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Clinical Focus on

Labor & Childbirth

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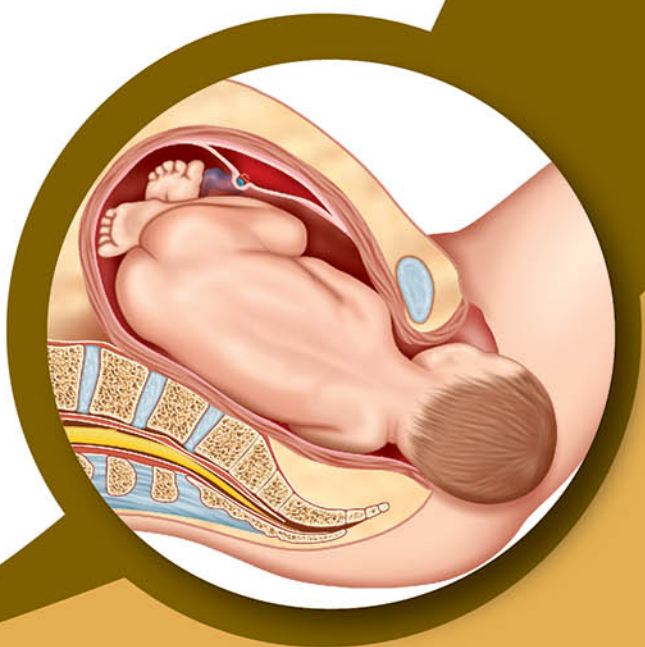

Foreword
Shyam Desai

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CHAPTER 2

Physiology of Labor

Richa Rathoria, Parul Sinha

INTRODUCTION

Term labor is a physiological process that involves a series of coordinated changes in the myometrium, decidua, and cervix that start slowly over a few days to a few weeks and quickly develop over a few hours, ending in the expulsion of the fetus and placenta.[1] Cervix biochemical connective tissue alterations begin before uterine spasms and cervical dilatation, which often take place before the fetal membranes breach. The uterus gradually returns to its prepregnancy form after birth.[2]

Term labor may be better viewed as a withdrawal of pregnancy's inhibitory effects on uterine tissue, rather than as an active process controlled by the release of uterine stimulants. Inhibitory and stimulatory processes, however, probably both work. In contrast, many occurrences of preterm labor appear to be the result of labor activation caused by infection, inflammation, hemorrhage, or other pathologic conditions that bypass or overwhelm the systems that keep the uterus quiescent.[2]

It is speculated that a "parturition cascade" ensues that eliminates the mechanisms that sustain the uterus dormant and recruits components that promote uterine activity. Given its conceptual significance, such a cascade would probably have several redundant loops to create a fail-safe mechanism for assuring pregnancy success. Each component of this model is sequentially coupled to the others, and many of the components exhibit positive feed-forward properties typical of cascade mechanisms.[3]

PHASES OF UTERINE ACTIVITY

Physiologically, the phases of uterine activity during pregnancy are classified into four phases: Quiescence (Inhibition) Myometrial, activation, stimulation, and involution **(Fig. 1)**.[4]

Phase 1: Quiescence (Inhibition)

Phase I starts from conception until a few weeks before the commencement of labor. The myometrium's usual phenotype is contractile, and its primary roles are to effectively remove the endometrial lining (menstruation) each month and constrict the uterine radial (penetrating) arteries to lessen menstrual blood loss. This contractile phenotype needs to be actively repressed throughout pregnancy to keep the myometrium in a functional quiescent condition. Several potential inhibitors are progesterone, prostaglandin I2 (prostacyclin), relaxin, parathyroid hormone related peptide, corticotropin-releasing hormone (CRH), human placental lactogen (HPL), nitric oxide (NO), calcitonin gene-related peptide, adrenomedullin, and vasoactive intestinal peptide (VIP).[2,4]

