3RD EDITION

OBSTETRIC AND GYNECOLOGIC DERMATOLOGY

Edited by

MARTIN BLACK

CHRISTINA AMBROS-RUDOLPH

LIBBY EDWARDS

PETER LYNCH

MOSBY
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MARTIN BLACK
MD FRCP FRCPATH
Emeritus Professor of Dermatological Immunopathology and Honorary Consultant Dermatologist
St. John's Institute of Dermatology
St. Thomas's Hospital
London, UK

CHRISTINA M. AMBROS-RUDOLPH MD
Associate Professor
Department of Dermatology
Medical University of Graz
Graz, Austria

LIBBY EDWARDS MD
Associate Professor of Dermatology
University of North Carolina, Chapel Hill;
Chief of Dermatology, Carolinas Medical Center
Mid-Charlotte Dermatology and Research
Charlotte, North Carolina, USA

PETER J. LYNCH MD
Program Director and Frederick G. Novy, Jr. Professor of Dermatology
Department of Dermatology
University of California, Davis
Sacramento, California, USA

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Preface

The first and second editions of this text, which we believe was the first ever to focus specifically on the full range of dermatologic problems encountered in obstetric and gynaecological practice, were very well received. For the third edition all chapters have been thoroughly updated and each presents a basic, sensible approach to a complex subject. We are particularly fortunate to have been able to recruit two eminent dermatologists who have long specialised in vulval dermatoses: Dr. Peter Lynch and

Dr. Libby Edwards have brought their considerable expertise together and have completely updated and largely re-written the sections on vulval dermatoses and tumours. All sections are profusely illustrated presenting numerous examples of common and unusual disorders or presentations. It is intended that the format of the illustrations and text will aid the user in comparing images of similar-appearing skin problems encountered in the clinic or office.

Further emphasis is also made in the text on practical suggestions for diagnostic approaches as well as outlining detailed and practical suggestions for therapy.

In addition I am grateful that my co-worker Dr. Christina Ambros-Rudolph has been able to join us as a co-editor and that Dr. Samantha Vaughan Jones has continued to work closely with us.

We believe that this atlas and detailed text will continue to be appreciated by consulting dermatologists and non-dermatologists alike. In particular we feel that this book will continue to be a useful reference for all clinicians who care about women's health.

Martin M. Black;
Christina Ambros-Rudolph;
Peter Lynch;
Libby Edwards.

List of Contributors

Christina M. Ambros-Rudolph MD

Associate Professor of Dermatology and Venereology
Department of Dermatology
Medical University of Graz
Graz
Austria

Martin M. Black MD FRCP FRCPath

Emeritus Professor of Dermatological
Immunopathology
Honorary Consultant Dermatologist
St. John's Institute of Dermatology
St. Thomas's Hospital
London
UK

Peter R. Braude PhD FRCOG F Medsci

Professor of Obstetrics & Gynecology
Chairman & Head of Department
United Medical & Dental Schools of Guy's &
St. Thomas's Hospital
London
UK

Libby Edwards MD

Associate Professor of Dermatology
University of North Carolina, Chapel Hill;
Chief of Dermatology
Carolinas Medical Center
Mid-Charlotte Dermatology and Research
Charlotte, NC
USA

Emma Fox FRCP

Consultant Virologist
Department of Infection
Guy's and St. Thomas' NHS Foundation Trust
Guy's Hospital
London
UK

Hope K. Haefner MD

Professor
Department of Obstetrics and Gynecology
The University of Michigan Hospitals
Ann Arbor, MI
USA

Diana Hamilton-Fairley MD FRCOG

Joint Head of Women's Health Services Delivery Unit
Guy's and St. Thomas' NHS Foundation Trust
St. Thomas' Hospital
London
UK

Rachel E. Jenkins BSc MD FRCP

Consultant Dermatologist
Dermatology Department
West Suffolk Hospital
Bury St Edmonds
Suffolk
UK

Diana N. J. Lockwood BSc MD FRCP

Consultant Physician & Leprologist
Hospital for Tropical Diseases, Copper Street
London
UK

Peter J. Lynch MD

Program Director and
Frederick G. Novy, Jr. Professor of Dermatology
Department of Dermatology
University of California, Davis
Sacramento, CA
USA

Eithne MacMahon MD FRCPI MRCPath

Consultant in Infectious Diseases and Virology
Department of Infectious Diseases and Virology
St. Thomas' Hospital
London
UK

Lynette Margesson MD

Assistant Professor
Division of Dermatology
Queens University
Kingston, ON
Canada

Catherine Nelson-Piercy MA FRCP MB BSc FRCOG

Consultant Obstetric Physician
Women's Health Services Directorate
St. Thomas' Hospital
London
UK

Daghni Rajasingham MBBS MRCOG

Consultant Obstetrician
Women's Health Services Directorate
St. Thomas' Hospital
London
UK

Jeff K. Shornick MD MHA

Private Practice
Groton, CT
USA

Catherine J. M. Stephens MBBS FRCP

Consultant Dermatologist
Poole Hospital NHS Trust
Poole
Dorset
UK

Samantha Vaughan-Jones MD FRCP

Consultant Dermatologist
Department of Dermatology
Ashford and St. Peter's NHS Trust
Chertsey
Surrey
UK

Dedications

To my wife, Aniko.

