Fetal acidemia and electronic fetal heart rate patterns: Is there evidence of an association?

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Abstract

Objective. Despite the ubiquity of electronic fetal monitoring, the validity of the relationship between various fetal heart rate (FHR) patterns and fetal acidemia has not yet been established in a large unselected series of consecutive pregnancies. The aim of this study was to examine the published literature for evidence of such a relationship.

Methods. Four hypotheses based on assumptions in common clinical use were examined. The literature was searched for relationships between certain aspects of FHR patterns (e.g., degree of FHR variability, depth of decelerations), and fetal acidemia, or fetal vigor (5-minute Apgar score ≥7). We also attempted to relate duration of these patterns to the degree of acidemia. Using standardized FHR nomenclature we defined patterns based on baseline FHR variability, baseline rate, decelerations, and accelerations.

Results. The following relationships were observed: (1) Moderate FHR variability was strongly associated (98%) with an umbilical pH ≥7.15 or newborn vigor (5-minute Apgar score ≥7). (2) Undetectable or minimal FHR variability in the presence of late or variable decelerations was the most consistent predictor of newborn acidemia, though the association was only 23%. (3) There was a positive relationship between the degree of acidemia and the depth of decelerations or bradycardia. (4) Except for sudden profound bradycardia, newborn acidemia with decreasing FHR variability in combination with decelerations develops over a period of time approximating one hour. Most studies identified were observational and uncontrolled (grade III evidence of US Preventive Services Task Force); however, there was general agreement amongst the various studies, strengthening the validity of the observations.

Conclusions. The validity of the relationship between certain FHR patterns and fetal acidemia and/or vigor, is supported by observations from the literature. In addition four assumptions commonly used in clinical management are supported. These conclusions need to be confirmed by a prospective examination of a large number of consecutive, unselected FHR patterns, and their relationship to newborn acidemia. Pending the completion of such studies, these observations can be used to justify certain aspects of current clinical management, and may assist in standardizing the diversity of opinions regarding FHR pattern management.

Keywords: Fetal pH, fetal monitoring, electronic FHR monitoring, fetal acidemia

Introduction

Electronic fetal heart rate (FHR) monitoring was introduced into clinical practice without appropriate studies on its reliability (intra- and inter-observer variability), validity (relationship of FHR patterns to fetal outcome), and causal relationship to outcome (ability of intervention to avoid metabolic acidemia) [1].

Recommendations for studies of each of these items were amongst the conclusions of the National Institute of Child Health and Human Development (NICHD) Research Planning Workshop, Electronic fetal heart rate monitoring: Research guidelines for interpretation [2]. In order to determine the validity of FHR monitoring, the suggestion was for a large descriptive epidemiological study of the frequency of different FHR patterns using the standardized definitions, and correlation of these patterns with several immediate outcome measures, including umbilical vessel blood gases and acid–base state (in particular metabolic acidemia) and Apgar scores.

The NICHD panel stated that studies of the reliability and validity of FHR monitoring should precede the development of a system of management of FHR patterns, because such studies would most
likely influence the protocols. However because of the ubiquity of electronic FHR monitoring in North America, we felt it was important to attempt to develop preliminary algorithms for managing patterns in our own institution based on the best available data.

Although a large epidemiological study referred to above has not yet been carried out in an unselected, consecutive series of patients, a number of authors over the past four decades have examined such a relationship in relatively small unselected series or in selected pregnancies, particularly in the earlier years. The aim of our study was to examine the literature to see if FHR patterns are related to fetal acidemia and/or Apgar scores in the series that have been reported, recognizing that these conclusions will be tentative, pending further more valid studies.

Methods

A single institution committee was formed to objectively evaluate the literature. There were representatives from obstetrics, midwifery, nursing, epidemiology, and quality improvement. We searched for papers relating fetal heart rate patterns to acid–base state of the fetus, or Apgar scores at birth, using Medline. Approximately 50 papers were reviewed, and following comparison with reference lists and personal files, 18 were selected for this study based on the inclusion of appropriate data that could be analyzed.

We rated the quality of evidence based on the criteria adopted by the US Preventive Services Task Force [3]. We used the standardized fetal heart rate nomenclature described by the NICHD Research Planning Workshop [2].

