*Original Article*

Reproductive Sciences
2019, Vol. 26(6) 858-863

DOI: 10.1177/1933719119833796
journals.sagepub.com/home/rsx
SAGE

# The Fetal Reserve Index Significantly Outperforms ACOG Category System in Predicting Cord Blood Base Excess and pH: A Methodological Failure of the Category System

**Mark I. Evans, MD[1,2,3], David W. Britt, PhD[1], Robert D. Eden, MD[1], Paula Gallagher, PhD[1], Shara M. Evans, MSC, MPH[1,4], and Barry S. Schifrin, MD[1]**

## Abstract

**Objective:** Electronic fetal monitoring (EFM) has been used extensively for almost 50 years but performs poorly in predicting and preventing adverse neonatal outcome. In recent years, the current "enhanced" classification of patterns (category I-III system [CAT]) were introduced into routine practice without corroborative studies, which has resulted in even EFM experts lamenting its value. Since abnormalities of arterial cord blood parameters correlate reasonably well with risk of fetal injury, here we compare the statistical performance of EFM using the current CAT system with the Fetal Reserve Index (FRI) for predicting derangements in base excess (BE), pH, and $pO_2$ in arterial cord blood. **Methods:** We utilized a research database of labor data, including umbilical cord blood measurements to assess patients by both worst CAT and last FRI classifications. We compared these approaches for their ability to predict BE, pH, and $pO_2$ in cord blood. **Results:** The FRI showed a clear correlation with cord blood BE and pH with BE being more highly correlated than pH. The CAT was much less predictive than FRI ($P < .05$). The CAT II cases had FRI scores across the spectrum of severity of FRI designations and as such provide little clinical discrimination. The $PO_2$ was not discriminatory, in part, because of neonatal interventions. **Conclusions:** The Fetal Reserve Index (FRI) provides superior performance over CAT classification of FHR patterns in predicting the BE and pH in umbilical cord blood. Furthermore, the CAT system fails to satisfy multiple fundamental principles required for successful screening programs. Limitations of CAT are further compounded by assumptions about physiology that are not consistent with clinical observations.

## Keywords

Fetal Reserve Index, electronic fetal monitoring, ACOG category system, statistical performance metrics, base excess, pH

## Introduction

Electronic fetal monitoring (EFM) was developed nearly 50 years ago to monitor fetal acidemia in labor in order to timely predict and prevent significant fetal compromise. In retrospect, its rapid, broad implementation was predicated on a limited number of publications that were not randomized control trials.[1] Subsequently, numerous publications have either confirmed or strongly denied the efficacy of this approach[2-5] but invariably advanced the notion that EFM is largely responsible for the rise in cesarean delivery rate (CDR).[6,7] Indeed, some of its fundamental principles (eg, lack of fetal acidosis and the absence of neurologic injury with category [CAT] II) have been shown to be erroneous.[2-4]

There are several important problems confounding the assessment of EFM starting with its mathematical foundation as a screening test.[5] Even ignoring basic principles of statistical screening (principally identifying a large portion of abnormal cases for a small proportion of false positives),[8] there are problems with the biological underpinnings and pathophysiology of the classifications of patterns.[5,9,10] At a practical level, there are problems with the reproducibility of interpretation—even among experts as well as widespread confusion among

[1] Fetal Medicine Foundation of America, New York, NY, USA
[2] Comprehensive Genetics, PLLC, New York, NY, USA
[3] Department of Obstetrics & Gynecology, Mt. Sinai School of Medicine, New York, NY, USA
[4] Department of Maternal and Child Health, Gillings School of Public Health, University of North Carolina at Chapel Hill, NC, USA

**Corresponding Author:**
Mark I. Evans, Comprehensive Genetics, 131 E 65th St, NY 10065, USA.
Email: evans@compregen.com

obstetrical providers, patients, and researchers as to whether EFM is a screening test or a diagnostic test.[9] Since interventions are provided in response to seemingly worrisome screening data, there were never enough natural history data to develop accurate and reproducible statistical performance metrics. Further, there is confusion about the importance of fetal acidemia/asphyxia as the exclusive end point of the classification, the delineation of decreased and absent variability, the meaning and significance of the various classifications of FHR decelerations, and the need for intervention for the breadth of abnormal FHR combinations.[10]

