Aesthetic Breast Surgery

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To all my national and international colleagues
whose contributions have made this book possible.

Preface

"Beauty is in the eye of the beholder" (Robert Browning). This new book has attempted to present the extensive experience of the foremost international authorities in the field of aesthetic breast surgery. Current techniques have been stressed by the editor. Included are discussions by the authorities of some of the possible pitfalls in their techniques, which they have described so well. An attempt has also been made to bring to the reader's attention useful information accumulated from contributions of many other prominent plastic surgeons who have had untoward results and difficult problems. Their description of the surgical management of these varying specific problems should be most useful to the readers in attempting to cope with the multiple problems in aesthetic breast surgery that they are likely to see in their practice.

Aesthetic surgery today is a constantly improving art and the most successful aesthetic surgeon will hold onto the older techniques he has learned over the years as long as the result continues to be good, but will invariably utilize new techniques when he achieves better results. This book has attempted to present a variety of the latest techniques to the aesthetic surgeon.

Acknowledgments

I continue to be most grateful to all my national and international colleagues who contributed so much of their time and skills to this book.

I wish to thank our dedicated Medical Center Library staff and our fine, hard-working secretaries who spent so much of their time making the "multilingual" manuscripts we received "palatable" to our readers, and to the staff of Williams & Wilkins who were so cooperative and helpful in the final delivery of this finished book.

To my associates and colleagues, Drs. Donald Serafin, Ronald Riefkohl, William Barwick, Kenneth McCarty, Jr., Edward Clifford, and, last but not least, my son, Gregory Georgiade, I thank them for their increasing assistance and suggestions.

Finally this book, as all my others, would not have been possible without the skills and dedication of my devoted wife, Ruth, who carefully shepherded this book from its conception to its fruition.

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Contents

CHAPTER 1

The Breast: Anatomy and Physiology*

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INTRODUCTION

Paramount to the understanding of those breast disorders commonly managed by a plastic surgeon is a knowledge of the basic embryological, morphological, and physiological factors involved in the development of this organ. This chapter will review these areas of breast biology and integrate these concepts with the clinical expression of breast pathology.

EMBRYOLOGY OF THE BREAST

The adult breast is composed of 16–18 lobes, consisting of ducts, ductules, and lobular-alveolar units in a fibrofatty stroma. Development (Table 1.1) begins with the milk line forming in the 5th week of fetal life, extending from each axilla to the inguinas. This line thickens to form the milk ridge by 6 weeks. The milk hill develops from the midthoracic portion of the ridge while the remainder of the ridge involutes. Persistence of additional portions of the mammary ridge occurs in 2–5% of women and 1–3% of men resulting in supernumerary nipples (2, 7). On occasion, ectopic breast tissue may be noted outside of the area of the mammary ridge (3). Supernumerary nipples or ectopic breast tissue is usually asymptomatic and most patients request excision for aesthetic reasons. Under the influence of luteal and placental hormones, the ectoderm of the milk hill extends into the underlying mesenchyme, stimulating the condensation of the mesenchyme about the mammary bud (5). By the 10th–14th week, the breast anlage has formed a sunken cone with a surface nipple groove. In the surrounding mesenchyme, fine mammary capillaries and the smooth muscle of the nipple and areola develop. At 12 weeks, three vascular zones can be identified. The inner zone, in intimate contact with the basal layer of anlage, is drawn deeply into the subcutaneous tissue as the epithelium sprouts. Fat and connective tissue accompanying these vessels form septae which separate the mammary gland into lobules. An intermediate group of larger vessels lies in the connective tissue surrounding the breast mound and supplies the cutis. The largest vessels of the outer zone supply the peripheral fat and glandular tissue (7).

At 15 weeks, the vestigial mammary epithelium sprouts into 15–25 stalks. Simultaneously, skin appendages differentiate forming hair and sebaceous glands. Selected sebaceous glands transform to milk glands, while others form the apocrine Montgomery glands (7). The remaining epithelial specializations regress.

At this stage of development (15 weeks), the breast is sensitive to testosterone. The duration of the sensitivity of the mesenchyme to testosterone is short (5). In the presence of testosterone, mesenchyme proliferates and strangles the epithelial stalk, isolating the mammary bud subdermally. Thus, with testosterone exposure, full breast development does not occur (5). Epithelial sprouts *not* exposed to testosterone in the critical period of sensitivity, proceed to canalize from 20–32 weeks to become milk ducts (7). Mesenchymally derived factors appear to determine the morphology of this ectodermally derived tissue (5). From 32 weeks until 40 weeks, the straight milk ducts branch into the lobular-alveolar system (7) and active secretion by the fetal mammary gland is stimulated by placental and maternal ovarian sex steroids. Maternal steroids and fetal prolactin secretion are adequate to maintain neonatal secretion of colostrum until 3–4 weeks of age. Subsequently, with hormonal withdrawal, the lobular-alveolar system reverts to a ductular organization (5). Unilateral aplasia or hypoplasia may be corrected at puberty by prosthetic augmentation to prevent disturbance in the body image during a critical period of emotional development. Further adjustments may be necessary later. Bilateral abnormalities present less difficulty to the patient and are usually managed at an older age.

