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A Neural Explanation of Fetal Heart Rate Patterns: A Test of the Polyvagal Theory

ABSTRACT: *The current study applies a neurophysiological model based on the Polyvagal Theory (Porges, 1995) to interpret fetal heart rate patterns. Beat-to-beat heart rate data from 7 fetuses monitored during the first and second stages of labor were analyzed. Transitory heart rate accelerations and reduced beat-to-beat variability reliably preceded heart rate decelerations. The data are interpreted within the context of the Polyvagal Theory, which provides a plausible explanation of the neurophysiological mechanisms that mediate fetal heart rate decelerations. Specifically, it is proposed that both the transitory heart rate accelerations and the depression of the respiratory rhythm in the beat-to-beat heart rate pattern reflect a withdrawal of the vagal tone determined by myelinated vagal pathways originating in the nucleus ambiguus. Functionally, withdrawal of vagal tone originating in the nucleus ambiguus results in the cardiac pacemaker becoming vulnerable to sympathetic influences and to the more-primitive unmyelinated vagal pathways originating in the dorsal motor nucleus of the vagus, which may contribute to clinically relevant bradycardia.* © 1999 John Wiley & Sons, Inc. Dev Psychobiol 35: 108–118, 1999*

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Based upon clinical observations and limited experimental manipulations, specific features of the fetal heart rate pattern have been accepted by the clinical community as having diagnostic utility. Despite the widespread acceptance of fetal heart rate monitoring, especially in high-risk pregnancies, there remains a limited understanding of the neurophysiological mechanisms mediating the specific features of the heart rate patterns used in the clinical assessment of the fetus.

The primary goal of fetal heart rate monitoring is the detection of fetal hypoxia at its earliest stage (Kruse, 1982). Specific characteristics of the heart rate pattern (i.e., early decelerations, variable decelerations, late decelerations, beat-to-beat variability, tachycardia, bradycardia, and sinusoidal patterns) have been identified and proposed as reflections of the neurophysiological regulation of the heart and thus, as sensitive indices of fetal condition. However, because the features of the fetal heart rate pattern are often unreliable diagnostic indi-

cators, controversy still exists over the use and interpretation of fetal heart rate monitoring.

Difficulties in the diagnostic application of fetal heart rate monitoring may be due, in part, to uncertain and contradictory neurophysiological explanations (Goodlin & Haesslein, 1977; Rosen & Dickinson, 1993). For example, beat-to-beat variability is assumed to be mediated by the vagus with high levels of variability indicating fetal well-being (Kruse, 1982; Paul, Suidan, Yeh, Schifrin, & Hon, 1975). However, heart rate decelerations, indicators of fetal compromise, are assumed also to be mediated by the vagus.

The current article attempts to reconcile this paradox by interpreting fetal heart rate patterns within the context of the Polyvagal Theory (Porges, 1995, 1997). The Polyvagal Theory focuses on the phylogenetic changes in the functional neuroanatomy of the vagus and how these modifications are represented in the neural regulation of heart rate patterns.

THE NEUROPHYSIOLOGY OF MAMMALIAN HEART RATE PATTERNS

The vagus, the 10th cranial nerve, conveys neural influences to the cardiac pacemaker (i.e., sinoatrial node). The release of acetylcholine on the sinoatrial node of the heart, triggered by vagal activity, not only inhibits cardiac pacemaker activity but also attenuates sympathetic influences on the heart (Levy, 1984; Vanhoutte & Levy, 1979). Therefore, heart rate slows with increases in vagal tone and heart rate speeds with decreases in vagal tone. Vagal output to the heart originates in two brainstem nuclei, the dorsal motor nucleus of the vagus and the nucleus ambiguus. Vagal pathways originating in both nuclei are capable of producing heart rate decelerations (Jones, Wang, & Jordan, 1995). The vagal fibers originating in the dorsal motor nucleus of the vagus are unmyelinated. During most states, the unmyelinated vagal fibers have little influence on heart rate levels (Ford, Bennett, Kidd, & McWilliam, 1990; Jones et al., 1995) and exhibit little or no spontaneous activity or variability (Ford et al., 1990). In contrast, the vagal fibers originating in the nucleus ambiguus are myelinated, influence both heart rate level and beat-to-beat variability (McAllen & Spyer, 1978), and maintain tonic influence over the heart.

