Preface

The volume of information that today's OB/GYN practitioner needs to know is staggering and growing rapidly because of new basic research and clinical discoveries made daily. There is a critical need for both medical students and practicing physicians to have easy access to relevant information in a concise and carefully organized manner. This goal is difficult to achieve because 21st century medicine is characterized by a vast quantity of clinically relevant information. That is why the goal of this textbook is to clearly and concisely present the *essential* knowledge needed to practice state-of-the art obstetric and gynecologic medicine.

Women's health and the field of obstetrics and gynecology are critically important to the overall health of every society. Indeed, the health of women and their newborn children are key determinants of the potential of a society for advancing. The Association of Professors of Gynecology and Obstetrics has developed consensus learning objectives for medical students on clinical rotations in obstetrics and gynecology. These objectives, modified to focus on the most important and common clinical problems, are the basis for the extensive amount of material presented in this handbook.

Each chapter is presented in a clear, consistent manner and, where appropriate, begins with the definition of a particular condition, its common manifestations, and how it is routinely diagnosed. This is followed by a discussion of the prevalence and epidemiology of the condition, its etiology and pathophysiology, detailed information about the methods and protocols for its screening and/or detection, and the most recent, evidence-based information about its management. Each chapter uses photographs, tables, and other figures to present critical data and ideas, and ends by emphasizing the key clinical points. Within most chapters, an "Evidence" box is included to provide readers with exposure to how high-quality research information is used to guide clinical diagnosis and treatment. These "Evidence" boxes provide the kind of clinical information that inform the modern practice of OB/GYN medicine.

Medical students do not learn by a bolus infusion of a massive quantity of information. Rather, they learn through a continuous cycle of reading, thinking, talking, and doing. The purpose of this handbook is to assist the student and practitioner in this constant cycle of life-long learning and, more importantly, to offer the most evidence-based approach to the diagnosis and treatment of real OB/GYN patient problems.

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The Menstrual Cycle

The menstrual cycle can be divided into three phases:

1. Follicular phase
2. Ovulation
3. Luteal phase

Follicular Phase

Follicular development is a dynamic process designed to allow the monthly recruitment of a cohort of follicles, and the selection of one dominant follicle that will release a single mature oocyte each month.

In humans, the average length of the follicular phase ranges from 10 to 14 days (**Fig. 4.1**), and variability in this length is responsible for most of the variation in total cycle length. The follicular phase initiates at the first day of the menses. At this time the levels of gonadal steroids are low, and with the demise of the corpus luteum follicle-stimulating hormone (FSH) levels begin to rise recruiting a cohort of follicles.

In response to FSH, the follicles initiate the secretion of estrogen, which increases through the follicular phase and is responsible for endometrial growth. The rise in estrogen exerts a negative feedback on FSH at the hypophysis (pituitary gland) level.

In addition, the growing follicles produce inhibin B, which also suppresses FSH secretion by the pituitary.

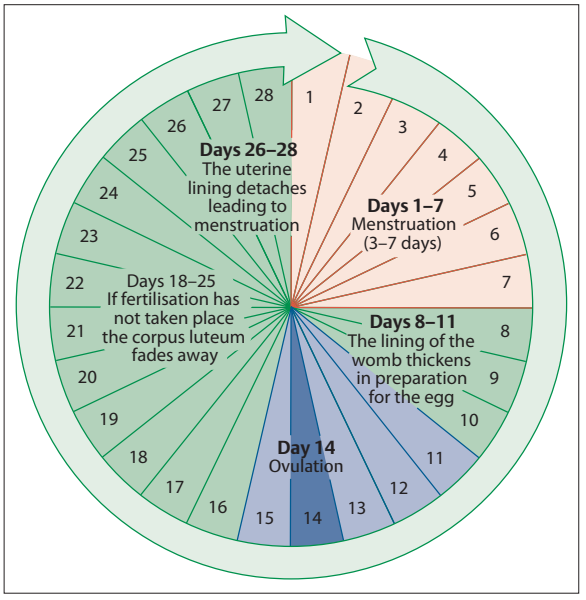

Fig. 4.1 A diagram of the menstrual cycle. The follicular phase constitutes the period beginning with menstruation and ending at ovulation, which is approximately 14 days in most women.

Conversely, the rise in estrogen levels at the beginning of the cycle produces a negative effect on the secretion of luteinizing hormone (LH), but late in the follicular phase LH levels increase dramatically.

During the follicular phase, hormonal feedback promotes the orderly development of a single dominant follicle, which is destined to ovulate from a period of initial growth of a primordial follicle through the stages of the preantral, antral, and preovulatory follicular growth (**Fig. 4.2**).

This phase corresponds to the proliferative phase in the uterus, in which there is building of the endometrial lining. In the middle of the follicular phase of menstrual cycle, after growth of a follicle has been achieved, local concentrations of prostaglandins and proteolytic enzymes induce the extrusion of the oocyte through the follicular wall, and ovulation occurs.

After ovulation, the menstrual cycle enters the luteal phase, the ruptured follicle becomes the corpus luteum and secretion of both progesterone and estrogen provide the adequate environment for the fertilized oocyte to implant in the endometrium. This phase corresponds to the secretory phase in the uterus.

If fertilization occurs, the secretion of human chorionic gonadotropin (HCG) by the embryo rescues the corpus luteum, allowing the continued secretion of progesterone and estrogen to sustain the pregnancy. If fertilization does not occur, the corpus luteum dies, which causes a drop in progesterone and estrogen levels and eventual shedding of the endometrium (menses).

Ovarian Follicular Development

Most oogonia are lost during fetal development, and the remaining follicles are recruited during the reproductive years until menopause occurs, in which the oocyte reserve is depleted.

During fetal development, the oogonia are arrested at the diplotene stage of the prophase in the first meiosis, the germ-cell process of reduction division. At this stage, a single layer of 8 to 10 granulosa cells surrounds the oogonia to form the primordial follicle. Those oogonia that fail to be properly surrounded by granulosa cells undergo atresia.

When the developing oogonia begin to enter the meiotic prophase I, they are known as primary follicles (**Fig. 4.3**), or oocytes, and remain arrested in this phase, until the time of ovulation, by a probable stasis mechanism involving an oocyte maturation inhibitor (OMI) produced by granulosa cells.

