A Good Cycle Chart. Bryn.

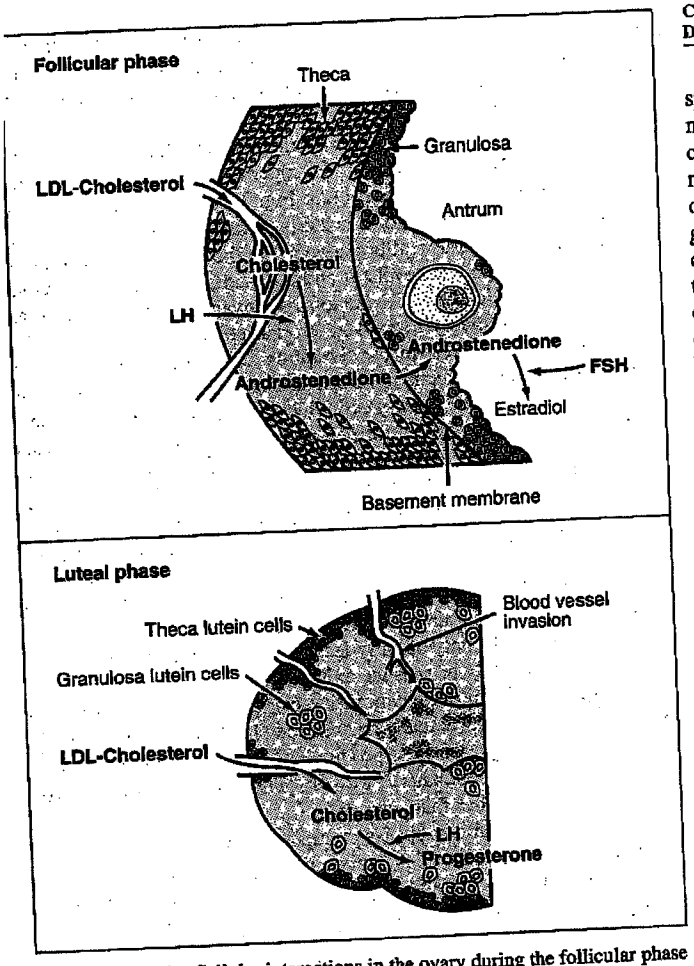


FIGURE 337-4 Cellular interactions in the ovary during the follicular phase (*top*) and luteal phase (*bottom*). LDL, low-density lipoprotein; FSH, follicle-stimulating hormone; LH, luteinizing hormone. *(From Carr et al, 1982.)*

After the onset of menses, follicular development continues, but FSH levels decrease. Approximately 8 to 10 days prior to the midcycle LH surge, plasma estradiol levels begin to rise as the result of estradiol formation by the granulosa cells of the dominant follicle. During the second half of the follicular phase, LH levels also begin to rise (owing to positive feedback). Just before ovulation, estradiol secretion reaches a peak and then falls. Immediately thereafter, a further rise in the plasma level of LH mediates the final maturation of the follicle, followed by follicular rupture and ovulation 16 to 23 h after the LH peak. The rise in LH is accompanied by a smaller increase in the level of plasma FSH, the physiologic significance of which is unclear. The plasma progesterone level also begins to rise just prior to midcycle and facilitates the positive feedback action of estradiol on LH secretion.

