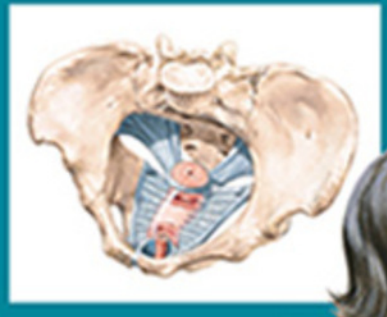

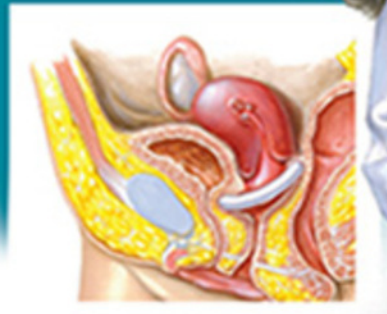

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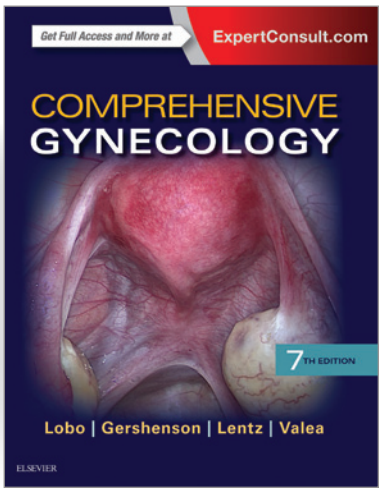


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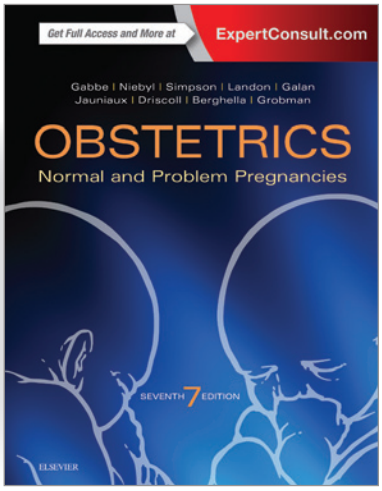


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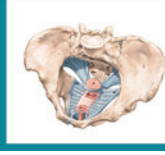

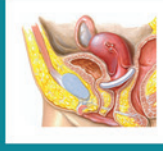


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# PREFACE

No student of medicine, past or present, is unaware of the extraordinary series of medical illustrations created by Dr. Frank Netter. It is an incredible body of work that has been carried forward by the talented Carlos Machado, MD, and John Craig, MD, since Dr. Netter's passing. Older physicians have looked with envy at these images, wishing they had been available when they were learning; established physicians return to them as comfortable sources of information; young physicians seek them out for the wealth of information they contain and their ability to make clear difficult clinical concepts. This spirit of concise reference and resource is the premise of this text.

This third edition maintains the same consistent format in presenting topics to facilitate rapid access—the same information is in the same location—that was so well received in the first and second editions. Chapters have been organized to provide a quick, concise resource for the diagnosis and treatment of common conditions encountered by anyone who provides care for women. In producing this third edition, more than 25 new topics have been added, including sections on embryology and anatomy, a more intuitive organization has been developed, an expanded section on commonly encountered procedures is included, new artwork has been developed, and subtle enhancements (such as indications of the level of evidence provided for references) have been made throughout the work.

It is our hope that this work will be both a useful resource and a celebration of the artistic richness that is clinical medicine.

**Roger P. Smith, MD**

# ABOUT THE ARTISTS

## FRANK H. NETTER, MD

Frank H. Netter was born in 1906 in New York City. He studied art at the Art Students League and the National Academy of Design before entering medical school at New York University, where he received his MD degree in 1931. During his student years, Dr. Netter's notebook sketches attracted the attention of the medical faculty and other physicians, allowing him to augment his income by illustrating articles and textbooks. He continued illustrating as a sideline after establishing a surgical practice in 1933, but he ultimately opted to give up his practice in favor of a full-time commitment to art. After service in the United States Army during World War II, Dr. Netter began his long collaboration with the CIBA Pharmaceutical Company (now Novartis Pharmaceuticals). This 45-year partnership resulted in the production of the extraordinary collection of medical art so familiar to physicians and other medical professionals worldwide.

In 2005, Elsevier purchased the Netter Collection and all publications from Icon Learning Systems. There are now over 50 publications featuring the art of Dr. Netter available through Elsevier, Inc. (in the US: www.us.elsevierhealth.com/Netter; outside the US: www.elsevierhealth.com).

Dr. Netter's works are among the finest examples of the use of illustration in the teaching of medical concepts. The 13-book *Netter Collection of Medical Illustrations*, which includes the greater part of the more than 20,000 paintings created by Dr. Netter, became and remains one of the most famous medical works ever published. The Netter *Atlas of Human Anatomy*, first published in 1989, presents the anatomical paintings from the Netter Collection. Now translated into 16 languages, it is the anatomy atlas of choice among medical and health professions students the world over.

The Netter illustrations are appreciated not only for their aesthetic qualities, but, more important, for their intellectual content. As Dr. Netter wrote in 1949, ". . . clarification of a subject is the aim and goal of illustration. No matter how beautifully painted, how delicately and subtly rendered a subject may be, it is of little value as a *medical illustration* if it does not serve to make clear some medical point." Dr. Netter's planning, conception, point of view, and approach are what inform his paintings and what make them so intellectually valuable.

Frank H. Netter, MD, physician and artist, died in 1991.

Learn more about the physician-artist whose work has inspired the Netter Reference collection:

http://www.netterimages.com/artist/netter.htm.

## CARLOS MACHADO, MD

Carlos Machado was chosen by Novartis to be Dr. Netter's successor. He continues to be the main artist who contributes to the Netter collection of medical illustrations.

Self-taught in medical illustration, cardiologist Carlos Machado has contributed meticulous updates to some of Dr. Netter's original plates and has created many paintings of his own in the style of Netter as an extension of the Netter collection. Dr. Machado's photorealistic expertise and his keen insight into the physician/patient relationship inform his vivid and unforgettable visual style. His dedication to researching each topic and subject he paints places him among the premier medical illustrators at work today.

Learn more about his background and see more of his art at:

http://www.netterimages.com/artist/machado.htm.

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# SEXUAL DIFFERENTIATION

# 1

Genetic sex is determined by the complement and function of sex chromosomes (X and Y) that are present at the time of conception. A Y chromosome carrying specific genes is necessary for the development of testes. The testes are responsible for the organization of the sexual duct system into a male configuration and for the suppression of the paramesonephric (Müllerian) system responsible for female anatomic structures. In the absence of a Y chromosome, specifically required genes, or a functioning gonad, the development will be female. General phenotypic development of the female is viewed as a default event, although new evidence of a more complex process is emerging.

