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Maternal Hyperventilation During Labor

THE POSSIBILITY that maternal hyperventilation might have an adverse effect on fetal oxygenation and acid-base state is of particular interest to the anesthesiologist.

Many women hyperventilate during labor in response to painful contractions or undue apprehension. Shallow panting breathing is an integral part of auto-hypnosis in Lamaze's technique for painless childbirth, and may lead to overventilation with tingling, dizziness or even carpal spasm. During elective cesarean section, active hyperventilation is practiced by some anesthesiologists, more in Great Britain than in the United States, in the belief that it is of benefit to the infant.

The first evidence that maternal hyperventilation might be harmful for the fetus was obtained in 1960 when attempts were being made to improve the oxygenation and acid-base state of human infants delivered at elective cesarean section by lowering maternal Pa_{CO_2}.[1] Experiments on pregnant guinea pigs confirmed these findings,[2] but the mechanisms involved were not all delineated. More recently Motoyama and his coworkers have extended the investigation of this phenomenon[3] and their findings published in this issue of the JOURNAL provide new and interesting information. These authors suggest that alteration of *p*H rather than Pa_{CO_2} might be the controlling factor, influencing placental vascular resistance and intraplacental shunting as well as the maternal and fetal oxygen dissociation curves.

Evidence questioning the harmful effect of maternal hypocapnea on the fetus has recently been published in the Lancet.[4] Following relatively short periods of hyperventilation when the maternal Pa_{CO_2} decreased to a mean level of 15.7 mm. of mercury and the *p*H increased to 7.62, 18 infants were delivered by elective cesarean section, apparently in excellent condition. The authors, however, ignored the greater degree of acidosis and low oxygen levels in infants whose mother's *p*H was 7.65 or higher. These infants were already clearly adversely affected, although not to a degree to cause clinical depression. Had the hyperventilation continued for longer, their condition would undoubtedly have deteriorated.

It is worthwhile to point out that in the original study of Moya and his co-workers only 2 infants of 61 were depressed (25 patients were studied before the first depressed infant was observed). Although severe degrees of maternal alkalosis were not seen during spontaneous breathing, this has now been observed.[5] A very apprehensive mother was discovered to have a Pa_{CO_2} of 16 mm. of mercury and a *p*H of 7.64. This was not ameliorated by epidural anesthesia. The initial capillary sample from the fetal scalp had a *p*H of 7.32. During the next 2 hours fetal *p*H fell to 7.13 followed by the passage of meconium. Emergency mid-forceps delivery was performed and the infant had an Apgar score of 1.

While a brief period of maternal alkalemia might not adversely affect the fetus there is no evidence that this is beneficial and at the same time there is considerable evidence of its potential danger. In the light of present knowledge it would seem prudent to avoid willful hyperventilation at all times. Until all the mechanisms involved in this phenomenon have been clearly defined, the skeptical physician should satisfy his curiosity by studying experimental animals.

A number of unanswered questions still remain, particularly the effect of maternal *p*H or Pa_{CO_2} on uterine vascular resistance and

blood flow. Ideally these questions should be investigated with the fetus intact *in utero* and with monitoring devices chronically implanted in the maternal and fetal circulations because it is almost impossible to avoid some deterioration when the fetus is exteriorized in an acute preparation. Maternal hyperoxia should be avoided, and care should be taken to use only sedation or anesthetic agents which do not influence uterine blood flow.

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Anesthesia

DESYNCHRONIZED SLEEP Desynchronized electroencephalographic patterns of deep sleep in the ipsilateral hemisphere in cats appear with lesions of the medial as well as the lateral tegmental structures of the midbrain. These findings seem to indicate that: (1) there is not a single pontine structure or group of structures of crucial importance for the EEG desynchronization of deep sleep; the whole rostral pons appears to contribute to the EEG-desynchronizing influence; (2) this influence runs rostrally through the midbrain tegmentum, without following any known fiber pathway. (*Candia, O., Rossi, G. F., and Sekio, T.: Brain Stem Structures Responsible for the Electroencephalographic Patterns of Desynchronized Sleep, Science 155: 720 (Feb.) 1967.*)

MANDIBULAR NERVE BLOCK Owing to varying degrees of anomalous arterial supply of the eye and orbit by branches of the middle meningeal branch of the maxillary artery, it is possible, by forceful injection and retrograde arterial flow, to cause anesthesia of these structures during block of the inferior dental nerve. A temporary amaurosis and ophthalmoplegia may accompany anesthesia over the maxillary area. This undetected blindness may be dangerous in ambulatory subjects. (*Blaxter, P. L., and others: Transient Amaurosis after Mandibular Nerve Block, Brit. Med. J. 1: 681 (March) 1967.*)