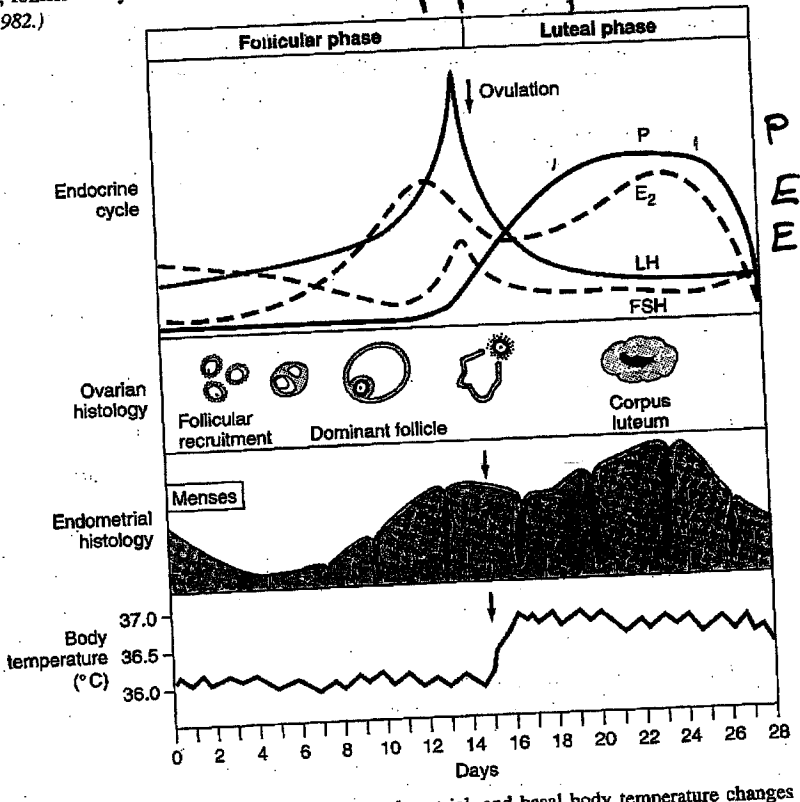
At the onset of the luteal phase, plasma gonadotropins decrease, and plasma progesterone increases. A secondary rise in estrogens causes further gonadotropin suppression. Near the end of the luteal phase, progesterone and estrogen levels fall, and FSH levels begin to rise to initiate the development of the next follicle (usually in the contralateral ovary) and the next menstrual cycle.

The endometrium lining the uterine cavity undergoes marked alterations in response to the changing plasma levels of ovarian hormones (see Fig. 337-5). Concurrent with the decrease in plasma estrogen and progesterone and the decline of corpus luteum function in the late luteal phase, intense vasospasm occurs in the spiral arterioles supplying blood to the endometrium, causing ischemic necrosis, endometrial desquamation, and bleeding. This vasospasm is caused by locally synthesized prostaglandins. The onset of bleeding marks the first day of the menstrual cycle. By the fourth to fifth day of the cycle, the endometrium is thin. During the proliferative phase, glandular growth of the endometrium is mediated by estrogen. After ovulation, increased progesterone levels lead to further thickening of the endometrium, but the rapid growth slows. The endometrium then enters the secretory phase, characterized by tortuosity of the glands, curling of the spiral arterioles, and glandular secretion. As corpus luteum function begins to wane in the absence of conception, the sequence of events leading to menstruation is again set into action.

Biphasic changes in basal body temperature are characteristic of the ovulatory cycle and are mediated by alterations in progesterone levels (see Fig. 337-5). An increase in basal body temperature by 0.3 to 0.5°C begins after ovulation, persists during the luteal phase, and returns to the normal baseline (36.2 to 36.4°C) after the onset of the subsequent menses.

Cellular Interactions in the Ovary during the Normal Cycle LH stimulates thecal cells surrounding the follicle to form androgens, and androstenedione diffuses across the basement membrane of the follicle into granulosa cells, where it is aromatized to estrogen (see Figs. 337-3 and 337-4).

The increase of FSH late in the preceding menstrual cycle stimulates growth and recruitment of the primary follicles by enhancing granulosa cell proliferation, resulting ultimately in the formation of the dominant follicle. In the granulosa cells, FSH also stimulates estrogen synthesis. Enhanced secretion of estradiol causes an increase in the number of estradiol receptors and further proliferation of granulosa cells. In the late follicular phase, FSH, in concert with estradiol, causes induction of LH receptors on the granulosa cells. LH acts via these receptors to increase progesterone secretion at midcycle. The amount of progesterone formed by the follicle is believed to be limited by the availability of cholesterol to serve as substrate for steroidogene-



P. Progesterone
E_2 Estriadol

FIGURE 337-5 The hormonal, ovarian, endometrial, and basal body temperature changes and relationships throughout the normal menstrual cycle.