Sexual differentiation genes are located on the Y chromosome, the primary of which is the *SRY* gene, also called the testis-determining factor. The *SRY* gene is found on the short arm of the Y chromosome and influences Sertoli cell differentiation, mesonephric ridge cell development, and male architectural development of the gonad, including blood vessels and other structures of the testes. Several other genes, including those that express

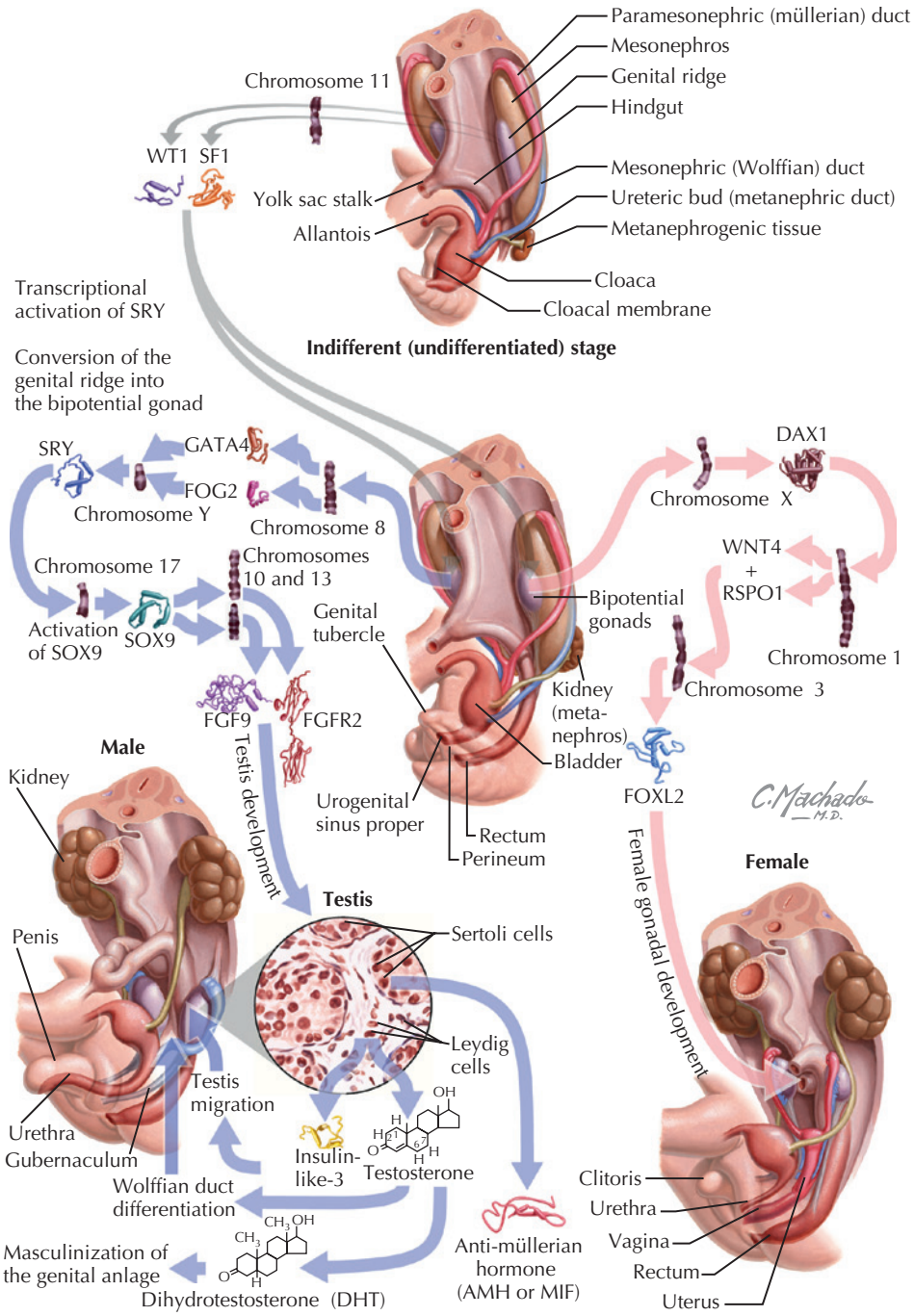



**Figure 1.1** Genetics and biology of early reproductive tract development

steroidogenic factor-1, WT1, and DAX1, on other chromosomes, are also necessary for normal testicular development. To date, multiple mutations of the *SRY* gene have been reported and all are associated with sex reversals (female phenotype).

As discussed earlier, genes in other locations are also important for complete male sexual differentiation. DAX1, a nuclear hormone receptor, can alter *SRY* activity during development by suppressing genes downstream to *SRY* that would normally induce testicular differentiation. A second gene, *WNT4*, largely confined to the adult ovary, may also serve as an "antitestis" gene. In very rare male individuals a Y chromosome may be absent, but the *SRY* gene may be present on another chromosome, most commonly the X chromosome, resulting in a male phenotype. It is becoming apparent that genes, such as *WNT4* and *DAX1*, can proactively induce female gonadal development even in the presence of *SRY*, thereby further complicating the picture. This may account for individuals who are exceptions to the normal sexual dichotomy (eg, males with a uterus or females with an XY karyotype) or who exhibit biologic and/or behavioral characteristics of both sexes.

Male gonadal development precedes female development, and the early secretion of testosterone and anti-Müllerian hormone (AMH) steers the further development of the genital tracts away from the default female phenotype. At a critical point, AMH, produced by Sertoli cells, and testosterone, secreted by Leydig cells, must be produced in sufficient amounts. AMH acts locally, thus suppressing the Müllerian duct system. Testosterone acts systemically, thus causing the differentiation of the mesonephric duct system and male development of the urogenital tubercle, urogenital sinus, and urogenital folds. Enzymes involved in testosterone biosynthesis and conversion to dihydrotestosterones are regulated by genes located on autosomes. The ability to secrete AMH is a recessive trait coded on either an autosome or the X chromosome, and genes for the development of cytoplasmic receptors of androgens seem to be coded on the X chromosome.

The development of the ovary occurs at approximately the 11th or 12th week of gestation, although the primordial germ cells migrate several weeks earlier to the germinal ridge. Two functional X chromosomes are necessary for the optimal development of the ovary. Thus, in 45,X and 46,XY females the ovaries are almost invariably devoid of oocytes. In contrast, germ cells in the testes do best when only one X chromosome is present; rarely do they survive in the XX or XXY condition.

When non-Y-bearing oocytes enter the differentiating gonad, the primary sex cords break up and encircle the oocytes in the cortex of the gonad (in contrast to the structure of the XY gonad). This occurs at approximately 16 weeks of gestation, and the isolated cell clusters are called primordial follicles. No new oogonia form after birth and many of them degenerate well before birth. Those that remain grow and become primary follicles to be stimulated following puberty.

# 2 UPPER GENITAL TRACT DEVELOPMENT

Phenotypic gender is determined by a complex tissue differentiation process that begins in the medial genital thickening or ridges on the posterior surface of the embryonic body cavity. Once gonadal sexual differentiation has begun, several other events must occur for normal male or female phenotypic differentiation to occur. During the fifth week after conception, coelomic epithelium, later known as germinal epithelium, thickens in the area of the medial aspect of the mesonephros. As germinal epithelial cells proliferate, they invade the underlying mesenchyme, producing the gonadal ridge. In the sixth week after conception the primordial germ cells, which formed at approximately the fourth week after conception, in the wall of the yolk sac, migrate up the dorsal mesentery of the hindgut and enter the undifferentiated gonad. These cells will differentiate into testes or ovaries based on the gene functions noted in Chapter 1, Sexual Differentiation.