M.B.

To my family for encouraging me to go into medicine and to my husband, Johannes, for his love and continuing support.

C.A.-R.

To my wonderful niece, Amanda Coombs.

L.E.

I would like to express my gratitude to Eduard G. Friederich, Jr., Raymond W. Kaufman, and Donald J. Woodruff. Their role in helping me to understand genital disease in women cannot be overestimated. They have in addition served as valued colleagues, important mentors, and warm friends.

P.L.

CHAPTER 1

Hormonal Changes during Puberty, Pregnancy, and the Menopause

Peter Braude
Diana Hamilton-Fairley

Introduction

This chapter summarizes the hormonal changes that occur during puberty, the menstrual cycle, pregnancy, and the menopause, and how these changes affect the skin physiologically.

All children go through the bewildering hormonal changes that the transition from child to adult necessitates. However, it is only the female who will continue to experience a changing hormonal milieu – either cyclically, with the monthly production of an egg followed by menses, or the effects of pregnancy if conception takes place. Then, for the last third of their lives, women face the consequences of a reduction in estrogen levels following the menopause. Although the hormonal events immediately preceding the menopause are turbulent, once the climacteric is reached, it too may cause its problems.

Most women are aware of the changes taking place in their skin at these different stages in their lives.

Hypothalamic–pituitary axis

An understanding of the interrelationship between the hypothalamus, the pituitary gland, and the ovary is imperative if the cyclical and long-term hormonal changes occurring in women are to be appreciated.

Situated above the pituitary gland, the hypothalamus initiates the release of the polypeptides that regulate ovarian function. The ovary cannot produce mature fertile oocytes (eggs) if the signals from the pituitary gland never start, cease prematurely, or are disordered. The female reproductive cycle is regulated precisely via biologic feedback mechanisms from the ovary, which alter the activity of the hypothalamus and pituitary. Normal physiologic changes in the functioning of the hypothalamic–pituitary axis result in the hormonal changes that occur during the four main reproductive endocrine phases of

a woman's life: puberty, menstruation, pregnancy, and the climacteric.

As menarche (the first period) is one event during the years of puberty, so menopause (the final period) marks one event during the years of declining reproductive function (the climacteric or menopause).

Puberty

Puberty describes the physiologic, morphologic, and behavioral changes that occur in a child as the gonads mature from the infantile to the adult state that affects most of the organs of the body in both sexes.

These physiologic changes can be divided into two main groups: growth and hormonal. Although the changes start at different chronologic ages in different individuals, the sequence of events is similar. In girls the start of puberty is strongly weight-related, with the mean body weight being 47 kg at menarche. Although the age of menarche has declined from 17 years in 1840 to 13.5 years in the 1940s, and is now 12.5 years in the United States, the mean body weight at menarche seems to have remained constant.

Growth spurt

The adolescent growth spurt is an acceleration of growth in most skeletal dimensions. The peak height velocity (PHV) is 9–10 cm per year and lasts for about 2 years. There is no difference in the PHV of girls and boys, and both sexes grow between 25 and 28 cm during puberty. However, girls start their growth spurt 2 years earlier than boys, at which time they are 10 cm shorter than when boys start theirs. This accounts for the difference in adult height between the sexes.

This large increase in height is mediated by an increase in growth hormone (GH) production by the pituitary gland. The greatest increase in the frequency and amplitude of GH takes place at night in a similar fashion to luteinizing hormone (LH) pulses (Figure 1.1).

Hormonal changes

The hormonal changes of puberty produce two main effects: maturation of the ovary so that reproduction can occur, and development of secondary sexual characteristics (breasts, axillary and pubic hair).

During childhood, serum levels of the gonadotrophins – LH and follicle-stimulating hormone (FSH) – are low. During early to mid-puberty, however, there is a striking increase in the magnitude and frequency of LH pulses at night during sleep (see Figure 1.1). In late puberty, there is an increase in magnitude during the day, but not as marked as at night. Only when puberty is complete do the LH pulses lose their diurnal variation and settle into an adult pattern, with pulses approximately every 90 minutes during the follicular phase, and between 120 and 180 minutes in the luteal phase.

These events are probably initiated by the maturation of the hypothalamus and the onset of secretion of gonadotrophin-releasing hormone (GnRH). However, it is impossible to prove the exact sequence of events initiating puberty because the experiments required would be unethical in humans.

The increase in both LH and FSH activity has a trophic effect on the ovary, stimulating the production of estradiol. The primordial follicles (the oocyte and surrounding support cells), present from birth, begin to mature into antral follicles lined by granulosa cells. This process of maturation takes about 10 weeks. LH acts mainly on the theca cells which surround the follicles, causing them to produce testosterone, which is then converted by an aromatase into estradiol in the granulosa cells under the influence of FSH.