The outcome measures used depended on the data reported. Measures of metabolic acidemia were only available in two publications, one using a base excess cutoff of −12 and the other −16 mEq/L. The pH of fetal umbilical arterial or scalp blood was more commonly reported, with either a threshold of 7.15 or 7.20 to define acidemia. We used the lower value when available. When neither base excess nor pH were reported, we used the Apgar score, defining newborn depression as a 5-minute score of <7.

We evaluated four hypotheses regarding the relationships between the FHR patterns and acidemia and/or Apgar score. They were based on assumptions in common clinical use [2,4–6]. These hypotheses were:

1. Moderate (normal) FHR variability is associated with the absence of acidemia and the presence of a non-depressed (vigorous) neonate.

2. Minimal or undetectable FHR variability in the presence of late decelerations or variable decelerations, is associated with the presence of acidemia and/or a depressed neonate.

3. The depth of decelerations is directly related to the degree of acidemia and/or neonatal depression.

4. In the presence of progressive decelerations in a fetus with an initially normal FHR pattern, and the absence of catastrophic events, the development of significant acidemia evolves over a significant period of time.

Results

The relationship of moderate FHR variability to the non-acidemic vigorous neonate

Five relevant publications were identified where FHR pattern descriptions, and fetal blood gases and acid–base state from scalp samples or Apgar scores were available [7–11]. All of the tracings used had moderate FHR variability, and we did not exclude series where there were concomitant decelerations or bradycardias. The tracings were examined in the last 30 minutes before delivery, or at the time when fetal blood samples were taken.

These papers included a total of 1551 fetuses or newborns that were appropriate for inclusion in the analysis. Of these, 1518 were either non-acidemic, with pH >7.20 or had 5-minute Apgar scores >7, depending on the endpoint used by the authors. Thus 98% of the fetuses with moderate FHR variability, whether or not there were decelerations or bradycardia, were either non-acidemic or vigorous. It was not possible to relate base excess values in fetal blood to the fetal heart rate patterns in these publications.

A further paper was identified that related FHR variability scored by five different methods to outcome, but individual numbers could not be determined for inclusion in the above analysis. In 1968 pregnancies the presence of moderate variability had >99% negative predictive value for adverse outcome, defined as 5-minute Apgar score <7, and >85% negative predictive value for umbilical arterial blood pH <7.2 [12]. In a further publication from the same group, it was concluded that in 2200 consecutive deliveries, fetuses with a normal FHR trace, or mild variable decelerations, or decreased FHR variability, or mild bradycardia, or accelerations present, in the last 30-minute segment recorded before delivery, had an Apgar score ≥7 in 99.7%, and umbilical arterial cord pH ≥7.15 in 96.9% of cases [13]. More recently in a study of 488 term fetuses with monitor tracings for two hours before delivery and including at least part of the last 30 minutes, the authors state that the fetuses with the presence of normal variability, even with late or
variable decelerations, had a cord pH of $\geq 7.0$ in $\geq 97\%$ of cases [14].

The relationship between minimal or undetectable FHR variability in the presence of late decelerations or variable decelerations, and the presence of acidemia and/or a depressed neonate

Eight papers were identified which included a total of 588 appropriate patients, all of which had at most minimal FHR variability and decelerations [7–10,15–18]. We were not always able to distinguish between undetectable or minimal variability in the published data because they were sometimes grouped together. In this combined series, 137 patients had fetuses or newborns with pH $< 7.2$, or a base deficit $> 12$ mEq/L in fetal blood, or 5-minute Apgar scores $< 7$. That is, approximately 23% of the babies with reduced FHR variability and decelerations had acidemia or newborn depression.

Further support for the hypothesis was qualitatively evident in a study of 71 term newborns with umbilical artery base deficit $> 16$ mEq/L and 71 case controls. Ten of 11 with absent FHR variability, usually accompanied by late or variable decelerations, were in the asphyxia group [19].

