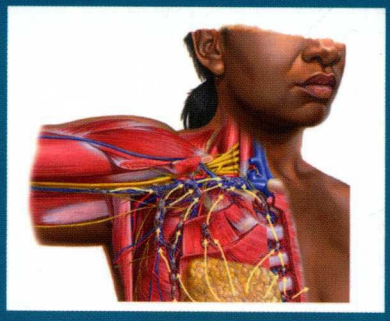

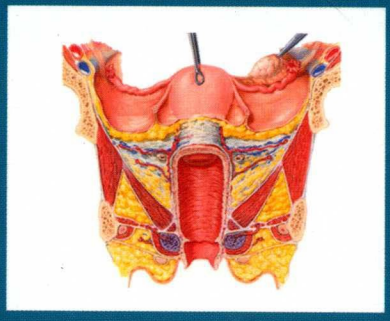

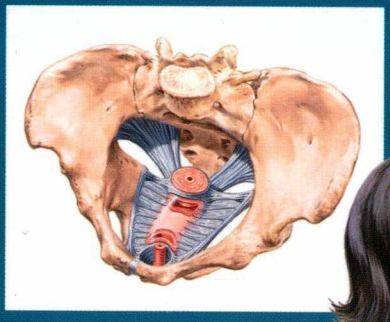

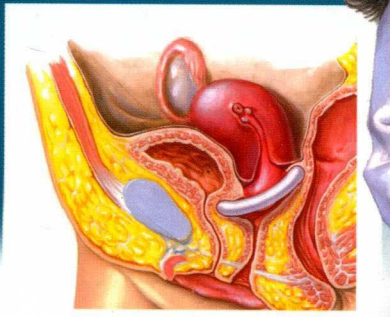

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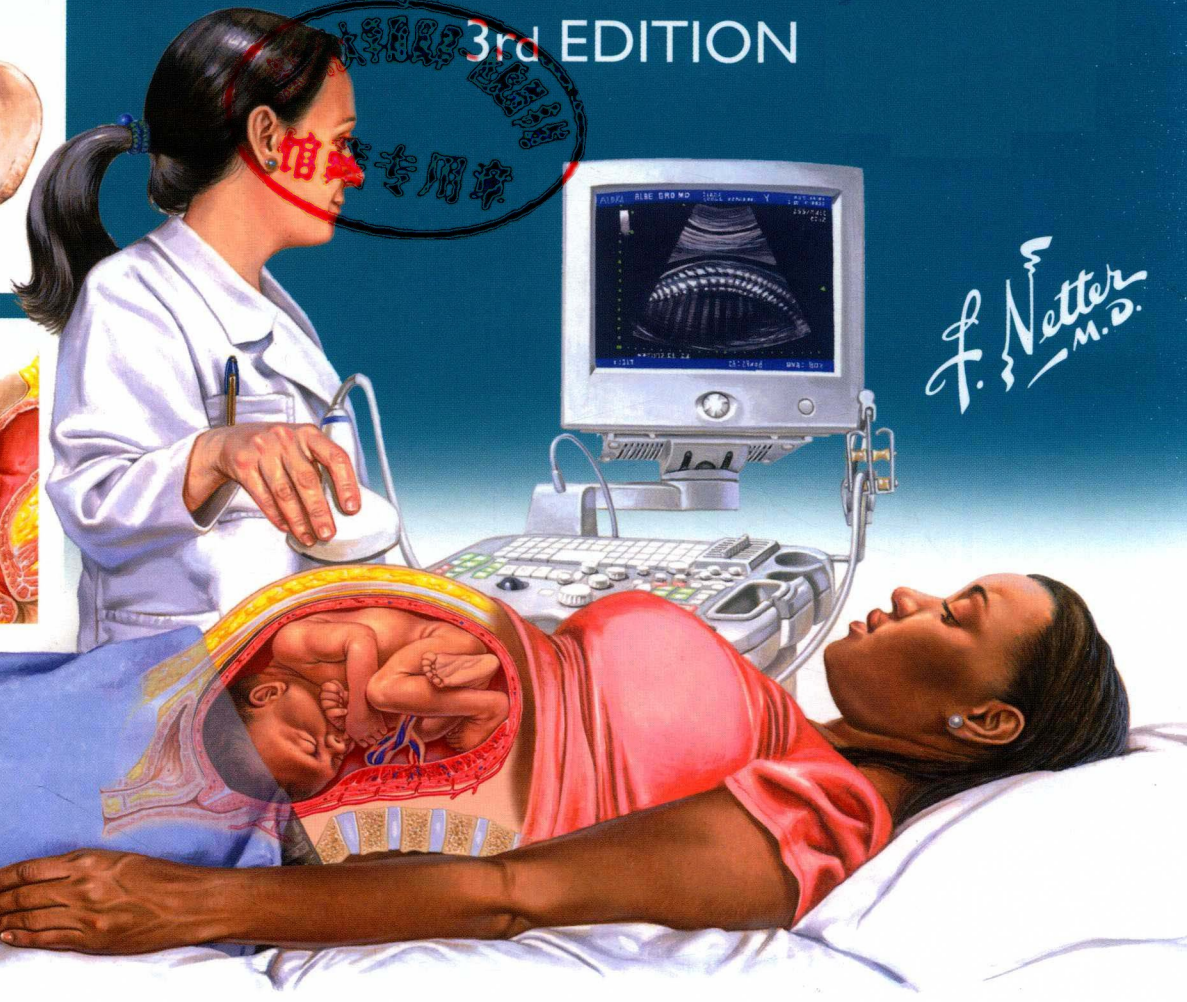