Signaled by the arrival of primordial germ cells in the fifth week after conception, two sets of paired genital ducts, the mesonephric or nephric (wolffian) ducts and the paramesonephric (müllerian) ducts, develop. The mesonephric system is the precursor to the male genital system and the paramesonephric to the female reproductive structures. The mesonephros is a prominent excretory structure that consists of a series of mesonephric tubules. The tubules connect with the elongating mesonephric (wolffian) ducts as the latter extend caudally, terminating in the urogenital sinus on each side of the midline. Derived from the evagination of the coelomic epithelium, the paramesonephric ducts develop lateral to each of the mesonephric ducts. The cephalward ends of these ducts open directly into the peritoneal cavity, whereas the distal ends grow caudally, fuse in the lower midline, and form the uterovaginal primordium. They join the urogenital sinus as an elevation, known as the müllerian tubercle, which separates the urogenital area from the more posterior gut. Under the influence of the *SRY* gene in the prototestis the mesonephric (wolffian) ducts are maintained during development. As the developing male Sertoli cells begin to differentiate in response to SRY, they secrete a glycoprotein hormone, müllerian-inhibiting substance (MIS) or antimüllerian hormone (AMH), which causes the paramesonephric (müllerian) ducts to regress rapidly between the 8th and 10th fetal weeks. Without testosterone and AMH the mesonephric ducts degenerate and disappear, and the paramesonephric ducts develop into a uterus, fallopian tubes, and upper vagina. Leydig cells synthesize insulin-like-3 (coded by the *INSL3* gene) to promote transabdominal testicular descent into the scrotum. Mutations in this gene may lead to cryptorchidism. In females a structure similar to the gubernaculum develops in the inguinal canal, giving rise to the round ligaments that suspend the uterus in the adult.

Primary sex cords condense and extend to the medullary portion of the developing testes. They branch and join to form the rete testis. The testis therefore is primarily a medullary organ. Eventually the rete testis connects with the tubules of the mesonephric system and joins the developing epididymal duct. Müllerian duct remnants in the male include the appendix testis (hydatid of Morgagni) and the prostatic utricle. In females, MIS is not present, so müllerian ducts remain and the mesonephric tubules and ducts degenerate in the absence of androgens. This often results in remnant epoöphoron and paroöphoron cystic structures within the ovarian mesentery and Gartner duct cysts within the anterolateral vaginal wall. These structures are clinically important because they may develop into sizable and symptomatic cysts (see Chapter 105, Vaginal Cysts).

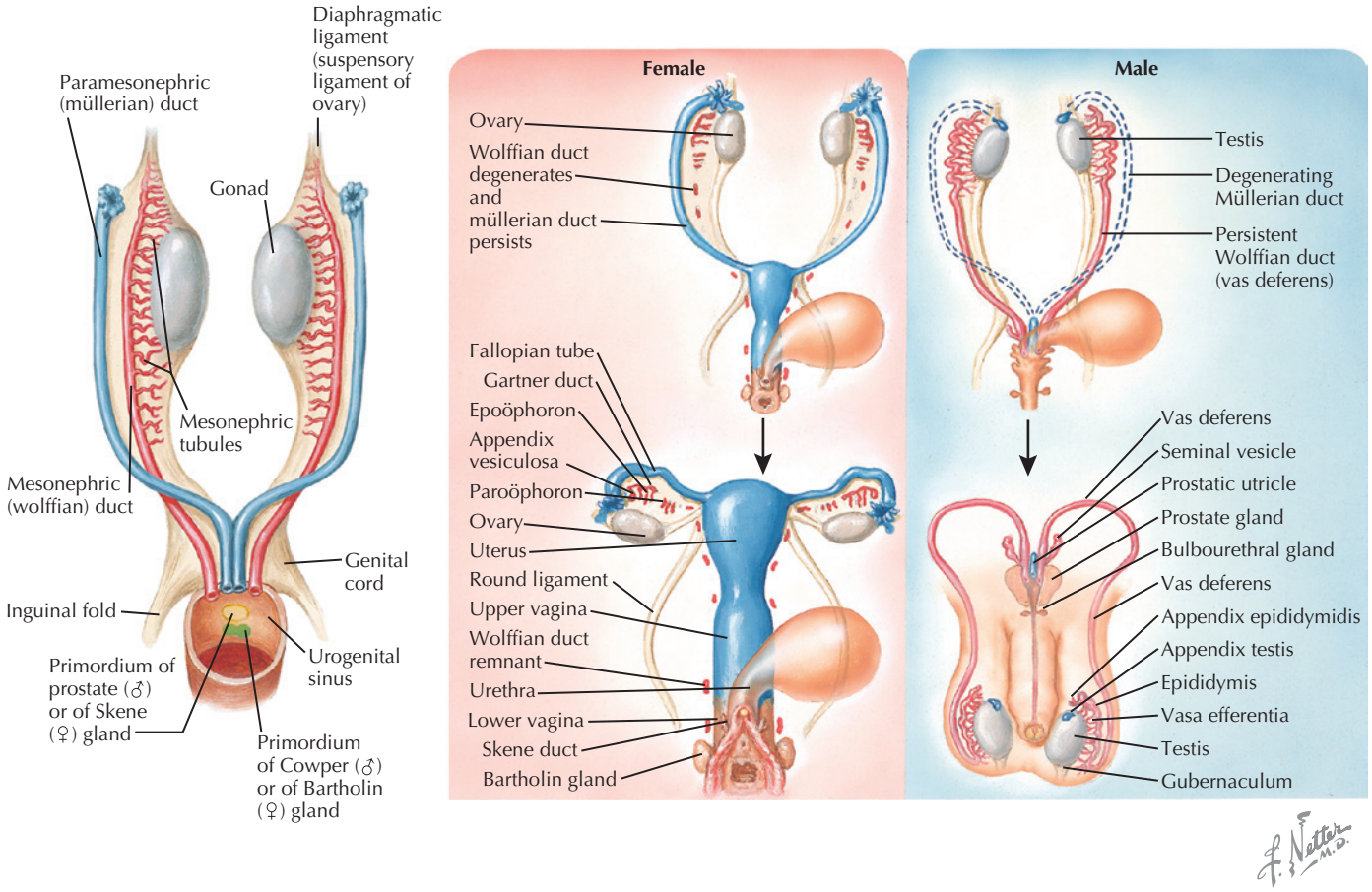



**Figure 2.1** Homologs of the internal genitalia

The process of development and loss of the müllerian and wolffian systems begins at approximately the sixth week after conception and proceeds in a cephalad to caudal fashion. The more cephalad portions of the paramesonephric ducts, which open directly into the peritoneal cavity, form the fallopian tubes. The fused portion or uterovaginal primordium gives rise to the epithelium and glands of the uterus and cervix. Endometrial stroma and myometrium are derived from adjacent mesenchyme. Failure of the development of the paramesonephric ducts leads to agenesis of the cervix and uterus. Failure of fusion of the caudal portion of these ducts may lead to a variety of uterine anomalies, including complete duplication of the uterus and cervix or partial duplication of a variety of types (see Section VI, Chapter 136, Uterine Anomalies: Bicornuate, Septate, and Unicornuate Uterus). Peritoneal reflections in the area adjacent to the fusion of the two paramesonephric ducts give rise to the broad ligaments. Mesenchymal tissue here develops into the parametria.

The remnants of the mesonephric duct in the female include a small structure called the appendix vesiculosa, a few blind tubules in the broad ligaments (the epoöphoron), and a few blind tubules adjacent to the uterus (collectively called the paroöphoron). Remnants of the mesonephric duct system are often present in the broad ligaments or may be present adjacent to the uterus and/or vagina as Gartner duct cysts. The epoöphoron or paroöphoron may develop into cysts. Cysts of the epoöphoron are known as paraovarian cysts.

