# Antepartum heart rate patterns in small-for-gestational-age third-trimester fetuses: Correlations with blood gas values obtained at cordocentesis

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Antepartum fetal heart rate records were made immediately before cordocentesis and blood gas analysis in 58 small- and 29 appropriate-for-gestational-age fetuses at 27 to 38 weeks' gestation. All appropriate-for-gestational-age fetuses had blood $Po_2$ and pH values within the normal ranges for gestation, and in 27 of 29 cases the heart rate pattern was reactive; in two it was nonreactive. Abnormal heart rate patterns were present in 15 of the 19 small-for-gestational-age fetuses that were found to be hypoxemic, acidemic, or both. The abnormalities included decreased baseline variation, absence of accelerations, presence of decelerations, and increased baseline heart rate. A repetitive decelerative pattern best identified the hypoxemic fetuses. Fetal $Po_2$ values in the lower normal range, present in many of the small-for-gestational-age fetuses, were in general associated with a reactive fetal heart rate pattern. (AM J OBSTET GYNECOL 1990;162:698-703.)

**Key words:** Fetal heart rate monitoring, cordocentesis, fetal blood gases, fetal hypoxemia, small for gestational age

Antepartum fetal heart rate (FHR) monitoring is widely used to assess the fetal condition. Studies correlating antepartum FHR patterns with cord blood gas results at elective cesarean section have demonstrated that decelerative patterns are associated with fetal hypoxemia.[1, 2] Furthermore, "terminal" FHR patterns, with absence of heart rate variability and the presence of decelerations after every Braxton Hicks contraction, are associated with fetal acidemia.[3, 4]

Cordocentesis has now made it possible to measure fetal blood gases in utero,[5, 6] thus avoiding possible effects of anesthesia and surgery. We have previously reported on FHR patterns in normoxemic, hypoxemic, and anemic fetuses at 20 to 26 weeks' gestation.[7] In this article we describe the relationship between FHR patterns and blood gases in appropriate- (AGA) and in small-for-gestational-age (SGA) fetuses, between 27 weeks and term.

## Patients and methods

Fetal heart rate monitoring was performed immediately before cordocentesis on 96 occasions in 87 patients at 27 to 38 weeks' gestation and the blood samples were analyzed for pH and $Po_2$. The FHR records were taken between 10 AM and 6 PM with the patient in a semirecumbent position. Recordings were made at a paper speed of 1 cm/min (Hewlett-Packard cardiotocograph model 8041A, Boblingen, West Germany). The duration of recording was 30 to 60 minutes; in cases of nonreactive or decelerative patterns the recording time was always extended to 60 minutes.

Cordocentesis was performed as an outpatient procedure without maternal fasting and sedation, or fetal paralysis. The umbilical cord vessel was identified as artery ($n$ = 17) or vein ($n$ = 79) by the ultrasonographically detected turbulence produced after the intravascular injection of 200 to 400 μl of normal saline solution.[5] Fetal blood (200 μl) was collected into heparinized syringes and blood gas analysis was performed with a Radiometer ABL 330 blood gas analyzer (Copenhagen, Denmark). The temperature at which the blood gases were measured was 37° C; this is similar to the temperature of fetal subcutaneous tissues.[8]

Blood oxygen tension in both the umbilical artery and vein decreases with gestation in normal fetuses.[6] Therefore the individual $Po_2$ values obtained in this study were expressed as the number of standard deviations by which the values differed from the normal mean for gestation for the appropriate vessel sampled.[6] Umbilical venous and arterial pH do not change with gestation and therefore acidemia was defined as a pH >2 SD below the mean (normal range for the umbilical artery, 7.33 ± 0.07 [2 SD] and for umbilical vein, 7.38 ± 0.07 [2 SD]).[6]

