

Excerpts from
THE PLACENTA: TO KNOW ME TO IS LOVE ME

Maternal Indication: Preterm Labor

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Preterm labor (PTL) is the onset of regular uterine contractions accompanied by cervical effacement and/or dilation and fetal descent in a gravid woman at greater than 20 weeks and less than 37 weeks completed gestation (less than 259 days from the first day of the last menstrual period). It accounts for only 6-8% of all deliveries, but is associated with 75% of perinatal deaths. About 10% of survivors of preterm birth suffer from a permanent major handicap.

In most cases, the cause of preterm labor is unknown. However, **fetal etiologic factors** include: 1) premature rupture of membranes; 2) amniotic fluid infection - chorioamnionitis (group B Streptococcus and normal cervicovaginal flora such as aerobic and anaerobic bacteria, Mycoplasma, Chlamydia and Candida); 3) multifetal gestation (average duration of pregnancy decreases with each fetus); 4) polyhydramnios (about 1/3 have a preterm delivery); 5) some fetal anomalies, especially those associated with polyhydramnios (anencephaly) or with oligohydramnios (renal agenesis). **Maternal etiologic factors** include: 1) maternal disease (preg-

nancy induced hypertension, renal disease, appendicitis); 2) uterine structural abnormalities; 3) women whose mothers received DES while pregnant have a higher incidence of midtrimester preterm labor; 4) cervical incompetence; 5) history of preterm labor - 25-50% recurrence risk with each succeeding preterm delivery; 6) history of induced abortion, socioeconomic status (poor nutrition, inadequate prenatal care, low maternal age, heavy work); 8) maternal cigarette smoking.

Placental etiologic factors include: 1) antepartum hemorrhage (placental previa and abruptio placentae increase the risk for PTL by 4-5 times); 2) placental insufficiency. Grossly, the placenta may show **abruptio placentae, circumvallate placenta, small placenta, edematous umbilical cord, single umbilical artery, changes suggestive of placenta previa, infarcts, chorioamnionitis, and multifetal gestation**. Microscopically, one may also see changes associated with chronic low uteroplacental blood flow, uneven accelerated placental maturation and chronic villitis.

Chorioamnionitis is thought to cause preterm labor due to the liberation of high

levels of intrauterine prostaglandin by the decidual and amniotic epithelial cells in response to a cascade of events triggered by the interaction of bacterial products and the maternal immune system. In other words, it is the host response to infection that initiates labor, not the infection itself. Chorioamnionitis-associated PTL is a complex biochemical event which is often refractory to tocolytic therapy. Maintenance of the physical and biological integrity of the cervical mucus plug may best protect against chorioamnionitis and its complications.

Tocolysis is the pharmacological control of preterm labor. Most commonly, this is attempted using terbutaline sulfate (Brethine) and magnesium sulfate, and occasionally with Procardia and Indocin. Before any attempt is made to arrest labor, it is imperative to ascertain if prolongation of the pregnancy is likely to benefit the fetus without detriment to the mother.