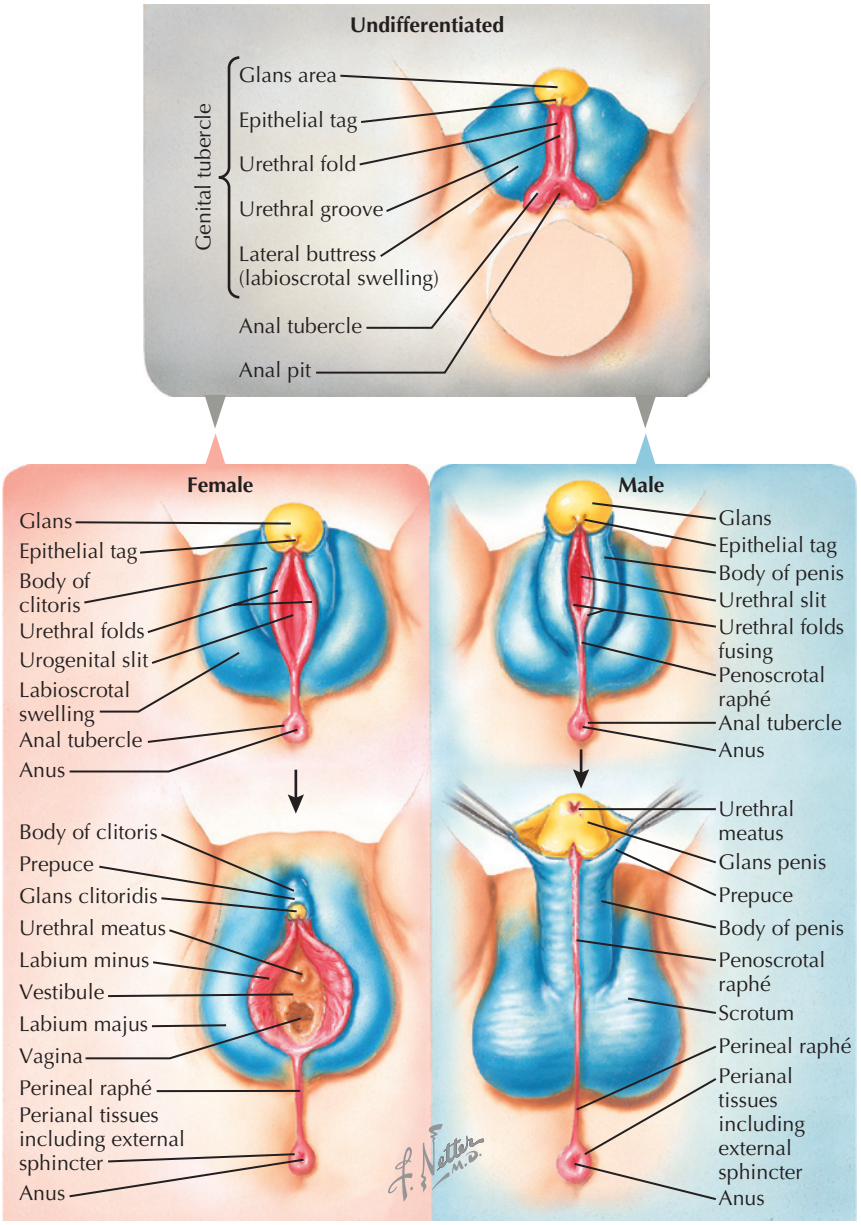



**Figure 3.1** Homologs of external genitalia

imperforate until late in the embryonic life. However, occasionally, perforation does not completely occur (imperforate hymen). Failure of the sinovaginal bulbs to form leads to agenesis of the vagina. The precise boundary between the paramesonephric and urogenital sinus portions of the vagina has not been established.

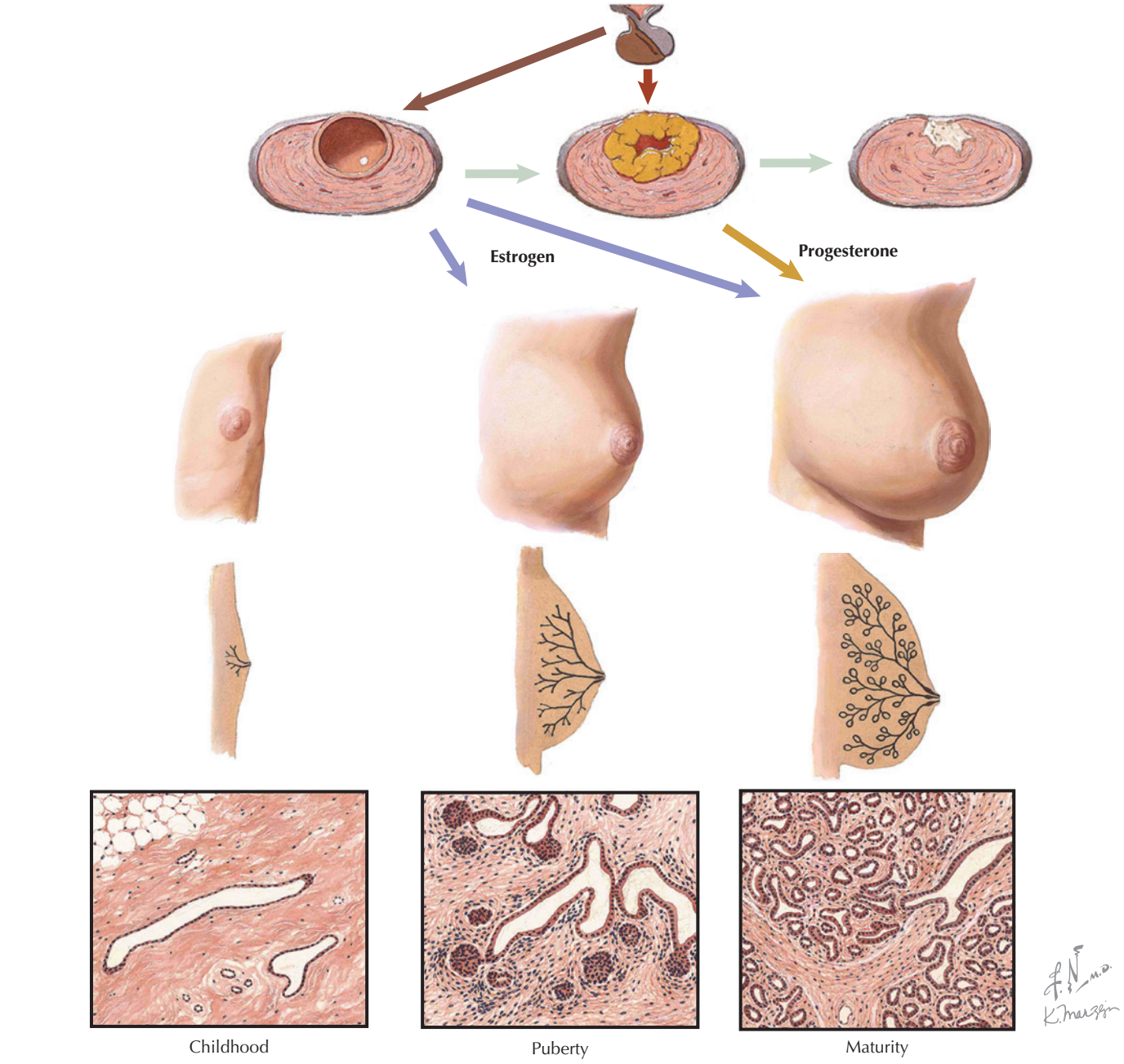
Beginning in the fourth week after conception, the genital tubercle develops at the ventral tip of the cloacal membrane, with the labioscrotal swellings and urogenital folds developing soon after on either side of the cloacal membrane. In both sexes the genital tubercle subsequently elongates to form a phallus. By the end of the sixth week, the cloacal membrane is joined by the urorectal septum. This septum separates the cloaca into the urogenital sinus ventrally and the anal canal and rectum dorsally. The point on the cloacal membrane where the urorectal septum fuses will become the site of the perineal body. The cloacal membrane, now in two parts, then ruptures, opening the vulva and anal canal. Failure of the anal membrane to rupture results in an imperforate anus. With the opening of the urogenital membrane a urethral groove forms on the undersurface of the phallus, completing the undifferentiated portion of external genital development. Differences between male and female embryos can be observed as early as the ninth week, but the distinct final forms are not found until 12 weeks of gestation.