The increase in estradiol secretion stimulates breast development. The five stages of breast development take about 4 years to complete (Figure 1.2). Menarche (the first menstruation) usually occurs once breast development is quite well advanced – between stages III and IV[1]. Rapid breast development or increase in breast size, common during pregnancy and less common on the oral contraceptive pill, may cause stretch marks to develop, especially in the lateral margins of the breast, which may be of concern, particularly to younger women.

Several other changes also occur, which are important in understanding the physiologic changes in the skin. The first is adrenarche. This

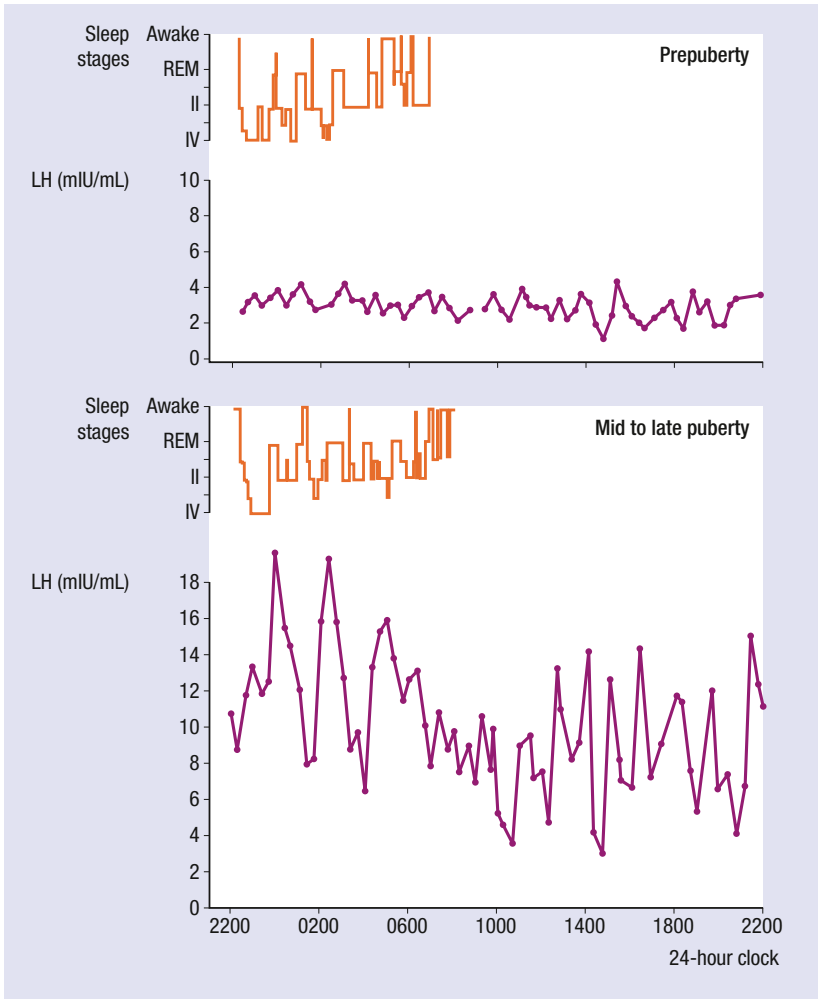

Figure 1.1 Luteinizing hormone (LH) levels during puberty. Changes in the pulse frequency and amplitude of luteinizing hormone during puberty. REM, rapid eye movement.

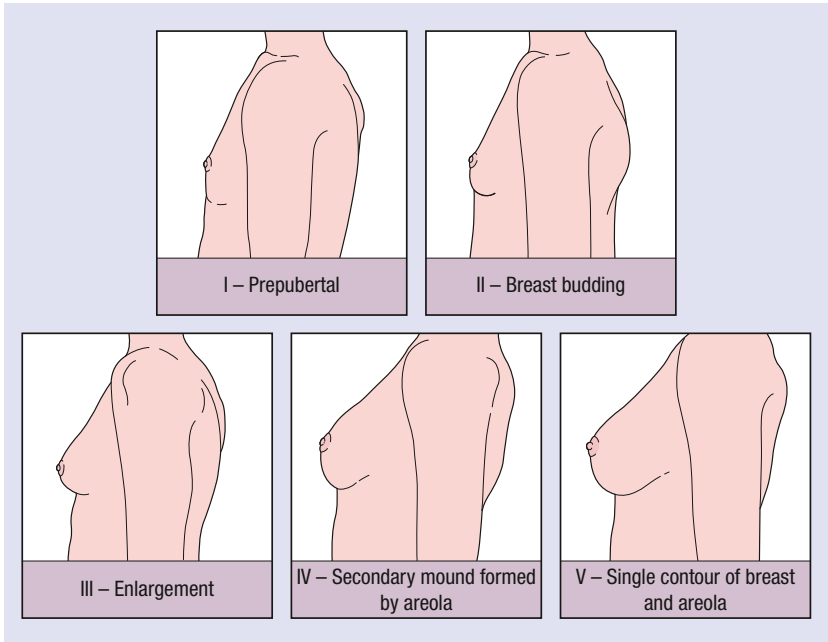

Figure 1.2 Breast development. The Tanner stages I–V of breast development.