Now, after 50 years of virtually universal acceptance and use, it would be considered highly unethical, and with considerable medicolegal exposure, to ignore ominous EFM data; thus, such primary investigations can never be obtained. The best that can be done is to obtain "strong inference" from both animal models and extrapolate from what clinical data are available.[6] In an effort to standardize EFM interpretation of and improve its performance, American College of Obstetricians and Gynecologists (ACOG) published Monographs in 2003 and 2014 citing criteria necessary to determine whether intrapartum events were responsible for neonatal encephalopathy (NE).[7,8] ACOG then established and implemented the classification of FHR patterns with the "CAT I-III" system in 2008 to "improve patient management," but the introduction was without prospective evidence for its metrics, or facilitating its introduction into clinical practice.[11]

To meet these challenges, we developed the Fetal Reserve Index (FRI) by combining the traditional EFM variables with risk factors (maternal, obstetrical, and fetal) and uterine contraction characteristics. The FRI has high performance metrics in the identification of cases entering labor with apparently normal status and an unharmed fetus, who: (1) went on to have a baby with cerebral palsy (CP), (2) would require an emergency operative delivery (EOD), (3) its real-time use has appeared to lower the risk of EOD and the overall CDR.

Here, we compared the ability of the FRI and CAT to predict umbilical cord blood base excess (BE) and pH. The continued reliance on the CAT (I-III) system may lie in part on its appearance of high positive predictive values and some dubious claims of the relationship of CAT II (no risk of acidemia or injury) and CAT III (high risk of acidemia).[11] The CAT system appears to do this only by defining "at risk" (of fetal acidemia) as CAT III and excluding this diagnosis for CAT I and CAT II when they likely represent a continuum.[12,13] At best, the false-positive rate (acidemia present) of CAT III is about 50%, while the more important false-negative rate (those erroneously believed to be nonacidemic or without risk of harm) of CAT II tracings permits the continued observation of the threatened fetus.[10,14,15] Conversely, using the CAT II as an at-risk marker is completely impractical since the majority of patients are classified as CAT II (70%-80%). To analyze this issue directly, we compared the relative capacity of the CAT system and FRI to predict the occurrence of a BE value of $\leq -12$ MIU/mL.

## Methods and Materials

We retrospectively analyzed 248 singleton, term cases from a prospectively created research database, not previously included in our studies, of randomly selected high-risk, singleton, term patients having a trial of labor, and all entered labor with reassuring tracings. All cases had had close medical supervision by experienced faculty and had good outcomes. Data collected included standard demographic and clinical outcome measures. Laboratory measurements included cord blood arterial pH, BE, and $pO_2$.

As we have previously published, the FRI divides the EFM into quantifiable components (heart rate, variability, accelerations, decelerations, and uterine activity). It contextualizes these components by quantifying uterine contractions along with standard medical (eg, diabetes), obstetrical (eg, intrauterine growth restriction), and fetal risk factors (eg, meconium) which we have previously delineated.[16-20] All parameters, except uterine contractions, used standard ACOG definitions. We have defined increased uterine activity as $\geq$5 contractions per 10 minutes—similar to Association of Women's Health, Obstetric, and Neonatal Nurses, instead of $\geq$6/10 minutes averaged over 30 minutes as per ACOG. Each parameter scores 1 point if normal, and 0 if not, collectively forming an 8-point scale. For example, all normal, 8/8 = 100%, and 2/8 = 25%. Scores are then further categorized into green zone ($\geq$51%), yellow (50%-26%), and red ($\leq$25%). We further subdivided the "RED zone" cases into ruby (25%-12.5%, and crimson (0%). We then compared both scores to our outcome variables from umbilical cord blood gases (BE, pH, and $pO_2$).[16-20] We used SPSS version 25 to compute the cross tabs, perform the means comparisons, and create receiver operating curve (ROC) curve analyses. "Medical" was used to check the screening-characteristic computations.