In a more recent study of tracings of 488 term fetuses, 31% had an umbilical artery pH $\leq 7.0$ when the FHR variability was minimal or absent in the last hour before delivery [14]. In those with decreased FHR variability and late decelerations, 24% had pH $\leq 7.0$, and in those with decreased FHR variability and variable decelerations, 13% had a pH $\leq 7.0$. Base deficit in the same study group was $< 16$ mEq/L in 39% of those with decreased variability, in 32% with decreased FHR variability and late decelerations, and in 13% of those with decreased FHR variability and variable decelerations [14]. These incidences of acidemia are in the same range as those noted in the combined series described above.

The relationship of the depth of decelerations to the severity of acidemia and/or neonatal depression

It is commonly accepted clinically that the deeper the decelerations the greater the likelihood of acidemia and/or depression. Three papers contained information addressing this subject, and they included a total of 703 patients with deceleration patterns having variable degrees of FHR variability, not always stated [8,16,20].

To determine if there was a dose–response relationship between the depth and/or duration of decelerations and fetal or neonatal acidemia or depression, we compared the outcome with mild versus severe decelerations, defined by the same standard [20]. Severe late decelerations were associated with a pH $< 7.15$, or an Apgar score $< 7$ at 5 min, in 35 of 55 cases (64%), whereas mild decelerations were associated with 3/119 (3%). If we consider only those cases where there was reduced FHR variability with severe late decelerations, 28 of 28 cases (100%) had pH $< 7.15$. With reduced variability and mild late decelerations 0 of 33 were acidemic [8] (Table I).

Severe variable decelerations were associated with fetal acidemia or low 5-minute Apgar scores in 22 of 201 patients (11%) whereas mild decelerations were associated in 10 of 287 cases (3%) (Table I).

The relationship between acidemia and depth of variable decelerations is evident in another study where FHR variability was not stated. The mean fetal pH with variable decelerations of $> 60$ bpm below baseline was 7.16, whereas with decelerations of $30–60$ bpm it was 7.25, compared with a control value of 7.32 [21].

The time course of development of significant acidemia in the presence of decelerations when there is an initially normal FHR pattern, and absence of catastrophic events

In a study of term fetuses with an initially normal FHR tracing and normal scalp blood pH, but who subsequently developed an abnormal tracing based on a scoring system, it was found that they remained non-acidemic (scalp blood pH $> 7.25$), for at least 90 min of the abnormal pattern [22].

Low et al. [23] showed that there was an approximately one-hour window from the start of FHR patterns containing minimal baseline variability and late or prolonged decelerations, which preceded fetal asphyxial decompensation and newborn morbidity.

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<td>Severe</td>
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<td>Late decelerations</td>
<td>64% (35/55)</td>
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<td>Variable decelerations</td>
<td>11% (22/201)</td>
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FHRV = fetal heart rate variability. Data from references 8, 16, and 20.
In a case-control study of fetuses with umbilical artery pH < 7.05, Ingemarsson et al. [24] concluded that the four-year follow-up developmental screening test was worse when the pathologic tracing lasted for more than 60 minutes.

These three studies support the concept that in the absence of catastrophic events, in a fetus with an initially normal FHR pattern, the development of significant acidemia in the presence of variant FHR patterns evolves over a significant period of time, of the order of at least one hour.

Most of the above studies were judged to be grade III evidence (observational and uncontrolled studies) of the US Preventive Services Task Force [3], although two were case-control studies (grade II). However within each hypothesis each of the authors came to similar conclusions, which tends to strengthen the quality of the evidence.

**Discussion**

Fetal heart rate monitoring is being used in virtually all delivery suites within North America. Interpretations and management decisions are often being made in a non-standardized and sometimes subjective way, unsubstantiated by evidence-based observations. Ideally, the relationship between specific FHR patterns and fetal acidemia should be determined in a prospectively gathered series of unselected cases that includes the full range of different FHR patterns recorded up until the time of birth, and measurements of umbilical cord arterial blood gases and acid–base state, and other measures of newborn outcome. This would enable the determination of the validity of a relationship between specific patterns and fetal acidemia. However, in the absence of such a series, we are forced to use the observational data that are available.

