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Obstetrics

ARREST OF PREMATURE LABOR Adrenergic β-mimetic compounds are potent supressors of uterine contraction. However, these compounds also affect the cardiovascular system and may cause marked hypotension and tachycardia, thereby limiting their clinical usefulness. Ritodrine hydrochloride, a new β-mimetic compound, infused iv at rates of 100 to 300 μg/min, completely inhibited uterine contractility in five of six premature labors. Moderate increases in pulse pressure and heart rate occurred. Effective inhibition of the intensity of uterine contractions accompanied by minor cardiovascular changes would seem to make ritodrine valuable for the treatment of premature labor. (*Bieniarz, J., Motew, M., and Scommegna, A.: Uterine and Cardiovascular Effects of Ritodrine in Premature Labor. Obstet. Gynecol. 40: 65, 1972.*)

HYPOXIA AND FETAL HEART RATE Fetal bradycardia which commences during a uterine contraction and persists for 30 to 60 seconds *after* the contraction is over is called "late deceleration." This pattern of bradycardia during labor is usually associated with an infant severely asphyxiated and depressed at birth. An experimental model was developed in the subhuman primate to monitor directly the cardiovascular and acid–base state of the near-term fetus during labor. The relationship between late deceleration of the fetal heart rate, acid–base state, and level of oxygenation was studied in 30 experiments. Late deceleration was accompanied by a decrease in fetal oxygen levels. Fetuses well oxygenated during uterine contractions showed no change in heart rate or late deceleration. The latter was abolished or suppressed when fetal oxygenation was improved by administration of a high concentration of oxygen to the mother. (*James, L. S., and others: Mechanism of Late Deceleration of the Fetal Heart Rate. Am. J. Obstet. Gynecol. 113:578, 1972.*)