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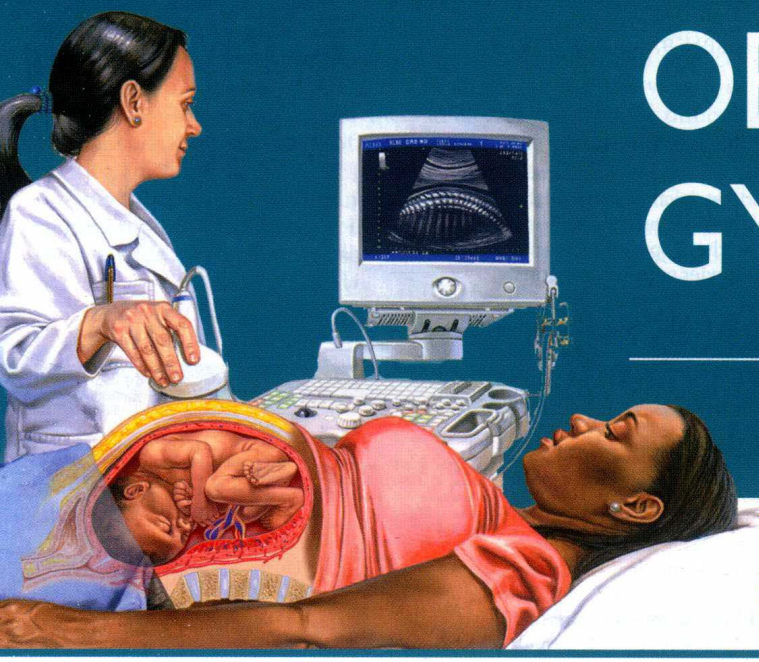

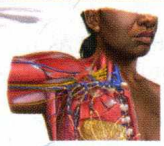

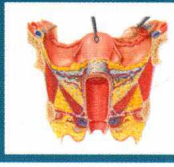

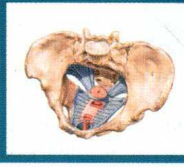

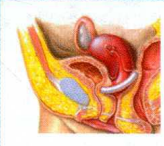

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PREFACE

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 AUTHOR

Although Roger P. Smith, MD, has spent much of his career in academic medicine and has a *curriculum vitae* that is appropriately long, he regards himself as a clinician. Dr. Smith received his undergraduate education at Purdue University and his medical education, internship (in General Surgery), and residency at Northwestern University in Chicago. He then spent almost 10 years in a multidisciplinary group practice at the Carle Clinic in Urbana, Illinois, before moving to the Medical College of Georgia in 1985, where he was Chief of the Section of General Obstetrics and Gynecology. In 1999, Dr. Smith joined the University of Missouri–Kansas City, where he served as Vice Chair and Residency Program Director until 2008. In 2011, he became the Robert A. Munsick Professor of Clinical Obstetrics and Gynecology and Director of the Division of General Obstetrics and Gynecology at Indiana University. Since 2016, he has served as the Assistant Dean for Graduate Medical Education and Professor of Clinical Biologic Sciences, Charles E. Schmidt College of Medicine, Florida Atlantic University, in Boca Raton, Florida. In addition to his other duties, he is currently President of the Association of Professors of Gynecology and Obstetrics (APGO) and the Central Association of Obstetricians and Gynecologists (CAOG).