In the pubescent female, the rising estrogen levels in the

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presence of either growth hormone or prolactin restimulate ductal growth. Budding of mammary ducts, thickening of ductal epithelium, and stromal growth continue until a characteristic ductal spacing is achieved. Alveolar differentiation requires the presence of progesterone (5). With cyclic hormonal fluxes, further ductular-lobular-alveolar structure development continues until approximately 30 years of age (7) (Table 1.2).

Table 1.1.
Embryology of the human breast

5 weeks	— Milk line develops
6 weeks	— With further differentiation and proliferation, milk ridge develops
6–10 weeks	— Milk hill forms and extends into mesenchyme. Remainder of ridge regresses
10–14 weeks	— Breast anlage completely submerged with surface nipple groove
12 weeks	— Vascular development begins
15 weeks	— Mammary epithelium branches and begins to differentiate. Sensitive to testosterone inhibition
20–32 weeks	— Ductular development
32–40 weeks	— Lobular-alveolar development

ANATOMY OF THE MATURE BREAST

The mature female breast may vary in size from under 30 grams to over 500 grams. The breasts are normally unequal in size but symmetric in contour with each breast extending over ribs 2–7 from near the sternal edge to the anterior axillary line with extension of the glandular tail of Spence toward the axilla.

The nipple is ectodermally derived, located at approximately the level of the 4th intercostal space. The dark pigmentation of the areola is enhanced by estrogen influence. While major ducts of the underlying breast extend into the nipple to form the milk ampullae, the majority of the nipple areola glands are sebaceous and apocrine. The dermis is innervated extensively with free nerve endings, Ruffinilike bodies and Krausian end bulbs, and is, therefore, highly sensitive to mechanical stimuli (7). The milk duct ampullae

Table 1.2.
Major hormonal influences on the human breast

Hormone	Effects
1. Estrogen	Necessary for ductal, alveolar growth Necessary to prime glandular tissue for insulin, glucocorticoid, prolactin stimulation of lactation Not necessary for maintenance of lactation Stimulates pituitary production of thyroid stimulating hormone, prolactin Stimulates casein, lactose synthesis with prolactin + thyroid hormone
2. Progesterone	Necessary for alveolar differentiation Not necessary for ductal formation Cannot inhibit established lactation
3. Testosterone	Causes mesenchymal destruction of gland organization during critical period of testosterone sensitivity
4. Glucocorticoid	Necessary for maximal growth of ducts Stimulates rough endoplasmic reticulum formation, differentiation of Golgi apparatus Enhances lobuloalveolar growth Necessary for critical mitosis leading to prolactin sensitivity.
5. Insulin	Stimulates mitoses of alveolar epithelium Not necessary for ductal growth Necessary for secretory activity Stimulates rough endoplasmic reticulum formation Responsiveness to insulin predominantly during pregnancy and lactation.
6. Prolactin	Necessary for lactogenesis and maintenance of lactation after epithelial exposure to cortisone/insulin/estrogen Stimulates epithelial growth after parturition Stimulates colostrum production
7. Human placental lactogen	Can substitute for prolactin in epithelial differentiation. Present in 2nd half of pregnancy; stimulates alveolar growth and lactogenesis
8. Growth hormone	Necessary for ductal growth in adolescence
9. Thyroid hormone	Increases epithelial secretory response to prolactin
10. Oxytocin	Contraction of myoepithelial cells