is an increase in the production of adrenal androgens, dehydroepiandrosterone (DHEA) and its sulfate (DHEAS), which starts at about 8 years of age and continues until 13–15 years of age in both sexes. This increase is thought to stimulate the development of axillary and pubic hair, as hair growth and changes in sebum secretion are modulated predominantly by androgens in both sexes. Pubic and axillary hair growth usually starts before the breasts change following the increase in adrenal androgen levels, but reaches the mature stage at around the same time. The testosterone level increases in girls, as in boys, under the influence of LH, but most of it is converted into estradiol.

During puberty, the concentration of the main binding protein of the sex hormones (sex hormone-binding globulin, SHBG) declines in both sexes, despite the increase in estradiol concentrations in girls[2]. SHBG has a greater affinity for testosterone than for estradiol, with the result that, in most girls, more than 90% of circulating testosterone is bound to SHBG, thus limiting the effect that testosterone may have peripherally. The decrease in SHBG seems to be mediated by an increase in insulin concentration, which has been demonstrated in both sexes[3].

Polycystic ovary syndrome

There is a group of girls who produce an excess of testosterone accompanied by morphologic changes in their ovaries, a phenomenon known as polycystic ovaries (PCO)[4]. Typically these girls never establish regular menstruation and have increased hair growth, usually of a male pattern, with an abdominal escutcheon, moustache, or other facial hair growth (Figure 1.3). They may also develop acne. Many girls with acne and/or hirsutism have polycystic ovary syndrome. These girls also have higher insulin concentrations and lower SHBG concentrations than their weight-matched contemporaries[5].

A lower SHBG concentration, together with an increased circulating testosterone level, will lead to an increased free testosterone level. It is the fraction of free testosterone that is thought to be active peripherally on the skin, sebaceous glands, and hair follicles.

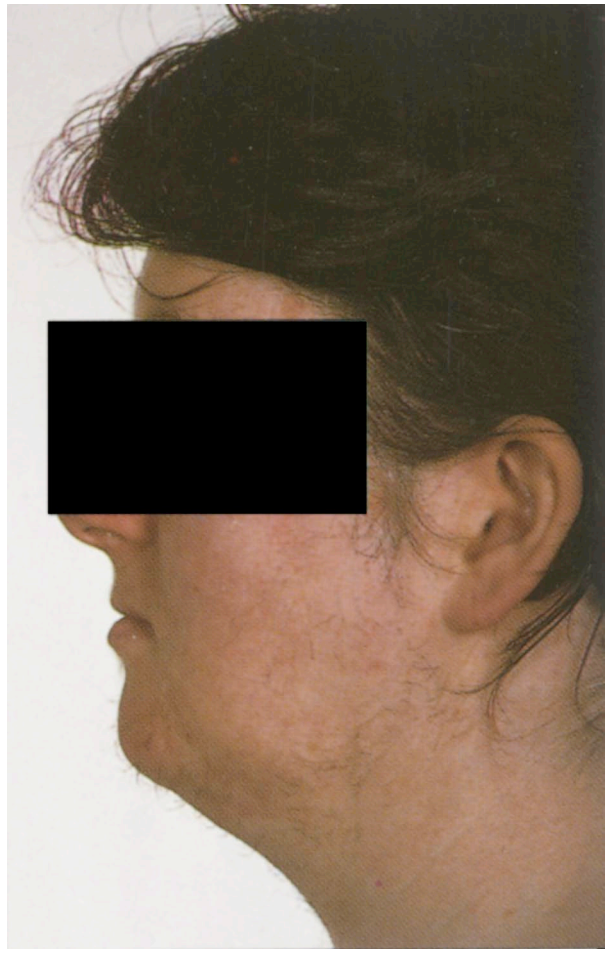

Figure 1.3 Polycystic ovary syndrome. Facial hirsutism associated with polycystic ovary syndrome.

Acne

Testosterone has major effects on the hair follicle and sebum secretion. Acne vulgaris[6] (Figure 1.4) and hirsutism are never seen in prepubertal children with normal adrenal function, providing further evidence that puberty-related changes trigger these events. Although there is no evidence of increased androgen production in men with acne, most women with acne do have increased ovarian androgen production and a reduced SHBG concentration. Undoubtedly, genetic factors also play an important part in determining which girls will suffer and which will not. The pilosebaceous gland becomes more differentiated, increases in size, and changes its sebum composition. These changes are most marked on the scalp and around the nose, chin, and cheeks, as well as on the upper chest and

