases they almost about the basal lamina of the epidermis. By 9 weeks EGA, innervation (the appearance of nerve endings) of the epidermis has begun to occur, although there are some Merkel cells in the epidermis that are not yet associated with axons. In embryos older than 10 weeks EGA, Merkel cells are predominant in the developing epidermis, and their related axons and neurofilaments are present in the dermis (Moore and Munger, 1989, p 127; Smith and Holbrook, 1986).

The dermis becomes distinguishable from deeper subcutaneous tissue due largely to a horizontal network of developing blood vessels. From 8 to 12 weeks EGA, vessels organize from dermal mesenchyme and bring much-needed oxygen and hormones to the underside of the developing epidermis. Unlike other epidermal structures, blood vessels continue to alter with aging, as some capillary loops are lost and new ones arise from the interpapillary network. This continues into late adulthood (Figure 3–5) (Smith and Holbrook, 1986).

A second vascular network forms deep in the reticular dermis by about 12 weeks EGA. Unlike the developing primary ridges, the vascular network is not a permanent structure. There is significant reorganization of capillary beds during the period 8–20 weeks EGA to keep pace with skin growth; even after birth, microcirculation continues to form and remodel (Holbrook, 1991b, p 100; Smith and Holbrook, 1986).

FIGURE 3–6

A reconstruction of the first three-dimensional undulations that occur on the underside of the fetal volar epidermis at the epidermal–dermal junction. (Artwork by Brandon Smithson. Re-drawn from Hale (1952), p 152.)

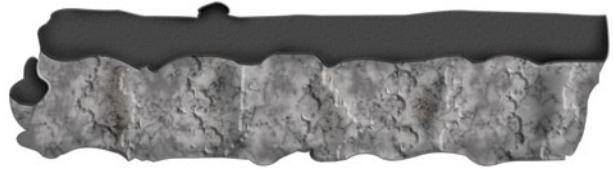
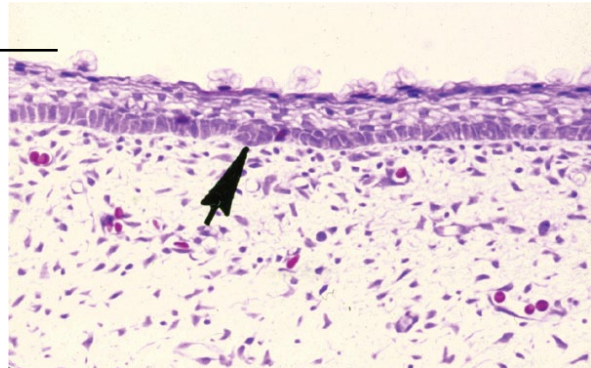


FIGURE 3–7

A histological cross section of 10.5-week EGA fetal volar skin at the onset of rapid localized cellular proliferation. (Image provided by William Babler.)



3.5 Primary Ridge Formation

3.5.1 Initiation of Primary Ridge Formation

At around 10–10.5 weeks EGA, basal cells of the epidermis begin to divide rapidly (Babler, 1991, p 98; Holbrook and Odland, 1975, p 17). As volar epidermal cells divide, shallow “ledges” (Hale, 1952) can be seen on the bottom of the epidermis. These ledges delineate the overall patterns that will become permanently established on the volar surfaces several weeks later (Babler, 1991, p 101; Evatt, 1906). Primary ridges are the first visual evidence of interaction between the dermis and epidermis and are first seen forming as continuous ridges (Figure 3–6).

The prevailing theory of events before the visualization of primary ridge structure involves centers of active cell proliferation (Figure 3–7), which will become the centers of sweat gland development (Babler, 1991, p 98).

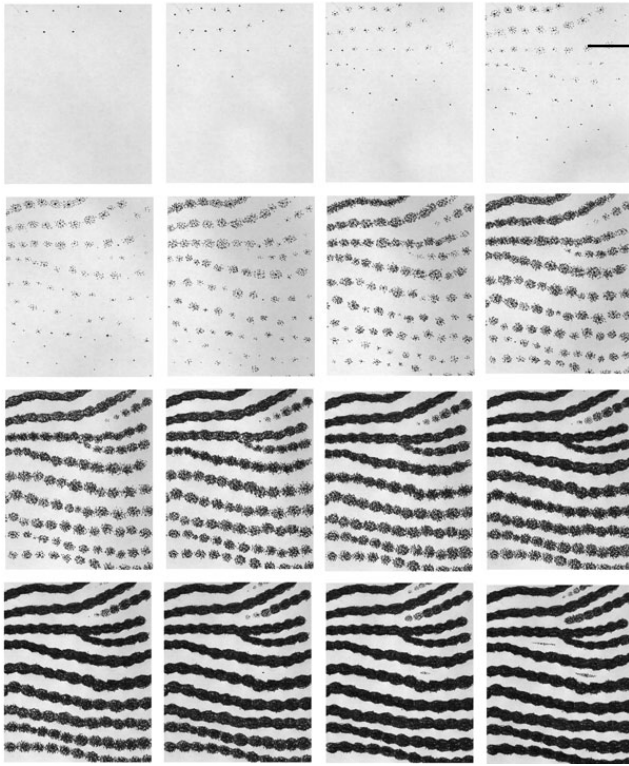
According to this theory, the “units” of rapidly multiplying cells increase in diameter, somewhat randomly, growing into one another (Figure 3–8) along lines of relief perpendicular to the direction of compression.

Furthermore, according to this theory, as the series of localized proliferations “fuse” together, the resulting linear ridges of rapidly dividing epidermal cells fold into

the dermis, creating the first visible ridge structure at the epidermal–dermal junction (Ashbaugh, 1999, p 79). Another plausible theory is that developing nerves may interact with epidermal cells to stimulate clustered interactions that blend together in the early stages of ridge development.

At the time of embryonic friction ridge formation, the central nervous and cardiovascular systems are undergoing a critical period of development (Hirsch, 1964). Researchers have reported innervation at the sites of ridge formation immediately preceding the appearance of friction ridges and suggest that innervation could be the trigger mechanism for the onset of proliferation (Bonnievie, 1924; Dell and Munger, 1986; Moore and Munger, 1989). Several researchers even postulate that the patterning of the capillary–nerve pairs at the junction of the epidermis and the dermis is the direct cause of primary ridge alignment (Dell and Munger, 1986; Hirsch and Schweichel, 1973; Moore and Munger, 1989; Morohunfolo et al., 1992).

Early research on pattern distribution established “developmental fields,” or groupings of fingers on which patterns had a greater tendency to be similar (Meier, 1981; Roberts, 1982; Siervogel et al., 1978). Later discoveries confirmed the neurological relation of spinal cord sections C–6, C–7, and C–8 to innervation of the fingers (Heimer, 1995). Specifically, Kahn and colleagues (2001) reported that a

**FIGURE 3–8**

These drawings represent the theory that just before ridge formation, localized cellular proliferations grow together into what will appear as ridges at around 10.5 weeks EGA. (Reprinted with permission from Wertheim and Maceo (2002), p 49.)

large ridge-count difference between C-8-controlled fingers 4 and 5 may predict a larger waist-to-thigh ratio and, therefore, an increased risk of some major chronic diseases such as heart disease, cancer, and diabetes. Other interesting hypotheses have been published regarding the connection between innervation and friction ridge patterning, but the main consideration for the purposes of friction ridge formation is that specific parts of the nervous system are undergoing development at the same time that ridges begin to appear on the surface of the hands.