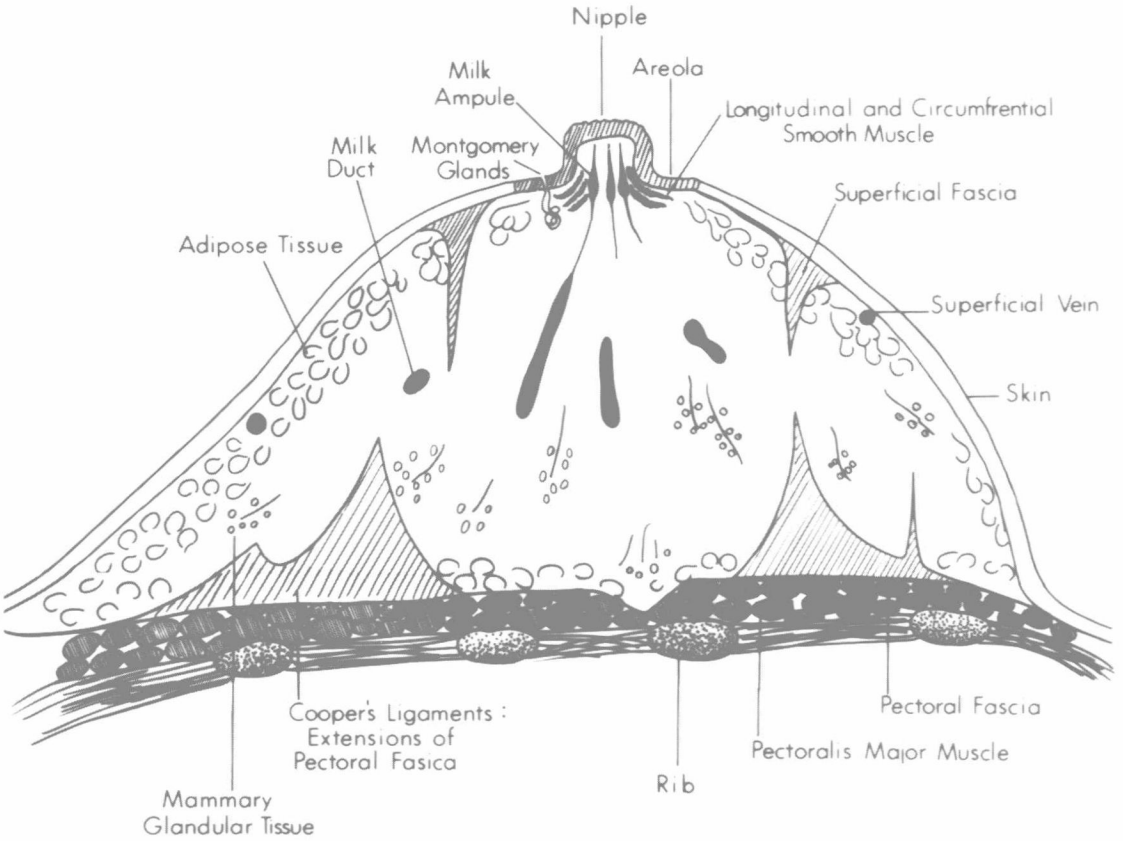

Figure 1.1. Cross section of a mature human breast showing normal anatomic organization. Breast secretions are formed in the mammary glandular tissue and are carried to their milk ampullae within the nipple via the milk ducts. Surrounding structures of the breast include the skin and superficial fasciae superiorly and the pectoralis muscle and fasciae inferiorly. Extensions of the pectoral fasciae, Cooper's ligaments lend support to the protuberant breast tissue.

(0.4–0.7 mm) open into the nipple separately and, in the lactating breast, serve as a milk reservoir. When the breast is not actively lactating, these ampullae are often filled with epithelial debris. Fibrous and elastic connective tissue lie among and encircle the milk ducts. Most of the nipple is comprised of circumferential and radial muscle fibers which insert into the base of the dermis. Their contraction and the resultant venous stasis cause nipple erection. Connective tissue and smooth muscle secure lactiferous ducts; sebaceous and sweat glands and serve as a milk duct sphincter (7).

The areola typically measures from 15–60 mm in diameter. Areolar tissue surrounds openings to sweat glands and small sebaceous glands, lanugo hair, and Morgagni's tubercles, into which open Montgomery glands (Fig. 1.1). Montgomery glands are large sebaceous glands with accompanying small milk glands, derived from apocrine sweat glands. They enlarge and secrete with pregnancy and involute postmenopausally (7).

The skin overlying the breast thins beyond the areola and is thinnest as it approaches the axilla. This skin contains a valveless dermal lymphatic network. Beneath the skin, a delicate superficial fascia, continuous with the cervical fascia and Cooper's superficial abdominal fascia, encloses the entire breast (Fig. 1.1). This fascia defines an avascular plane, under which arteries lie as close as 0.5 cm from the skin surface, below the more superficial veins (2). Abnormalities in the formation of this fascia may be responsible for the tuberous breast configuration. Cooper's suspensory ligaments (retinacula cutis mammae) connect the deep layers of the epidermis to the superficial fascia overlying the breast, fixing the skin undersurface and the nipple to the breast. Fibrosis or displacement by mass may cause traction on these fibrous bands and result in skin dimpling or nipple retraction. Ptosis of the breast occurs when the underlying connective tissue and elastic fibers of the skin become stretched. The breast loses its youthful shape and descends to a lower than optimal level. The normal tension lines of the skin, Langer's lines, lie concentric to the nipple and are important landmarks for incision placement when biopsy is needed (3).