It is believed that the inhibitory action of this substance is achieved via gap junctions connecting the oocyte to its surrounding granulosa cells. When the LH surge at midcycle occurs, the gap junctions are disrupted, and the connection between the oocyte and granulosa cells is interrupted, allowing meiosis I to resume.

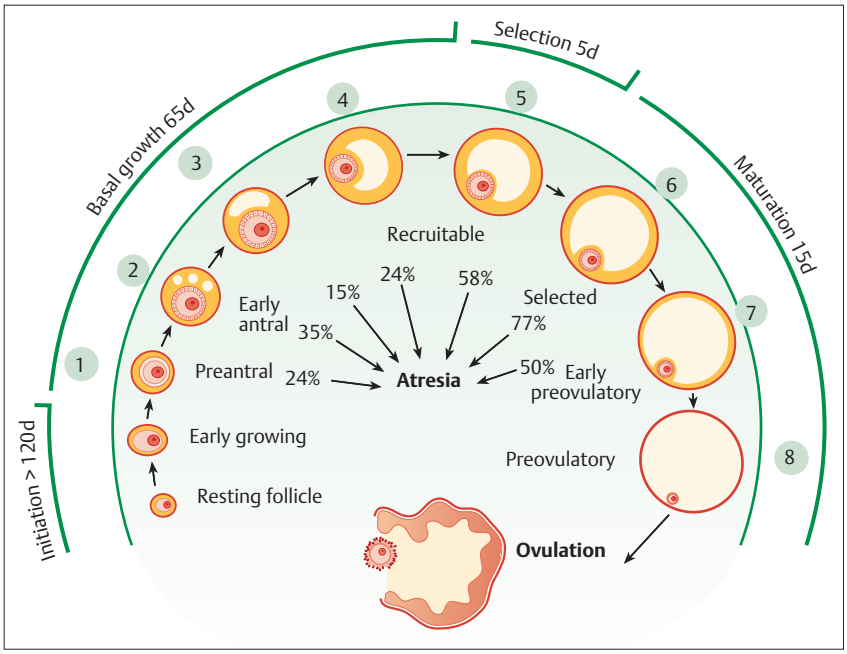

Fig. 4.2 Folliculogenesis and the classes of growing follicles in the human ovary. The early stages of folliculogenesis proceed very slowly, and it has been estimated that in humans the process can take more than 300 days. Even in the preantral stage (class 1), many growing follicles fail to survive, and degenerate through a process termed follicular atresia. Growing follicles enter class 2 usually in the late luteal phase, class 3 between late luteal and early follicular phases, class 4 during late follicular phase, and become recruitable class 5 follicles during late luteal phase.

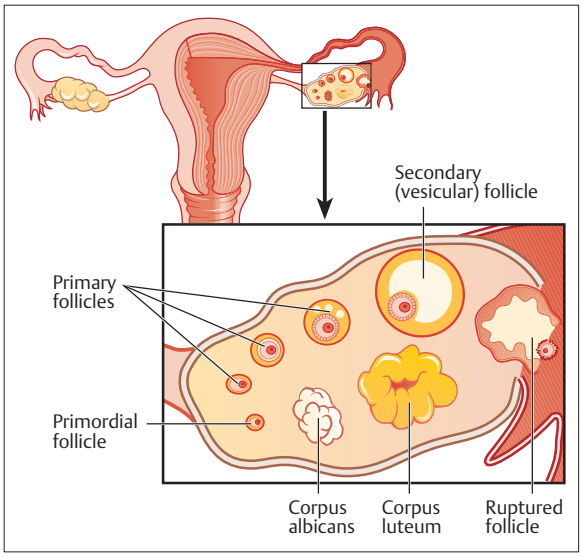

Fig. 4.3 The life cycle of a follicle.

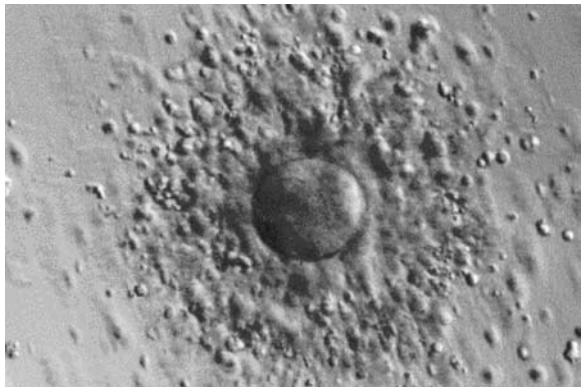

Fig. 4.4 A human oocyte with surrounding granulosa cells, after aspiration.

Primordial Follicles

In each cycle there is growth of a cohort of oocytes. The initial recruitment and growth of the primordial follicles is independent of gonadotropin and affects a cohort over several months. The factor(s) responsible for the recruitment in each cycle is unknown. After initial recruitment, control of follicular growth and differentiation shifts from gonadotropin-independent to gonadotropin-dependent growth, presumably by FSH. The action of FSH promotes growth of the oocyte and expansion of the granulosa cells (**Fig. 4.4**) from a single layer to a multilayer of cuboidal cells.

Preantral Follicle

Driven by the stimulus of FSH, the zona pellucid, a glycoprotein-rich substance, is formed, which separates the oocyte from the surrounding granulosa cells. Simultaneously with the proliferation of granulosa cells, there is proliferation of theca cells in the stroma bordering the granulosa. Both granulosa and theca cells function synergistically to produce estrogen, which is then secreted into the circulation. One of the follicles attains dominance over the rest of the cohort, which undergo atresia.

The mechanism for selection of the dominant follicle is still not clear, but follicular development can be explained by the "two-cell, two-gonadotropin theory," which states that during follicle development, steroid hormone synthesis takes place in a compartmentalized

6 Pap Smear and Human Papilloma Virus Testing

Benjamin Piura and Ruthy Shaco-Levy

After breast cancer, cervical cancer is the second most common malignancy diagnosed in women worldwide. Approximately 500000 new cases of cervical cancer are diagnosed each year, and nearly 250000 women die of this disease each year worldwide.