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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CONTENTS

SECTION

Embryology

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

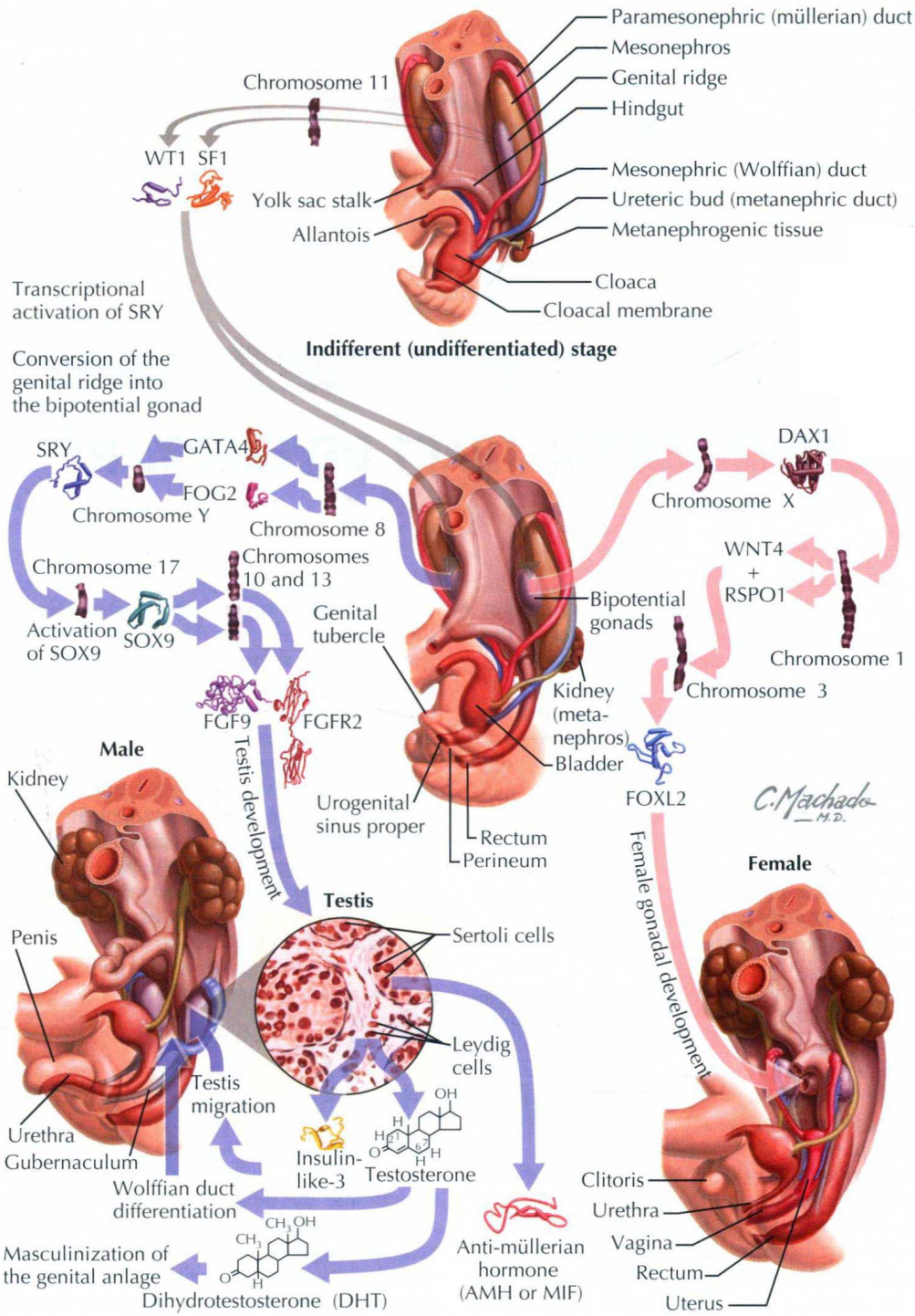

Figure 1.1 Genetics and biology of early reproductive tract development

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).