Feminization of the undifferentiated external genitalia occurs in the absence of androgenic stimulation. The embryonic phallus remains quiescent and becomes the clitoris. The urogenital folds remain unfused except in front of the anus, forming the posterior fourchette. The unfused urogenital folds form the labia minora, while the labioscrotal folds remain as the labia majora. The labioscrotal folds fuse anteriorly to form the mons pubis. A portion of the urogenital sinus between the level of the hymen and the labia develops into the vestibule of the vagina, into which the urethra, the vagina, and the ducts of Bartholin glands enter. Beyond 12 weeks of gestation, the labioscrotal folds will no longer fuse when exposed to androgens, although other manifestations of masculinization may occur.

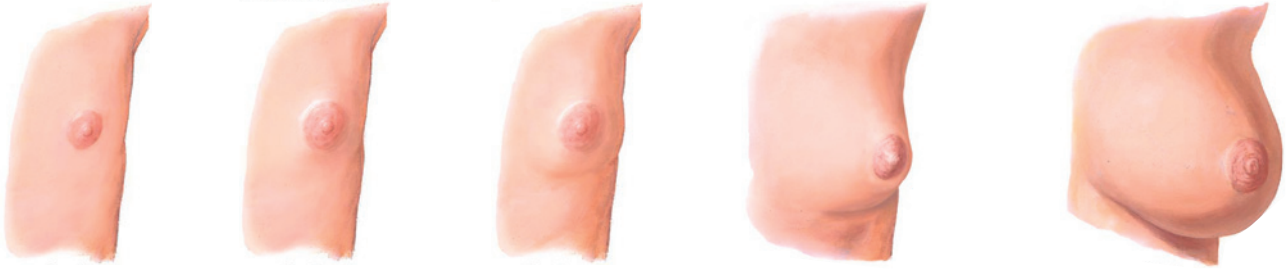
In contrast to the long-held belief that the development of the female genital was passive and occurred in the absence of androgens, the large number of estrogen receptors found in the genital tissues suggests that there is a role for maternal estrogens in the development of the female external genitalia. Female external

genital structures also contain androgen receptors. The distribution of androgen receptors resembles that of the male, which explains why the female genitalia can be masculinized if exposed to high androgen levels early in gestation.

The auxiliary genital glands of the female genitalia form from buds that grow out of the urethra. These buds come from the surrounding mesenchyme and form the urethral and paraurethral glands (Skene glands). These glands correspond to the prostate gland in males. Similar outgrowths of the urogenital sinus form the vestibular glands (Bartholin glands), which are homologous to the bulbourethral glands in the male.




## Tanner Stages of Breast Development



**Stage 1**
Elevation of papilla only

**Stage 2**
Breast bud: elevation of breast and papilla as a small mound and enlargement of areolar diameter

**Stage 3**
Additional enlargement of breast and areola with no separation of their contours

**Stage 4**
Areola and papilla project from surface of breast to form secondary mound

**Stage 5**
Mature stage with projection of papilla only with recession of the areola to the general contour of the breast

**Figure 4.1** Developmental stages of the breast

# SECTION II

## Anatomy



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# EXTERNAL GENITALIA



The perineum is bound in front by the mons veneris, behind by the buttocks, and laterally by the thighs. More deeply it is limited by the margins of the pelvic outlet; namely, the pubic symphysis and arcuate ligament, ischiopubic rami, ischial tuberosities, sacrotuberous ligaments, sacrum, and coccyx. The vulva includes the portions of the female genital tract that are externally visible in the perineal region.

The mons veneris is a fatty prominence that overlies the symphysis pubis. It is covered by curly sexual (pubic) hair that functions as a dry lubricant during intercourse. From the mons veneris the labia majora extend in elliptical fashion to enclose the vulval cleft. They contain an abundance of adipose tissue and sebaceous and sweat glands and are covered by hair on their upper outer surfaces. Posteriorly a slightly raised connecting ridge, the posterior commissure or fourchette, joins them. Between the fourchette and the vaginal orifice a shallow, boat-shaped depression, the fossa navicularis, is evident. The labia minora are thin, firm, pigmented, redundant folds of skin, which split anteriorly to enclose the clitoris. Laterally, they bound the vestibule and diminish gradually as they extend posteriorly. The skin of the small labia is devoid of hair follicles, poor in sweat glands, and rich in sebaceous glands.

The clitoris, a small, cylindrical, erectile organ situated at the lower border of the symphysis, is composed of two crura, a body and a glans. The crura lie deeply in close apposition with the periosteum of the ischiopubic rami. They join to form the body of the clitoris, which extends downward beneath a loose prepuce and is capped by the acorn-shaped glans. Generally, only the glans of the clitoris is externally visible between the two folds formed by the bifurcation of the labia minora.

The vestibule becomes apparent on separation of the labia. Within it are found the hymen, the vaginal orifice, the urethral meatus, and the opening of Skene and Bartholin ducts. The external