Cervical cancer is the most common malignancy encountered in women in developing countries. The majority of these cases occur in countries with limited or no effective screening programs using the Papanicolaou (Pap) smear test[1] for detecting cervical cellular abnormalities, which places women at a greater risk for developing cervical cancer. In the United States, Finland, Sweden, Iceland, and other developed countries where Pap smear screening is widely used, rates of cervical cancer have noticeably dropped up to 50% over the past 20–30 years (see Evidence Box 6.1).

However, health disparities prevent more lives from being saved with Pap smear screening, even in developed countries. Indeed, despite the test's widespread availability in the United States, for example, more than 10000 new cases of cervical cancer are diagnosed each year, and almost 4000 women die each year unnecessarily from this preventable disease. About 50% of women with cervical cancer in the United States did not have a Pap smear test in the preceding 3 years, and an additional 10% had not been screened in the past 5 years.

Nevertheless, more than 50 million Pap smears are performed each year in the United States alone, and 7% (3.5 million) of these give abnormal test results. It has been estimated that women who never had a Pap smear have a 3.5% risk of developing cervical cancer, whereas the risk is reduced to 0.8% with Pap smear screening. Since infection with human papilloma virus (HPV) has been found in almost all cervical cancers, testing for the presence of high-risk HPV types in cervical samples has now become a part of routine clinical work-up in women with equivocal Pap smear test results.

[1] In 1928, George Papanicolaou began sampling vaginal cells, speculating that the presence of any atypical cells might predict the development of cervical cancer. It was only in 1943 when he and Herbert Traut published a monograph on the topic that the Papanicolaou (Pap) smear became the standard cervical cancer screening test. In 1947, Ayers introduced a specially designed wooden spatula (Ayer spatula) for the direct collection of cells from the uterine cervix.

Pap Smear Terminology

There are a number of outdated terminologies regarding Pap smear results (**Table 6.1**). The lack of a common terminology initially resulted in widespread confusion about what really constitutes an abnormal test result. This confusion necessitated further action. In December 1988, a National Cancer Institute workshop held in Bethesda, Maryland, provided for the first time a consensus, now known as the Bethesda System, on how to properly read Pap smears. The result was initial guidelines designed to decrease the variability among laboratories reporting the results.

The three most important contributions of this Bethesda System were:

1. Establishing a special category of abnormal squamous cells of undetermined significance (ASCUS)
2. Organizing the four grades of atypical squamous cells (mild, moderate, severe, and carcinoma in situ) of the old classifications into two distinctive groups: low-

Table 6.1 Historical Pap smear terminology

Papanicolaou	United States	United Kingdom
Normal	Normal	Normal
Inflammatory	Inflammatory	Inflammatory
Atypical cells	Mild atypia Moderate atypia	Mild dyskariosis Moderate dyskariosis
Carcinoma in situ	Severe atypia	Severe dyskariosis
Carcinoma	Carcinoma	Carcinoma

grade squamous intra-epithelial lesion (LSIL) (**Fig. 6.4**), and high-grade squamous intra-epithelial lesion (HSIL) (**Fig. 6.5**). LSIL (previously referred to as "mild atypia") is compatible with grade 1 cervical intraepithelial neoplasia (CIN 1) and HSIL (encompassing "moderate atypia," "severe atypia," and "carcinoma in situ") is compatible with grade 2 or 3 cervical intra-epithelial neoplasia (CIN 2, 3) and carcinoma in situ (CIS)

3. Establishing a new category of abnormal glandular cells of undetermined significance (AGCUS)

Key Terminology Changes in the 2001 Bethesda System

The Bethesda System was revised in 1991 and 2001. The 2001 Bethesda System (**Table 6.2**) reflects the most current knowledge about the biology of Pap test abnormalities and addresses new screening technologies such as the liquid-based, thin-layer Pap smear and HPV testing. It recommends dividing the category of atypical squamous cells (ASCs) into two subcategories: a) atypical squamous cells of undetermined significance (ASCUS), and b) atypical squamous cells that cannot exclude high-grade intra-epithelial lesion (ASC-H).

Overall, among all women with ASC, the risk of developing invasive cancer is low (0.1–0.2%). Nevertheless, the prevalence of CIN 2, 3 confirmed by biopsy among women with ASC is 7–12%, whereas the prevalence of CIN 2, 3 confirmed by biopsy among women with ASC-H ranges from 26% to 68%. Rates of high-risk HPV DNA positivity are 40–51% among women with ASCUS, whereas they are 74–88% among women with ASC-H. Consequently, ASC-H should be considered to represent equivocal HSIL and a productive HPV infection. Thus, the performance of HPV testing allows for clear statements regarding the meaning of an ASC interpretation.

The 2001 revisions to the Bethesda System also eliminated the category of AGCUS (atypical glandular cells of undetermined significance) and identified three subcategories of atypical glandular cells (AGCs):

- AGC not otherwise specified
- AGC favoring neoplasia
- adenocarcinoma in situ (AIS)

Table 6.2 The 2001 Bethesda System

Specimen adequacy
Satisfactory for evaluation (8000–12 000 well-visualized squamous cells for conventional smears and 5000 squamous cells for liquid-based preparations (*note presence/absence of endocervical/transformation zone component*—there should be at least 10 well-preserved endocervical or squamous metaplastic cells)
Unsatisfactory for evaluation (specimens with >75% of epithelial cells obscured)

Table 6.2 Continued

General categorization
Negative for intra-epithelial lesion or malignancy
Epithelial cell abnormality
Other
Interpretation/result
Negative for intra-epithelial lesion or malignancy
• **Organisms** – *Trichomonas vaginalis* – Fungal organisms morphologically consistent with *Candida* species – Shift in flora suggestive of bacterial vaginosis – Bacteria morphologically consistent with *Actinomyces* species – Cellular changes consistent with herpes simplex virus
• **Other non-neoplastic findings** Reactive cellular changes associated with: – inflammation (includes typical repair) – radiation – intrauterine contraceptive device Glandular cells status posthysterectomy Atrophy
Epithelial cell abnormalities
• **Squamous cell** – Atypical squamous cells (ASC) – of undetermined significance (ASC-US) – cannot exclude HSIL (ASC-H) (*5–10% of ASC cases overall*)
• **Low-grade squamous intra-epithelial lesion (LSIL)** (generally a transient infection with HPV) encompassing: human papilloma virus/mild dysplasia/cervical intra-epithelial neoplasia (CIN) 1
• **High-grade squamous intra-epithelial lesion (HSIL)** (more often associated with HPV persistence and higher risk of progression) encompassing: moderate and severe dysplasia, carcinoma in situ; CIN 2 and CIN 3
• **Invasive squamous cell carcinoma**
• **Glandular cell**
• **Atypical glandular cells (AGC)** (*specify endocervical, endometrial, or glandular cells not otherwise specified*)
• **Atypical glandular cells (AGC)** (*specify endocervical, endometrial, or glandular cells not otherwise specified*)
• **Endocervical adenocarcinoma in situ (AIS)**
• **Invasive adenocarcinoma**
Other
Endometrial cells in a woman ≥40 years of age