All umbilical artery samples were assessed for internal consistency in relationship to standard limits (including $pO_2$-$pCO_2$—the measured values) and as well as to their consistency with simultaneously obtained values from the umbilical vein.[21] Cases with samples with inconsistent values (other than switching of "artery" and "vein") were excluded.

We have accepted in accordance with general consensus that a cutoff point of $-12$ BE has clinical meaning—a value that represents the bottom 15% of our sample. Our samples did not have a very good representation of very low pH value scores (this is also true in the general population), we used a cutoff of the bottom 20% in our sample. We used the same cutoff for $pO_2$. However, results for $pO_2$ have a fundamental confounding; since in the course of obstetrical/anesthetic management, some parturients receive $O_2$ therapy and some do not. Irrespective, we could not clearly separate out the 2 categories. Hence, the $O_2$ scores are in general unrelated to outcome measures.

One of us (M.I.E.) has patents on the FRI approach; others are pending. Demographic, EFM, and clinical maternal, obstetrical, fetal, and neonatal variables were deidentified and as

**Table 1.** Categorization of Cases by Both Fri and Category Systems (Means and [SD]).[a]

| Parameters | Green (>50%) | Yellow (26%-50%) | Ruby (12.5-≤25%) | Crimson (0%) | *P* | CAT I | CAT 2 | *P* |
|---|---|---|---|---|---|---|---|---|
| N | 11 | 109[b] | 100[b] | 25 | | 15 | 231[b] | |
| BE | −6.78 (1.27) | −7.18 (2.55) | −9.48 (2.97) | −10.89 (2.63) | <.000 | −6.28 (1.14) | −8.62 (3.04) | 0 |
| Ph | 7.31 (.05) | 7.29 (.06) | 7.24 (.07) | 7.22 (.08) | <.000 | 7.3 (.05) | 7.25 (.07) | .02 |
| $pO_2$ | 18.61 (3.64) | 18.36 (4.81) | 17.92 (4.65) | 18.2 (3.50) | NS | 17.89 (3.29) | 18.24 (4.68) | NS |

Abbreviations: BE, base excess; CAT, Category; FRI, Fetal Reserve Index; NS, not significant.
[a]FRI provides higher level of discrimination for level of risk than does the CAT system.
[b]Ns vary slightly due to missing data.