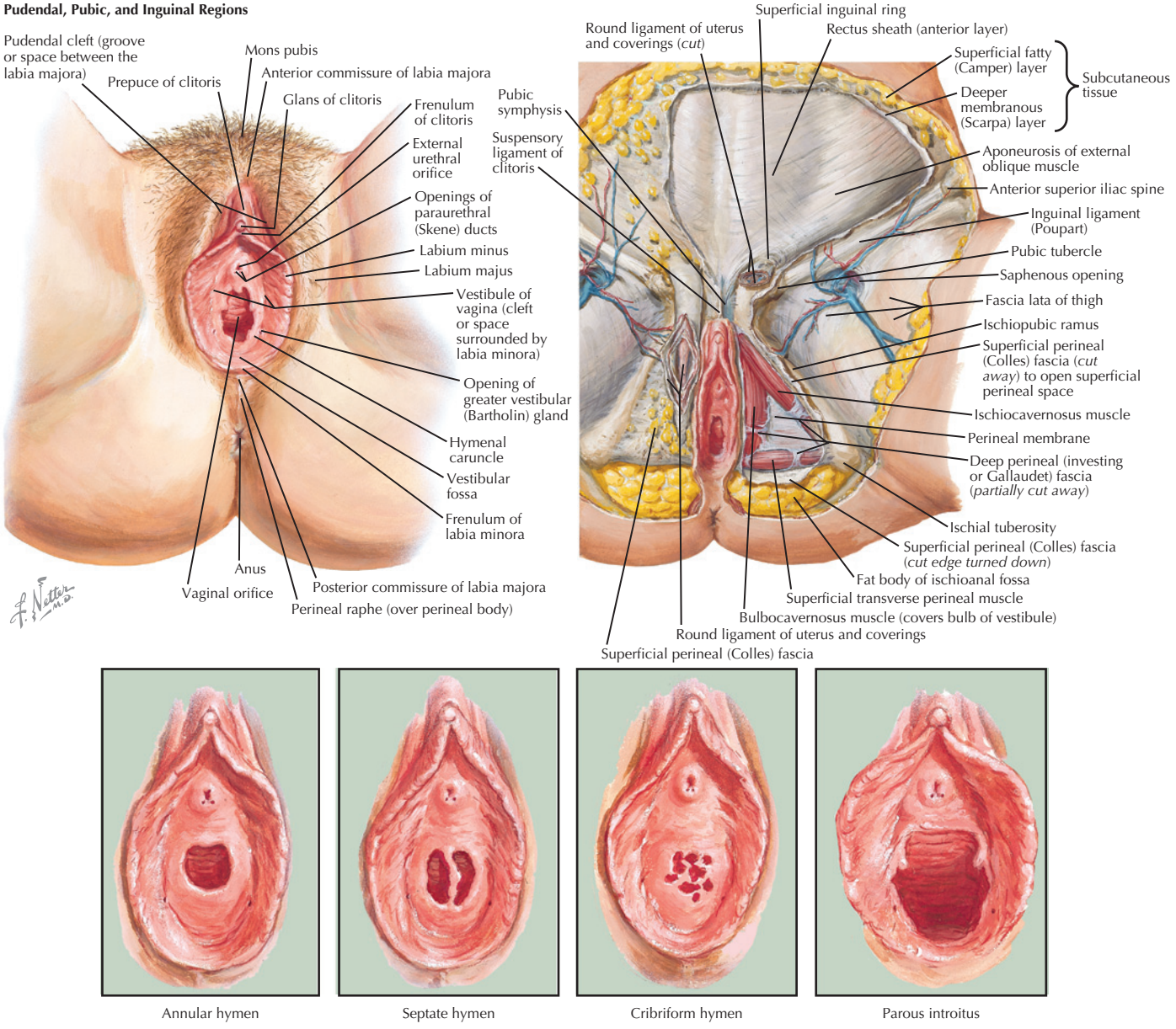


**Figure 5.1** External genitalia

urethral meatus is situated on a slight papilla-like elevation, approximately 2 cm below the clitoris. In the posterolateral aspect of the urinary orifice lie the openings of Skene ducts. They run below and parallel to the urethra for a distance of 1–1.5 cm. Bartholin ducts are visible on each side of the vestibule, in the groove between the hymen and the labia minora, at about the junction of the middle and posterior thirds of the lateral boundary of the vaginal orifice. Each duct, approximately 1.5 cm in length, passes inward and lateral to the deeply situated vulvovaginal glands. The Bartholin glands are situated posterior to the 3 and 9 o'clock locations, which is important clinically when a Bartholin gland abscess is considered in patients with labial swelling.

The hymen is a thin, vascularized membrane or its remnants (the hymenal ring), which separates the vagina from the vestibule. It is covered on both sides by stratified squamous epithelium. As a rule, it shows great variation in thickness and in the size and shape of the hymenal openings (annular, septate, cribriform, crescentic, fimbriate, etc.). After tampon usage, coitus, and childbirth, the shrunken remnants of the hymen are known as carunculae hymenales or hymenal caruncles. The presence or absence of the hymen is insufficient to determine the presence or absence of past sexual activity.