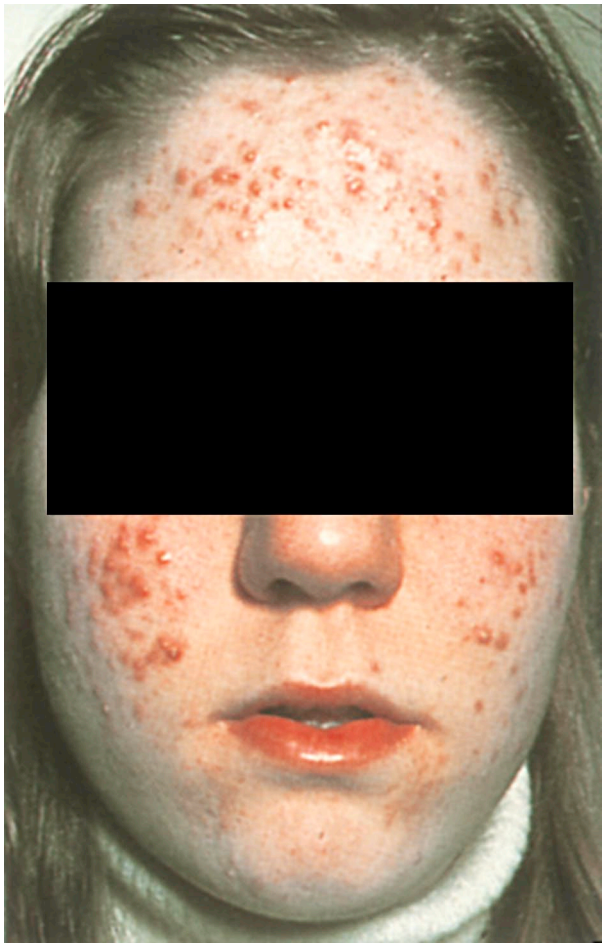

Figure 1.4 Acne vulgaris.

back (see Chapter 2). Acne tends to reach a peak during puberty and before sexual maturity. It is therefore thought that the adrenal glands provide the initial stimulus.

The menstrual cycle

The menstrual cycle is divided into two phases which are named as viewed from two different standpoints:

- The follicular and luteal phases – according to events in the ovary
- The proliferative and secretory phases – according to changes that take place in the endometrium

As endometrial changes are dependent on the hormonal changes occurring in the ovary, the terms "follicular phase" and "luteal phase" will be used in this chapter.

Follicular phase

A few days before the onset of menstruation, the level of FSH starts to rise under the stimulation of GnRH secreted from the hypothalamus (Figure 1.5). This causes several antral follicles to start producing estradiol. As these follicles fill with fluid produced by the granulosa cells, which lie as a single layer around each follicle, they become visible on an ultrasonographic scan. In order to produce estradiol, the granulosa cells utilize testosterone produced by the theca cells, which lie as a second monolayer around the follicle.

In the early follicular phase, the granulosa cells carry receptors for FSH, whereas the theca cells are stimulated by LH. The estradiol produced by the ovary is released into the circulation. The pituitary has an abundance of estradiol receptors; their activation results in the inhibition of both LH and FSH production in the mid follicular phase. The granulosa cells also produce inhibin, a protein that augments the negative feedback of estradiol on FSH. This protein is also produced by the corpus luteum following ovulation. As a result of this effect, the smaller follicles stop growing and undergo atresia.

By this stage, only one follicle (but, occasionally, two or more) has reached a diameter of about 10–12 mm, and is called the dominant follicle. The granulosa cells of the dominant follicle develop LH receptors and so become receptive to both LH and FSH. The follicle increases in diameter by 2 mm/day. The estradiol concentration rises faster and the granulosa cells begin to accumulate in several layers over the oocyte.

Oocyte release

When the follicle reaches a diameter of around 18–20 mm, and the estradiol concentration reaches 800–1000 pmol/L, the biofeedback on the pituitary is reversed. This results in a rapid rise in hormone concentrations, predominantly of LH and to a lesser extent of FSH. In turn, this leads to a luteinization of the granulosa cells and consequently they begin to produce progesterone in preference to estradiol. This change leads to the rupture of the follicle wall, and the oocyte is released into the peritoneum about 24–36 hours