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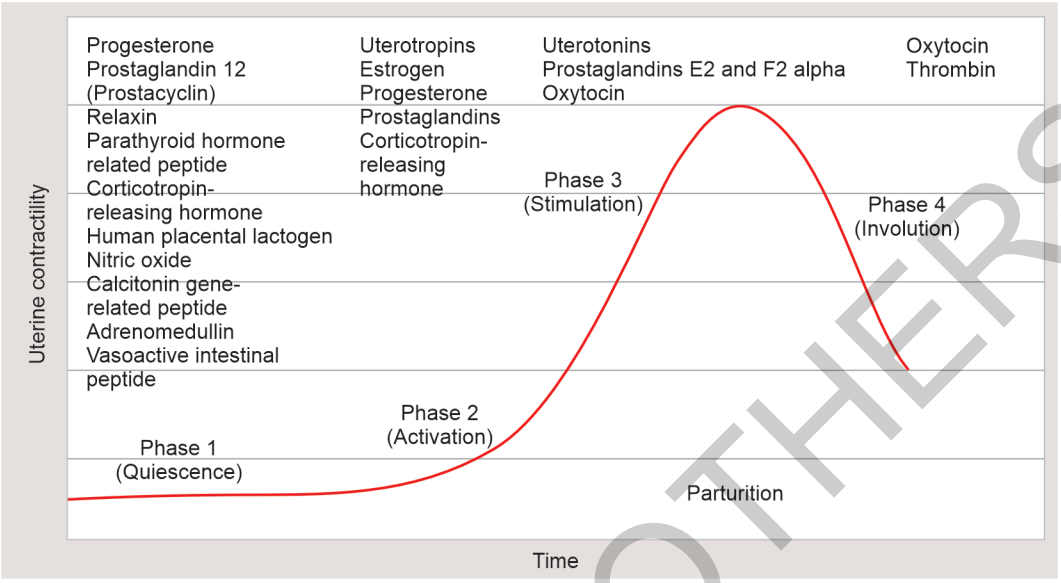

Fig. 1: Phases of uterine activity and their regulation.

Phase 2: Myometrial Activation (Priming)

Women lack a systemic progesterone withdrawal before the start of labor, but as term approaches, the uterus experiences a functional decrease in progesterone activity. The contraction-associated proteins (CAPs), which include myometrial receptors for prostaglandins and oxytocin, are expressed more frequently as a result of the withdrawal of progesterone's inhibitory effects on myometrial cells in conjunction with an increase in the circulating levels of uterotropins (such as estrogen). Additionally, certain ion channels are activated, and connexin-43, a crucial gap junction component, is expressed more frequently. Contraction coordination is made feasible by the myometrium's improved electrical synchronization as a consequence.[2,4]

Phase 3: Stimulation

The primed uterus contracts once the myometrium has been activated by endogenous and exogenous uterotonic agonists including oxytocin and the stimulatory prostaglandins E2 and F2 alpha, which finally result in delivery.[2,4]

Phase 4: Involution

Within a few days and weeks after delivery, the uterus involutes. The main mediator of this process is oxytocin.[2,4]

DETERMINANTS OF LABOR INITIATION

The maternal and fetoplacental variables that promote and maintain human labor at term are still unclear despite earlier investigations that focused on endocrine, biochemical, molecular mechanisms, and genetic regulation.[2] Several major hormones and neuropeptides involved in this process are explored further below.

Prostaglandins

Prostaglandins are mostly paracrine/autocrine hormones. Despite having almost

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similar structures, PGE2 and PGF2 alpha signal through separate receptors and have frequently antagonistic effects. The increased production of the prostaglandins of the E and F series by the decidua and fetal membranes predominantly appears to represent a final common pathway for the commencement of labor in humans, whether at term or preterm.[2,5]

The removal of fetal-paracrine support for the dormant uterus causes "decidual activation," which is followed by the production of PGF2 alpha and the onset of spontaneous labor. PGE2 seems to be more vital for cervical ripening and fetal membrane rupturing than for uterine contractility.[2,5]

Progesterone

Early miscarriage may easily be caused by the administration of a progesterone receptor antagonist or by removing the corpus luteum before 7 postmenstrual weeks of gestation, proving that ovarian progesterone production is essential for maintaining early pregnancy. After 5–7 postmenstrual weeks, the placenta takes over as the primary producer of progesterone, and the corpus luteum begins to recede.[2]

Progesterone's function in late pregnancy is less clear. Humans do not experience systemic progesterone withdrawal before labor, and average circulating progesterone concentrations during labor are comparable to those observed 1 week earlier. Progesterone receptor antagonists solely are not a successful approach to induce labor at term (although they may enhance cervical ripening), and progesterone administration late in pregnancy does not postpone the beginning of labor.[2]