Reproduced with permission from Salomon D et al. The 2001 Bethesda System: terminology for reporting results of cervical cytology. JAMA 2002; 287:2116.

Types of Pap Smear, Their Sensitivity, and Screening Guidelines

There are two main types of Pap smear: a conventional Pap smear, and liquid-based Pap smear.

Conventional Pap Smear

For a conventional Pap smear, the cell specimen on the collection instrument is spread across a glass slide and fixed to it by either spraying a fixative on the glass slide (**Fig. 6.1**) or placing it in a vial containing an ethanol fixative.

Ideally, samples for this type of a Pap smear should be obtained from three locations: (i) the endocervical canal (E), (ii) the exocervix (including the entire transformation zone) (C), and (iii) posterior vaginal pool (posterior fornix) (V). The samples can be smeared separately on three glass slides that are marked with the letters E, C, or V, respectively.

Some investigators, however, do not advocate collecting samples from the posterior vaginal pool. Nevertheless, for screening, all three samples can be smeared and mixed on one glass slide. The smear should be thick enough that it is not transparent.

Pap smears on a glass slide should be evaluated by a trained laboratory technician or cytopathologist, utilizing a regular light microscope (**Fig. 6.2**). In 1997, the US Food and Drug Administration (FDA) approved two systems for routine use as quality control (rescreening) devices. Although studies have shown that these systems can catch problems not detected on a microscopic evaluation of Pap smears, such technical triumphs have been overshadowed by conflicting opinions about their cost-effectiveness and accuracy among cytopathology professionals, clinicians, patients, and device manufacturers.

Liquid-Based, Thin-Layer (ThinPrep) Pap Smear

A ThinPrep Pap smear involves rinsing or dropping the collection instrument into a vial containing a liquid fixative (**Fig. 6.3**). The cells obtained are filtered, placed on a

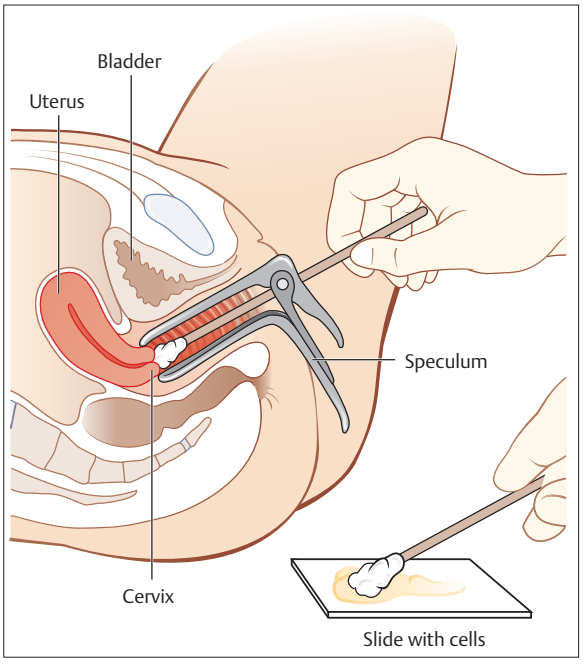

Fig. 6.1 The proper technique for obtaining a sample of cells for a Pap smear.

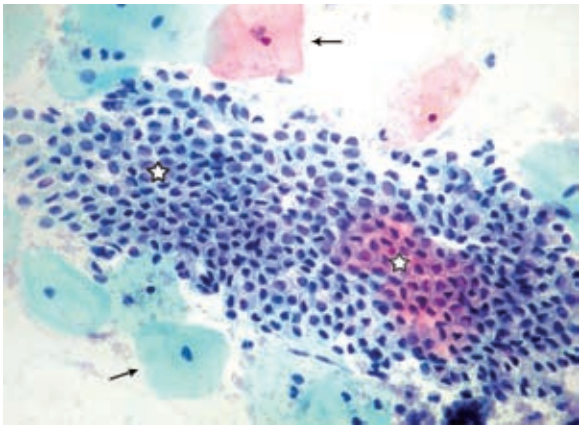

Fig. 6.2 The normal Pap smear. Benign superficial squamous cells (arrows) and endocervical cells (white stars) can be visualized by light microscopy.

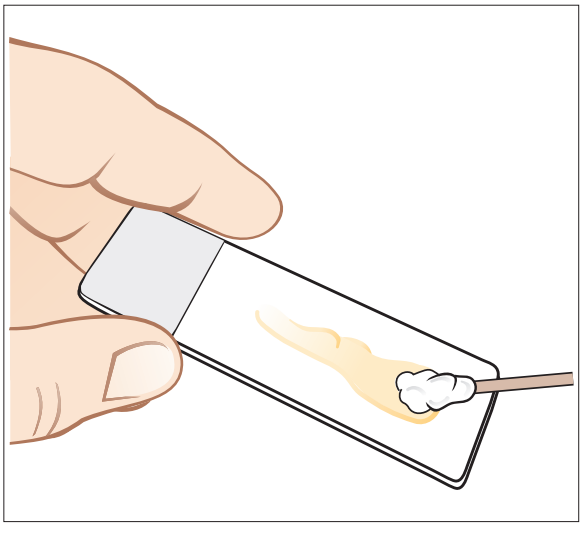

Fig. 6.3 A liquid-based Pap smear requires the collection instrument to be inserted into a vial containing a liquid fixative.