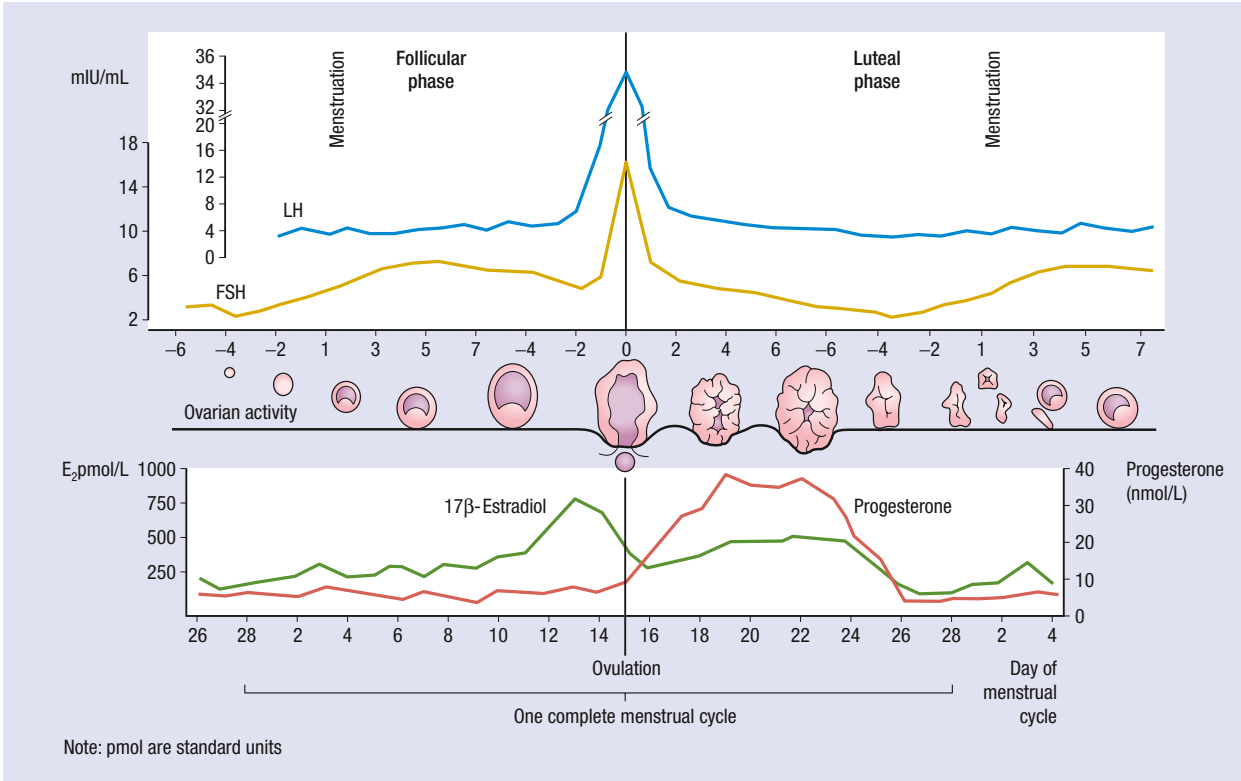

Figure 1.5 Hormonal changes of the menstrual cycle. LH, luteinizing hormone; FSH, follicle-stimulating hormone.

after the LH surge. The follicular phase ends with release of the oocyte, and varies in length from 12 to 16 days.

Luteal phase

Oocyte release marks the start of the luteal phase. Following release of the oocyte, the granulosa cells reseal the defect in the wall within a few hours, forming the corpus luteum. The granulosa (now luteal) cells produce progesterone, and this reaches a peak concentration 5–8 days after ovulation. The effect of progesterone on the endometrium is to increase the surface area of the endometrial glands and their blood supply by causing them to become spiral. They also start to produce large amounts of glycogen, an essential nutrient for the early days of embryo development if fertilization takes place.

If the oocyte is fertilized and implantation occurs, then progesterone levels remain high. These levels are maintained by human chorionic gonadotrophin (hCG) produced by the trophoblastic elements of the embryo as the primitive placenta invades the endometrium. If fertilization does not occur, the concentration of LH is insufficient to maintain production of progesterone by the corpus luteum. The levels decline and the endometrium becomes ischemic; its superficial layers slough off. This, together with bleeding from the spiral arterioles that supplied the endometrium, produces menstruation.

Thus, the cycle has come full circle to the hormonal and endometrial states found at its beginning. The whole process then begins again.

Skin changes

Skin changes during the menstrual cycle are usually temporary and of minor importance. They include an increase in sebum production before menses, which may lead to acneiform eruptions on the face and occasionally on the back.

Knowledge of the hypothalamic, pituitary, and ovarian hormonal changes is useful in understanding therapies to modify or ablate the hormonal mileu. There is a range of superactive GnRH analogs (buserelin, goserelin, nafarelin, etc.), which can be given by daily nasal spray or by monthly depot injections, whose effect (after a brief

stimulation of FSH output) is competitively to block the GnRH receptor and thus abolish FSH and LH production. This effectively renders the woman reversibly menopausal, such that events attributable to the cyclicity of the menstrual cycle can be investigated, such as cyclic pain, eruptions, or progesterone sensitivity (see Chapter 2). This regimen should not be employed for more than 6 months at a stretch because of the estrogen-depleting effect and thus its potentially adverse effect on bone loss.

Pregnancy

During the first few weeks of pregnancy, progesterone concentrations increase. Progesterone is initially produced by the corpus luteum, which is maintained by the production of hCG from the trophoblast of the conceptus.

hCG has been found in the maternal circulation almost immediately after fertilization and rises to a peak by 60–90 days of gestation. The concentration of hCG doubles every 2–3 days until this time, then gradually declines to a plateau level for the remainder of the pregnancy.