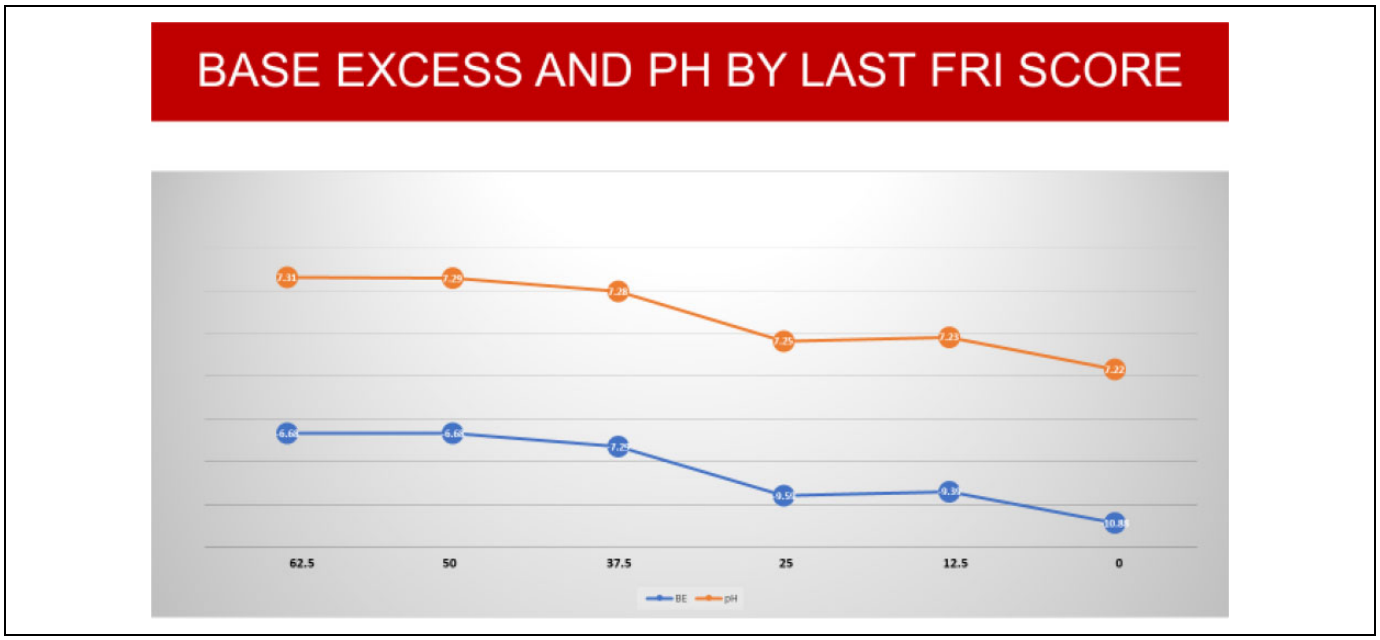


**Figure 1.** Decreasing BE and pH as correlates of the FRI score. Base excess has tighter correlation than pH, but both are significantly better ($P < .005$) than that demonstrated by CAT I and CAT II. BE indicates base excess; CAT, category; FRI; Fetal Reserve Index.

such qualified for exemption by the institutional review board of Biomedical Research Association of NY (#16-12-180-429).

## Results

The last FRI score before delivery correlates significantly with BE and pH (both $P < .01$) (Table 1, Figure 1) but not with $pO_2$, which, as expected, showed no correlation. Using ≤−12 BE as threshold (Table 2), CAT II has a 100% sensitivity as it detected at birth all 31 such babies. However, there was an enormous false-positive rate (FPR; 87%; CAT II n = 232, TP = 31, FP = 201). Such a large proportion of false-positives yields a positive likelihood ratio (sensitivity/1-specificity) of about 1. The FRI, using a simple dichotomy (yellow-green vs ruby-crimson), had a sensitivity of 90%, with a much lower FPR (Table 3). This is a more clinically reasonable construct, reflected by a positive likelihood ratio of almost 2:0. In our sample, 232 cases were identified as being high risk in terms of BE using CATII and therefore predicted to be at risk, but only 31 of these cases were actually, clinically at risk. The relative superiority of FRI lies in its capacity to make finer distinctions among those cases while CAT system lumps them into CAT II. These 235 CAT II cases were actually arrayed along the entire FRI continuum. Of those, 107 (46%) were classified by FRI as being at minimal risk (green-yellow). Three of these actually were ≤−12 BE but that still leaves FRI with a 90% sensitivity and a huge reduction in false positives.

To focus more graphically on the trade-off between true and false positives, we performed a ROC curve analysis of the relative effectiveness of these 2 screening mechanisms (Figure 2), which highlights the comparatively poor performance the CAT system. The area under the curve (AUC) for CAT is quite modest, appearing in the graph as almost an alternative reference line (AUC = .535 with a standard error of .053) versus FRI (AUC = .759, with a standard error of .041). The 2 AUCs are significantly different from one another ($P < .005$; Figure 2).

**Table 2.** Cord Blood Base Excess Divided at ≤ 12 MIU/mL by CAT.[a]

| Category | At-Risk BE (≤−12) | Safe BE (≥−12) | Total |
|---|---|---|---|
| CAT 2 | 31 | 201 | 232 |
| CAT 1 | 0 | 15 | 15 |
| Total | 31 | 216 | 247 |

Abbreviations: BE, base excess; CAT, Category.
[a]Sensitivity = 100%; specificity = 7%; PPV = 13%; NPV = 100%; positive likelihood ratio = 1; negative likelihood ratio = 0.

