181 **FETAL HEART RATE PARAMETERS PREDICTIVE OF NEONATAL OUTCOME IN THE PRESENCE OF FETAL BRADYCARDIA** KEITH WILLIAMS P[1], FRANCE GALERNEAU[1], [1]Yale University, OBSTETRICS & GYNECOLOGY, New Haven, CT

OBJECTIVE: To correlate the presence of variability and the duration of bradycardia in intrapartum fetal heart rate tracings with the development of neonatal acidemia.

STUDY DESIGN: We identified 186 patients who had continuous electronic fetal monitoring for at least 2 hours during labor, with an identified bradycardia with umbilical artery cord blood analysis done and delivery within 30 minutes of that bradycardia. One investigator blinded to the cord gas outcome reviewed the last 2 hours of the tracing using the NICH guidelines for fetal heart rate monitoring. In particular, variability was defined as abnormal when the amplitude was ≤5 beats/min. We assessed the presence or absence of variability prior to the bradycardia and recovery or nonrecovery of the bradycardia and placed the patients into 4 groups: Group 1 Normal variability and recovery, Group 2 Normal variability and nonrecovery, Group 3 Decreased variability and recovery and Group 4 Decreased variability and nonrecovery. The relationship between the various groups was assessed using ANOVA and the chi-squared test.

RESULTS: The presence of decreased variability and nonrecovery of the fetal heart rate after a bradycardia was associated with the lowest ph 6.83 ± .16 and a 78% incidence of significant acidosis (Table). Decreased variability prior to fetal heart rate bradycardia was the fetal heart rate parameter most significantly correlated with low pH.

CONCLUSION: The most significant factors predicting the development of pathologic neonatal acidemia and indicating the need for urgent delivery in the presence of a bradycardia are decreased variability prior to the bradycardia and nonrecovery.

Table

	NOR VAR & RECOV	NOR VAR & NO RECOV	DEC VAR & RECOV	DEC VAR & NO RECOV	*P*
Number	128	40	9	9	
pH	7.17 ± .09	7.13 ± .15	7.11 ± 10.5	6.83 ± .16	<.000
Base deficit	-6.54 ± 3.9	-7.15 ± 5.1	-10.32 ± 3.68	-20.17 ± 6.0	<.000
pH<7.0	2%	18%	50%	77.8%	<.000
pH<7.1	22%	33%	65%	88.9%	<.000
Base Def.<16	1%	8%	11.1%	77.8%	<.000
Base Def.<12	4.7%	12.8%	22.2%	88.9%	<.000

182 **C/SECTION AS AN APPROPRIATE INTERVENTION FOR ABNORMAL INTRAPARTUM FETAL HEART RATE TRACINGS** KEITH WILLIAMS[1], FRANCE GALERNEAU[1], [1]Yale University, Obstetrics & Gynecology, New Haven, CT

OBJECTIVE: Pathologic acidemia has been defined as an umbilical artery ph < 7. We evaluated the extent to which C/Section for fetal distress based on an abnormal intrapartum fetal heart rate pattern indicates appropriate intervention prior to the development of pathologic acidemia.

STUDY DESIGN: A 3 year review (1997-1999) of all cases of laboring women undergoing C/Section for fetal distress and for failure to progress in which umbilical artery cord gases at birth was done. Fetal distress in this institution is defined as an abnormal fetal heart rate tracing with the presence of decreased variability, variable decelerations, or late decelerations. We compared the ph, base deficit and 1 and 5 minute Apgars at birth in patients with a diagnosis of C/Section for fetal distress with patients who underwent a C/Section for failure to progress. We classified patients into different groups based on ph and base deficit (i) prepathologic acidemia defined as a ph < 7.2 and ≥ 7.0 or a base excess > 8 or ≤16, (ii) pathologic acidemia with a ph < 7 or a base deficit < 16. Comparison was done between the fetal distress and the failure to progress groups using student test and the chi-squared test.

RESULTS: The ph was lower and the base deficit was higher in the C/Section for fetal distress group compared with the failure to progress group (Table). The incidence of prepathologic acidemia, a ph < 7.2 but > 7.0 in the C/Section for fetal distress group was 28% compared to 5.8% in the failure to progress group. (Table) Of all patients who underwent C/Section for fetal distress, in 69.5% no acidemia existed.

CONCLUSION: Intrapartum fetal heart rate parameters clinically used to determine the need for C/Section for fetal distress do not appropriately predict a prepathologic acidemic state in which the intervention of C/Section would be expected to reduce the fetal long term morbidity and mortality caused by the development of pathologic acidemia.