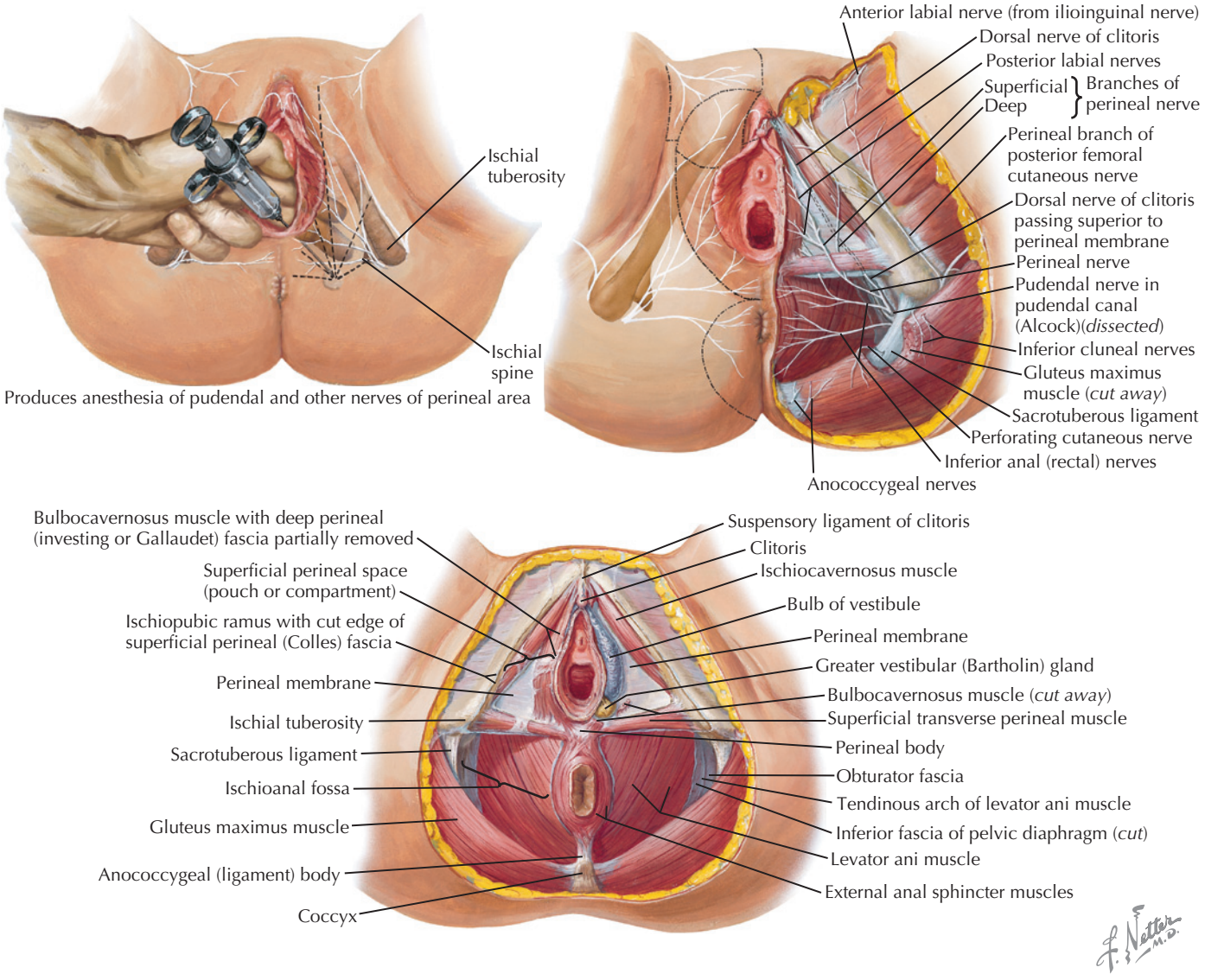


**Figure 6.1** Innervation of external genitalia and perineum

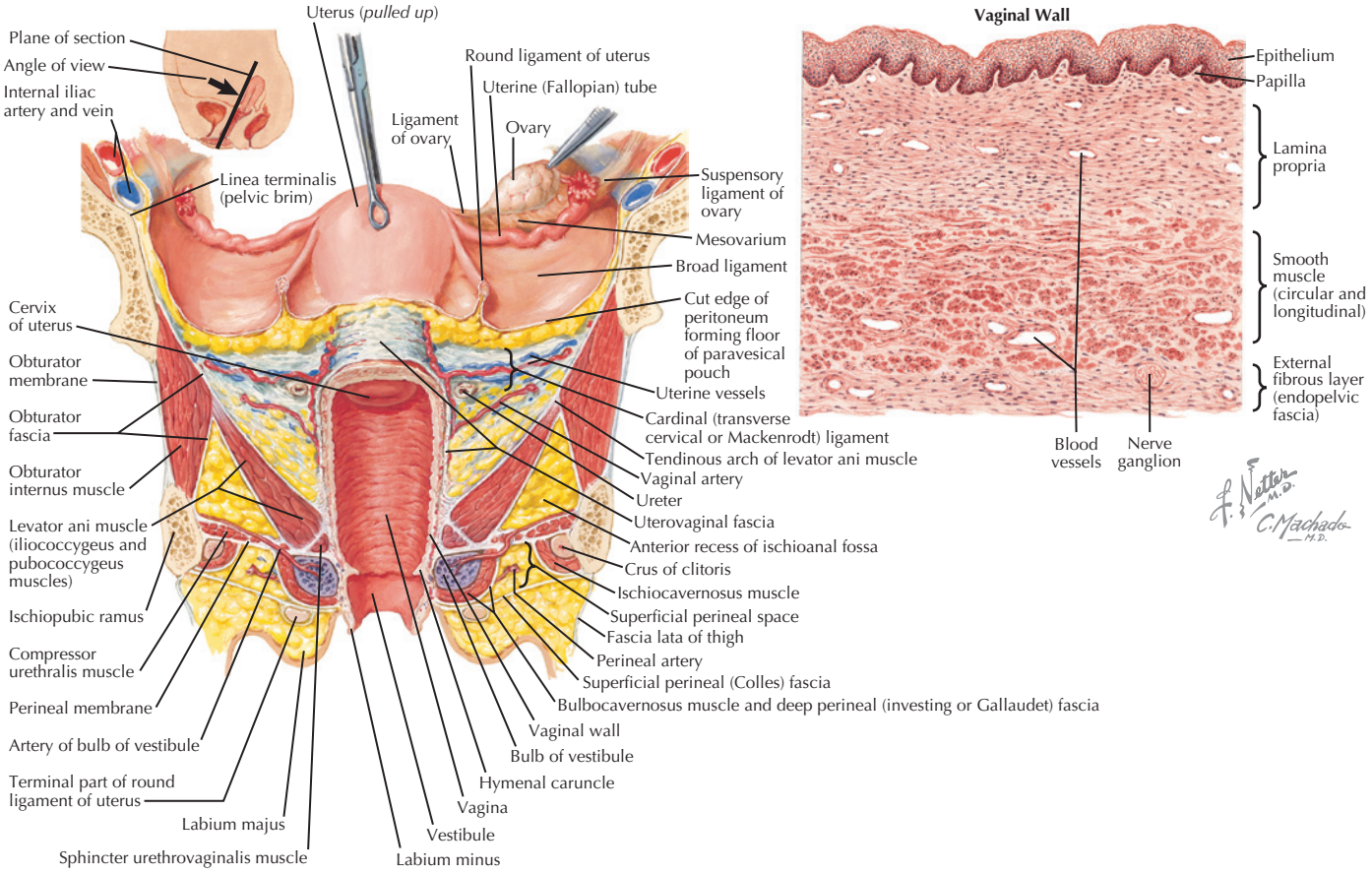


**Figure 7.1** Support of pelvic viscera

sends more and larger papillae into the underlying connective tissue, giving the basement membrane an undulating outline. These papillae are more numerous on the posterior wall and near the vaginal orifice. Beneath the epithelium, which has a thickness of 150–200 μm, a dense connective tissue layer known as the lamina propria is supported by elastic fibers crossing from the epithelium to the underlying muscle. These elastic fibers, here and throughout the pelvis, are critical for pelvic support and function. The lamina propria becomes less dense as it approaches the muscle, and in this area, it contains a network of large, thin-walled veins, giving it the appearance of erectile tissues. The smooth muscle beneath this layer is divided into internal circular and external longitudinal groups, the latter being thicker, stronger, and continuous with superficial muscle bundles of the uterus. No dividing membrane or fascia separates these two interlacing muscle groups. The adventitial coat of the vagina is a thin, firm, fibrous layer arising from the visceral or endopelvic fascia. In this fascia and in the connective tissue between the fascia and the muscle runs another large network of veins and a rich nerve supply.

In its distal extreme the vagina opens to the vulva at the hymenal ring, opening at the caudal end of the vulva, behind the opening of the urethra. When upright the vaginal tube points in an upward–backward direction with the axis of the upper portion of the vagina close to the horizontal plane and curving toward the hollow of the sacrum. In most women an angle of at least 90 degrees is formed between the vagina and uterus. The cervix is directed downward and backward to rest against the posterior vaginal wall. The spaces between the cervix and attachment of the vagina are called fornices, with the posterior fornix considerably larger than the anterior fornix.

Although there is wide variation, the length of the vagina is approximately 6–9 cm (2.5–3.5 in.) along the anterior wall and 8–12 cm (3–4.5 in.) along the posterior wall. During sexual arousal, the upper portion of the vagina elongates and widens through a relative upward movement of the uterus and cervix. This is considered to facilitate capture and retention of sperm to improve the chance of conception.

Throughout most of its length the vagina lies directly on top of the descending rectum, separated by the rectovaginal septum. The upper one-fourth of the vagina is separated from the rectum by the rectouterine pouch (posterior cul-de-sac). The urethra and base of the urinary bladder lie above the anterior vaginal wall, separated by the thin layers of endopelvic fascia. As they enter the bladder, the ureters pass forward and medialward close to the lateral fornices.

The vagina is held in position by the surrounding endopelvic fascia and ligaments. The lower third of the vagina is surrounded and supported by the urogenital and pelvic diaphragms. The levator ani muscles and the lower portion of the cardinal ligaments support the middle third of the vagina, while the portions of the cardinal ligaments and parametria support the upper third.

The vagina is supplied with an extensive anastomotic network of vessels that surround its length. The vaginal artery originates either directly from the uterine artery or as a branch of the internal iliac artery arising posterior to the origin of the uterine and inferior vesical arteries. There is an anastomosis with the descending cervical branch of the uterine artery to form the azygos arteries. Branches of the internal pudendal, inferior vesical, and middle hemorrhoidal arteries also contribute to the interconnecting network from below. These can be a significant source of bleeding with obstetric lacerations. They are also important in the development of vaginal transudate during sexual arousal, when the vagina produces lubrication to aid in penetration.

While the muscular hammock of the levator plate provides the caudal (inferior) floor for the pelvic viscera, the organs of the pelvis have their own mechanisms of support. When either or both of these two support systems fail, it can result in clinical dysfunctions, including urinary incontinence, fecal retention, and dyspareunia. The viscera contained in the female pelvis minor include the pelvic colon, urinary bladder and urethra, uterus, uterine tubes, ovaries, and vagina.