12 Immediate and Postpartum Newborn Care

Julieta E. Irman and Gustavo F. Leguizamòn

The neonatal period comprises the four weeks following delivery. During this phase, the newborn must rapidly adjust to its extrauterine life. To survive and achieve normal development, it must make major physiologic changes, including increased respiratory gas exchange, switching from fetal to neonatal circulation, and taking over its own thermoregulation. This chapter addresses these major physiologic changes involved in the newborn's transition to its extrauterine life. It also offers the basics of medical interventions commonly used to assist newborns throughout this critical transition.

Definitions

Apgar score: The Apgar score provides an objective method of evaluating the physical condition of a newborn infant soon after delivery. The score takes into account the heart rate, respiratory effort, muscle tone, skin color, and responce to a catheter in the nostril. Each of these objective signs can receive 0, 1, or 2 points, and each test is performed at 1 minute and 5 minutes after delivery. A common mnemonic is APGAR: **A**ppareance, **P**ulse, **G**rimace, **A**ctivity and **R**espiration.

Hyperbilirubinemia: This term refers to excess bilirubin in the blood, usually characterized by jaundice or yellowing of the eyes.

Hyperthermia: Abnormally high body temperature that occurs when the body's metabolic heat production or environmental heat load exceeds the normal heat loss capacity (or when heat loss is impaired).

Hypothermia: A dangerous lowering of body-core temperature, caused by losing heat faster than it is produced by the body.

Postpartum: A term used to describe something that occurs after childbirth, usually involving the mother.

Tachypnea: This refers to an excessively rapid respiratory rate, defined as greater than 20 breaths per minute.

Transition: The transition period is referred to as 6–12 hours after birth, when the baby goes through physiological adaptation to extrauterine life.

Vaginal introitus: This is the anatomical term for the vaginal opening.

The Respiratory Transition

The alveoli of the fetus in utero are filled with fluid that must be cleared during the initial transitional period, so that the baby can breathe. In addition, to ensure effective pulmonary perfusion and to match perfusion to ventilation, the baby must be able to increase blood flow to the lungs.

During the last few weeks of pregnancy, there is a maturation and recruitment of sodium channels in the epithelial cells of the lungs in response to endogenous steroid and catecholamine surges that are triggered by the onset of labor. It is through these epithelial sodium channels that a significant part of the fluid in the fetus' lungs is purged. Liquid is also driven out of the fetus' lungs through the pulmonary epithelium into the vasculature as well as through the mechanical *squeeze* and Starling forces that occur during the process of labor and vaginal delivery.

Newborns sometimes have difficulty purging all the liquid from their lungs, particularly if they are late preterm babies. As a result, they may exhibit respiratory difficulties that require stabilization and immediate supportive therapy, such as supplemental oxygen and assist-

ed ventilation. They are also at increased risk for associated morbidities.

Wang et al. estimated that nearly one-third of late preterm newborns exhibit respiratory difficulties. Neonates who are born by cesarean delivery before labor begins are at increased risk for respiratory distress, as are late preterm males compared with late preterm females. Hemodynamic instability caused by hypothermia or hypoglycemia may worsen the newborn's underlying respiratory distress.

Acute respiratory distress syndrome (RDS) is the most common respiratory condition experienced in newborns. The condition, which is characterized by severe difficulty in breathing and related complications, occurs primarily in neonates born between 34 and 36 weeks' gestation. It is the fourth leading cause of death for neonates in the United States (**Fig. 12.1**).

Late preterm infants also are at increased risk of having low Apgar scores, transient tachypnea of the newborn (TTN), persistent pulmonary hypertension, and respiratory failure. TTN and RDS are both common respiratory conditions in the late preterm newborn and are related to difficulty in clearing fluid from the lungs or a surfactant deficiency, or both. The protocols for managing a child in respiratory distress are covered later in this chapter.

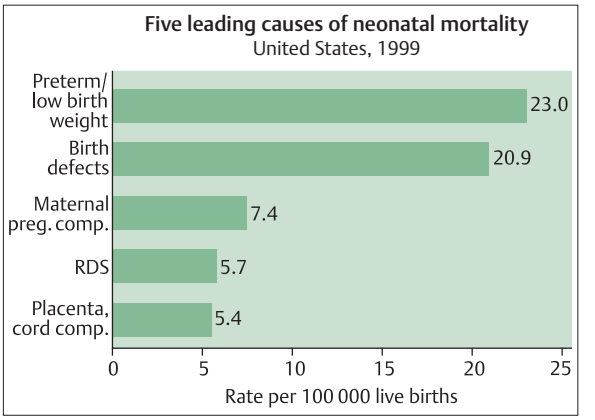

Fig. 12.1 Respiratory distress syndrome (RDS) is the fourth leading cause of death among neonates in the United States at approximately 6 per 100 000 live births. Adapted from the National Center for Health Statistics, 1999 period birth/infant death data; prepared by March of Dimes Perinatal Data Center, 2002

The Circulatory Transition

As described in Chapter 9, there is a substantial difference between the circulation of a fetus and a neonate. In the womb, well oxygenated blood from the placenta is delivered to the fetus through the umbilical vein. The umbilical vein gives off branches to the left lobe of the liver and then continues as the ductus venosus (**Fig. 12.2**). The left hepatic vein fuses with the well oxygenated ductus venosus and flows into the inferior vena cava to reach the left atrium by crossing thorough the foramen ovale. Then, via the aorta and carotid circulation, this blood supplies the brain and upper body. The right ventricular output is directed through the ductus arteriosus to the descending aorta.

Since the pulmonary vascular resistance is high and the mean pulmonary artery pressures are higher than aortic pressures, the flow is mainly directed toward the ductus arteriosus, leaving the pulmonary circulation with 5–10% of the ventricular output. Finally, fetal blood returns to the placenta through the umbilical arteries.

However, when delivery occurs, clamping the umbilical cord provokes a sudden decrease in the amount of neonatal blood draining toward and away from the baby. This leads to a sudden increase pressure in the systemic circulation and a decrease in the right side circulation, reversing the right-to-left shunt and leading to the closure of the ductus arteriosus. Prostaglandins play a pivotal role in maintaining patency of the ductus arteriosus in utero and in its closure during early neonatal life.