The fetuses were divided into two groups. In the

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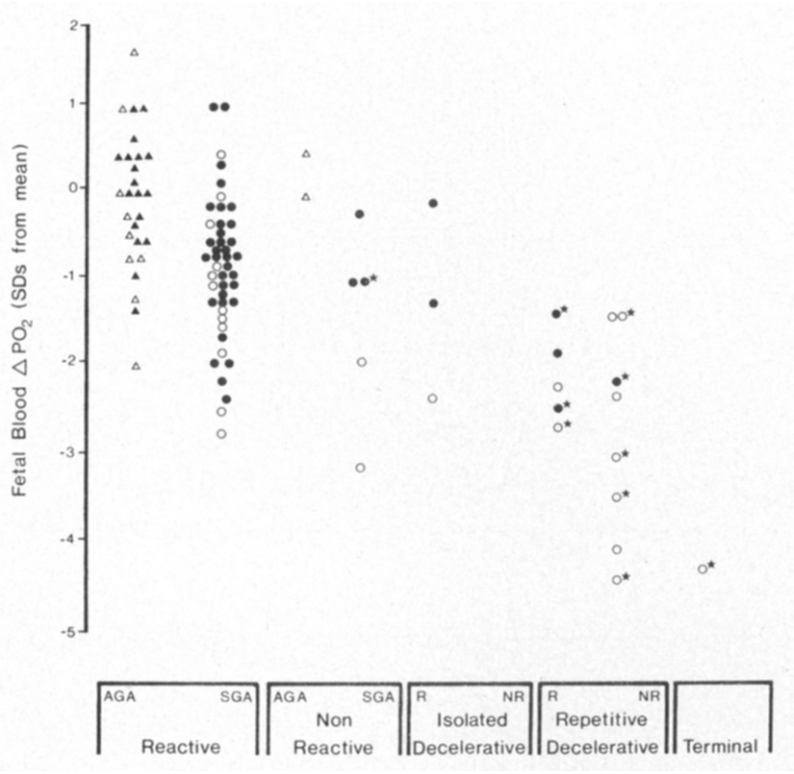


**Fig. 1.** Relationship between antepartum FHR patterns and fetal blood Δ $P_{O_2}$. The isolated and repetitive deceleration patterns were subclassified as reactive *(R)* or nonreactive *(NR)*. Appropriate and SGA fetuses are indicated by ▲ and ●, respectively. The *open symbol* relates to pregnancies of 27 to 32 weeks' gestation and the *closed symbol* to pregnancies of 33 to 38 weeks' gestation. The *asterisk* indicates acidemic cases (pH > 2 SD below mean)

**Table I.** Relationship between the different fetal heart rate patterns and presence or absence of hypoxemia (Δ $P_{O_2}$ < 2 SD below mean) or acidemia (Δ pH < 2 SD below mean) in 67 traces from 58 SGA fetuses

| *FHR pattern* | *n* | *Fetal blood $P_{O_2}$ and pH* | | | |
|---|---|---|---|---|---|
| | | *Normal* | *Hypoxemia* | *Acidemia* | *Hypoxemia and/or acidemia* |
| Reactive | 45 | 41 | 4 | — | 4 |
| Nonreactive | 5 | 3 | 1 | 1 | 2 |
| Isolated decelerations | 3 | 2 | 1 | — | 1 |
| Repetitive decelerations | 13 | 2 | 9 | 8 | 11 |
| Terminal | 1 | — | 1 | 1 | 1 |
| Total | 67 | 48 | 16 | 10 | 19 |

AGA group cordocentesis was performed for prenatal diagnosis ($n$ = 29). The indications were karyotyping in malformed fetuses (unilateral or minor bilateral renal abnormalities, $n$ = 15; diaphragmatic hernia, $n$ = 2; duodenal atresia, $n$ = 1; talipes, $n$ = 1; exomphalos, $n$ = 1) or polyhydramnios ($n$ = 2); fetal blood grouping because of disputed paternity ($n$ = 1); and measurement of fetal platelet count in cases of alloimmune or idiopathic thrombocytopenia ($n$ = 6). Cases with oligohydramnios, fetal hydrops, fetal cardiac or chromosomal defects were excluded from this group.

The second group was comprised of SGA fetuses with a fetal abdominal circumference >2 SD below the normal mean for gestation ($n$ = 67). All these cases had a normal karyotype and no malformations. The amniotic fluid volume was subjectively assessed by ultrasonography as normal in 20 cases, and reduced in 47; in 28 of the latter cases there was oligohydramnios.