Table

	C/S-DISTRESS	C/S-FTPROGRESS	*P*
Number	220	156	
Ph	7.23 ± 0.1	7.27 ± .04	.00
Base deficit	-3.90 ± 4.36	-3.02 ± 2.17	.02
Apgar 1 min	6.76 ± 2.37	7.86 ± 1.33	.00
Apgar 5 min	8.44 ± 1.56	9.08 ± .47	.00
ph<7.0	2.3%	0%	.14
ph<7.2≥7.0	28.2%	5.8%	.00
Base deficit<16	1.4%	0%	.33
Base deficit<8 ≥16	11.6%	1.9%	.33

183 **THE SIGNIFIANCE OF THE DURATION OF INTRAPARTUM FETAL HEART RATE VARIABILITY IN PREDICTING THE DEVELOPMENT OF SIGNIFICANT NEONATAL ACIDOSIS** KEITH WILLIAMS[1], FRANCE GALERNEAU[1], [1]Yale University, Obstetrics and Gynecology, New Haven, CT

OBJECTIVE: To correlate the duration of abnormal variability in intrapartum electronic fetal heart rate patterns with the development of significant neonatal acidosis.

STUDY DESIGN: We identified 437 pregnant women at a gestational age of > 36 weeks who had electronic fetal monitoring prior to delivery and umbilical artery cord gas analysis done. One investigator, blinded to the neonatal outcomes, reviewed the last 2 hours of tracings using the NICHD guidelines for fetal heart rate monitoring. In particular variability was defined as abnormal when the amplitude was ≤ 5 beats/min. All patients with bradycardia were removed from further analysis. The duration of normal or abnormal variation was evaluated on an hourly basis over the last 2 hours and patients placed into 3 groups: 1. Normal variability for the entire 2 hours. 2. Decreased variability for 1 hour. 3. Decreased variability for the entire 2 hours. The relationship between the duration of abnormal variability with the outcome variables of umbilical artery ph and Base Deficit (BD) was assessed using ANOVA.

RESULTS: Increasing duration of decreased variability in the fetal heart rate pattern was directly correlated with the incidence of pathologic neonatal acidemia. A decreased variability for > 2 hours resulted in a 20 fold increase in the incidence of significant acidemia over normal variability (Table).

CONCLUSION: The presence of decreased variability of one hour's duration represents the upper limit for conservative management beyond which the likelihood of development of a significant neonatal acidosis is high.

Table

	NOR-VAR	DEC-VAR ≥1 HR	DEC-VAR ≥ 2 HRS	*P*
Number	391	23	23	
Ph	7.18 ± .09	7.15 ± .14	7.01 ± .19	.0001
Base deficit	-6.04 ± 3.73	-7.38 ± 5.3	-13.02 ± 7.86	.05
ph<7.0	2.8%	8.7%	47.8%	.000
ph<7.1	15.6%	34.8%	60.9%	.000
BD<16	1%	8.7%	39.1%	.000
BD<12	4.6%	17.4%	47.8%	.000

184 **LATE DECELERATIONS AND SEVERE VARIABLES ARE PREDICTIVE OF FETAL HYPOXIA** RICHARD LEE[1], MEAGAN MOORE[1], WENDY BREWSTER[2], POONEH HENDI[1], CAROL PATTILLO[1], A ZIOGAS[3], THOMAS GARITE[1]; [1]University of California, Irvine, Obstetrics & Gynecology, Orange, CA; [2]University of California, Irvine, Obstetrics & Gynecology, Irvine, CA; [3]University of California, Irvine, Biostatistics, Irvine, CA

OBJECTIVE: To determine the physiologic significance of non-reassuring fetal heart rate (FHR) patterns with fetal oxygenation as determined by fetal pulse oximetry (FPO).

STUDY DESIGN: An analysis of FHR tracings with fetal oxygen saturation (FSpO2) from the US RCT of FPO (AJOG 2000;183:1049-58) was performed. Each FHR tracing was divided into 2-hour epochs, in which the worst 40 minutes was evaluated. Late, moderate variable, and severe variable decelerations were correlated with FSpO2 <30% (measure of fetal hypoxia) and actual FSpO2. Variability and fetal tachycardia were also evaluated. Simple regression, multivariate analysis, and logistic regression were performed.

RESULTS: 493 epochs from 261 FHR tracings with >50% registration time of FPO recorded were reviewed. Frequency of late decelerations ($P < .01$) and frequency of severe variables ($P < .01$) were predictive of the number of episodes of FSpO2 <30% in a multivariate model which also controlled for time. Frequency of moderate variables, variability, and fetal tachycardia were not predictive in this model. In another multivariate model also controlled for time, frequency of late decelerations ($P < .05$), frequency of severe variables ($P < .05$), variability ($P < .05$) were significantly associated with a lower FSpO2 nadir. Frequency of moderate variables and tachycardia were not. Late decelerations occurring more frequently than every 10 minutes were more likely to be associated with episodes of FSpO2 <30% than late decelerations occurring less than every 10 minutes (RR 1.75, 95% CI 1.45-2.13).

CONCLUSION: Late decelerations and severe variables are predictive of fetal hypoxia. Fetal tachycardia and variability alone do not predict fetal hypoxia, but decreased variability is associated with a lower FSpO2 nadir. This is the first human study that confirms known physiologic data from animal studies regarding the relationship of fetal hypoxia and abnormal fetal heart rate patterns. It also supports the validity of FPO in measuring fetal hypoxia.