**Table 3.** Cord Blood Base Excess Divided at ≤12 MIU/mL by FRI.[a]

| FRI Groupings | At-Risk BE (≤−12) | Safe Threshold BE (>−12) | Total |
|---|---|---|---|
| Ruby-crimson | 28 | 98 | 126 |
| Green-yellow | 3 | 117 | 120 |
| Total | 31 | 215 | 246 |

Abbreviations: BE, base excess; FRI, Fetal Reserve Index.
[a]Sensitivity = 90%; specificity = 54%; PPV = 22%; NPV = 98%; positive likelihood ratio = 1.98; negative likelihood ratio = .18.

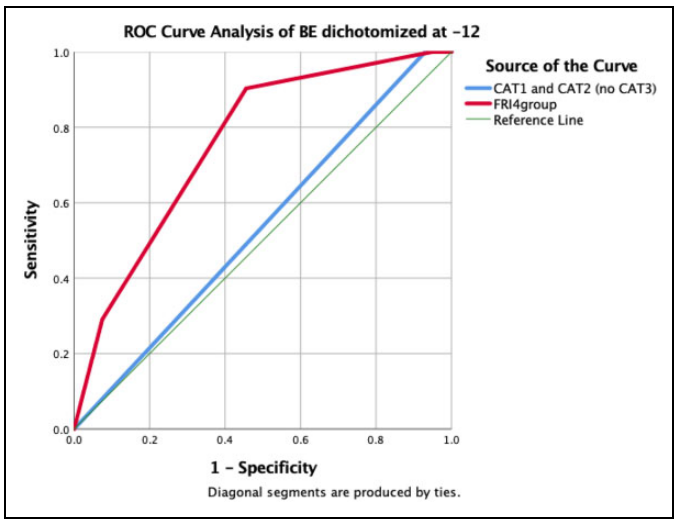


**Figure 2.** Receiver operating curve (ROC) demonstrates that the FRI score has substantially larger area under curve than CAT system and has better ability to detect low base excess (BE) levels associated with increased risks for neurological damage at multiple levels of false-positive rates (=1-specificity). CAT indicates category; FRI; Fetal Reserve Index.

The pH results are similar, but not as powerful as BE, reflecting the more important metabolic component of acidotic and clinical risk. The lowest pH observed was 7.03, so we used the bottom 20% as a cutoff point.

Table 4 extends this analysis to pH and $pO_2$. In all cases, when using CAT, the sensitivity is 100%, but the false-positive rate is almost that high—even using $pO_2$ as an outcome variable, for whom the results should be about the same as a coin flip. Fetal Reserve Index performs very well for BE, somewhat less well for pH in our sample (though still identifying about twice as many true positives as false positives), and is of little use for $pO_2$—which is exactly what should be expected if the data for $pO_2$ are confounded. It is only because of the very high sensitivity at first glance that CAT may look reasonable, but as one digs a bit deeper to examine the false-positive rate, the flaws in CAT become more evident.

**Table 4.** Screening-Test Characteristics for FRI and CAT.

| Method Statistics | BE | pH | p<SC>O</SC>$_2$ |
|---|---|---|---|
| FRI | | | |
| Sensitivity | 90 | 76 | 56 |
| Specificity | 54 | 55 | 50 |
| PPV | 2.0 | 1.9 | 1.1 |
| NPV | 2 | .45 | .87 |
| CAT | | | |
| Sensitivity | 100 | 100 | 100 |
| Specificity | 7 | 3 | 4 |
| PPV | 1.1 | 1.0 | 1.0 |
| NPV | 0 | 0 | 0 |

Abbreviation: BE, base excess; CAT, Category; FRI, Fetal Reserve Index; NPV, Negative predictive value; PPV, Postive predictive value.

## Discussion

Compared to ACOG categories, the FRI more accurately predicts cord blood BE and pH and is more sensitive to both early and late decrements in fetal reserve, that is, how much more can the fetus tolerate before decompensation. The essentially linear relationship of FRI score with worsening BE and pH suggests a physiologic underpinning of the FRI's and a correlation with both normal and adverse outcomes not present in the CAT formulation. Further investigation is needed to validate this quantitative, physiologically pertinent approach to fetal surveillance with an eye toward improving outcome (reducing injury) and lowering the CDR—a necessary ingredient of assessing FHR benefits.