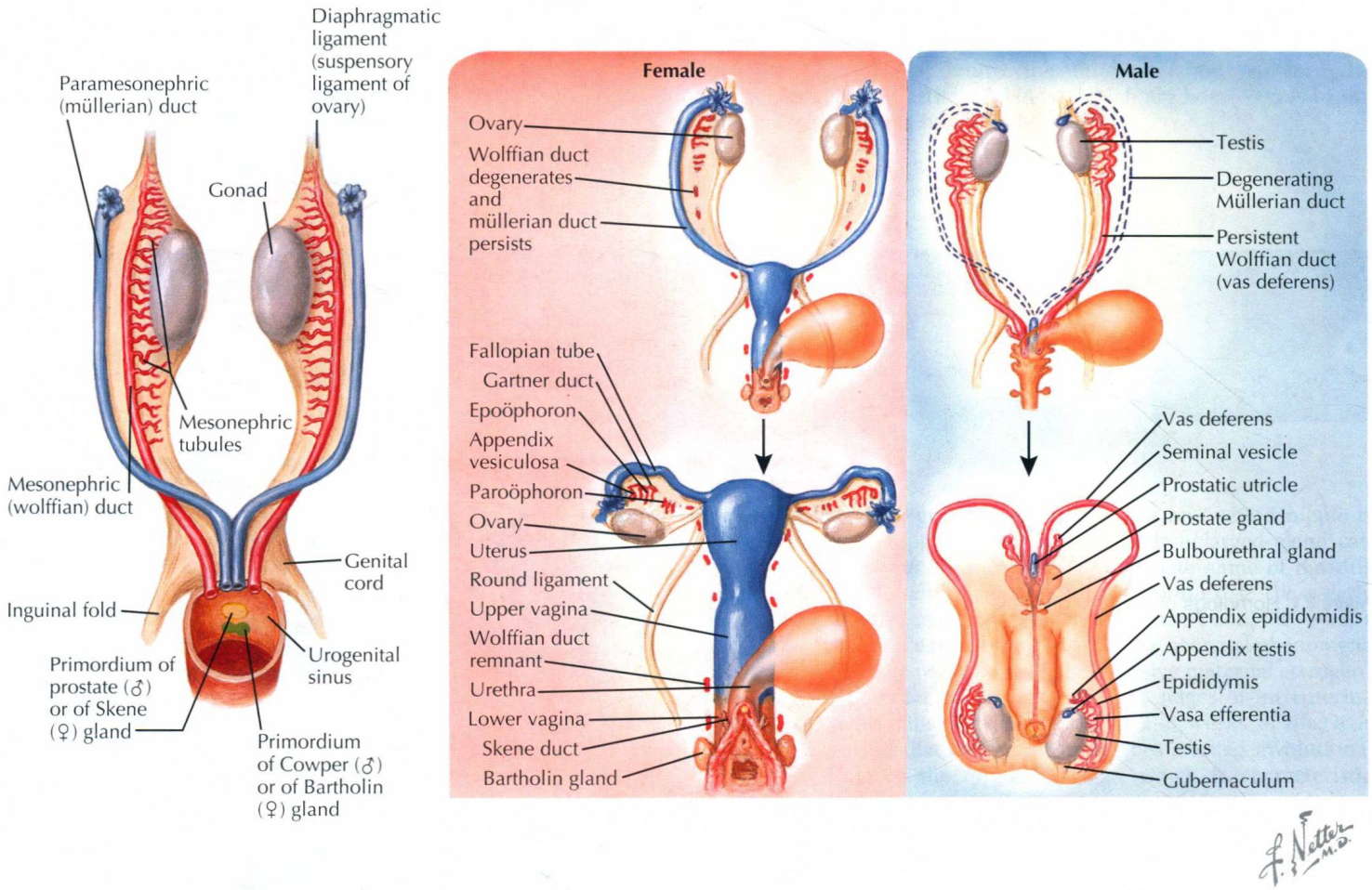

Figure 2.1 Homologs of the internal genitalia

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.