The Thermoregulatory Transition

Thermoregulation is a critical physiologic function that is closely related to the transition and survival of the infant. An understanding of transitional events and the physiologic adaptations that must be made is essential in helping the neonate to maintain its thermal stability. This section reviews neonatal thermal regulation, heat loss and gain, and infant thermoregulatory behavior. Measures to ensure thermal stability for the neonate are discussed later in this chapter.

Neonatal Thermoregulation

Babies are not as adaptable as adults to temperature change. A baby's body surface is about three times greater than an adult's, relative to the weight of the body. Babies can lose heat rapidly, as much as four times more quickly than adults. Premature and low birth weight babies usually have little body fat and may be too immature to regulate their own temperature, even in a warm environment. Even full-term and healthy newborns may not be able to maintain their body temperature if the environment is too cold.

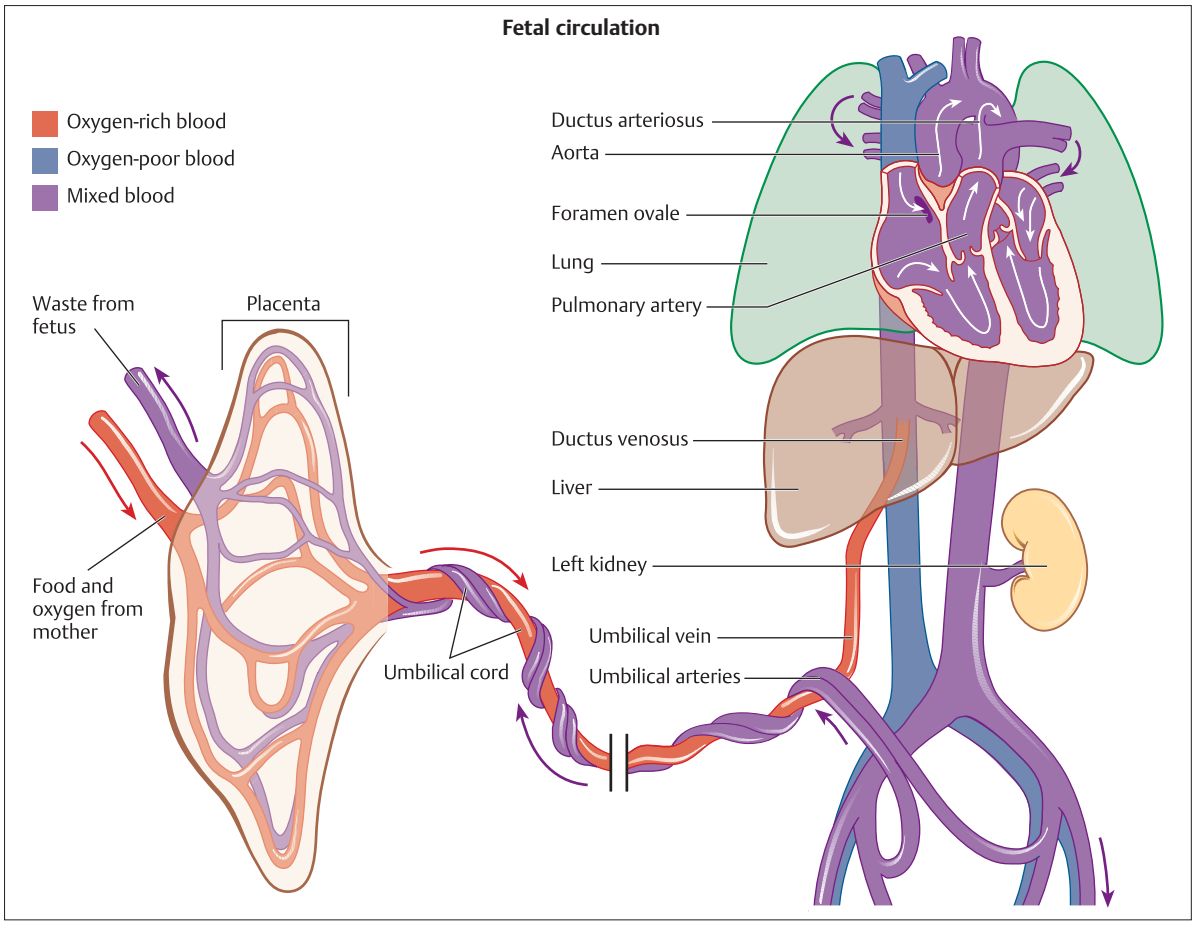

Fig. 12.2 The fetus receives all the necessary nutrition, oxygen, and life support from the mother's placenta via the blood vessels in the umbilical cord. Waste products and carbon dioxide from the fetus are sent back through the umbilical cord and placenta to the mother's circulation to be eliminated.

When babies are stressed by cold, they use energy and oxygen to generate warmth. If skin temperature drops just one degree from the ideal 36.5 °C (97.7 °F), a baby's oxygen use can increase by 10%. Keeping babies at optimal temperatures, neither too hot nor too cold, enables them to conserve energy and build up reserves. This is especially important when babies are sick or premature.

For optimal thermal stability the baby's temperature must be kept within the Thermal Neutral Zone (**Fig. 12.3**). Once stabilized within the Thermal Neutral Zone, the baby's energy expenditure and oxygen consumption are minimized, promoting optimal growth.

Thermoregulation is controlled by the hypothalamus, a region of the central nervous system responsible for certain metabolic process and other activities of the autonomic nervous system. Thermal stimuli from the skin and body's deep (central) thermal receptors provide information on body temperature to the hypothalamus (**Fig. 12.4**). It is in the hypothalamus that sensory information describing thermal status throughout the body is processed and compared against the temperature set

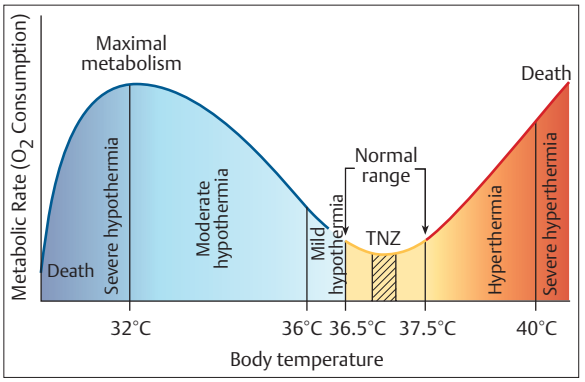

Fig. 12.3 Thermal stability is essential for every baby, especially those with limited metabolic capacity due to illness, prematurity, or low birth weight. Research has shown that the Thermal Neutral Zone for preterm babies less than 30 weeks old is less than 0.5 °C.