The presence of nerves and capillaries in the dermis before friction ridge formation may be necessary for friction ridge proliferation. It would seem that complex simultaneous productions such as friction ridge formation would benefit from being in communication with the central nervous system or the endocrine and exocrine (hormone) systems (Smith and Holbrook, 1986). However, it is doubtful that nerves or capillaries independently establish a map that directly determines the flow of the developing friction ridges. It seems more likely that the alignment of the nerves and capillaries is directed by the same stresses and strains on the developing hand that establish ridge alignment (Babler, 1999; Smith and Holbrook, 1986). It is well recognized in cell biology that physical pressure on a cellular system can trigger electrochemical changes within

that system. Merkel cells occupy the epidermis just prior to innervation along those pathways (Holbrook, 1991a), suggesting that even before ridge formation, the stresses created by the different growth rates of the dermis and epidermis are causing differential cell growth along invisible lines that already delineate pattern characteristics (Loesch, 1973). Regardless of the trigger mechanism controlling the onset of the first primary ridge proliferations, the propagation of primary ridges rapidly continues.

3.5.2 Propagation of Primary Ridge Formation

Primary ridges mature and extend deeper into the dermis (Figure 3–9) for a period of approximately 5.5 weeks, from their inception at 10.5 weeks EGA until about 16 weeks EGA. The cell growth during this phase of development is along the primary ridge, in what has been labeled the “proliferative compartment.” The proliferative compartment encompasses basal and some suprabasal cells, ultimately governed by stem cells, and is responsible for new skin cell production of the basal layer of skin (Lavker and Sun, 1983).

3.5.3 Minutiae Formation

Although the exact mechanisms for formation of minutiae are unclear, the separate accounts of many researchers



FIGURE 3-9

Histological cross section of fetal volar skin between 10.5 and 16 weeks EGA. During this time, primary ridges (as marked by the arrow) increase in depth and breadth. (Image provided by William Babler.)

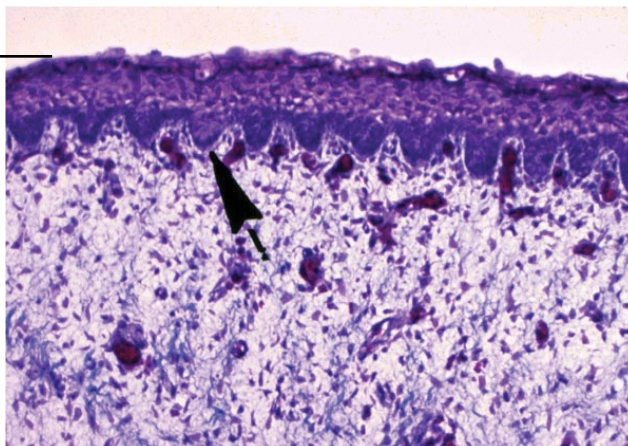
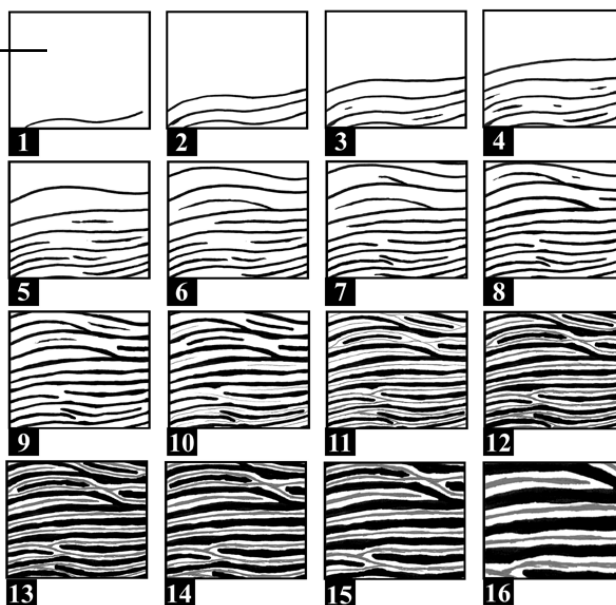


FIGURE 3-10

Drawings that illustrate the theoretical formation of minutiae arising from expansion of the volar surface during the critical stage (frames 1–10) and continuing to increase in size after secondary ridge formation (frames 11–16). (Reprinted with permission from Wertheim and Maceo (2002), p 51.)

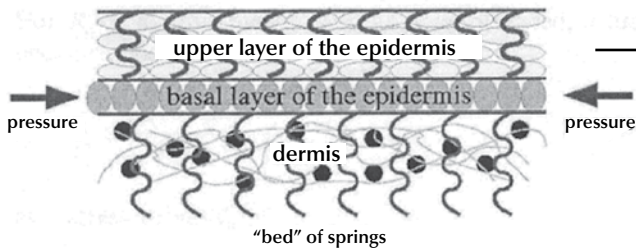


who have examined fetal tissue allow for a fairly accurate reconstruction of the morphogenesis of friction ridges in successive stages of the development process. Figure 3-10 illustrates the process of minutiae formation as hypothesized by a general consensus of the literature.

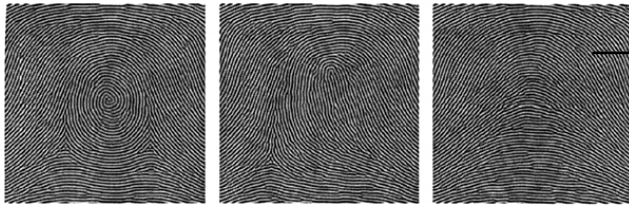
Many events happen during this rapid period of primary ridge growth. The finger rapidly expands, new primary ridges form across the finger, and the existing primary ridges begin to separate because of growth of the digit. As existing ridges separate, the tendency of the surface to be continually ridged creates a demand for new ridges. Hale reports that new ridges pull away from existing primary ridges to fill in these gaps, creating bifurcations by

mechanical separation. Ending ridges form when a developing ridge becomes sandwiched between two established ridges. According to this theory, “fusion between adjacent ridges [which have already formed] seems improbable, although there is no evidence for or against this process” (Hale, 1952, p 167).

Other models explain ridge detail in nature as a chemical reaction-suppression scheme in which morphogens react and diffuse through cells, causing spatial patterns (Murray, 1988, p 80). According to these models, hormones circulate first through newly formed capillaries just before ridge formation in the epidermis, offering another potential factor in the genesis of ridge formation (Smith and Holbrook, 1986).

**FIGURE 3–11**

A drawing that represents the state of the epidermal–dermal boundary just before ridge formation. (Reprinted with permission from Kücken and Newell (2005), p 74.)

**FIGURE 3–12**

Computer simulations demonstrating that bounded stress fields across a three-dimensional spherical surface produce fingerprintlike patterns. (Reprinted with permission from Kücken and Newell (2005), p. 79.)

A recent model of the process of friction ridge morphogenesis has been likened to mechanical instability (Kücken and Newell, 2005). Building on the folding hypothesis of Kollmann (1883) and Bonnevie (1924), Kücken and Newell (2005) consider the basal layer as “an overdamped elastic sheet trapped between the neighboring tissues of the intermediate epidermis layer and the dermis,” which they mathematically model as “beds of weakly nonlinear springs” (Figure 3–11).

Their computer program models the results of forcing enough compressive stress to cause a buckling instability on a virtual three-dimensional elastic sheet constrained by fixed boundaries on two sides. The resulting ridge patterns are similar to all three major fingerprint pattern types oriented by the upper fixed boundary of the nailbed and the lower fixed boundary of the distal interphalangeal flexion crease (Figure 3–12).