The term endopelvic fascia (actually a pseudofascia) refers to the reflections of the superior fascia of the pelvic diaphragm on the pelvic viscera. At the points where these hollow organs pierce the pelvic floor, tubular fibrous investments are carried upward from the superior fascia as tightly fitting collars, which blend with and may even become inseparable from their outer muscle coat. Thus three tubes of fascia are present, encasing the urethra and bladder, the vagina and lower uterus, and the rectum. These fascial envelopes, with interwoven muscle fibers, are utilized in the repair of cystoceles and rectoceles anteriorly and posteriorly. It is also within this fibrous tube investing the lower uterine segment that the so-called intrafascial hysterectomy is performed in an effort to protect the support of the remaining vaginal cuff. The vesical, uterine, and rectal layers of endopelvic fascia are continuous with the superior fascia of the pelvic diaphragm, obturator fascia, iliac fascia, and transversalis fascia.

Uterine support is maintained directly and indirectly by a number of peritoneal, ligamentous, fibrous, and fibromuscular structures. Of these the most important are the cardinal ligaments and pelvic diaphragm with its endopelvic fascial extensions. The vesicouterine peritoneal reflection is sometimes referred to as the anterior ligament of the uterus and the rectouterine peritoneal reflection as the posterior ligament. These are not true ligaments, and they provide only limited additional support. The round ligaments are flattened bands of fibromuscular tissue invested with visceral peritoneum that extend from the angles of the uterus downward, laterally, and forward, through the inguinal canal to terminate in the labia majora. These are analogous to the gubernaculum in males.

The sacrouterine (uterosacral) ligaments are true ligaments of musculofascial consistency that run from the upper part of the cervix to the sides of the sacrum. At the uterine end, they merge with the adjacent posterior aspect of the cardinal ligaments and endopelvic fascial tube. The broad ligaments consist of wing-like double folds of peritoneum reflected from the lateral walls of the uterus to the lateral pelvic walls. Their superior margins encase the uterine tube and round ligaments. They then continue as the infundibulopelvic ligaments as they progress laterally and superiorly. Inferiorly the ensheathed uterine vessels and cardinal ligaments may be felt. Within the two peritoneal layers are found loose areolar tissue and fat, the fallopian tube, the round ligament, the ovarian ligament, the parametrium, the epoöphoron, paroöphoron and Gartner's duct, the uterine and ovarian vessels, lymphatics, and nerves.

The cardinal or transverse cervical ligaments (of Mackenrodt) are composed of condensed fibrous tissue and some smooth muscle fibers. They extend from the lateral aspect of the uterine isthmus in a tent-like fashion toward the pelvic wall, to become inserted, fan-shaped, into the obturator and superior fasciae of the pelvic diaphragm. This triangular septum of heavy fibrous tissue includes the thick connective tissue sheath, which invests the uterine vessels.

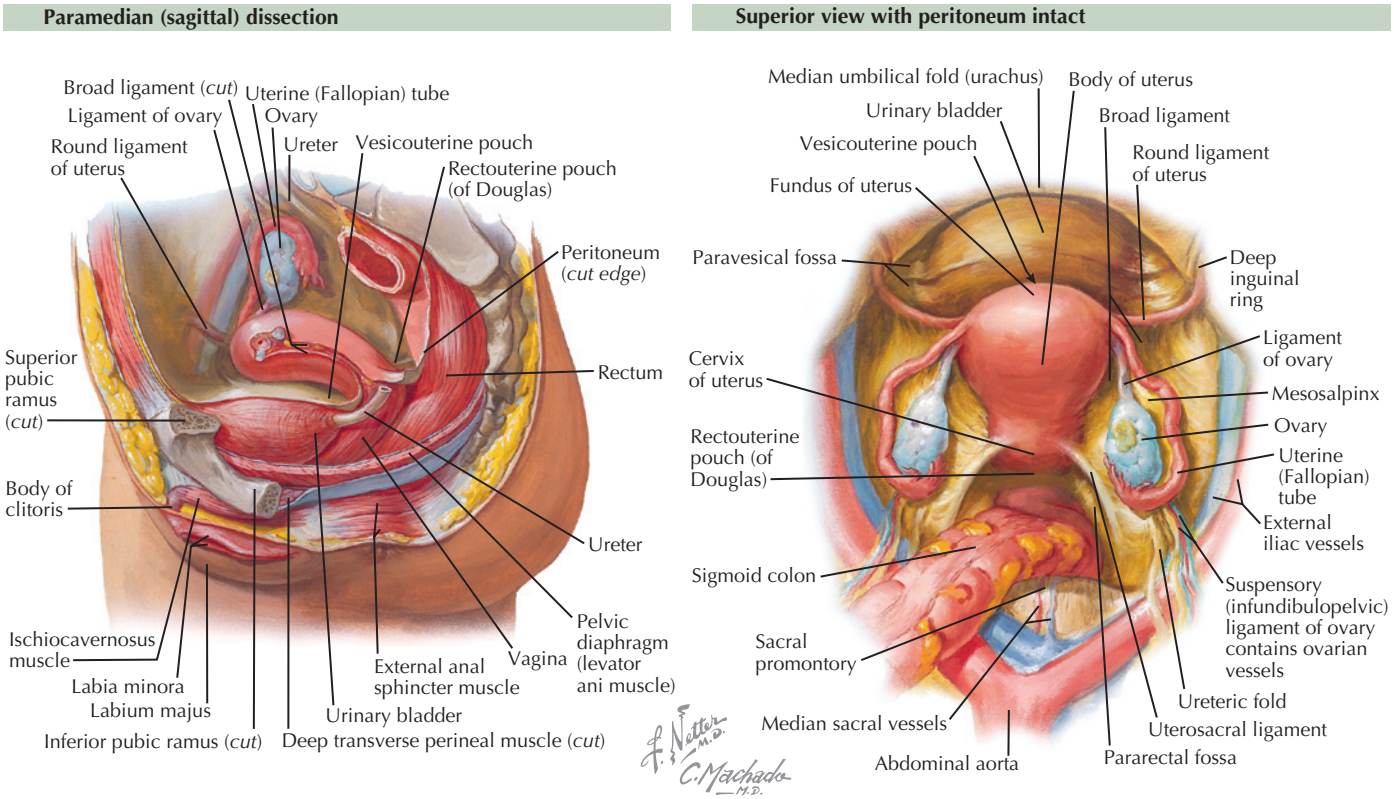


**Figure 8.1** Paramedian dissection and super views of pelvic visera

Mesially and inferiorly the cardinal ligaments merge with the uterovaginal and vesical endopelvic fascial envelopes. Posteriorly, they are integrated with the uterosacral ligaments.

The vesical and rectal endopelvic fasciae maintain bladder and rectum support, respectively.

With the exception of the ovarian, superior hemorrhoidal, and middle sacral arteries the hypogastric divisions of the common iliac arteries supply the pelvic viscera. The uterine artery arises from the anterior division of the hypogastric artery close to or in common with the middle hemorrhoidal or vaginal artery. It courses slightly forward and medialward on the superior fascia of the levator ani muscle to the lower margin of the broad ligament. It arches over the ureter approximately 2 cm from the uterus. At the level of the isthmus, it gives off a descending cervical branch, which surrounds the cervix and anastomoses with the branches of the vaginal artery. The main uterine vessels follow a tortuous course upward along the lateral margin of the uterus, giving off spiral branches to the anterior and posterior surfaces of the uterus. The uterine artery terminates in a tubal branch within the mesosalpinx and an ovarian ramus, which anastomoses with the ovarian artery in the mesovarium.