Estrogens

During pregnancy, the placenta acts as the main source of the production of estrogen. Placental estrogen production requires C19 steroid precursors since the human placenta is an insufficiently steroidogenic organ (it cannot produce estrogen from progesterone).[2] Dehydroepiandrosterone (DHEAS), a plentiful C19 estrogen precursor, is produced by the fetal adrenal directly in its intermediate zone. The fetal DHEAS and 16-hydroxy (OH) DHEAS are converted to estradiol and estriol.[6]

Progesterone suppresses the estrogen receptors in the uterus during the whole pregnancy, but at term, the so-known functional withdrawal of progesterone eliminates this suppression and permits the uterotonic action of estrogens. Estrogen reduces resting potential and increases muscular contractility, while progesterone causes the muscle to become quiescent and increases the membrane potential. Estrogen promotes gap junction development, whereas progesterone inhibits it. Estrogens are known to labile the intracellular lysosomes and enhance the secretion of phospholipase A2, which is required for prostaglandin production, whereas progesterone has a reverse effect. Estrogen increases oxytocin receptors in the myometrium, whereas progesterone inhibits them.[6]

Oxytocin

Oxytocin is a peptide hormone produced in hypothalamus and pulsatile discharged from the posterior pituitary. The placenta also produces it. It has a biologic half-life of 3–4 minutes in the maternal circulation and undergoes inactivation in the liver and kidney, but during pregnancy, placental oxytocinase is principally responsible for its degradation.[6]

At term, the myometrial decidua has an increase in oxytocin receptors. In a previously sensitized uterus, oxytocin causes

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contractions and makes the uterus that is currently contracting contract stronger and more frequently. It raises intracellular calcium levels, which amplifies myometrial cell electrical discharges. Ferguson reflex is the discharge of maternal oxytocin from the posterior pituitary in reaction to cervix and/or vaginal dilation. Oxytocin promotes the uterine decidua's production and release of prostaglandins (PGE2 and PGF2a), which causes myometrial contractility. Beginning about the 20th week of pregnancy, oxytocin receptor development continues unaltered until the start of labor. However, estrogens cause an increase in myometrial sensitivity to oxytocin. Oxytocin receptors increase during late pregnancy, active labor, the third stage of puerperium, and breastfeeding. Oxytocin does not serve an integral role in phase 2, but it is of greater significance in phases 3 and 4 and during the puerperium. It is essential for reducing postpartum hemorrhage, enabling uterine involution, and encouraging breast milk letdown.[6]

Vasopressin

Vasopressin is an octapeptide produced by hypothalamus and stored in the posterior pituitary. It is structurally related to oxytocin and has strong oxytocin-like properties in humans.[6]

Relaxin

Relaxin is a peptide hormone that activates collagenases and may contribute to cervical ripening before labor.[6]

Cytokines

Interleukin 1 (IL-1), IL-6, and tumor necrosis factor (TNF)-alpha levels appear to rise in the mother's peripheral circulation before the start of spontaneous term labor. By activating ERK1/2, IL-1 beta inhibits the expression of progesterone receptors in decidual cells. Additionally, physical and hormonal indications from the developing fetus may encourage macrophage migration to the uterus, producing cytokines, and transcription factor activation associated with inflammation. During labor, IL-8 concentrations in the human myometrium, decidua, and fetal membranes rise (but not IL-2 or TNF-alpha). The main target of the powerful chemotactic cytokine IL-8 are neutrophils which could increase collagenase enzyme activity that causes membranes to spontaneously rupture or induce cervical ripening. Cytokines and eicosanoids work in concert to accelerate one another's production in a cascade-like manner, leading to additional rises in prostaglandin synthesis. Additionally, it has been suggested that the heightened inflammatory response encourages uterine contractility by directly activating genes involved in contractility [such as cyclooxygenase-2 (COX-2), the oxytocin receptor, and connexin] and/or impairing progesterone's ability to control uterine quiescence.[2,7]

FETAL HYPOTHALAMIC-PITUITARY-ADRENAL AXIS FUNCTION

The large levels of cortisol are released from the intermediate (fetal) zone of the fetal adrenal glands when the fetal hypothalamic–pituitary–adrenal axis is activated in the latter stages of pregnancy. Contrary to their detrimental impact on hypothalamic CRH synthesis, glucocorticoids (including cortisol) are powerful stimulants of fetal membrane, decidual, and placental CRH production. Placental CRH production is also induced by inflammatory cytokines, catecholamines, acetylcholine, and oxytocin, whereas