The FHR traces were examined by an observer (G. H. A. V.) who was unaware of the indications for fetal blood sampling and of the pH and blood gas results. The FHR records were classified as reactive, nonreactive, flat, decelerative, or terminal.

A reactive trace was one in which there were two or

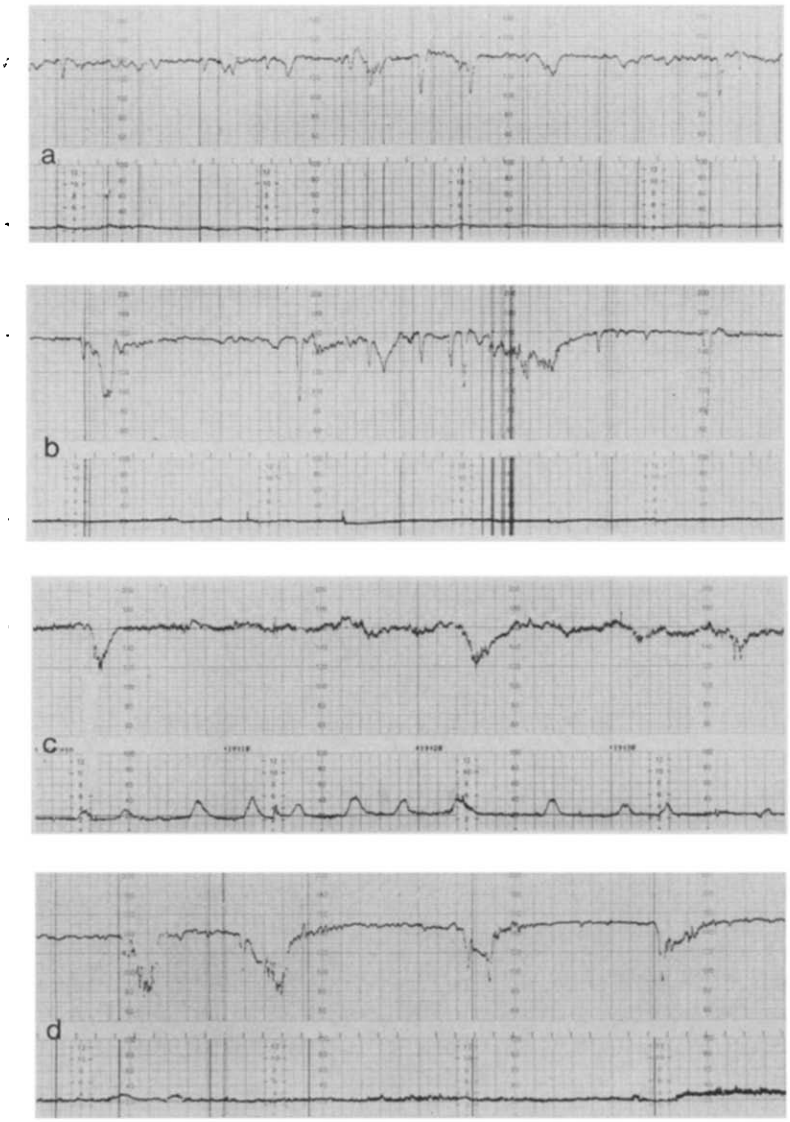


**Fig. 2.** Examples of repetitive decelerative FHR patterns. Cases *a* and *b* were moderately hypoxemic ($Po_2$ = 2.5 SD below mean), case *c* was severely hypoxemic ($Po_2$ = 4.2 SD below mean), and case *d* was severely hypoxemic ($Po_2$ = 4.5 SD below mean) and moderately acidemic (umbilical vein pH = 7.20). Paper speed was 1 cm/min. The *vertical bars* indicate fetal movement as felt by the mother.

more accelerations (amplitude ≥10 beats/min and duration ≥15 seconds) in a period of ≤30 minutes at ≤36 weeks' gestation or ≤40 minutes after 36 weeks; there were no decelerations.