RESPIRATORY SINUS ARRHYTHMIA IN THE FETUS

The vagal efferent fibers originating in the nucleus ambiguus have a respiratory rhythm (Richter & Spyer, 1990). This central respiratory rhythm is an emergent property of the brainstem with significant contributions from the interneuronal communication between the nucleus ambiguus and the nucleus of the solitary tract. The functional impact of the nucleus ambiguus vagal pathways on the sinoatrial node is to produce a respiratory rhythm in the heart rate pattern. The amplitude of these rhythmic increases and decreases in heart rate (i.e., respiratory sinus arrhythmia, RSA) is, thus, a valid indicator of vagal outflow from the nucleus ambiguus. Even in the absence of actual breathing, there are reports of beat-to-beat changes in fetal heart rate that track a rhythm similar to the spontaneous breathing of a neonate (Donchin, Caton, & Porges, 1984; Groome, Mooney, Bentz, & Wilson, 1994).

BRADYCARDIA

Across vertebrate species, the control of cardiac function exhibits a phylogenetic trend towards greater neural involvement. The Polyvagal Theory (Porges, 1995) focuses on the specific phylogenetic changes in the vagal efferents and the medullary nuclei from which these pathways originate. Vagal efferent pathways originate in two medullary nuclei, the nucleus ambiguus and the dorsal motor nucleus of the vagus. According to the theory, because these two vagal systems have different phylogenetic and embryologic origins, they have different and potentially contradictory response strategies when challenged. The unmyelinated vagal fibers that originate in the dorsal motor nucleus of the vagus evolved before the myelinated vagal fibers of the nucleus ambiguus. The dorsal motor vagal system evolved as a passive avoidance system and responds to environmental challenges by sending inhibitory impulses to the heart, which reduces cardiac output in an effort to conserve metabolic resources. In reptiles, the dorsal motor vagal system is the predominant vagal system in the regulation of cardiopulmonary processes. This system promotes apnea and bradycardia by constricting the bronchi and by inhibiting the cardiac pacemaker. These physiological responses are neurophysiologically consistent with the commonly observed reptilian avoidance strategies of submerging or immobilizing. In mammals, the nucleus ambiguus vagal system is the predominant vagal system in the regulation of cardiopulmonary processes. This phylogenetically more-recent system has myelinated vagal fibers that enable a rapid and dynamic regulation of cardiac output via transitory increases and decreases in tonic vagal output to the heart. Although both nuclei are capable of slowing heart rate in mam-

mals, stimulation of vagal fibers originating in the dorsal motor nucleus of the vagus has been proven ineffective in producing large bradycardia (Jones et al., 1995).

Research examining the relative influence of dorsal motor nucleus vagal fibers in promoting the clinically relevant bradycardia observed in fetal distress is not conclusive at this time (Hopkins, Bieger, de Vente, & Steinbusch, 1996). However, it is possible that hypoxia itself potentiates the cardioinhibitory actions of vagal fibers on the sinoatrial node. Potter and Mc-Closkey (1986) report a feedback system between duration of hypoxia and vagal efferent discharge, which results in a potentiation of the vagal output on the heart. Although there is a massive decline in vagal firing during hypoxia, this system functions to maintain bradycardia by potentiating the influence of vagal firing on the sinoatrial node. Under these conditions, the magnitude of the bradycardia is determined, in part, by peripheral mechanisms. Therefore, it may be possible that bradycardia could be mediated by a relatively small number of vagal fibers originating in the dorsal motor nucleus of the vagus potentiated in the periphery by hypoxia.