Regardless of the exact mechanism of minutiae formation (mechanical or static; fusion or chemical), the exact location of any particular bifurcation or ridge ending within the developing ridge field is governed by a random series of infinitely interdependent forces acting across that particular area of skin at that critical moment. Slight differences in the mechanical stress, physiological environment, or variation in the timing of development could significantly affect the location of minutiae in that area of skin.

3.6 Secondary Ridge Formation

3.6.1 Initiation of Secondary Ridge Formation

By 15 weeks EGA, the primary ridges are experiencing growth in two directions: the downward penetration of the sweat glands and the upward push of new cell growth. Generally, the entire volar surface is ridged by 15 weeks EGA. Okajima (1982) shows a fully ridged palm of a 14-week-old fetus (Figure 3–13).

Between 15 and 17 weeks EGA, secondary ridges appear between the primary ridges on the underside of the epidermis (Babler, 1991, p 98). Secondary ridges are also cell proliferations resulting in downfolds of the basal epidermis. At this time in fetal development, the randomly located minutiae within the friction ridge pattern become permanently set (Hale, 1952, pp 159–160), marking the end of new primary ridge formation (Figure 3–14) (Babler, 1990, p 54).

3.6.2 Propagation of Secondary Ridge Formation

As the secondary ridges form downward and increase the surface area of attachment to the dermis, the primary ridges are pushing cells toward the surface to keep pace with the growing hand. These two forces, in addition to cell adhesion, cause infolding of the epidermal layers above the attachment site of the secondary ridges (Hale, 1952). As

FIGURE 3-13

Image of a 14-week EGA fetal palm stained with toluidine blue. (Reprinted with permission from Okajima (1982), p 185 (no magnification given).)

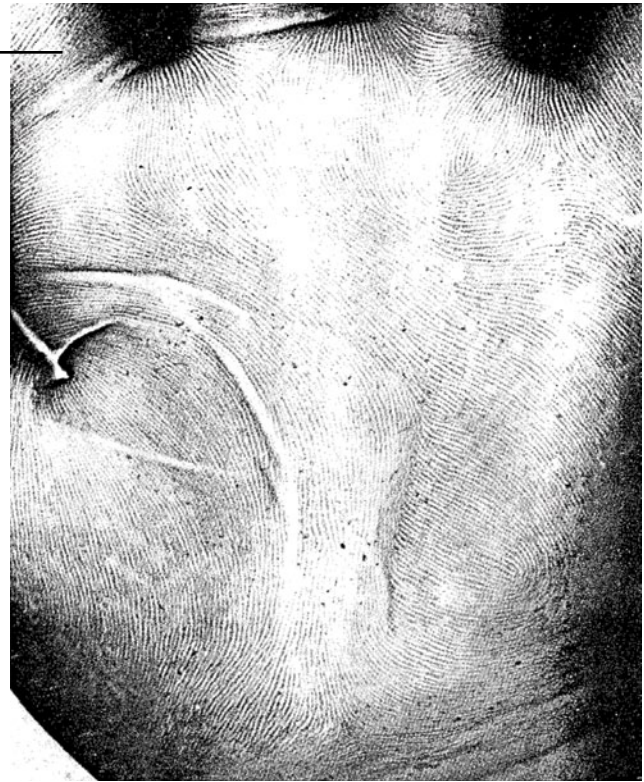
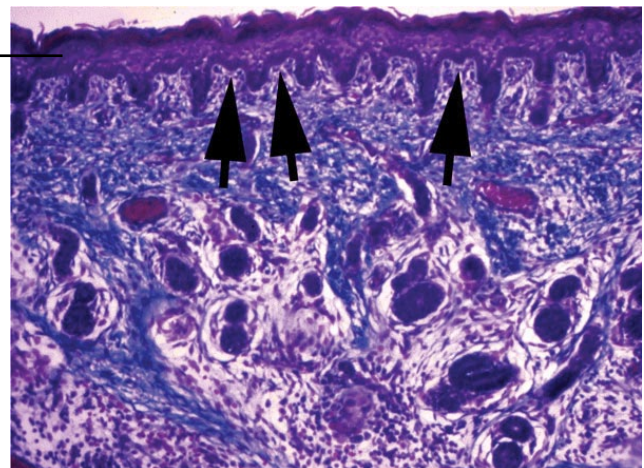


FIGURE 3-14

A histological cross section of fetal volar skin representing the onset of secondary ridge formation between maturing primary ridges (as marked by the arrows) at about 16 weeks EGA. (Image provided by William Babler.)



secondary ridges continue to mature from 16 to 24 weeks EGA, this structure is progressively mirrored on the surface of friction ridge skin as the furrows (Burd et al., 1979, pp 25-38) (Figure 3-15).

3.6.3 Formation of Dermal Papillae

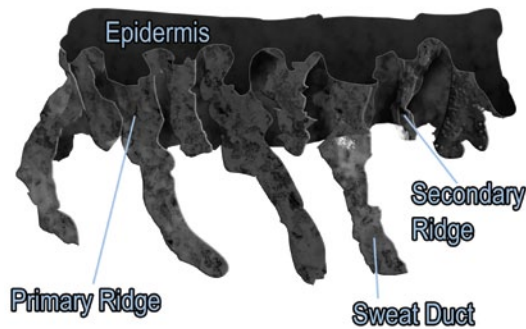
Dermal papillae are the remnants of dermis left projecting upward into the epidermis when anastomoses bridge primary and secondary ridges (Figures 3-16 and 3-17). They begin to form at approximately 23 weeks EGA (Okajima,

1975) and continue to become more complex throughout fetal formation and even into adulthood (Chacko and Vaidya, 1968; Misumi and Akiyoshi, 1984).

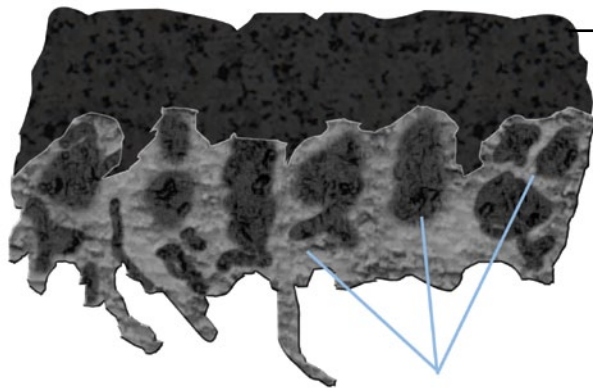
3.7 Pattern Formation

3.7.1 Shape of the Volar Pad

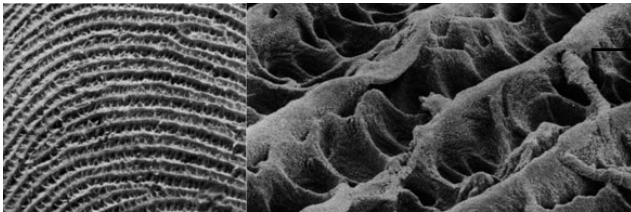
It is observed throughout the physical world that ridges tend to align perpendicularly to physical compression across a surface (Figure 3-18).

**FIGURE 3–15**

A reconstruction of the secondary ridges continuing to form on the underside of the fetal volar epidermis between existing primary ridges with sweat ducts. (Artwork by Brandon Smithson. Re-drawn from Hale (1952), p 153.)

**FIGURE 3–16**

A reconstruction of the underside of the epidermis of fetal volar skin that represents anastomoses bridging primary and secondary ridges and cordoning off sections of dermis that remain protruding upward as “dermal papillae” or “papillae pegs”. (Artwork by Brandon Smithson. Re-drawn from Hale (1952), p 154.)