The correlations noted in our results are impressive. Hypothesis 1, the generally accepted clinical belief [2,4–6] that the presence of FHR variability, even in the presence of decelerations, is highly associated with the absence of significant metabolic acidemia is supported. The 98% association noted in this study should be considered a minimal value, and the actual predictive value may be somewhat higher, because a number of the FHR patterns were not available immediately before delivery, and subsequent acute events might have worsened the degree of acidemia or newborn depression actually recorded at birth. In some cases the acidemia may have been respiratory rather than a metabolic acidemia, and it is generally believed that respiratory acidemia is a much less important index of morbidity. A further reason for the lack of a higher correlation might be that there are degrees of metabolic acidemia in the fetus that reflect maternal acidemia, and not intrinsic anaerobic metabolism within the fetus. It is not possible to determine the influence of this potential limitation without concomitant maternal acid–base values.

Although it is generally accepted (hypothesis 2) that there is an association between acidemia and minimal or undetectable FHR variability in the presence of decelerations [2,4–6], we found that the overall correlation was only 23%. With absent FHR variability and severe decelerations the association is probably much higher [8]. This lower correlation with simply reduced FHR variability may reflect the fact that we were unable to separate out minimal from undetectable variability cases in most of the publications. Thus the strength of the relationship between absent variability with periodic changes may have been diluted, and the true relationship will have to await confirmation from a large prospective series. However, the ability of investigators to accumulate such cases will be limited, as most clinicians will not tolerate such patterns before intervening.

The relationship between the depth of the decelerations and likelihood of acidemia (hypothesis 3) is greater with severe late decelerations than with severe variable decelerations. With severe late decelerations 64% of fetuses were acidemic or depressed, whereas only 3% of those with mild late decelerations were so. In one study the relationship in the presence of reduced FHR variability was even stronger, in that no fetuses with mild late decelerations were acidemic, whereas 100% of those with severe late decelerations were. With severe variable decelerations only 11% were acidemic or depressed, and 3% of those with mild decelerations were so [8]. These observations are of potential importance in management, because they support the approach of following the evolution of the tracing based on the deepening of decelerations to indicate risk of acidemia. Together with an evolving reduction of FHR variability this may serve as a trigger for action. The dose–response relationship is sufficiently impressive to use at least tentatively as an indicator of the acceptability of this form of management.

The fact that with evolving patterns acidemia develops over a reasonably long period of time (hypothesis 4), approximately one hour, is further support for the practice of following the evolution of FHR patterns to predict acidemia, in that it suggests that one has time, at least in theory, for obstetric decision-making, attempts at amelioration of patterns, and appropriate intervention before serious acidemia sufficient to damage the fetus has occurred. However this raises another important aspect of FHR pattern management, that is the intervention time, or 'decision–delivery' time, which may be vastly different in various institutions. The logistical realities
of each individual labor and delivery suite will determine at what stage of the evolution of the pattern the intervention should occur.

This study has a number of limitations. Firstly, the individual cases were mostly selected by the various authors, generally on the basis of the ‘abnormality’ of the FHR pattern. Secondly, in only rare cases did we have the full fetal or umbilical cord acid–base panel, including base excess, and instead we had to rely mostly on pH. Thirdly the pHs were mostly grouped according to a preconceived view of ‘abnormality’. This has been defined by most authors as a threshold of <7.15 or <7.2. In a number of cases blood gases were not available so we had to use the Apgar score in a similar way to the way in which we used pH as a surrogate for metabolic acidemia.

The findings in this review form a sufficient basis, we believe, for strongly recommending that a prospective clinical survey be performed, correlating fetal heart rate patterns and presence of acidemia. Meanwhile, we believe the tentative findings of this paper justify the setting up of standardized algorithms for management of FHR patterns, with the aim of avoiding serious metabolic acidemia and minimizing unnecessary obstetric intervention. We believe these algorithms are needed now, even while studies of the reliability and validity of FHR monitoring are pending, because of the almost universal use of monitoring in North America, and the diversity of clinical management schemas being used [2]. We also believe that standardization of management is required even while awaiting agreement with regard to the results of trials of ancillary techniques, such as pulse oximetry [25,26], EKG ST segment analysis [27,28], and computerized decision support tools [29], because of the substantial lag time which often accompanies the clinical acceptance or rejection of new obstetrical technologies, even after the publication of results of randomized controlled trials [30].

Once algorithms based on reasonable evidence have been formulated they should then be subjected to prospective examination by appropriately designed trials. This would then allow a much more rational usage of FHR monitoring in contemporary obstetric practice.

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References