Connective tissue makes up a large portion of the mature mammary gland and its surrounding structures. It attaches lobes to fat deep within the breast, encloses each lobule with a collagen rich capsule, and provides a well-defined basement membrane surrounding each acinus and collecting duct. The pectoralis fascia, lying beneath the superior ⅔ of the breast, encases the pectoralis muscle, extends laterally to the contralateral pectoralis fascia, and blends rostrally with the deltoid and clavicular fasciae. Breast parenchyma extends through this fascia into the pectoralis muscle. Breast tissue not overlapping the pectoralis lies upon the fascia covering the serratus magnus, external oblique, and rectus

abdominus muscles. Thus, to justify subjecting a patient to subcutaneous mastectomy, as thorough a glandular resection as is done during a modified radical mastectomy is mandatory. The mobility of the breast on the chest wall is due to a layer of loose connective tissue lying interposed between the breast and muscle fasciae. An axillary fascia encloses and separates the pectoral muscles and stretches from the deltoid muscle and clavicle to the chest wall. Its costocoracoid fascia portion guards the nerves, vessels, and lymphatics of the axilla as well as enclosing the pectoralis minor muscle (3).

The breast receives sensory and sympathetic autonomic innervation only (7). Skin of the upper breast is supplied with sensory innervation by the supraclavicular nerves, formed from the 3rd and 4th branches of the cervical plexus. The anterior cutaneous divisions of intercostal nerves II–VII emerge through the chest wall paralleling perforating branches of the internal thoracic artery. Small medial branches of the anterior cutaneous nerve supply the medial and inferior aspects of the breast. Lateral branches travel subcutaneously to the areola and midclavicular line. There are conflicting reports regarding the degree of sensation after reduction mammoplasty. Probably the determinants are the preoperative status, the amount of tissue resected, and the type of technique used. The lateral cutaneous nerve from T4 is the dominant innervation to the nipple in many women—damage to this nerve during augmentation mammoplasty may result in anesthesia or dysesthesia of the nipple. It should be emphasized that because of the routing of these nerves, pain may be experienced in breast disease along the lateral chest or the back, (via the intercostal nerves) or above the scapula, on the medial arm, or in the neck (via intercostobranchial and supracervical nerves) (7).

Sympathetic β-adrenergic stimulation causes contraction of smooth muscles of the nipple and blood vessels. Although myoepithelial cells of the alveoli lack innervation, they respond indirectly to nervous excitation. Manipulation of the nipple, such as sucking, causes release of pituitary prolactin and oxytocin via a cholinergic sensory reflex arc. Oxytocin causes contraction of the myoepithelial cells and, in the lactating gland, milk ejection. β-adrenergic stimulation releases norepinephrine causing relaxation of myoepithelial cells. Malfunction of the pituitary reflex pathway or presence of excessive circulating catecholamines may impair milk secretion into the alveoli either by decreasing oxytocin or increasing norepinephrine or may impair both synthesis and secretion via decreasing prolactin.

Blood flows to the breast by three main routes: via the internal thoracic (internal mammary) artery, the lateral thoracic artery, and the anterior and lateral branches of the posterior intercostals (2) (Fig. 1.2). Each courses subcutaneously toward anastomoses about the areola and its subdermal plexus. The internal thoracic artery lies near the sternum immediately beneath the cartilage of the first 6 ribs and sends perforating branches through the intercostal spaces to the medial breast supplying 60% of mammary blood flow (7). The lateral thoracic artery branches either from the axillary artery or less commonly from the thoracoacromial or subscapular artery, or is absent (2, 3). It descends laterally around the pectoralis major muscle to supply the upper-outer and lateral portions of the breast with up to 30% of breast blood flow. The anterior and lateral