**FIGURE 3–17**

A scanning electron microscope view of the complex understructure of human epidermis as the dermis has been removed (inverted). Magnification (approximate) = 8 X (left) and 80 X (right). (Reprinted with permission from Montagna and Parakkal (1974), pp 34–35.)

Ridges also form transversely to the lines of growth stress in friction skin. The predominant growth of the hand is longitudinal (lengthwise) and ridges typically cover the volar surface transversely (side to side). This phenomenon is seen in the ridge flow across the phalanges.

Bonnevie first hypothesized in 1924 that volar pad height affects friction ridge patterns (Bonnevie, 1924, p 4). Disruptions in the shape of the volar surfaces of the hands and feet create stresses in directions other than longitudinal. The ridges flow in a complex manner across these three-dimensional structures.

The distinction between the size, height, and shape of the volar pad, and the effects of differences in each of these elements on a friction ridge pattern, is a difficult topic to study (Chakraborty, 1991; Jamison, 1990; Mavalwala et al., 1991). However, almost all research points to the conclusion that the shape of the volar pad influences the stress across the skin that directs ridge alignment. One contrary viewpoint to this conclusion exists. In 1980, Andre G. de Wilde proposed a theory that pattern formation is directed much earlier in fetal life, before volar pads form, while the hand is still in a paddlelike shape (De Wilde, 1980). He

FIGURE 3-18

When tension is applied across the top of a semiflexible membrane, forces of compression occur on the bottom. The natural relief of compression forces creates ridges forming transversely to the stress. (Reprinted with permission from Wertheim and Maceo (2002), p 57.)

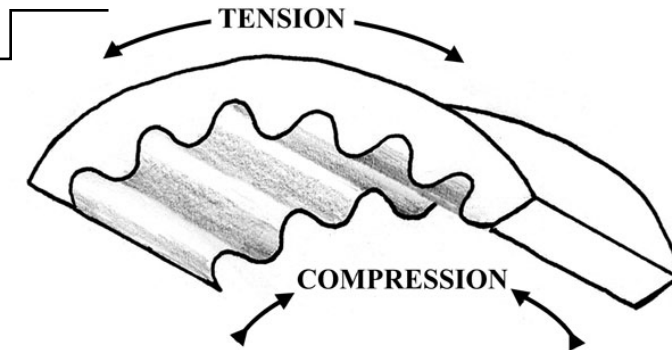
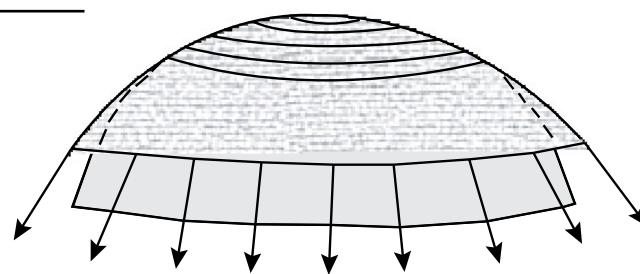


FIGURE 3-19

The loxodrome results when an elastic film is stretched evenly over a hemisphere. Ridges form concentrically around the apex of the membrane disruption. The mathematical formula for this pattern can be found in tensor calculus, a field that offers much promise in predicting ridge formation across volar surfaces. (Reprinted with permission from Wertheim and Maceo (2002), p 62.)



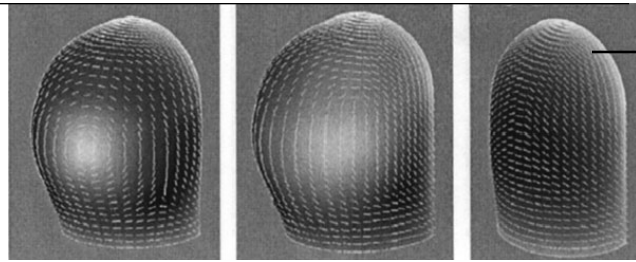
hypothesized that ridges direct the size and shape of the volar pads. However, no other theoretical or empirical support for this theory could be found. All other research indicates that friction ridges align according to volar pad shape and symmetry at approximately 10.5 weeks EGA.

3.71.1 Symmetrical Volar Pad. The growth and regression of the volar pads produce variable physical stresses across the volar surface that affect the alignment of the ridges as the ridges first begin to form. Whether ridge flow will conform to a whorl or a loop pattern appears highly correlated with the symmetry of the stress across the surface of the finger. If the volar pad and other elements of finger growth are symmetrical during the onset of primary ridge formation, then a symmetrical pattern (a whorl or an arch) will result. Ridges will form concentrically around the apex of a volar pad that is high and round when the generating layer of friction ridge skin first begins to rapidly produce skin cells. The ridge flow from a symmetrical volar pad conforms to the navigational pattern of the loxodrome (Figure 3-19) (Mulvihill and Smith, 1969; Elie, 1987). Research in both the medical and mathematical fields suggests that this same physical model applies across the entire volar surface of the hands and feet (Cummins, 1926, 1929; Loesch, 1973; Penrose and O’Hara, 1973).

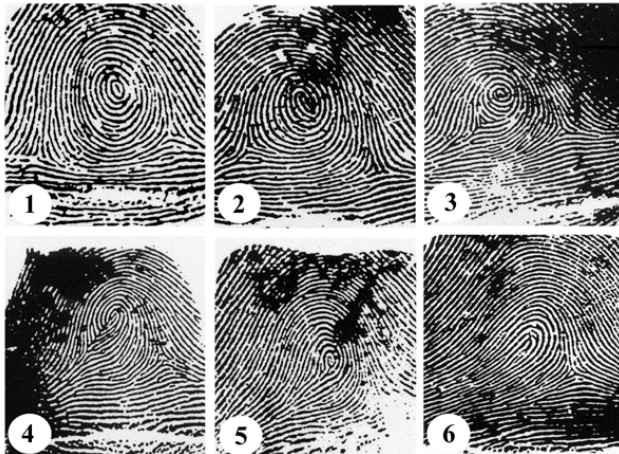
3.71.2 Asymmetrical Volar Pad. The degree of asymmetry of the finger volar pad when ridges first begin to form determines the asymmetry of the pattern type. Many researchers have reported that asymmetrical “leaning” pads form looping patterns and that low or absent volar pads form arch patterns (Cummins, 1926, p 138). Babler perhaps conducted the most scientific validation of the correlation between pad symmetry and pattern type through extensive examination of fetal abortuses (Babler, 1978).

Cummins published an extensive analysis of malformed hands to demonstrate the effect of the growth and topology of the hand on ridge direction (Cummins, 1926). Cummins also concluded that ridge direction is established by the contours of the hands and feet at the time of ridge formation. Penrose examined friction ridge pattern formation from a mathematical perspective, arriving at the same conclusion (Loesch, 1973; Penrose and Plomley, 1969). More recently, Kücken and Newell (2005) modeled stress fields across bounded three-dimensional, spherical virtual surfaces, creating relatively accurate-appearing ridge patterns (Figure 3-20).

If the volar pad and other growth factors of the finger are asymmetrical during the critical stage, then that same

**FIGURE 3–20**

Computer models demonstrating directional field points (tic marks) stretched in the direction of stress. The white spot illustrates the degree of compressive stress and the location where ridge formation takes place first (center of the white portion represents the apex of the pad). (Reprinted with permission from Kücken and Newell (2005), p 79.)