35 Pelvic Relaxation, Urinary Incontinence, and Urinary Tract Infection

Jon I. Einarsson

Pelvic Relaxation

Incidence

Pelvic relaxation or pelvic organ prolapse is a common condition, with a recent study finding the prevalence of stage 2 and greater pelvic organ prolapse to be 37% in a population of 1004 women seeking care at a gynecologic clinic. While some of these women are asymptomatic, genital prolapse remains one of the most common reasons for gynecologic surgery in women after the fertile period.

Subtypes and Anatomy

Pelvic organ prolapse is often divided into three main subtypes, depending on where the prolapse is located (**Fig. 35.1**). In anterior prolapse (cystocele), the urinary bladder protrudes into the vagina. In posterior prolapse (rectocele), the rectum and/or the rectosigmoid protrudes into the vagina. Apical prolapse occurs when the cervix or the vaginal apex (following a hysterectomy) falls down through the vagina. Anterior and posterior prolapse are usually defects in the endopelvic fascia, which is a thick layer of connective tissue in the vagina, that is, the muscularis layer of the vaginal wall. This connective tissue can become detached from the normal attachments to the pelvic sidewall and/or become weakened or torn during childbirth or through wear and tear such as with chronic cough or constipation. Apical prolapse is more commonly associated with detachment or tearing of the uterosacral ligaments and the cardinal ligaments that connect to the pericervical ring to maintain the vaginal apex in a normal anatomic position.

Symptoms

Pelvic organ prolapse causes pelvic pressure, pain, painful intercourse and significantly impacts the quality of life of those who suffer from it. A cystocele can cause frequent urination or urinary retention, and a rectocele can cause difficulty in emptying the rectum during bowel movements.

Etiology

Several etiologic factors that contribute to pelvic organ prolapse have been identified such as higher age, increased parity, obesity, and heritability.Recent evidence also suggests that women with pelvic organ prolapse have smaller amounts of collagen in the fibrous connective tissue of the endopelvic fascia.

Diagnosis

Diagnosis is made during a detailed pelvic exam. It is important to distinguish between prolapse in different vaginal compartments, since surgical treatment varies. A standardized system to evaluate and report pelvic organ prolapse, called the POP-Q system, is widely used in research and clinical practice. Some consider this to be too complex and prefer a simpler system, such as the Baden–Walken halfway system. In this system, 1st degree prolapse extends halfway to the introitus, 2nd degree prolapse extends to the introitus, 3rd degree prolapse extends halfway outside the introitus and 4th degree prolapse means that there is complete eversion of the pelvic organs (procidentia).

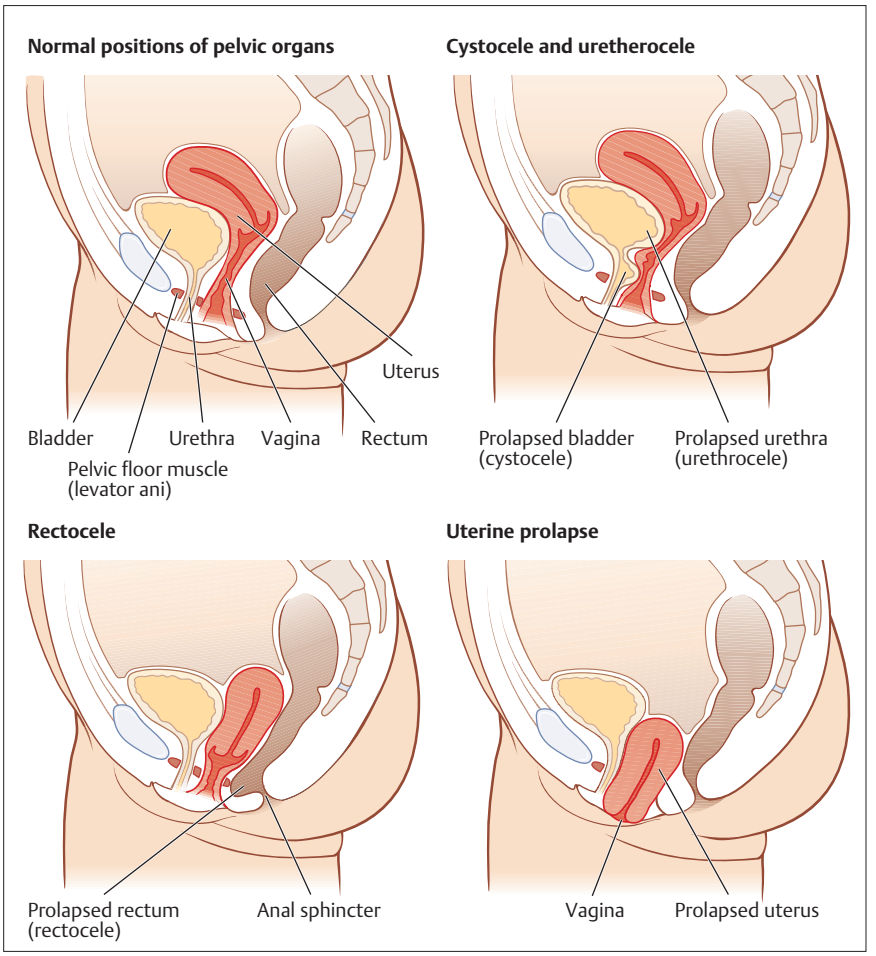

Fig. 35.1 Anatomy of the most common types of pelvic organ prolapse.