A nonreactive trace was one in which there was an oscillation amplitude of >5 beats/min but less than two accelerations in a period of ≤30 minutes at ≤36 weeks' gestation or ≤40 minutes after 36 weeks; there were no decelerations.

A flat trace was one in which the oscillation amplitude was ≤5 beats/min, with less than two accelerations and no decelerations.

Decelerative traces were either repetitive, if there was more than one, or isolated, if there was only one deceleration. The decelerations considered to be significant were the U-shaped "late" decelerations after Braxton Hicks contractions, and the V-shaped "variable" decelerations that were not necessarily related to antepartum contractions. All late decelerations with a decrease from the baseline of ≥10 beats/min were included. Variable decelerations were defined as a decrease from the baseline of ≥20 beats/min lasting ≥60 seconds. The decelerative traces were subdivided into a reactive subgroup (baseline variability >5 beats/min, ≥2 accelerations, or both) and a nonreactive subgroup (baseline variability ≤5 beats/min and no accelerations).

The terminal FHR pattern was defined as being com-

**Table II.** Relationship between the different components of the FHR pattern and presence or absence of hypoxemia or acidemia in 67 traces from 58 SGA fetuses*

| *Components of FHR pattern* | *n* | *Fetal blood $Po_2$ and pH* | | | |
|---|---|---|---|---|---|
| | | *Normal* | *Hypoxemia* | *Acidemia* | *Hypoxemia and/or acidemia* |
| Accelerations | | | | | |
| Present | 48 | 41 (85%) | 6 | 2 | 7 |
| Absent | 19 | 7 | 10 | 8 | 12 (63%) |
| Baseline variation | | | | | |
| >5 beats/min | 58 | 47 (81%) | 9 | 4 | 11 |
| <5 beats/min | 9 | 1 | 7 | 6 | 8 (89%) |
| Decelerations | | | | | |
| Absent | 50 | 44 (88%) | 5 | 1 | 6 |
| Present | 17 | 4 | 11 | 9 | 13 (76%) |
| Heart rate | | | | | |
| <150 beats/min | 52 | 42 (81%) | 9 | 5 | 10 |
| >150 beats/min | 15 | 6 | 7 | 5 | 9 (60%) |

*The negative and positive predictive values are in parentheses.

pletely flat with late decelerations after each Braxton Hicks contraction.

This classification was based on previously published reports.[3, 4, 7] Modifications were made to consider fetal rest-activity cycles and a gestational age effect on FHR patterns.[9] Because accelerations >15 beats/min are not a consistent phenomenon in normal pregnancies of <36 weeks,[9] the presence of accelerations of at least 10 beats/min was considered to indicate a reactive pattern.

## Results

The blood $Po_2$ and pH values were within the normal ranges for gestation in all AGA fetuses (Fig. 1). The fetal heart rate patterns were either reactive ($n$ = 27) or nonreactive ($n$ = 2). Both nonreactive traces were from fetuses <33 weeks' gestation and a change from nonreactive to reactive patterns occurred after 40 and 50 minutes of recording, respectively, in both.

In the SGA group, 45 records were classified as reactive, five as nonreactive, three as isolated decelerative, 13 as repetitive decelerative, and one as "terminal" (Table I). There were no cases with a flat trace. In 41 of 45 cases with a reactive FHR trace, the $Po_2$ value was in the (lower) normal range for gestation. Four fetuses were hypoxemic with a $Po_2$ >2 SD below the normal mean for gestation; this group included three of the 10 cases in which an arterial and one of 35 in which a venous sample had been taken. In none of the cases with a reactive FHR trace was the fetus found to be acidemic. Of the five cases with a nonreactive FHR pattern, one was hypoxemic and one acidemic (umbilical vein pH = 7.29; Fig. 1). In the latter case the nonreactive pattern continued for the entire 60 minutes of recording; in the other four cases a change from nonreactive to reactive occurred after 40 to 55 minutes of recording.

All three records with an isolated deceleration were reactive, with a baseline variation >5 beats/min. In one case there were accelerations; this was the only hypoxemic fetus in this subgroup. None of these three cases were acidemic.