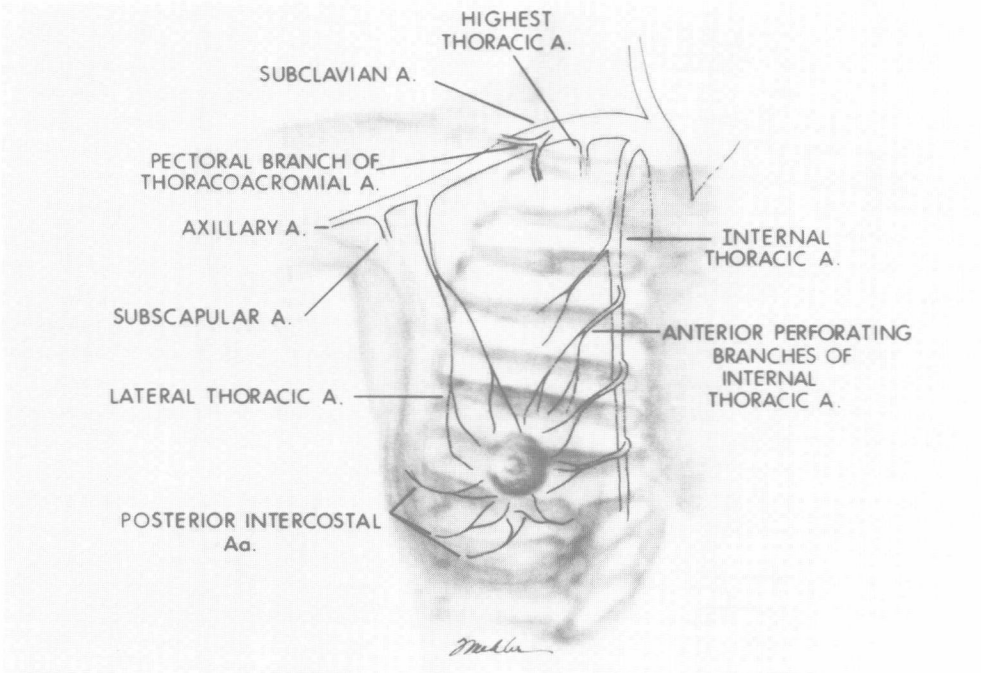

Figure 1.2. Arterial supply to the breast. Blood travels to the breast primarily via the internal thoracic, lateral thoracic, and posterior intercostal arteries with lesser input from the highest thoracic, thoracoacromial, and subcapsular arteries. The vessels travel subcutaneously and anastomose around the nipple.

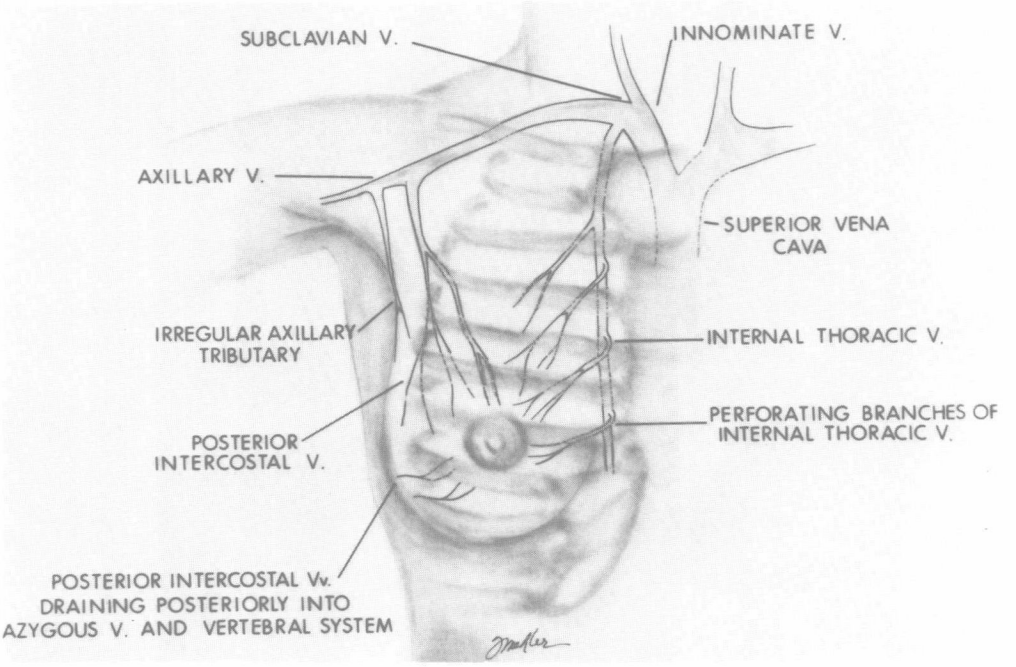

Figure 1.3. Venous pathways of the breast. Superficial veins run subcutaneously and drain into superficial lower muscle veins and the internal thoracic vein. Deep drainage is provided by the perforating branches of the internal thoracic vein, branches of the axillary vein, and the posterior intercostal veins.

branches of the 3rd–5th posterior intercostal arteries supply the lateral lower breast quadrant. Minor sources of arterial flow may include the axillary artery branches, the highest thoracic artery, the pectoral branch of the thoracoacromial artery, and the subcapsular artery (7). Variations are common and there is considerable overlapping and collateralization. Much controversy exists regarding the design of reduction mammoplasty that preserves the maximum circulation to the nipple and areola, yet results in a pleasing contour with minimal scars.