**FIGURE 3–21**

Six different fingerprint patterns from different individuals, representing the continuum of volar pad symmetry at the onset of friction ridge proliferation, ranging from (1) nearly symmetrical to (6) very displaced. (Reprinted with permission from Wertheim and Maceo (2002), p 69.)

degree of asymmetry will be reflected in the ridge flow of the resulting pattern. This biological process cannot be thought of as limited to the extremes of volar pad regression, occurring either completely symmetrically or asymmetrically (leaning all the way to one side). In fact, there is a continuum involved from whorl patterns to loop patterns. Figure 3–21 illustrates several patterns from different individuals whose volar pads were theoretically the same approximate size at the critical stage (i.e., the volar pads had similar ridge counts), but differed in the degree of their symmetry.

Subtle variations in the symmetry of a volar pad could affect the formation of a whorl pattern versus a central pocket loop whorl pattern, or a central pocket loop whorl pattern versus a loop pattern. Any one of the numerous genetic or environmental factors present during the critical stage could cause a slight deviation in the normal developmental symmetry of the volar pad and, therefore, affect the resulting pattern type.

3.7.2 Size of the Volar Pad

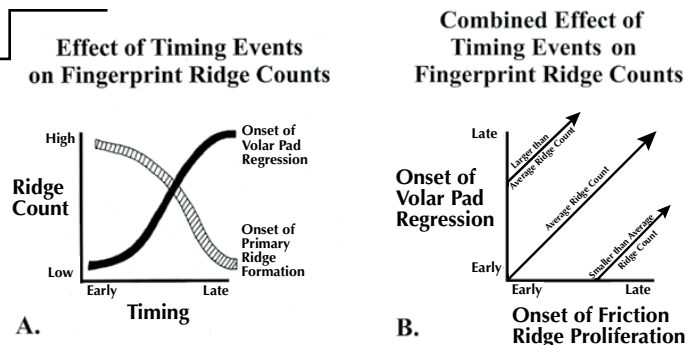
3.7.2.1 Pattern Size. The size, particularly the height, of the volar pad during primary ridge formation affects the ridge count from the core to the delta of normal friction ridge

patterns (Bonnievie, 1924; Mulvihill and Smith, 1969; Siervogel et al., 1978). Researchers have observed that ridges that form on high, pronounced volar pads conform to the surface as high-count whorl patterns. Conversely, ridges that form on a finger with a low or absent volar pad create low-count or arch-type patterns (Babler, 1987, pp 300–301). Holt (1968) reported that the total finger ridge count (TFRC) of all 10 fingers, taken by adding the ridge counts from the core to the delta in loops, or the core toward the radial delta in whorls, is the most inheritable feature in dermatoglyphics. This combined information points directly to the conclusion that timing events related to volar pad and friction ridge formation affect friction ridge patterns.

3.7.2.2 Timing Events. The ridge count of a friction ridge pattern is related to two different events: the timing of the onset of volar pad regression and the timing of the onset of primary ridge formation. Differences in the timing of either event will affect the ridge count of that particular pattern. For example, early onset of volar pad regression would lead to a volar pad that was in a more regressed state at the time of the onset of primary ridge formation, and a relatively low-ridge-count pattern (or arch) would likely result. Conversely, overall late onset of volar pad regression would mean that the pad was still relatively large

FIGURE 3–22

Chart A illustrates the effects of two independent timing events on the resulting ridge count of a friction ridge pattern. Chart B illustrates their combined effects on pattern ridge count. (Reprinted with permission from Wertheim and Maceo (2002), p 65.)



when primary ridges began forming, and a high-ridge-count pattern would more likely result (Figure 3–22). This theory is supported by a study that found that “late maturers” had higher-than-average ridge counts, and “early maturers” had lower-than-average ridge counts (Meier et al., 1987).

If the onset of volar pad regression occurred at the normal time, then earlier-than-average onset of primary ridge formation would occur on a larger-than-average volar pad, leading to a higher-than-average ridge count. Likewise, later-than-average onset of primary ridge formation would occur on a smaller-than-average volar pad, leading to a lower-than-average ridge count (Figure 3–22A). When both early and late timing of both factors are taken into account, the results become even more complex (Figure 3–22B).

To make matters even more complex, the size of the volar pad with respect to the finger is also affected by many factors. Diet and chemical intake of the mother (Holbrook, 1991b), hormone levels (Jamison, 1990), radiation levels (Bhasin, 1980), and any other factors that affect the growth rate of the fetus during the critical stage could all indirectly affect the ridge counts of the developing friction ridges on the finger. It is important to remember that anything that affects the tension across the surface of the finger could affect the resulting ridge alignment and pattern type. However, Holt’s findings seem to indicate that timing events, rather than environmental factors, play the dominant role in determining TFRC (Holt, 1968).

3.72.3 Delta Placement. The onset of cellular proliferation, which begins primary ridge formation, occurs first in three distinct areas: (1) the apex of the volar pad (which corresponds to the core of the fingerprint pattern); (2) the distal periphery, or tip of the finger (near the nailbed); and

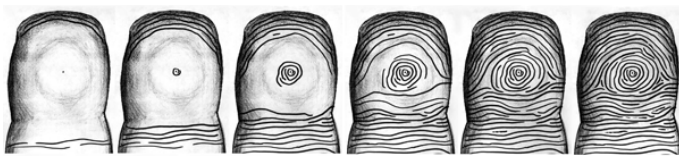
(3) the distal interphalangeal flexion crease area (below the delta(s) in a fingerprint) (Figure 3–23).

As ridge formation continues, new proliferation occurs on the edges of the existing ridge fields in areas that do not yet display primary ridge formation. These three “fields” of ridges converge as they form, meeting in the delta area of the finger. This wavelike process of three converging fields allows for the visualization of how deltas most likely form (Figure 3–24).

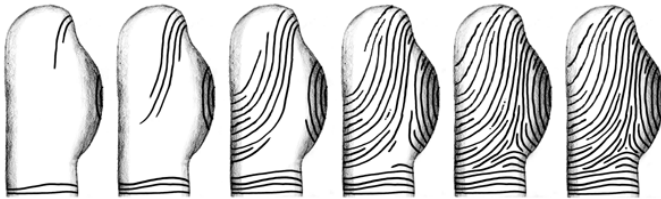
The concept of “converging ridge fields” also offers a way to visualize the difference between the formation of high-versus low-ridge-count patterns. If ridges begin forming on the apex (center) of the pad first and proceed outward before formation begins on the tip and joint areas, then by the time the fields meet, a relatively large distance will have been traversed by the field on the apex of the pad; in that instance, a high-count pattern will be formed (Figure 3–25). However, if the ridges form first on the two outermost portions and proceed inward, and formation begins at the last instant on the apex of the pad, then only a few ridges may be formed by the time the fields meet; in that instance, a very low-count pattern is observed (Figure 3–26). The combined observations of different researchers examining friction ridges on the finger during the critical stage of development further support the validity of this model (Babler, 1991, 1999; Dell and Munger, 1986; Hirsch and Schweichel, 1973).