TACHYCARDIA

In humans and other mammals, under most conditions, the primary vagal control of the heart is mediated via nucleus ambiguus pathways. During these states, the nucleus ambiguus vagal tone potentially may protect the mammalian heart from both the inhibitory surges from the dorsal motor nucleus of the vagus and the excitatory surges from the sympathetic nervous system. However, when metabolic demands are high, the adaptive response of the mammal is to increase heart rate by reducing the nucleus ambiguus vagal tone to the pacemaker. Because, at the level of the sinoatrial node, the cholinergic vagal system is inhibitory on the pacemaker and the sympathetic adrenergic system (e.g., Levy, 1984; Vanhoutte & Levy, 1979), the removal of cholinergic influences (i.e., the withdrawal of nucleus ambiguus vagal tone) results in a potentiation of sympathetic activity. Thus, the withdrawal of nucleus ambiguus vagal influence results in an instantaneous tachycardia.

FETAL DISTRESS

According to the Polyvagal Theory, prolonged withdrawal of nucleus ambiguus vagal tone creates a physiological vulnerability to other vagal mechanisms that may produce clinically significant bradycardia. Two neurophysiological factors contribute to this possibility. First, nucleus ambiguus vagal tone may protect the pacemaker from the influence of fibers originating in the dorsal motor nucleus. Thus, removal of nucleus ambiguus vagal tone would create a vulnerability of the pacemaker to the cholinergic influences of the vagal fibers originating in the dorsal motor nucleus. Second, because hypoxia potentiates cholinergic action on the pacemaker, stimulation of only a few vagal fibers during hypoxic states might be sufficient to produce massive bradycardia. Potter and McCloskey (1986) demonstrated that during progressive asphyxic hypoxia in dogs, not only was cardiac vagal activity elicited, but also the sensitivity of the sinoatrial node to vagal efferent influences was potentiated. Thus, during hypoxic conditions, the withdrawal of nucleus ambiguus influence on the heart (i.e., indexed by low-amplitude RSA) and the increased sensitivity of the sinoatrial node to vagal efferent influences may create a situation in which vagal output from the dorsal motor nucleus of the vagus might be sufficient to produce clinically relevant bradycardia.

APPLICATION OF THE POLYVAGAL THEORY

The current article applies the Polyvagal Theory as a model to interpret fetal heart rate patterns. Based on the theory, several predictions are made. First, the initial transitory response to withdrawal of nucleus ambiguus vagal tone would be a transitory heart rate acceleration. Second, withdrawal of nucleus ambiguus vagal tone would leave the pacemaker vulnerable to activity from vagal pathways originating in the dorsal motor nucleus of the vagus. If the above were observed, heart rate decelerations would be anticipated by a transitory heart rate acceleration in a background of low-amplitude RSA. Third, recovery from the heart rate decelerations would be characterized by a return to, and in many cases, an increase in RSA reflecting the dynamic adjustment of nucleus ambiguus vagal tone. Fourth, chronic fetal distress would be characterized by low beat-to-beat heart rate variability and the massive and frequent heart rate decelerations that characterize the clinical bradycardia indicative of fetal distress. Hypothetically, this response profile might be due to low nucleus ambiguus vagal tone and a pacemaker that is vulnerable to surges in the dorsal motor nucleus of the vagus.

The current study was conducted to test the Polyvagal Theory by statistically examining heart rate patterns in archived fetal heart rate data. Based on the

above four predictions, three hypotheses were tested: (a) Heart rate decelerations will be preceded by transitory accelerations reflecting the withdrawal of nucleus ambiguus vagal tone, (b) recovery from deceleration will be characterized by an increase in RSA, and (c) chronic low-amplitude RSA will be associated with more-frequent and severe heart rate decelerations.