Venous drainage of the breast is provided by superficial and deep venous systems (Fig. 1.3). Two different types of superficial drainage systems, transverse and longitudinal, are observed (3). Transverse vessels run medially subcutaneously, then dorsally, to join perforating vessels and empty into the internal thoracic veins. Alternatively, longitudinally oriented veins ascend to the suprasternal notch to empty into superficial lower neck veins. These superficial systems often anastomose across the chest midline and may dilate when there is underlying disease in the breast (3). Thrombophlebitis of a superficial vein (Mondor's disease) may resemble breast cancer clinically, since skin edema and retraction overlying an ill-defined firm mass may occur in both entities. The largest vessels of the deep draining system are the perforating branches of the internal thoracic vein, which drain into the innominate vein. The axillary vein and its irregular tributaries drain blood from the chest wall, pectoral muscle, and the deep breast tissue. The most important deep drainage runs within the chest wall posteriorly from the intercostal veins to the vertebral veins, azygous vein, and superior vena cava. Breast cancer may metastasize via these venous systems to the lungs, or may metastasize directly to bones of the pelvis, femur, shoulder, spine, or skull through the vertebral venous plexuses. These plexuses drain the intercostal veins and perforate the vertebral column connecting each vertebral segment with intercostal veins (3).

The average mammary gland is associated with approximately 35 lymph nodes. The lymphatic vessels follow vascular routes (Fig. 1.4). If nodes are blocked, as by neoplasm, retrograde lymph flow will occur in this valveless system (2). Four major lymph pathways drain the breast: cutaneous, internal thoracic, posterior intercostal, and axillary (2). Autoradiographs of surgical specimens have revealed no specific relationship between breast quadrant and drainage route (3). Dermal lymphatics include a superficial mesh which may be continuous with contralateral lymphatics; a deeper valved perilobular network which follows mammary ducts in the subareolar region; and Gerota's pathway, draining from the lower breast border to the epigastric plexus in the rectus abdominus sheath. Breast lymph flow across the abdominal wall to the liver or abdominal plexus may occur via Gerota's pathway.

The internal thoracic lymph channels normally carry from 3% (demonstrated post radical mastectomy with colloidal gold uptake studies) to 25% (shown by autoradiography on surgical specimens) of breast lymph flow (3). The 3 or 4 nodes per side lie within 3 cm of the sternal edge. They carry lymph a short distance before emptying into the venous circulation either: 1) via the thoracic duct on the left and right lymphatic duct, 2) via lower cervical nodes, or 3) directly into the jugular-subclavian confluence.

The axillary nodes, divided into 6 groups are exposed to most of the mammary lymph flow (3). The external mam-

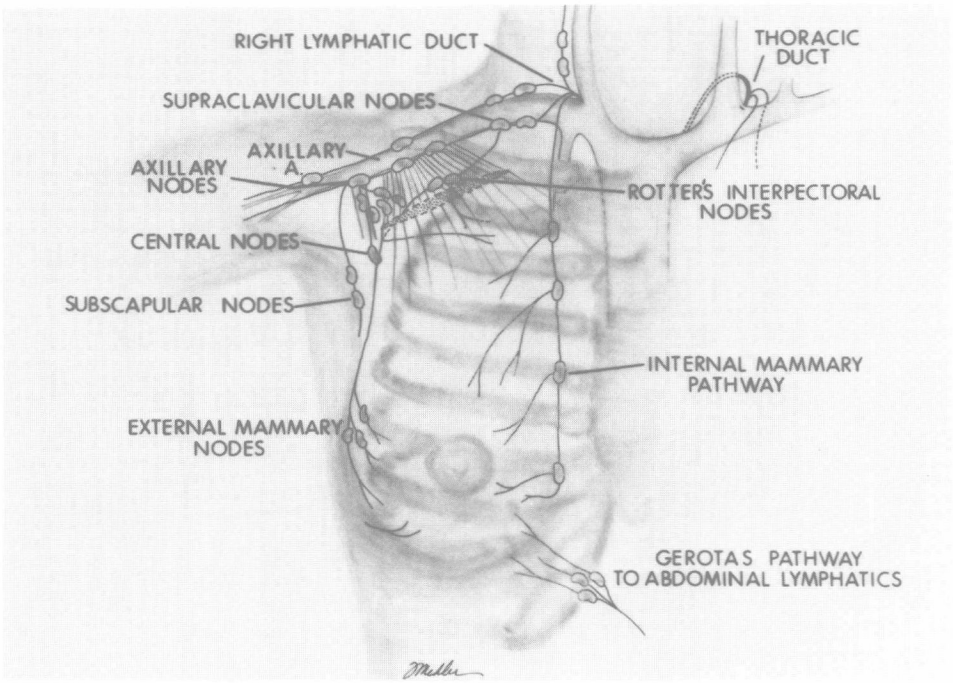

Figure 1.4. Lymphatic pathways of the breast. The lymphatic system of the breast is valveless and is divided into four major pathways: cutaneous, internal thoracic, posterior intercostal, and axillary. Dermal lymphatics may be continuous with contralateral lymphatics and are contiguous with a deep perilobular network following the mammary ducts.