3.7.3 Combined Effect of Timing and Symmetry on Ridge Formation

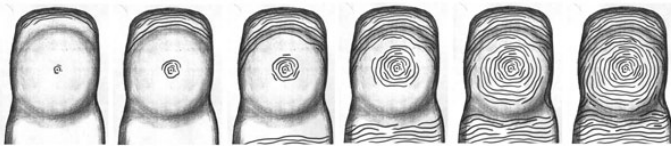
When it is understood that timing and symmetry control two very different elements of ridge flow, it becomes easy

**FIGURE 3-23**

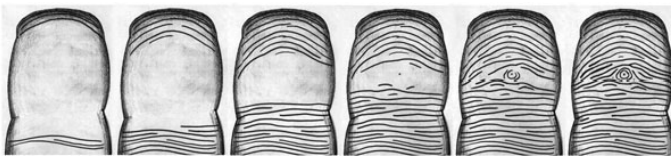
A drawing depicting the normal starting locations of ridge formation and subsequent coverage across the surface of a finger. (Reprinted with permission from Wertheim and Maceo (2002), p 66.)

**FIGURE 3-24**

A drawing that depicts an easy way to visualize how deltas form from three converging ridge fields. (Reprinted with permission from Wertheim and Maceo (2002), p 66.)

**FIGURE 3-25**

A drawing that depicts the likely progression of ridges on a high-ridge-count pattern. (Reprinted with permission from Wertheim and Maceo (2002), p 67.)

**FIGURE 3-26**

A drawing that depicts the likely progression of ridges on a low-ridge-count pattern. (Reprinted with permission from Wertheim and Maceo (2002), p 67.)

to see how both small and large loop and whorl patterns form. A finger pad that regresses symmetrically will form a whorl pattern, regardless of early or late timing of friction ridge formation with respect to volar pad regression. If the timing of the onset of primary ridge formation in this situation is early in fetal life, then the volar pad will still be high on the finger, and the whorl pattern will have a high ridge count. If timing is later in fetal life, after the pad has almost completely been absorbed into the contours of the finger, then a low-count whorl pattern will result. With further regression, an arch pattern will form (Figure 3-27).

Likewise, asymmetrical finger pads will form loop patterns and will also be affected by timing. If ridges begin forming early with respect to volar pad regression on an asymmetrical pad, then the pad will be large, and a high-count loop will result. Later timing leads to a low-count loop or arch-type pattern (Figure 3-28). Again, volar pad placement

is not simply symmetrical or asymmetrical; a continuum of volar pad symmetry occurs and accounts for the variety of pattern types observed.

A regression scheme seems to exist whereby the volar pad is symmetrical at the onset and becomes progressively more asymmetrical as it regresses. This is supported by general fingerprint pattern statistics that show that more than one-half of all fingerprint patterns are ulnar loops. More specifically, this scheme is supported by fetal research that has determined that early timing of primary ridge formation leads to a higher percentage (95 percent) of whorls (Babler, 1978, p 25). Also, low- and high-ridge-count patterns occur less frequently than average-count patterns (Cowger, 1983). All research tends to indicate that volar pads regress from an early symmetrical position to an asymmetrical position later in fetal life. Although this is the norm, it is certainly not without exception, because whorl patterns with extremely

FIGURE 3–27

These different fingerprint patterns (bottom) were formed on completely different, but symmetrical, volar pads (top). The drawings on the top illustrate the likely fetal condition of the symmetrical volar pad that produced the resulting print below it. From left to right, the images show the results of the combined timing of the onset of friction ridge proliferation versus volar pad regression. (Reprinted with permission from Wertheim and Maceo (2002), p 71.)



FIGURE 3–28

These different fingerprint patterns (bottom) were formed on different, asymmetrical volar pads (top). The drawings on the top illustrate the likely fetal condition of the asymmetrical volar pad that produced the resulting print below it. From left to right, the images show the results of the combined timing of the onset of friction ridge proliferation versus volar pad regression. (Reprinted with permission from Wertheim and Maceo (2002), p 71.)



low ridge counts and loop patterns with extremely high ridge counts can both be found with relative ease in even small collections of recorded fingerprints.

3.8 Genetics

3.8.1 Introduction to Genetic Diversity and Friction Ridge Skin

In 1904, Inez Whipple presented research that provided a detailed theory of evolutionary progression of the volar surface (Whipple, 1904). Ashbaugh succinctly summarizes Whipple’s proposition of the evolutionary genesis of friction ridges:

Early mammals were covered with a scale-like skin surface. Each scale had one hair protruding from it and an accompanying oil or sebaceous gland. On volar areas, which are the bottoms of the hands and feet, hairs slowly disappeared due to surface use. The pore that was related to the hair changed from a sebaceous gland to a sweat gland. Its purpose, to keep the surface skin damp which enhanced the grip of the volar surface.

Starting in all likelihood as a mutation, scales started to line up in rows and fuse together. This further assisted the grip of the skin surface by increasing friction. Through natural selection, this mutation became prevalent. Scales slowly evolved into wart-like units with pore openings near the centre. The fusing of these wart formations into rows is the predecessor to the friction ridge, the individual wart being the equivalent of a ridge dot (Ashbaugh, 1991, p 27).

Fourteen years after Whipple’s phylogenetic (evolutionary history) theory was presented, researchers diverged from her theory and presented an ontogenetic (individual developmental or embryonic history) model, suggesting that fusion of warts into ridges occurs during embryonic development (Wilder and Wentworth, 1918). In 1926, Cummins refuted the ontogenetic scheme (Cummins, 1926, p 134). However, Hale later included the ontogenetic model in his conclusions (Hale, 1952). Literature since that time has been mixed. Multiple researchers have demonstrated that the first visual evidence of interaction between the dermis and the epidermis is ridges, not a series of units, protruding into the dermis (Figure 3–6, p 3-8). Perhaps



with advances in technology, the theory that localized cell proliferations grow together into linear ridges before the appearance of the ridge as a structure will be demonstrated. Until then, fusion of units into ridges remains a possible model of development that could provide individuality before the appearance of the first ridge structures. The term “ridge unit” might be limited to a description of an adult sweat pore and surrounding ridge (Ashbaugh, 1999, pp 25, 35), with the term “localized proliferation” being used to describe theoretical events of fetal formation (Babler, 1987, p 298).

3.8.2 The Role of Genetics

Every aspect of the growth and development of a single cell into a fully formed human is initiated by a genetic blueprint. The capacity to form friction ridges is inherent within the developing embryo. The patterns that these ridges form, however, are limited by nature and are defined by the fingerprint community as whorls, loops, arches, combinations and transitions of these basic patterns, or lack of a pattern (Hirsch, 1964). Although genetics may direct when and where ridges will form by providing the blueprint for proteins, nature provides the boundaries for patterning through physical mechanisms (Ball, 1999).

Proteins direct cellular activity by facilitating biochemical processes within the cell. These processes depend not only on the protein derived from the gene but also on the many other nonprotein components of the cell such as sugars, lipids, hormones, inorganic elements (e.g., oxygen), inorganic compounds (e.g., nitric oxide), and minerals. Additionally, the physical environment around and within cells, including surface tension, electrical charge, and viscosity, contributes to the way the cell functions (Ball, 1999).

Genetic information directs cellular function, serves as a link between generations, and influences an individual's appearance. Some aspects of appearance are similar for each individual of that species (i.e., those characteristics that define the species). However, within the species, for each aspect of an individual's appearance, many genes and external factors affect the final outcome of physical appearance. The genes involved with a specific attribute (e.g., skin color) produce the appropriate proteins, which in turn react with each other and with the many nongenetic components of the cell in complex biochemical pathways during the growth and development of the fetus (Ball, 1999). These biochemical pathways proceed under the omnipresent influence of external factors.

Although DNA is crucial for providing the blueprint for the development of a particular model, there are so many steps between the genesis of the DNA-encoded protein and the final product that even two individuals who originated from the same DNA would produce two completely unique models.