The corpus luteum continues to produce progesterone, 17-hydroxyprogesterone, estrone, and estradiol, producing a rise in the concentration of all these hormones. In addition, hCG is responsible for the production of inhibin and relaxin by the corpus luteum. Inhibin reduces FSH concentrations so that folliculogenesis is arrested once the embryo has become implanted into the endometrium. It may also act as a growth factor for the early embryo. Relaxin is thought to act in synergy with progesterone to reduce the contractility of the uterine myometrium.

The concentrations of both of these hormones rise in parallel with the concentration of hCG, but they are produced only for a limited period by the ovary. From around 7 weeks' gestation, they are produced by the decidual fetal membranes and placental tissues. Similarly, ovarian steroid hormone production declines from 7 weeks' gestation, with the placental unit taking over this function. This explains why pregnancies fail if the corpus luteum is removed before 8 weeks, but continue unharmed if the pregnancy has reached 9 weeks' gestation.

Table 1.1 Hormones Produced by the Fetal–Placental Unit

Peptides	Inhibin
	Relaxin
	Human placental lactogen
Neuropeptides	Gonadotrophin-releasing hormone
	Corticotrophin-releasing hormone
	Thyroid-releasing hormone
Steroid hormones	Progesterone
	Androgens
	Estradiol
	Estrone
	Estriol
Peptide growth factors	Insulin-like growth factors I and II

The placenta

The placenta is a complex organ. Not only does it provide nutrients and excrete waste products from the fetus, but it also modifies the maternal metabolism at various stages of pregnancy via hormones. The placenta reaches structural maturity by the end of week 12 of pregnancy. The functional unit is the chorionic villus, which consists of a central core of loose connective tissue and abundant capillaries. These connect to the fetal circulation and provide a large surface area in contact with the maternal uterine circulation. Around this central core are two layers of trophoblast, an outer syncytium (syncytiotrophoblast), and an inner layer of discrete cells (cytotrophoblast).

The fetus and placenta form an interdependent partnership which regulates the endocrine-metabolic processes during pregnancy. This fetal-placental unit therefore becomes an endocrine system, producing a large number of different hormones (Table 1.1).

Several placental products have been measured over the years in the search for a marker for placental insufficiency. These include estriol and human placental lactogen (hPL), the concentrations of which rise steadily throughout pregnancy. But, as

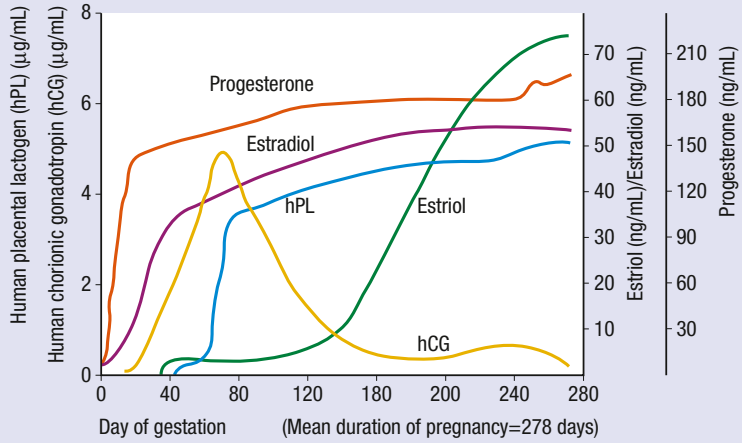

Figure 1.6 Hormonal changes during pregnancy. Changes in the production of progesterone, estradiol, estriol, human chorionic gonadotropin, and human placental lactogen.

their normal ranges are very large, they have not proved clinically useful in predicting the outcome of pregnancy.

Following the baby's birth, all the hormone levels return to normal within a few days. The production of hCG, progesterone, estriol, estradiol, and hPL during pregnancy is shown in Figure 1.6. Despite our ability to measure these hormones during pregnancy, the role that they play in maintaining pregnancy and/or initiating parturition is still poorly understood.

Skin changes in pregnancy

Striae gravidarum

Striae gravidarum are linear purple-red lesions which over time lose their pigmentation and atrophy, leaving scar-like tissue. They can cause itching and discomfort and affect 50–90% of women during pregnancy. The underlying etiology has always been unclear, with two principal theories. The first is the association with stretching of the skin causing disruption to the collagen fibers and elastin in the dermis as the uterus grows. However a small study has shown that a significant number of women develop striae gravidarum before 24 weeks[7]. The other is hormonal change, as in Cushing's syndrome and steroid therapy. There is little research in this area but the strongest association with the development of striae gravidarum is the presence of striae on the thighs or breasts prior to pregnancy. It appears that the group at highest risk of developing severe striae is teenagers[8].

Melasma

Melasma (chloasma) is known as the "mask of pregnancy" as it causes an increase in pigmentation of facial skin, particularly over the cheeks. It is associated with hormonal change as it occurs in 70% of women during pregnancy, in 5–34% of those using the oral contraceptive pill, and has been reported in women using hormonal creams, some cosmetic products, and certain types of drugs. It usually clears once pregnancy is over, confirming its hormonal origins[9].

