I. INTRODUCTION

A. Luteal Phase (metestrus + Diestrus; Figure 9-1)

1. The period from ovulation until CL regression
2. Longer in length compared to the follicular phase (~ 80% of the length of the estrous cycle)
3. Growth and maturation of the corpora lutea (CL; the dominant ovarian structures)
4. Rise in blood progesterone from the CL, and relatively low FSH and LH concentrations (see figure 9-2)

II. THREE MAJOR EVENTS OF THE LUTEAL PHASE
(Figures 9-3 through 9-6)

A. Development of the luteal structure: corpus hemorrhagicum

1. When the follicle ruptures at ovulation, blood vessels break resulting in a structure with a red appearance: corpus hemorrhagicum
2. Corpora hemorrhagica may be observed from the time of ovulation until about day 3 of the estrous cycle.

B. Growth and Maturation of the luteal structure: the corpus luteum (CL)

1. Between day 3 to 5, the CL begins to increase in size and increases in mass until the middle of the cycle
2. Maximal size of the CL will coincide with maximal production of progesterone (approximately during the middle of the estrous cycle)

C. Regression and degradation of the luteal structure

1. During the end of the luteal phase, luteolysis occurs and the CL regresses
   The lysed CL will become a corpus albicans (CA)

CA may be observed for a long period of time
CA appears as a white scar-like structure

II. LUTEINIZATION (DEVELOPMENT OF THE LUTEAL TISSUE)
A. The corpus luteum originates from the ovulatory follicle

B. Luteinization is a process in which the follicular cells are transformed into luteal tissue

1. Begins prior to ovulation

2. Theca interna and granulosa cells undergo a dramatic transformation

3. Transformation is governed by LH

4. Theca and granulosa cells, under LH influence, become luteal cells large cells
   - Small cells originate from cells of the theca interna (Figure 9-7)
   - Both large and small luteal cells produce progesterone (Figure 9-7)

C. Progesterone from the CL goes into systemic circulation and affects: (Figure 9-8)

1. The hypothalamus (surge and tonic center)
   - Under progesterone influence, GnRH is secreted in a low frequency high amplitude fashion, as is FSH and LH
   - This allows follicles to develop during the luteal phase, but these follicles do not reach preovulatory size until progesterone decreases

2. The endometrium
   - Secretions from these glands help support the development of the early embryo

3. The mammary gland by causing final alveolar development prior to parturition for subsequent lactation

III. LUTEOLYSIS: DESTRUCTION OF THE CL

A. Occurs during a few days at the end of the luteal phase and results in cessation of progesterone production, structural regression of the CL, and subsequent follicular development

B. Luteolysis is controlled by two hormones

1. Oxytocin from the CL (and more likely not from the posterior pituitary)

2. Prostaglandin F2α from the uterine endometrium

C. Uterus is required for successful luteolysis. (Figure 9-10)

1. Ewes with an intact uterus: the CL lifespan is normal and the length of the estrous cycle is normal (15 to 17 days)
2. Ewes with a total hysterectomy: lifespan of the CL is long (145 days) and animal does not cycle for 5 months

3. Partial hysterectomy (contralateral to the CL = opposite side to the CL)
   - Lifespan of the CL is normal (15 to 17 d estrous cycle)
   - Therefore, removal of the portion of the uterus on the opposite side of the CL has no effect

4. Partial hysterectomy (ipsilateral to the CL = on the same side of the CL)
   - Lifespan of the CL is extended to about twice the normal estrous cycle length (35 days)
   - Therefore, removal of the portion of the uterus on the same side of the CL has an effect

**There is a local effect of the uterus on the ovary and CL**

### D. Luteolysis (mechanism of action by the uterus, PGF2α and oxytocin) (Figure 9-11)

1. **Oxytocin from the CL stimulates PGF2α synthesis** (Figures 9-12 and 9-13)

2. Prostaglandin F2α is produced by the uterine endometrium → venous drainage from the uterus by the utero-ovarian vein carries PGF2α towards the ovarian artery
   - (the ovarian artery wraps around the utero-ovarian vein) → PGF2α is passed from the utero-ovarian vein to the ovarian artery by countercurrent exchange → this ensures that a high amount of PGF2 alpha produced by the uterus will be transported directly to the ovary and CL without being diluted and metabolized in the systemic circulation

**Possible intracellular mechanisms that cause luteolysis:**

1) Reduced blood flow (vasoconstriction) by PGF2α which causes ischemia (weak theory)

2) Capillary degeneration and loss of blood flow to the CL thus CL destruction

3) Apoptosis (programmed cell death):
   a) Lymphocytes and macrophages produce materials called cytokines. Cytokines are capable of reducing progesterone synthesis and disrupt the normal integrity of the CL
   b) Cytokines may cause apoptosis which is accompanied by tissue necrosis and massive influx of intracellular Ca, collectively causing the destruction of the CL.