Perhaps Jamison best describes the interplay between genes and the environment in friction ridge skin:

Since dermatoglyphic formation cannot be derived solely from either genetic or environmental factors, it must result from an interaction of the two types of factors. This interaction is probably far from being simple and it most likely involves a multiple step reciprocal positive feedback relationship (Maruyama, 1963) in which either a genetically or an environmentally-based factor causes a change in the uterine environment, leading to a genetic response (perhaps in the form of a “switch mechanism”, as in Roberts (1986)), which then leads to an increasingly complex series of genetic-environmental interactive responses (Jamison, 1990, p 103).

The ultimate example of the role of the environment in friction ridge formation is monozygotic twins, who share identical genetic information and very similar intrauterine environments, but on many occasions have very different patterns. The role of genetics is currently understood by the indication that several main genes, in conjunction with a number of modifying genes, may be responsible for volar patterning, but it is well established that friction ridge patterning is also affected by the environment (Chakraborty, 1991; Hirsch, 1964; Loesch, 1982, 1983; Slatis et al., 1976; Weninger et al., 1976).

Like many traits, genetics influences pattern formation indirectly by contributing to the timing of the onset of friction ridge skin, the timing of the onset of volar pad regression, the growth rate of the fetus, and other factors. Stresses across small areas of skin are not inherited, but rather they represent one of many environmental factors that influence pattern formation.

Until recently (Chakraborty, 1991; Mavalwala et al., 1991), most researchers in the field of genetics and physical anthropology have traditionally viewed TFRC as evidence of direct genetic control of fingerprint pattern formation (Bonnievie, 1924; Holt, 1968). The research of Sara Holt (1968) regarding the inheritability of TFRC is a significant

finding that supports the two-tiered development scheme suggested by this and other literary reviews of fingerprint pattern formation. Logic also supports this scheme. Genetically controlled timed events would be less susceptible to environmental variations, and, therefore, TFRC would be more inheritable than pattern type. Additionally, the wide range of patterns found on the palms (Malhotra, 1982) demonstrates the complex nature of factors that affect ridge alignment. Patterning and ridge counts are indirectly inherited and are not affected by only one developmental factor. However, ridge flow and ridge count are both affected by tension across the surface of growing fetal skin.

3.8.3 Familial Studies

3.8.3.1 Ethnic Variation. Thousands of anthropological studies have been conducted on distinct populations to identify trends in fingerprint pattern formation. Perhaps one of the most comprehensive reviews of this tremendous body of research was conducted by Jamshed Mavalwala, resulting in a 300-page bibliography of dermatoglyphic references (Mavalwala, 1977). The major result from this body of work was the demonstration that intratribal variations in friction ridge pattern frequencies were greater than intertribal variations. Likewise, intraspecies variations in primates were greater than interspecies variations. The body of literature on ethnic variation suggests that multiple genes affect pattern formation and that those genes interact with respect to final pattern characteristics.

3.8.3.2 Abnormalities. The medical community has been, and continues to be, interested in dermatoglyphics (Durham et al., 2000; Kahn et al., 2001; Schaumann and Opitz, 1991) and creases (Kimura, 1991) as indicators of abnormal fetal development during the critical stage. Although there is evidence that interest has waned in recent decades (Reed, 1991), it was reported in 1991 that significantly more than 3,500 articles in the international literature dealt with different aspects of dermatoglyphics (Mavalwala, 1977). Although many articles relate certain medical conditions to statistically significant occurrences of abnormal ridge pattern combinations, many researchers still heed the warning that “dermatoglyphics may be of uncertain, if any, diagnostic value due to the lack of a specific dermatoglyphic stereotype in individual patients” (Schaumann, 1982, pp 33–34).

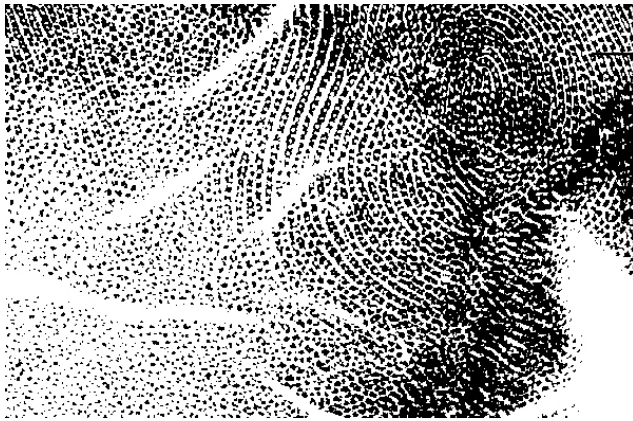
Harold Cummins was perhaps one of the most prominent researchers on the specific reasons behind abnormal friction ridge pattern development (Cummins, 1923, 1926).

From dozens of developmental-defect case studies, he concluded that “whatever the nature of the defect, the [ridge] configurations occur as systems partly or wholly unlike the normal, but obviously conforming to the irregularities of the part” (Cummins, 1926, p 132). Later in his career, Cummins established that the absence of dermal ridges can be caused by chromosomal abnormalities (Figure 3–29) (Cummins, 1965). Other research (Schaumann and Alter, 1976) has attributed a more pronounced condition, dysplasia, to localized deviation in normal nerve branching during fetal development (Figure 3–30).

A third and much more extreme (and rare) condition involves the complete lack of ridge features on the fingers and palms of the hands as well as the toes and soles of the feet. Cummins hypothesizes that in epidermolysis, or the death and dissolution of the epidermis, the disintegrated epidermis sloughs, and the denuded surface is gradually covered by a growth of skin cells arising from the dermis after the capacity has gone for the epidermal–dermal junction to produce ridges (Cummins, 1965). Other researchers indicate that this condition, also known as aplasia, appears to stem from a chromosomal abnormality linked to the complete lack of nerve development in the epidermis at the time ridges are supposed to form. In a 1965 article, Cummins postulates that epidermolysis can be inherited, citing three generations of a family, 13 of whom lacked ridges over fingers, palms, toes, and soles (Cummins, 1965). Schaumann and Alter (1976) reproduce a family tree showing 16 of 28 family members from four generations having congenital ridge aplasia, and go on to reference other evidence of the inheritance of ridge anomalies (Figure 3–31).

Goradia and colleagues (1979) make a convincing argument that there is a continuum between normal epidermal ridges, disassociated ridges, and aplasia. They cite cases of overlap in the same person between normal and disassociated ridges as well as overlap between disassociated ridges and areas with no discernible pattern. Additionally, the authors bring to light that certain chromosomal abnormalities have been found to be associated with both disassociation and aplasia.

Although not a typical abnormality, incipient ridges, also described as “rudimentary,” “interstitial,” or “nascent” ridges, are not present in the majority of friction ridge impressions. When they are present on an individual, studies have shown them to be hereditary (Penrose and Plomley, 1969). In 1979, Okajima examined incipient ridges and affirmed earlier research indicating that these structures are

**FIGURE 3–29**

An impression showing normal ridges (top right), mildly disassociated ridges (middle), and severely disassociated ridges (bottom left) in a patient with a chromosomal abnormality. (Reprinted with permission from Schaumann and Alter (1976), p 98.)

**FIGURE 3–30**

Impressions of epidermis displaying mild (left) and severe (right) dysplasia. (Reprinted with permission from Schaumann and Alter (1976), pp 94–96.)

