

Are Obstetrical Personnel Required for Intraoperative Fetal Monitoring during Nonobstetric Surgery?

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OBJECTIVE:

To determine if the scientific literature supports the practice of electronic monitoring of the fetal heart rate (FHR) during nonobstetric surgery.

STUDY DESIGN:

A search of the literature from 1966 to 1995 was performed using MEDLINE.

RESULTS:

No fetal hypoxic mortality or morbidity has been documented from non-obstetric surgery without occurrence of a maternal hypoxic complication regardless of the use of FHR monitoring or whether alterations of the FHR occurred.

CONCLUSIONS:

Fetal monitoring is an indirect assessment of maternal anesthetic and surgical management that is not as specific or effective as direct assessment of the maternal parameters to detect respiratory compromise. Current clinical evidence obtained does not substantiate the need for obstetric personnel to monitor FHR changes during surgical procedures because no change in fetal outcome has been documented.

Continuous intraoperative electronic fetal heart rate (FHR) monitoring has been recommended during nonobstetric surgery in two prominent anesthesia textbooks.^{1,2} However, when Kendrick et al.³ surveyed the use of such monitoring, more than 40% of responding hospitals did not comply with this practice. When our anesthesia service insisted on obstetricians to monitor the fetus during such cases, we also had reservations regarding its utility. We then reviewed the literature to see if the practice had scientific support. If such support existed, we wished to determine if there was any indication for having physicians or nurses present in the operating room for interpretation of the continuous FHR monitoring.

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Study Design

We reviewed all articles published from 1966 to 1995 using MEDLINE that discussed FHR monitoring during nonobstetric surgery. The key words used were "surgery," "fetal monitoring," "fetal heart rate monitoring," and "uterine monitoring."

Results

There are five articles that describe monitoring in a total of nine pregnant patients undergoing general surgical procedures.⁴⁻⁸ Five of these patients had no change in baseline FHR. Katz et al.⁴ described a transient tachycardia associated with maternal desaturation. Strong and coworkers⁵ noted a transient bradycardia with positioning of the patient in the right recumbent position with flexion, which resolved with repositioning. In 1985, Liu et al.⁸ reported on five patients who demonstrated loss of variability following induction of anesthesia with two of the five demonstrating a reduction in the baseline FHR with no detectable maternal changes. These changes were related to the effect of the inhalational anesthetic on the fetus.⁸

There are seven articles that deal with a total of eight patients undergoing open heart or great vessel surgery.⁹⁻¹⁵ One author described bradycardia during a repair of a coarctation; the other articles described onset of bradycardia with the FHR dropping to <100 bpm when extracorporeal circulation was established. Although significant FHR bradycardia ensued in each report, no long-term damage was detectable. The pregnancies progressed to term and resulted in grossly normal infants.

Discussion

When compared with the obstetric experience of the FHR monitoring in labor, this review of the literature is remarkable for the absence of documentation of fetal compromise or death. Each paper documents a transient change in the FHR without sequelae even though bradyarrhythmias lasting several hours were described with bypass. The literature does not record any cases of fetal demise or sequelae from 20 years' experience of fetal monitoring in nonobstetric surgical procedures. This is surprising, until one reviews the fetal physiology and the utility of the fetal monitor.

Hypoxia is the essential fetal risk of nonobstetric surgery. It may occur as hypoxic hypoxia from inadequate maternal oxygenation or circulatory hypoxia from inadequate perfusion of the intervillous



space. The former occurs secondary to maternal respiratory failure, which leads to rapid maternal oxygen desaturation. The latter results from decreased maternal circulatory volume or from vasoconstriction of the uterine vasculature.

During the interval between diagnosis of desaturation and correction, the fetus has three modes of protection. The first mode is manifested by the oxygen-dissociation curve of fetal hemoglobin that resides significantly to the left of the curve for maternal adult hemoglobin.¹⁶ Consequently, mild to moderate decreases in maternal arterial P_{O_2} do not usually impair oxygen delivery to the fetus because (1) the percentage of saturation falls slower than does maternal P_{O_2} ; (2) fetal hemoglobin has a stronger oxygen affinity than does maternal hemoglobin; and (3) the concentration of hemoglobin in the fetal blood is approximately 3 g/dl higher than that in maternal blood.