mary nodes lie beneath the pectoralis major and run with the lateral thoracic artery to the axilla. The interpectoral (Rotter's) nodes lie between the pectoralis major and minor muscles and infiltrate the pectoralis major, necessitating the muscle's removal when these nodes must be excised. The scapular nodes lie applied to the subcapsular vessels and the intercostobrachial nerve. Removal of these usually necessitates sacrifice of this nerve. Axillary vein nodes lie on the lateral, caudal, and ventral aspects of the vein, separated from it by a fascial plane. The 10–12 central axillary nodes lie embedded in the central axillary fat. The axillary nodes eventually drain into the 3 or 4 subclavicular nodes, which are the highest, most medial group along the ventrocaudal surface of the axillary vein, extending from the apex of the axilla medially to the thoracoacromial vein. One or more large lymphatic trunks conduct subclavicular lymph medially to the junction of the jugular and subclavian veins. Haagensen (3) remarked that if the subclavicular lymph nodes are involved with cancer "we know that her disease is incurable by surgery" (p. 642). The posterior intercostal lymph nodes become significant when other pathways are blocked (3).

Microscopically, the breast parenchyma consists of glands surrounded by basement membrane, stroma with connective tissue and vessels, and fat (Fig. 1.5). The mammary ducts are lined with an epithelial layering of cuboidal basal cells and low cylindrical surface cells with sparce mitochondria and endoplasmic reticulum. Epithelium of the alveoli is specialized into A, B, and myoepithelial cells, which undergo hormone induced changes. Luminal A cells are columnar basophilic secretory cells. B cells, thought to be precursors of A cells and myoepithelial cells, are polygonal cells with clear cytoplasm and round nuclei. Myoepithelial cells have branching cytoplasm which circumscribe and undermine the alveoli and small excretory milk ducts. The myofilaments contract in response to oxytocin (7). These epithelial elements are surrounded by a specialized structure; the epithelial-stromal junction. This is comprised of the plasma membranes of the epithelial and myoepithelial cells, a lamina lucida, basal lamina and a delimiting fibroblast (Figs. 1.5 and 1.6).

ENDOCRINOLOGY OF THE BREAST

The histological appearance of the mammary gland changes with the menstrual cycle, pregnancy, and menopause (Table 1.2). These changes must be considered in evaluating disease states. The collagenous stromal tissue (with its blood and lymphatic capillaries, fibroblasts, and mononuclear infiltrates) the epithelium of the ducts and acini, and the lumens they define undergo predictable morphological changes with the 5 phases of the menstrual cycle (6) and with pregnancy and lactation. Specific elements involute with the menopause.

Under the influence of rising estrogen, during days 3–7 of the menstrual cycle, the epithelium proliferates to produce 2–3 layers of B cells which obscure the lumen. In this proliferative phase, the stroma is dense with plump fibroblasts and frequent plasma cell infiltrates. The collagen of the epithelial stromal junction is evident (Fig. 1.7*A*) (6).

During the phase of follicular differentiation, days 8–14, three types of epithelial cells may be distinguished. The

most basal myoepithelial cells have small dense nuclei and clear cytoplasm. B cells of the proliferative phase persist, with round, central nuclei, prominent nucleoli, homogenous pale eosinophilic cytoplasm and polygonal shape. The third type of epithelial cell, the A cell, circumscribes the lumen. It has a dense, basal nucleus, a basophilic cytoplasm, due to high ribonucleic acid (RNA) and ribosome content, and a columnar shape. The follicular phase stroma is more collageneous and has less inflammatory infiltrate than does the proliferative phase (6).

With ovulation and increasing progesterone and prolactin levels, breast tissue enters the luteal phase of differentiation. Changes of this period include vacuolization and ballooning of the basal cell layer due to progesterone-induced increase in glycogen content and evidence of some secretion into the lumen but no evidence of proliferation. The stroma loosens and the basal lamina becomes less prominent (6).

During days 21–27, under the influence of estrogen, progesterone, and prolactin, the secretory phase occurs. Progesterone exposure is associated with apical budding, and apocrine secretion from the luminal epithelial cells. The acinar lumen dilates. Estrogen, insulin, and glucocorticosteroids are associated with a marked increase in RNA and ribosome content of the luminal epithelial cells, producing more basophilic staining qualities in these cells. Stroma in this phase changes from the dense compactness of the proliferative phase to an edematous tissue with prominent fluid filled spaces and venous congestion. The engorgement is thought to be due to a steroid-induced histamine effect (Fig. 1.7*B*). The menstrual phase, days 28–2, is associated with withdrawal of estrogen and progesterone. Apocrine budding ceases, but the lumen remains distended with eosinophilic granular secretion. Basal cells remain ballooned with glycogen. The stroma returns to its compact, well-demarcated state, and an increased plasma cell infiltrate is seen (6).