**FIGURE 3–31**

Impressions (left) of fragmented or absent ridges from a subject with aplasia (Reprinted with permission of the March of Dimes from Goradia et al., 1979). Overall image (right) of the hands of a mother and daughter with aplasia. (Reprinted with permission from Schaumann and Alter (1976), p 91.)

permanent, although they carry no sweat glands (Okajima, 1979) (Figure 3–32).

3.9 Uniqueness: Developmental Noise

3.9.1 Ridge Path

The uniqueness of friction skin is imparted from the permanent base structure through a myriad of random forces, which, themselves, are affected by a seemingly infinite

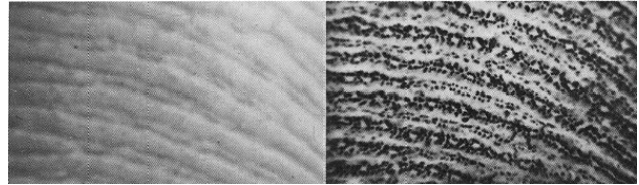
number of factors. The fetal volar pads play a major role in affecting the tensions that directly influence pattern formation (volar pad symmetry) and ridge count (volar pad size), but minutiae formation occurs on a much smaller level. Localized stresses (tensions and compressions), resulting from growth of the tissue layers of the digit and interactions with existing ridge fields, create the foundations for second-level uniqueness.

3.9.2 Ridge Morphology

Ridge morphology (third-level detail) is the surface manifestation of a unique heterogeneous cellular community

FIGURE 3–32

A photograph (magnification = 13 X) of incipient ridges (left) and the dermal surface under the ridges (right) showing double-row arrangement of dermal papillae, marking them as permanent features of friction ridge skin. (Reprinted with permission of the March of Dimes from Okajima (1979), p 191.)



along the basement membrane, which constantly feeds the epidermis a three-dimensional portrait of its uniqueness. It is completely inconceivable that the physical stresses and cellular distributions that create that community could be exactly duplicated, on any level, in two different areas of developing fetal tissue. Each individual section of every ridge is unique. Therefore, any ridge arrangement, regardless of quantity, cannot be replicated. Wide variations in the amount of detail that is recorded from the three-dimensional skin to the two-dimensional impression during any given contact may result in the impossibility of individualization of some latent impressions, but the arrangement of features on the skin and the resulting details in the impression on a surface are still unique.

3.9.3 Maturation of the Skin

After maturation of the primary and secondary ridges at 24 weeks EGA, anastomoses begin to cross through the dermis (Hale, 1952), linking primary and secondary ridges and molding the upper portion of the dermis into papillae pegs. Papillae continue to change form even into late adulthood and become complex (Misumi and Akiyoshi, 1984). Although the shape of the epidermal–dermal boundary may change over time, the rate of skin cell production in the basal layer of skin does not become spatially incongruent. It is for this reason that changes in the shape of the basal layer “sheet” do not produce features that appear significantly different on the surface (Figure 3–33). The consistent rate of basal skin cell proliferation in neighboring areas of skin provides consistent unique detail to the surface of skin. The pattern increases many times over in size, but the sequence of ridges never changes throughout fetal and adult life, barring injury or disease that affects the basal layer of skin.

3.10 Summary: Keys to Uniqueness and Pattern Formation

3.10.1 Uniqueness

As the skin progresses through the entire process of ridge formation (Figure 3–34), many factors contribute to the end result: complete structural uniqueness, from ridge path to ridge shape. Although genetics has been shown to play a role in pattern formation, it does not determine the arrangement of minutiae or ridge shapes within the pattern. The morphogenesis of these finer details is a product of the unique developmental noise that occurs in that area of skin during the critical period of friction ridge formation.

3.10.2 Pattern Formation

The fetal volar pads play a major role in influencing pattern formation (volar pad symmetry) and ridge count (volar pad size), but the volar pads do not directly cause ridge alignment. Instead, the volar pads affect the topology of the surface and the overall tension and compression across the developing epidermal–dermal junction, which in turn directly affects friction ridge alignment during the critical stage of ridge development. Any stress or strain on the developing finger during the critical stage (Figure 3–35) of friction ridge formation could affect ridge alignment.

3.11 Reviewers

The reviewers critiquing this chapter were Jeffrey G. Barnes, Patti Blume, Mary Ann Brandon, Brent T. Cutro, Sr., Lynne D. Herold, Michelle L. Snyder, and John R. Vanderkolk.

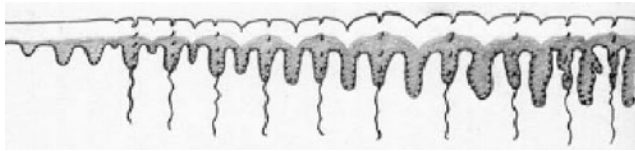


FIGURE 3–33

An illustration of the progression of the structure of volar skin from fetal life (left) through late adulthood (right). (Reprinted with permission from Wertheim and Maceo (2002), p 39.)

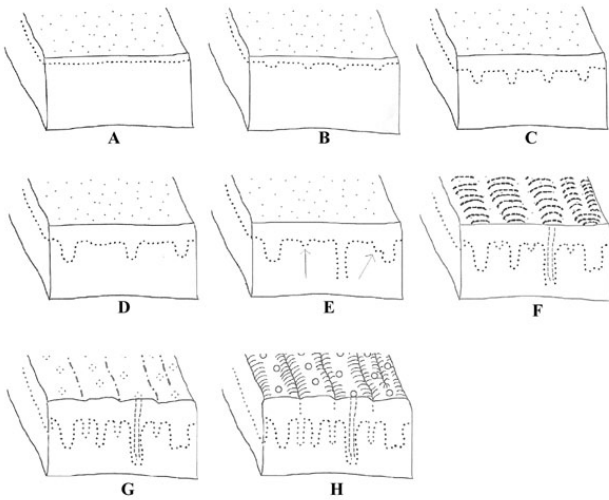


FIGURE 3–34

Drawings representing volar skin before (A), during (B–E), and after (F–H) the critical stage of friction ridge formation: (A) undifferentiated friction ridge skin; (B) initiation of primary ridge formation at the epidermal–dermal border; (C) primary ridges increasing in depth; (D) skin growth separating existing primary ridges; (E) new primary ridge growth between existing primary ridges (sweat ducts are forming); (F) initiation of secondary ridge growth between primary ridges; (G) secondary ridge maturation combined with surface ridge appearance; (H) entire system begins maturation process (approximately 24 weeks EGA). (Reprinted with permission from Wertheim and Maceo (2002), p 56.)

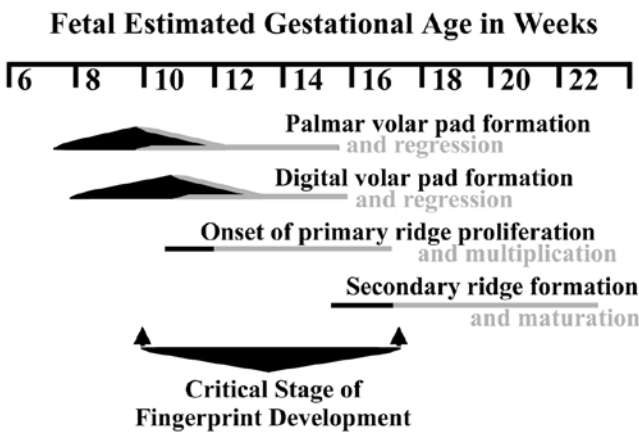


FIGURE 3–35

A chart showing the consensus of the literature regarding estimated time frames for the onset (becoming larger) and regression (becoming smaller) of the volar pads, as well as the onset and growth of the primary and secondary ridges.

3.12 References

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