The climacteric and the menopause

The menopause (cessation of menses) marks the end of a woman's reproductive life. The average age for the end of menses in the United Kingdom is 50.3 years. During the perimenopausal years (the climacteric) there is an increase in circulating FSH levels and a decrease in estradiol concentrations[10]. The negative feedback of estradiol on FSH still occurs, but the resting concentration of FSH is higher than in younger women. The concentrations of FSH at the mid-cycle surge and in the late luteal phase are also greater. LH levels tend to remain within the normal range until the cessation of menses. Ovulatory cycles may still occur with increased levels of FSH, providing evidence that the ovary gradually becomes less responsive to gonadotrophins. The timing of menses may become more irregular, and most cycles become anovulatory, as the ovaries become

depleted of antral follicles[11,12] and no longer respond to FSH.

Estradiol levels decline until they are so low that the endometrium no longer undergoes proliferation, and becomes atrophic. The endometrium is no longer shed and menses cease. As well as a decline in estradiol levels, androgen levels also decrease from an average of 1.6 to 0.5 nmol/L. This reduction in androgen levels has been used to explain the decrease in libido sometimes experienced by postmenopausal women.

As a secondary effect of reduced estradiol concentrations, FSH and LH levels rise, owing to a lack of negative feedback on the pituitary gland. While the postmenopausal ovary produces minimal estradiol, it continues to produce quite significant amounts of testosterone and, to a lesser extent, androstenedione, produced by the stromal cells of the ovary.

These androgens, predominantly adrenal androstenedione, are converted peripherally by aromatase into estrone. The extent to which this happens depends on age and weight (fat mass). Heavier women have higher conversion rates and circulating estrogen concentrations than slim women. The average percentage of conversion in menopausal women is 2.8%, double that found in premenopausal women. The relative change in balance between estrogen and androgen production in older women[13,14] may account for the increased incidence of hirsutism in this group.

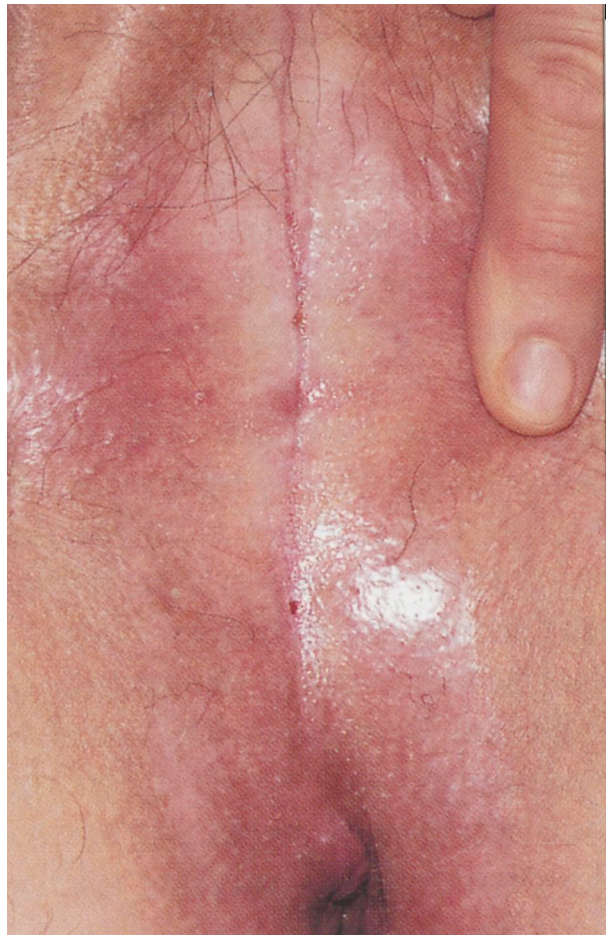

Figure 1.7 Fused labia. This condition occurred secondary to postmenopausal skin changes.

Skin changes after the menopause

The predominant process is progressive atrophy of the dermis and architectural changes leading to folds and wrinkles. The extent to which this occurs varies from individual to individual and depends on genetic and environmental factors. The generalized aging process of the skin involves the vagina and vulva too. Estradiol is essential to the maintenance of the elasticity and lubrication of the vagina. Most of the changes in the vulva and vagina associated with the menopause are secondary to low estradiol concentrations, as they can be reversed by the topical application of estradiol. In areas such as the vulva and vagina, which are protected from ultraviolet light, the epidermis becomes very thin. There is a reduced number of capillaries within the skin and elastotic changes occur in the arterioles. With time, the withdrawal of estrogens causes the vaginal skin to lose its folds, and the vaginal epithelium becomes thin and friable, making it more susceptible to trauma, with the result that it bleeds. This is a very common cause of postmenopausal bleeding, especially in the older woman.

The atrophic changes that affect the vulva predispose it to trauma, often secondary to excessive itching. This may lead to ulceration or scar formation as the wounds heal. In a few women, this leads to fusion of the labia majora (Figures 1.7 and 1.8).

Hormone replacement therapy (HRT) has been shown to increase surface lipid concentrations and the water-holding capacity of the skin. This change may improve the function of the skin as a