Treatment

It is important to point out that asymptomatic pelvic organ prolapse usually does not require any therapy. Since conservative measures such as Kegel exercises or physical therapy do not seem to be effective, the main therapeutic option for symptomatic pelvic organ prolapse is reconstructive surgery, where defects in the endopelvic fascia are identified and repaired. While these surgical therapies can be effective, relapse rates of up to 50–60% have been reported. In order to reduce relapse rates, synthetic polypropylene mesh has been used to improve long-term success. While effective, synthetic mesh is costly and can cause erosions and, rarely, pelvic infections. The anterior and posterior compartment are usually repaired by re-approximating the endopelvic fascia with or without a mesh overlay. The "gold standard" treatment for apical prolapse is abdominal sacrocolpopexy with mesh (**Fig. 35.2**). Less invasive vaginal approaches such as the sacrospinous ligament fixation are especially applicable to older or less active patients. Recently, laparoscopic apical repairs have become more commonplace, thereby combining long-term durability and low morbidity. These are especially applicable to younger active women, since sacrocolpopexy is associated with a lower rate of dyspareunia than the vaginal repairs.

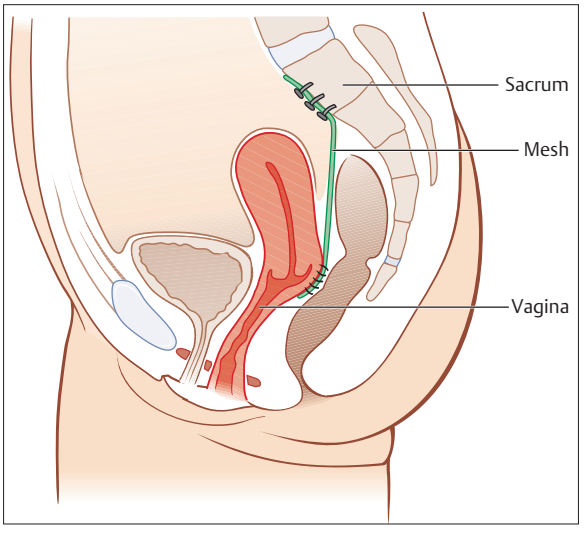

Fig. 35.2 Sacrocolpopexy involves attaching the vaginal apex to the promontory of the sacrum.

Urinary Incontinence

Incidence and Subtypes

Urinary incontinence, or involuntary leakage of urine, affects about 25% of premenopausal women and 40% of postmenopausal women.

The two most common types of urinary incontinence are stress incontinence and urge incontinence. Women who leak urine during coughing, sneezing, laughing, or lifting are considered to have stress urinary incontinence, and women who leak urine immediately following a strong urge to urinate have urge incontience. Urge incontinence is also associated with frequent urination (more than 12 times during the day) and nocturia (urinating more than once at night). Some women have mixed urinary incontinence, with components of both stress and urge incontinence.

Overflow incontinence is a rare type of urinary incontinence, where women do not have normal bladder sensation or are unable to adequately empty the bladder. This leads to frequent urination, urinary leakage, and frequent urinary tract infections because urine stays in the urinary bladder at all times.

Pathogenesis

If the urethral sphincter is weak, this can result in a subtype of stress urinary incontinence called intrinsic sphincter deficiency. The more common type is caused by too much mobility of the urethra (urethral hypermobility). The urethra is supported by connective tissue that can be weakened during childbirth, due to genetic defects in collagen or other connective tissue building blocks, or due to chronic cough or constipation. When the support of the urethra is weak or soft, the urethra sinks into the weakened connective tissue with increased intra-abdominal pressure, such as during cough or exercise. The pressure inside the bladder then becomes more than the pressure in the urethra leading to leakage of urine. This is similar to trying to stop a flow of water through a rubber hose by stepping on it. If the rubber hose is lying on soft grass, it is difficult to stop the flow of water by stepping on the hose, since the hose sinks into the grass and mud beneath it. However, if the hose is sitting on a hard surface, such as a sidewalk, it is much easier to stop the flow of water.

The causes of urge incontinence are more complex and often no specific cause is found. The most common cause is overactivity of the detrusor muscle of the bladder. The detrusor muscle is designed to be relaxed during bladder filling and only contract during bladder emptying. However, for a variety of reasons that are not fully understood, the bladder muscle sometimes becomes overactive, which leads to a strong urge to urinate. If the muscle contractions are very strong, this can lead to a sudden leakage of urine. Sometimes triggers can cause this, such as the sound of running water, but this may be completely unprovoked. The contractions of the bladder muscle are controlled by the autonomic nervous system. Therefore, women who have sustained an injury to the spine or have neurologic disorders such as multiple sclerosis and diabetes often have symptoms of urge incontinence.

Diagnosis

A detailed history is the first step in diagnosing urinary incontinence, inquiring about duration of symptoms, medications, fluid intake, when the leaking occurs and how much this affects quality of life. A urinary diary can be helpful both for the patient and the physician. The patient notes freqency and amount of urination as well as frequency and amount of fluid intake. She will also be asked to note any urine leakage, if there was a strong sense of urgency prior to the leak and what she was doing when it happened. A simple, validated three-item questionnaire can also be useful to discern between urge and stress incontinence in clinical practice (**Fig. 35.3**).

Simple urodynamics can be performed in the doctor's office with minimal equipment. The patient first produces a urine sample to check for an infection. The post-void residual can then be measured by using ultrasonography or by inserting a narrow catheter into the bladder. Normally, the bladder should empty completely during urination, but a post-void residual greater than 100 mL is generally considered abnormal.

Next, the bladder is filled with water through the catheter to determine whether the bladder distends normally. Most women will feel that their bladder is filling with urine at 100–150 mL. When the bladder is filled up to about 300 mL, most women would go to the bathroom if socially convenient. The maximum capacity of the bladder is normally about 400–450 mL, which is associated with an extreme urgency. If the bladder starts to contract during the filling process, this is an indication of an overactive detrusor muscle. After filling the bladder with about 300 mL the catheter is removed and the patient is asked to stand up and cough or jump to see if there is any leakage, which would indicate a diagnosis of stress incontinence.

A more sophisticated way to measure the function of the urinary bladder is with so called multichannel urodynamics. During this test, narrow catheters are placed into the bladder and the vagina. Additionally, small electrodes are placed on the inner thigh to measure the contractions of the pelvic muscles. The bladder is slowly filled with water, but here the amount of water is more accurately measured and the results are automatically drawn on a strip of

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Notes: Entries in *italics* refer to figures whilst those in **bold** refer to tables/boxed material.

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