To evaluate the efficacy of any screening approach for any disorder, a number of generic and specific issues need consideration. Firstly, one must understand the performance metrics of a new technique before it becomes routine. Neither EFM, as originally promulgated in the early 1970s, nor the CAT system in 2008 received underpinning from large scale, systematic evaluations before broad implementation.[11,14] In practice, there is often poor understanding for both patients and obstetrical care providers between the sensitivity/specificity and positive-/negative-predictive values for the CAT system making its interpretation even more tenuous.[15] Consider, for example, a stat delivery performed for a concerning tracing that produces a baby with Apgar's 9/9, pH of 7.1, and BE of −9 would be considered a clinical success. However, it is also a false positive—a screening failure. Conversely, if the same tracing produced a baby with Apgar's 2/3, pH of 6.9, and BE of −14, it would be a screening success (a true positive) but a clinical failure.

**Table 5.** Features of Category System.

| Baseline Features | Decelerations—Absent | Decelerations—Present |
|---|---|---|
| Normal | Category I | Category II |
| Abnormal | Category II | Category III |

Another issue plaguing the assessment of the value of EFM is the notion of preventable intrapartum injury. In 2000, ACOG commissioned a committee to evaluate the efficacy of EFM. One of us (M.I.E.) was part of the committee and an author of the report.[8] The report derived a set of requirements from demographically derived retrospective analysis for assigning injury that did not take advantage of nuanced interpretation of EFM or neuroradiological assessments. The report concluded that most cases of NE and CP were not related to the events of labor even when there was no other reasonable or plausible explanation. The pH < 7.00 threshold as a "critical marker" was not statistically vetted as an exclusion criteria for intrapartum hypoxic ischemic injury. Furthermore, a value < 7.00 is not pathognomonic for such injury.[2,6,7,18] In 2008, ACOG introduced the "Category System" in which CAT I represents a completely reassuring tracing, and CAT III suggests imminent danger and the need for immediate delivery. The CAT II shows "elements of concern" is "intermediate" (meaning nondiagnostic), but there is no specific, agreed understanding or course of action mandated other than "continued observation." Unfortunately, up to 80% of patients have CAT II tracings which renders it a logistical (and medicolegal) nightmare from a labor protocol perspective.

From a statistical perspective, this approach also violates fundamental mandates of a screening program. Its categorizations do not provide highly accurate prediction of adverse outcomes for a low percentage of false positives. Furthermore, many newborns with umbilical acidemia and a majority of babies who develop CP with intrapartum injury never reach CAT III or demonstrate severe umbilical acidemia[2,22-24] There is no clear demarcation between "affected" and "unaffected" categories of risk prediction—let alone actual diagnosis which again contradicts fundamental principles of a screening test. By ACOG definition, every CP baby that doesn't reach CAT III is a screening failure (false negative).

There are also serious underlying deficiencies in the conceptual makeup of the CAT system. Consider a simple 2 × 2 table in which the columns denote the presence or absence of decelerations (irrespective of type), while the rows denote the presence of normal or abnormal baseline features (rate and variability; Table 5). In its simplest terms, the analysis of decelerations rests with an assessment of their impact on baseline rate and variability with the understanding that in the normal fetus any deterioration in well-being must be accompanied by an alteration in the baseline features. Thus, to require, as the CAT system does, the complete absence of variability before the pattern can be called CAT III ignores the general ontogeny of these changes (deceleration with rising baseline and decreasing, but not absent, variability). Without recognizing these emerging changes in patients, there are no options for earlier intervention. On the other hand, abnormal baseline features without decelerations cannot represent an acute hypoxic ischemic event to the fetus. In Table 5, CAT II includes fetuses with either normal baseline features and decelerations (top right) or abnormal baseline features without decelerations (bottom left). The former represents fetuses under some threat, but who are adequately dealing with that threat. The latter represents fetuses with nonasphyxial problems (drugs, prematurity, injury, genetics, or anomaly). In the normal course of events, the FHR pattern cannot go back-and-forth within this category (bottom left—top right) without first returning to either CAT I or advancing to CAT III.