Of the 13 fetuses with repetitive decelerative patterns (Fig. 2), 11 were hypoxemic, acidemic, or both (Table I; Fig. 1). The other two fetuses had $Po_2$ values in the lower normal range (1.5 and 2 SD below the normal mean for gestation, respectively). The lowest $Po_2$ values were found in the nonreactive subgroup. In the reactive subgroup all records had a baseline variability >5 beats/min and in two cases accelerations were present; however, both fetuses were hypoxemic, acidemic, or both. With a repetitive decelerative pattern acidemia was mild or moderate with umbilical vein pH ranging from 7.20 to 7.31. In contrast, the fetus with a terminal FHR pattern was severely acidemic (umbilical vein pH = 6.90). Variable decelerations were observed in only three of the 16 records that were classified as decelerative. Two of these fetuses were hypoxemic and none were acidemic.

When the FHR pattern was classified as reactive ($n$ = 45) or otherwise ($n$ = 22) the sensitivity and specificity for the detection of normal (within 2 SDs of the appropriate mean for gestation) and abnormal (>2 SDs below the mean), blood gases and pH values were 79% and 85%, respectively, and the positive and negative predictive values were 68% and 91%, respectively. In Table II the different components of the FHR pattern are related to the data obtained at cordocentesis. The negative predictive value of the individual components varied from 81% to 88% and the positive predictive value from 60% to 89%. This also included FHR itself, which tended to be mildly increased (>150 beats/min) in cases of hypoxemia or acidemia (Table II; $\chi^2 = 7.62$, $p < 0.01$). A mean rate >160 beats/min was only observed in four cases, three of which were hypoxemic, acidemic, or both.

The predictive value of antepartum FHR patterns

with respect to $Po_2$ and pH values was not different in fetuses studied at 27 to 32 weeks compared with older fetuses (Fig. 1). Although there were more fetuses with abnormal FHR patterns in the former group, the incidence of abnormal blood gases was also higher.

## Comment

This study demonstrates that moderate to severe fetal hypoxemia, acidemia, or both are associated with changes in the FHR pattern. However, a fetal $Po_2$ value in the lower normal range, found in many of the SGA fetuses, is in general associated with a "normal" reactive FHR pattern. This indicates that changes in FHR pattern coincide with the occurrence of hypoxemia or acidemia. The lack of identification of fetuses with a $Po_2$ value in the lower normal range indicates that antepartum FHR monitoring is not a reliable screening method for a forthcoming impairment.

These data substantiate the findings of earlier studies in which antepartum FHR patterns were related to cord blood gases obtained after elective cesarean section. In those studies it was also found that the $Po_2$ value tends to be within the (lower) normal range in SGA fetuses with a normal FHR pattern,[1] whereas with decelerations hypoxemia and sometimes mild acidemia are present.[1,2] A "terminal" FHR pattern is associated with moderate to severe acidemia.[3,4]

With a reactive FHR pattern none of the SGA fetuses were acidemic, but four were hypoxemic ($Po_2$ >2 SD below the normal mean). Because the samples in these cases were predominantly arterial (artery, 3/10; vein, 1/35) it could be postulated that the hypoxemia was corrected through a single passage in the placenta and that therefore there was no venous hypoxemia to be detected by the chemoreceptors. Another possibility is that sampling from the umbilical artery might induce a vasospasm and therefore provide less reliable blood gas data. Retrospective reanalysis of the reactive FHR patterns and a change of the criterion of reactivity (accelerations >15 beats/min instead of >10 beats/min) did not improve the predictive value and only increased the number of false-positive tests. Because all reactive records had a baseline variability of >5 beats/min, inclusion of this criterion did not improve the diagnostic value.

