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The biological mechanism that maintain uterine quiescence through most of pregnancy and cause transition to labor at term are not well understood. The fetal membranes are interposed between fetal compartment and maternal corrpartment and are thus ideally located to regulate signals between fetus and mother. The amnion, chorion and maternal decidua are thought to be important sources for locally derived activators of uterine contractions and these stimulatory signals important for initiating labor at term (Patricia et al., 1993).

Collins et al., 1996 reported that signals among fetal membranes, decidua and uterus play an important role in the initiation of parturition in women. They reported also that fetal membranes inhibit uterine contractions by inhibition of L-type Ca^{++} channels.

The mechanisms regulating the timing of parturition are poorly understood. Parturition events may occur in a paracrine fashion within intrauterine tissue(amnionic, chorionic membranes and maternal decidua). Thus local alterations in the level of paracrine regulatory factors could occur without being reflected in the maternal circulation. Intrauterine tissues have been demonstrated to be the site of synthesis of estrogen and progesterone (Chibbar et al., 1986) and also prostaglandins that enhance myometrial contractions at labor (Rajni et al., 1993).

It is believed that oxytocin is locally synthesized within intrauterine tissues in late pregnancy and has a role in labor since

there is no evidence for increasing circulating maternal oxytocin concentration (Amico et al., 1984).

On the other hand, it is suggested that prostaglandins in amniotic fluid may not cross the fetal membranes. Therefore, amniotic fluid prostaglandine may not be the primary stimulators of uterine contractions (Patricia et al., 1993).

There is mounting evidence that calcium plays critical roles in a wide variety of cellular functions, including the contraction-relaxation cycle of muscle. In myomtrial smooth muscle, agonists that promote uterine contractions are believed to initiate such by raising intracellular of Ca^{++} (Miklos and Frank, 1990).

Calcium ion entry through voltage-dependent calcium channels (VDCCs) is responsible for excitation-contraction in response to depolarization of the plasma membrane as action potentials propagate over the surface of the muscle cell. The VDCC opening leads to a rapid influx of Ca^{++}, activation of myosin-light chain kinase, and initiation of muscle contractions. Becasue contractions of the myometrium are dependent on action potentials, VDCCs play a critical role in the regulation of uterine contractility (Naohiro et al., 1995).