Specific limitations of the CAT system also include (1) a lack of physiologic underpinning by demanding a specific, but arbitrary, level of pH (<7.0) and severe neonatal depression immediately after birth; (2) a monolithic dedication to the notion that all injury is mediated by asphyxia; and (3) that there is an unvarying path to that injury. The CAT approach defies physiologic principles. Considerable evidence, including ours with the FRI, suggests no such orthodoxy is required.[16-20] By waiting for CAT III, intervention is often only as a rescue perhaps after injury has occurred.[25] The CAT system also fails to account for fetal behavior and makes no attempt to make a neurologic diagnosis or entertain the notion of fetal injury.[25] This leads to uncertainty about the timing, mechanism, and as a result, the preventability of intrapartum injury.

Data from this study suggest that the FRI system correlates more strongly with cord blood pH and BE and has much better performance metrics than does the CAT system. We have previously shown that by focusing on limited interventions earlier in the course of fetal deterioration (especially those involved with pushing in the second stage of labor), we can diminish the need to rescue the fetus for heart rate patterns.[16-20]

The $pO_2$ is not a good measure of outcome because it has no memory. Imagine being suffocated. If the suffocation is relieved for 1 minute, the $pO_2$ rises to normal levels, but the BE keeps falling. It has a memory, and it cannot be recalibrated as quickly as $pO_2$. In this scenario, there is severe acidosis, but the $O_2$ level is normal.

Strengths of this study include the first correlations of the FRI approach with acid/base balance in arterial cord blood and that such correlations are more significant than seen with the CAT system. These data lay the foundation for a reevaluation of factors involved influencing various components of labor and how they affect risks and levels of metabolic acidosis. Weaknesses include enough data to establish significant correlations but not enough to perform subanalyses of individual parameters. Future studies must also capture the timing and level of interventions so as to permit the disentangling of emerging fetal problems from clinical interventions.

We now have several publications demonstrating physiological, statistical, and clinical alignment of the FRI with measurable improvements as compared to the category system.[16-20] We respectfully submit that the category system is severely methodologically flawed and question its continued use as the

"gold standard" for interpretation of EFM. However, as with all new approaches, we recognize that there will be considerable inertia to abandon any current methodology in favor of any new construct.[26] We suggest with further data and automation of the FRI (underway) that the FRI should produce more rigorous evaluation of "prospective" patient risk and therefore could become a better clinical tool for management.

## Authors' Note

This paper has been presented to the 2019 annual meeting of the Society for Reproductive Investigation.

## Declaration of Conflicting Interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: One of us (M.I.E.) has patents on the FRI approach and others are pending.

## Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

## References

1. Hon EH. Fetal monitoring for the practicing physician. *Calif Med*. 1970;113(6):46-47.
2. Cahill A, Macones G, McKinstry RC, Smyser CD, Mathur A. EFM and peripartum factors associated with brain injury in term infants. *AJOG*. 2015;212(1):S5.
3. Cahill AG, Roehl KA, Odibo AO, Macones GA. Association and prediction of neonatal acidemia. *Am J Obstet Gynecol*. 2012; 207(3):206.e1-206 e8.
4. Frey HA, Liu X, Lynch CD, et al. An evaluation of fetal heart rate characteristics associated with neonatal encephalopathy: a case-control study. *BJOG*. 2018;125(11):1480-1487.
5. Galen R, Gambino R. *Beyond Normality: The Predictive Value and Efficiency of Medical Diagnoses*. New Jersey, USA: Wiley biomedical publication. 1975:237.
6. Platt JR. Strong Inference. *Science*. 1964;146(3642):347-353.
7. ACOG. *Neonatal encephalopathy and neurologic outcome*. 2nd ed. Washington, DC: ACOG, 2014. doi:10.1097/01.AOG.0000445580.65983
8. ACOG. *Neonatal Encephalopathy and Cerebral Palsy: Defining the Pathogenesis and Pathophysiology*. Washington, DC: ACOG; 2003:94.
9. Grimes DA, Peipert JF. Electronic fetal monitoring as a public health screening program: the arithmetic of failure. *Obstet Gynecol*. 2010;116(6):1397-1400.
10. Clark SL, Hamilton E, Garite TJ, Timmins A, Warrick PA, Smith S. The limits of electronic fetal heart rate monitoring in the prevention of neonatal metabolic acidemia. *Am J Obstet Gynecol*. 2017;216(2):163.e1-163.e6.
11. Macones GA, Hankins GD, Spong CY, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. *J Obstet Gynecol Neonatal Nurs*. 2008;37(5):510-515.
12. Warrick PA, Hamilton EF, Precup D, Kearney RE. Classification of normal and hypoxic fetuses from systems modeling of intrapartum cardiotocography. *IEEE Trans Biomed Eng*. 2010;57(4): 771-779.
13. Schifrin BS. Graded classification of fetal heart rate tracings: association with neonatal metabolic acidosis and neurologic morbidity. *Am J Obstet Gynecol*. 2010;202(5):e11; author reply e-2.
14. ACOG Practice Bulletin No. 106: intrapartum fetal heart rate monitoring: nomenclature, interpretation, and general management principles. *Obstet Gynecol*. 2009;114(1):192-202.
15. Sartwelle TP. Electronic fetal monitoring: a bridge too far. *J Leg Med*. 2012;33(3):313-379.
16. Britt DW, Evans MI, Schifrin BS, Eden RD. Refining the prediction and prevention of emergency operative deliveries with the Fetal Reserve Index. *Fetal Diagn Ther*. 2019(in press). doi:10.1159/000494617
17. Eden RD, Evans MI, Britt DW, Evans SM, Schifrin BS. Safely lowering the emergency cesarean and operative vaginal delivery rates using the Fetal Reserve Index. *J Matern Fetal Neonatal Med*. 2019(in press). doi:10.1159/000494617
18. Evans MI, Eden RD, Britt DW, Evans SM, Schifrin BS. Re-engineering the interpretation of electronic fetal monitoring to identify reversible risk for cerebral palsy: a case control series. *J Matern Fetal Neonatal Med*. 2019(in press). doi:10.1159/000494617
19. Eden RD, Evans MI, Evans SM, Schifrin BS. Reengineering electronic fetal monitoring interpretation: using the Fetal Reserve Index to anticipate the need for emergent operative delivery. *Reprod Sci*. 2018;25(4):487-497.
20. Eden RD, Evans MI, Evans SM, Schifrin BS. The "Fetal Reserve Index": re-engineering the interpretation and responses to fetal heart rate patterns. *Fetal Diagn Ther*. 2018;43(2):90-104.
21. Pomerance J. *Interpreting Umbilical Cord Blood Gases*. Pasedena, CA: BNMG; 2004:132.
22. Cahill A, Macones G, McKinstry R, Smyser C, Mathur A. 7: EFM and peripartum factors associated with brain injury in term infants. *Am J Obstet Gynecol*. 2015;212(1):S5.
23. Cahill AG, Mathur AM, Smyser CD, et al. Neurologic injury in acidemic term infants. *Am J Perinatol*. 2017;34(7):668-675.
24. Frey HA, Tuuli MG, Shanks AL, Macones GA, Cahill AG. Interpreting category II fetal heart rate tracings: does meconium matter? *Am J Obstet Gynecol*. 2014;211(6):644 e1-644 e8.
25. Schifrin BS, Ater S. Fetal hypoxic and ischemic injuries. *Curr Opin Obstet Gynecol*. 2006;18(2):112-122.
26. Evans M, Hanft R. *The Introduction of New Technologies. ACOG Clinical Seminars*. Washington, DC: ACOG; 1997:3.