The implications of a nonreactive FHR record are unclear. If there is a spontaneous change to reactivity (within 30 minutes before 36 weeks or 40 minutes after 36 weeks), then the most likely explanation is that the nonreactive period is part of the rest-activity cycle and thus without adverse implications.[9] Although longer periods of nonreactivity may be a variant of this normal rest-activity cycle (flat traces of up to 80 minutes have been found in normal pregnancies), they may also be indicative of fetal brain damage or asphyxia.[10-12] The latter is demonstrated in this study: two of five SGA fetuses with a prolonged nonreactive pattern were hypoxemic or acidemic. In the only acidemic fetus in this subgroup there was no change to a reactive pattern during the entire hour of recording; this suggests that there is absence of cyclic variations in FHR in acidemia. However, a larger number of cases with longer durations of recording are necessary before this can be substantiated. The large variation in blood gas results within the group with a nonreactive FHR pattern suggests that a nonreactive FHR pattern would require investigation by further FHR recordings or other techniques, such as Doppler ultrasonography or cordocentesis, to determine the actual fetal condition.

With a repetitive decelerative FHR pattern, most fetuses were hypoxemic, acidemic, or both, whereas with isolated decelerations, two of the three fetuses had normal blood gas results. These data are compatible with the findings that with a repetitive decelerative FHR pattern asphyxia during labor is invariable, whereas with an isolated antepartum deceleration, the majority of fetuses withstand the stress of labor.[4] Therefore the finding of an isolated antepartum FHR deceleration requires further investigation.

There were accelerations in only two of the 13 records with repetitive decelerations and most records had a baseline variation <5 beats/min. This indicates that heart rate variation decreases and that accelerations disappear with the occurrence of decelerations. It has been shown in both a cross-sectional and a longitudinal study in which FHR patterns were assessed numerically that heart rate variation falls below the norm at about the same time as decelerations occur.[1,13] Table II shows that the predictive value of the different components of the FHR pattern are almost the same, which also suggests time-related changes. With a repetitive decelerative trace most fetuses were hypoxemic, acidemic, or both. Although in this study we did not measure fetal blood oxygen saturation, it is possible that differences in this measurement would have been greater than those in $Po_2$ between fetuses with normal and those with decelerative FHR patterns.

When should the SGA fetus be delivered? It may be inadvisable to await the development of FHR decelerations before delivery is undertaken because the neurologic outcome of such fetuses is poorer than that of SGA fetuses without decelerations.[12] However, at present there is no reliable method to identify those fetuses that would develop FHR decelerations or the time course of this event. Although unselected premature delivery of all SGA fetuses with nondecelerative FHR patterns may reduce the risk of intrauterine brain damage, the substantial rise in prematurity-related mortality or morbidity may limit such an approach.

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# Fetal assessment based on fetal biophysical profile scoring

## IV. An analysis of perinatal morbidity and mortality

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The relationship between the last biophysical profile score result and perinatal outcome was determined among a large referred population of high-risk pregnancies. A highly significant inverse linear correlation was observed for fetal distress, admission to neonatal intensive care unit, intrauterine growth retardation, 5-minute Apgar score <7, and umbilical cord pH <7.20 but not for the incidence of meconium or major anomaly. A highly significant inverse exponential (log 10) relationship was observed for perinatal mortality in total and by component parts and cause. These data strongly suggest the biophysical profile scoring method of fetal risk assessment is accurate and also provides insight into the extent of fetal compromise. (Am J Obstet Gynecol 1990;162:703-9.)

**Key words:** Biophysical profile score, perinatal morbidity, perinatal mortality

As in the past, the art of obstetrics currently remains essentially the weighing and balancing of relative risks, culminating in a clinical management strategy. This balancing equation, viewed from the perinatal perspective, must consider the risk of death (stillbirth) or damage in utero against the risk of death or damage as a result of iatrogenic prematurity and against the attendant maternal risks that are known to rise with interventional measures. The clinical equation must be further interpreted within the context of prevalence of disease within the perinatal population and the progression and variation in maternal risk factors. The dramatic and ongoing improvements in neonatal care that have resulted in a decline in neonatal mortality rates have had a profound impact on this clinical equation because intact survival may be anticipated at even earlier gestational ages. The advent of methods for monitoring of fetal biophysical activities and responses to stress in utero offer further means of refinement of relative perinatal risk. It follows that if such tests are to be used in clinical management decisions, their parameters of predictive accuracy of the methods must be defined. The purpose of this prospective clinical study was to

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