LOWER GENITAL TRACT DEVELOPMENT 3

The vagina develops from paired solid endoderm outgrowths of the urogenital sinus, the sinovaginal bulbs. These grow caudally as a solid core toward the end of the uterovaginal primordium. This core constitutes the fibromuscular portion of the vagina. The distal vagina develops as a diverticulum of the urogenital sinus near the müllerian tubercle, becoming contiguous with the distal end of the müllerian ducts. The sinovaginal bulbs then canalize to form the vagina. Roughly four-fifths of the vagina originates from the urogenital sinus and one-fifth is of müllerian origin. Abnormalities in this process may lead to either transverse or horizontal vaginal septa. The junction of the sinovaginal bulbs and the urogenital sinus remains as the vaginal plate, which forms the hymen. This remains

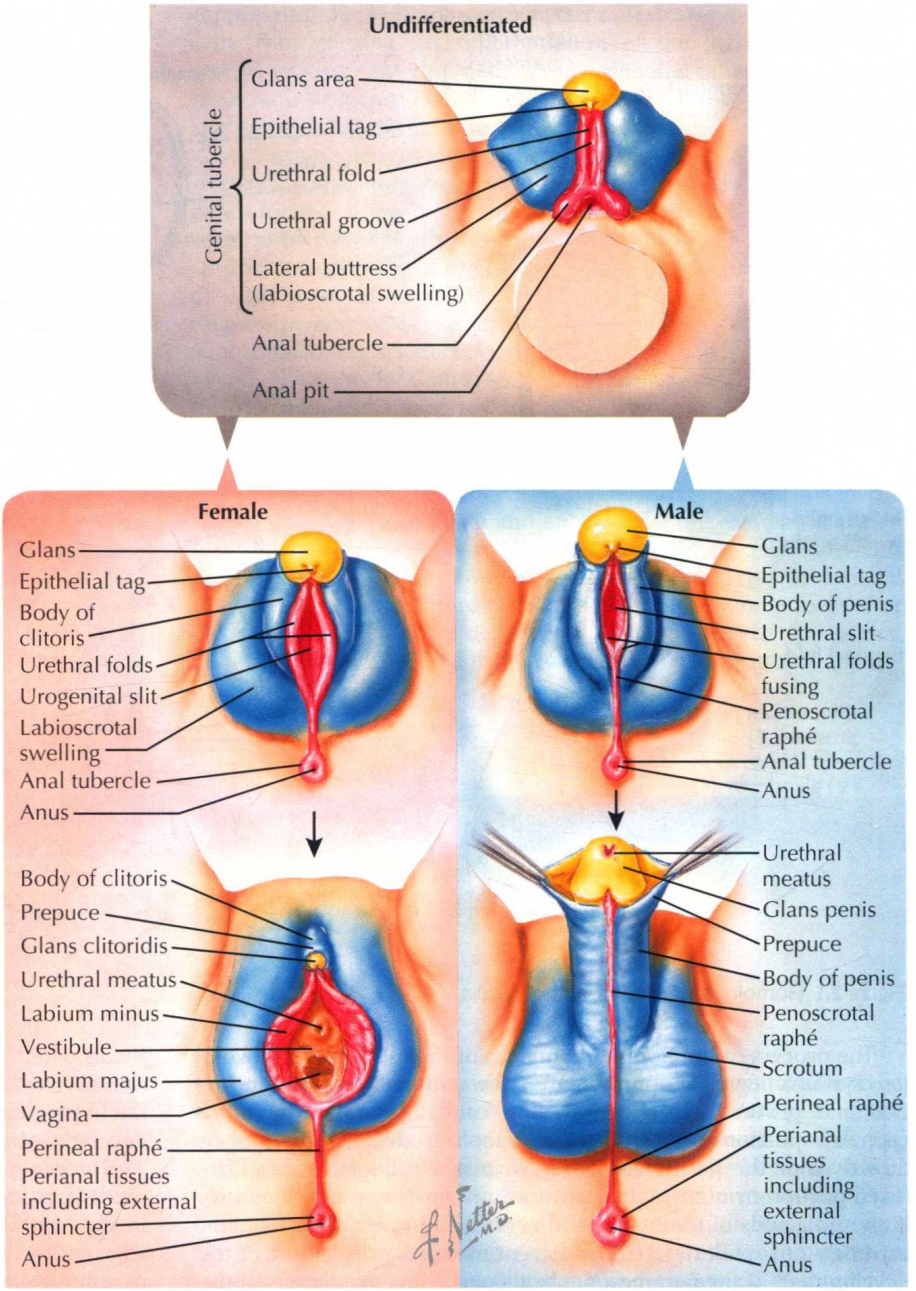

Figure 3.1 Homologs of external genitalia

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