In pregnancy, mammary epithelial cell replication and differentiation resumes such that lobular-alveolar structures displace the fibrofatty stroma. In the stroma, vasculature increases in response to epithelial growth and fat cells are depleted. Ductal sprouting and lobular-alveolar growth require estrogen, progesterone, prolactin, and growth hormone and are enhanced by adrenal corticosteroids, thyroid hormone, and insulin (5). During the 3–4 weeks after conception, mammary glandular tissue branches and proliferates extensively, replacing fat lobules. From weeks 5–8 of gestation, superficial veins dilate, areolar and nipple pigmentation intensifies, and fibroblasts and inflammatory cells become prominent in the stroma. By 3 months, colostrum has begun to collect in the alveoli. During midpregnancy, ductular branching continues at a slower rate. The alveolar cells form a single layer about lumens dilated with colostrum. New capillaries grow about the lobules. Fat and connective tissue appear relatively diminished (7).

The capacity for sustained secretion develops during the 3rd trimester in the presence of prolactin, human placental lactogen or growth hormone, and insulin and glucocorticoid

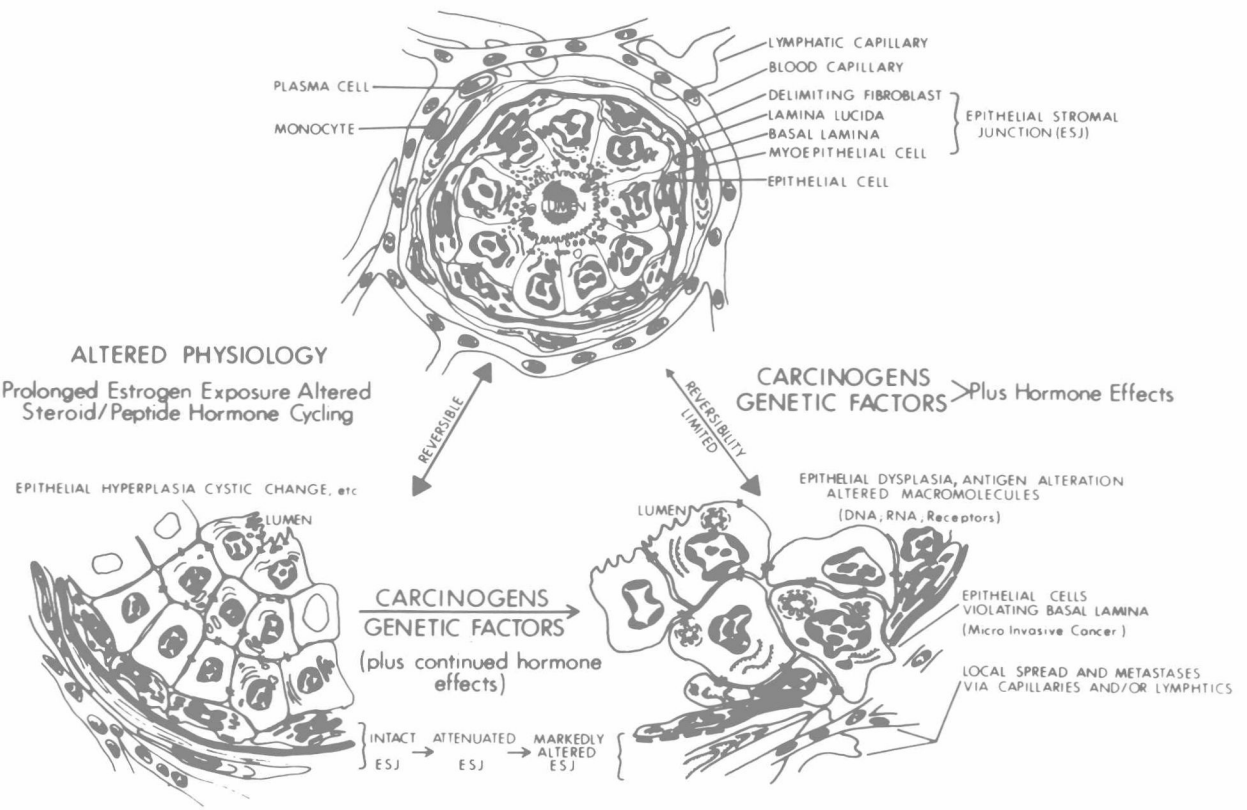

Figure 1.5. Histology of the mature breast acinus with proposed mechanisms of hyperplasia and dysplasia. Relationship of epithelium to the epithelial stromal junction (*ESJ*).