The second mode of fetal protection is triggered by an acute decrease in P_{O_2} , which causes a hyperbolic increase of blood flow to the central nervous system and the heart, although cardiac output and placental blood flow tend to remain constant. The limit of this defense mechanism is reached when the perfusion of the central nervous system is maximal.¹⁷ Underwood and coworkers¹⁸ found that the normal placenta can maintain adequate fetal oxygenation, as represented by no change in the fetal heart, for more than 7 minutes with the mother inhaling a mixture of 10% oxygen and 90% nitrogen. Fetal oxygen decreases to values as low as 60% of control during fetal hypoxia in the chronically instrumented fetal sheep with arterial oxygen tension of 10 mm Hg. This decrease is stable for up to 1 hour, is rapidly reversed on cessation of maternal hypoxia, and is accompanied by fetal bradycardia of about 30 bpm. This series of responses may be thought of as a temporary compensatory mechanism that enables the fetus to survive long periods (up to 1 hour) of limited oxygen supply without decompensation of vital organs.¹⁹

The third mode of prevention of fetal circulatory hypoxia occurs when the expansion of the mother's intravascular volume meets the demands of the hypervascularity of the uterus. This expansion protects the fetus against the deleterious effects of impaired venous return in the supine and erect positions and safeguards the mother against the adverse effects of blood loss.²⁰

Despite extensive use of fetal monitoring, it is a difficult tool to use for interpretation because the FHR changes in the absence of labor are nonspecific. Bradycardia associated with fetal hypoxia results in acidemia, which causes direct myocardial depression.²¹ It is not pathognomonic of hypoxia, which also occurs in a multitude of situations—supine hypotension, conduction anesthesia, head compression, maternal hypothermia, extended maternal exercise, and hemodialysis. In all of these nonhypoxic situations, it resolves spontaneously with cessation of the variable state. There are no recorded fetal deaths associated with these conditions, and there is no scientific evidence to support delivery in response to such a finding.²² The bradycardia seen with extracorporeal circulation may well be a similar phenomenon. Eileen et al.¹³ suggested that it may be secondary to the hypothermia. Other authors related it to low flow rates on bypass. It responded to increased flow for some, whereas others found no

change. Regardless, all the babies had normal outcomes despite the length of the bradycardia.^{11,12}

Recommendations for intraoperative monitoring imply that there is a window of fetal vulnerability that would not be detected by assiduous monitoring of the maternal physiology.¹ A review of the literature demonstrates no evidence to support this premise for noncardiovascular surgical procedures. The pregnant woman's physiology tolerates surgery just as it tolerates challenges by exercise demands and supine positioning, which embarrass oxygenation. The literature available would indicate that well-managed general anesthesia in which O_2 saturation is maintained above 90%, blood pressure remains stable, urine output is maintained in excess of 30 ml/hr, and hematocrit is $>30\%$ represents a minimal risk of fetal hypoxia for the fetus with normal placental function.^{23–25} As long as fetal circulation is maintained, the pulse oximeter will detect maternal desaturation before the fetus will be at risk from hypoxic hypoxia. Before significant change in the FHR, the anesthesiologist should be responding maximally to correct the maternal saturation deficit.

There is no diagnosable FHR response that would prompt delivery in a maternal crisis. Because actual acidosis occurs in only 20% of patients with ominous patterns, such as sustained bradycardia or late decelerations, no particular heart rate pattern will serve as a stimulus for immediate delivery.²⁶ Postmortem cesarean section is the only delivery described in the literature in which the surgeon has been confronted with an intraoperative maternal catastrophe in a nonobstetric surgery. This is because obstetric management dictates that stabilization of the maternal condition is in the best interest of the fetus until a hopeless situation is encountered.

Conclusions

The maternal and fetal physiology during pregnancy will protect the fetus from all but the most serious hypoxic insults during surgery. Fetal hypoxic hypoxia is directly related to the failure of maternal respiration. Fetal monitoring is an indirect assessment of maternal respiratory failure. From current evidence, the same would appear true for circulatory hypoxia in noncardiovascular surgeries.

Twenty years' experience has yielded no documented evidence that obstetric personnel are required to monitor FHR changes during nonobstetric surgical procedures.

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