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this is inhibited by progesterone and NO. Throughout the latter half of pregnancy, the amount of circulating maternal plasma CRH rises gradually, peaking in the last 6–8 weeks before the delivery. Additionally, monitoring high maternal plasma CRH levels between 28 and 30 weeks of gestation may help identify those who are more likely to give birth prematurely.[2]

Cortisol stimulates amnionic cyclo-oxygenase, which increases prostaglandin production, while inhibiting chorionic prostaglandin dehydrogenase activity, which prevents prostaglandin metabolism. Prostaglandin synthesis in the fetal membranes at term is directly increased by glucocorticoids. The immunophilin FKBP51, which inhibits progesterone receptor-mediated gene transcription in term decidual cells, is also expressed more highly by glucocorticoids, and both term and idiopathic preterm birth is linked to this enhanced expression.[8]

MEMBRANE RUPTURE

Collagen, fibronectin, and laminin are examples of extracellular membrane proteins that give fetal membranes their strength and integrity. A class of enzymes known as matrix metalloproteases (MMPs) increases collagen breakdown and reduces membrane strength. MMPs have a variety of substrate specificities. To preserve membrane integrity, tissue inhibitors of MMPs (TIMPs) attach to MMPs and stop proteolysis.

Due to limited MMP activity and high TIMP levels, the fetal membranes often remain intact until term. At term, peri-parturitional MMP activation may result in a chain of events that compromise fetal membrane integrity and encourage rupture. Also, it is likely that uterine contractions during labor also cause membrane rupture through stretch and shear pressures.[2,9]

MYOMETRIAL FACTORS

Whether the 'trigger' for parturition starts inside the fetus or outside of it, the end outcome is always characterized by a series of regular, phasic uterine contractions that are stronger and more frequent. Similar to other smooth muscles, the adenosine triphosphate (ATP)-dependent binding of myosin to actin is what causes myometrial contractions. Myosin light chain kinase, a calcium/calmodulin-dependent enzyme, phosphorylates the myosin light chain as a prerequisite for this interaction. Thus, a major regulator of myometrial contractility is the availability of free intracellular calcium.[10]

By connecting cell membrane receptors to effector enzymes and ion channels, GTP-binding proteins (G-proteins) play a crucial role in myometrial contractility. For instance, the G-alpha-s/adenyl cyclase/cyclic adenosine 3′,5′-monophosphate (cAMP) signal transduction pathway promotes myometrial relaxation when beta-adrenergic and/or PGE2 receptors are activated. On the other hand, oxytocin receptors pair to G-alpha-q/G-alpha-i/phospholipase C pathways, which results in an increase in inositol-1,4,5-trisphosphate and 1,2-diacylglycerol, which in turn activates protein kinase C. As a result, intracellular calcium levels rise and myometrial contractions increase.[10]

UTERINE INVOLUTION

The uterus goes through a remodeling and repair phase following birth. The physiological process through which the uterus returns to its typical prepregnant form, both physically and functionally, is known as uterine involution. Reduced uterine

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blood flow and endometrial vascularity, endometrial regeneration, and the restoration of ovarian function are its hallmarks. This is due to the apoptosis and autophagy of myometrial smooth muscle cells as well as a reduction in connective tissue. This process begins extremely quickly during the first postpartum week and is predominantly controlled by oxytocin; it then progresses and is often finished by the sixth postpartum week.[2,6]

CONCLUSION

The commencement of human labor is a complex physiological event that involves several factors. The myometrium, decidua, and uterine cervix of the mother undergo slow changes in the days to weeks leading up to labor onset. Prostaglandin production and release inside the uterus, myometrial gap junction development, and myometrial oxytocin receptor overexpression are a few examples of these alterations. At term, there probably occurs a "parturition cascade" that recruits factors that promote uterine activity and eliminates mechanisms that preserve uterine quiescence. Following the preparation of the myometrium and cervix, the pattern of myometrial activity changes from erratic contractions to regular, coordinated contractions due to endocrine and/or paracrine/autocrine hormones from the fetoplacental unit (mostly from the decidua). Cervical effacement and dilation, fetal membrane rupture, and the ejection of the fetus and placenta are the final outcomes. By coordinating the shift in myometrial activity through the generation of placental steroid hormones, mechanical uterine distension, the release of neurohypophyseal hormones, and other stimulators of prostaglandin synthesis, the fetus appears to influence the timing of labor at term. The uterus goes through an involution phase in the days and weeks following delivery, which is mostly mediated by oxytocin.

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