MATERIAL AND METHODS

Heart period data (i.e., R–R intervals) were collected at the Poriya Government Hospital in Israel. To insure accurate detection of beat-to-beat measures of heart period, data were collected during the first and second stages of labor using scalp electrodes. Electrodes were attached to the fetal scalp through the opening of the cervix during the initial stages of labor. Data from 11 fetuses were obtained. The number of subjects was governed by the clinician's decision to monitor the fetus with scalp electrodes. Ten cases were normal in terms of pregnancy, labor, clinical fetal heart rate patterns during labor, APGAR scores, umbilical cord blood pH, and maternal oxygen saturation. In addition, 1 case of intrauterine fetal death was analyzed. The intrauterine fetal death case was diagnosed prior to delivery and had pronounced fetal hydrocephalus and an abnormal heart rate pattern.

The ECG signal was digitized on-line in the delivery room using a Vagal Tone Monitor (Delta-Biometrics, Bethesda, MD). The Vagal Tone Monitor digitizes the ECG analog output of the fetal heart rate monitor at 1 kHz and identifies the peak of the R-wave and the R–R intervals with 1-ms accuracy. This degree of accuracy, which is uncommon in standard hospital monitors, is necessary for the accurate quantification of the low-amplitude RSA observed in the fetus (Donchin et al., 1984; Groome et al., 1994).

Of the 10 normal cases, 6 were used in the final analysis. Two cases contained excessive ECG artifact and could not be analyzed, and of the 8 remaining cases, only 6 contained at least one heart rate deceleration. Decelerations were operationally defined as a transitory decrease in heart rate slower than 85 beats per min (i.e., a heart period longer than 700 ms). From these six recordings, 28 heart rate decelerations were identified.

A computer graphics display of the beat-to-beat heart periods enabled a visual identification of the beginning and end of each heart rate deceleration. The beginning of the deceleration was operationalized as the point at which the heart period recording began to increase without a subsequent return to predeceleration (i.e., baseline) levels until after the 700-ms criterion was reached. The end of the deceleration was op-

erationalized as the point in time when the heart period stopped decreasing after reaching baseline levels.

Heart period levels immediately before the decelerations were evaluated to determine if a significant heart rate acceleration (i.e., decrease in heart period) occurred. Significant accelerations were statistically defined according to the following steps. First, for each deceleration, the range of heart period values was calculated during the preceding 60 s. Second, the lower 5th percentile of the range was calculated. Third, 95% confidence intervals were constructed around the heart period value representing the lower 5th percentile. The confidence intervals were estimated using a bootstrapping technique similar to the method described by Lunneborg (1986). This bootstrapping technique produces confidence intervals that are not necessarily symmetrical about the 5th percentile. Fourth, the heart period value immediately prior to the onset of deceleration was contrasted with the lower limit of the confidence intervals. If this heart period was outside the confidence interval, it was defined as a significant acceleration (See Figure 1).

To evaluate the hypothesized recovery of RSA following decelerations, a within subjects analysis of RSA contrasted the amplitude of RSA measured during the 60 s prior to and following the decelerations. Due to the temporal proximity of several decelerations, 9 decelerations did not have the required 60 s of heart period data following the deceleration; thus, the analysis of RSA was conducted on 19 of the 28 decelerations.

RSA was quantified using the Porges (1985) method, the steps of which are illustrated in Figure 2. Approximately 200 s of fetal heart period time sampled every 200 ms during a stable state are plotted in Figure 2a. To identify and quantify RSA, the data in Figure 2a were first smoothed with a 3rd order 21-point moving polynomial to create a template time series (Figure 2b) and the template time series is then subtracted from the data in Figure 2a to generate a residual time series that contains only the heart period variability in a frequency band above .3 Hz (Figure 2c). Note the high-frequency oscillations with amplitudes between ± 5 ms. These oscillations represent fetal RSA. To quantify the amplitude of these oscillations, the template time series is bandpassed to output a time series consisting only of variances associated with the frequency band from .3 to 1.3 Hz, the spontaneous breathing frequencies of a human newborn. The variance of the bandpassed time series is used as a measure of the amplitude of RSA. To stabilize the statistical estimates of the amplitude of RSA, the natural logarithms of these variance measures were used in the subsequent analyses.

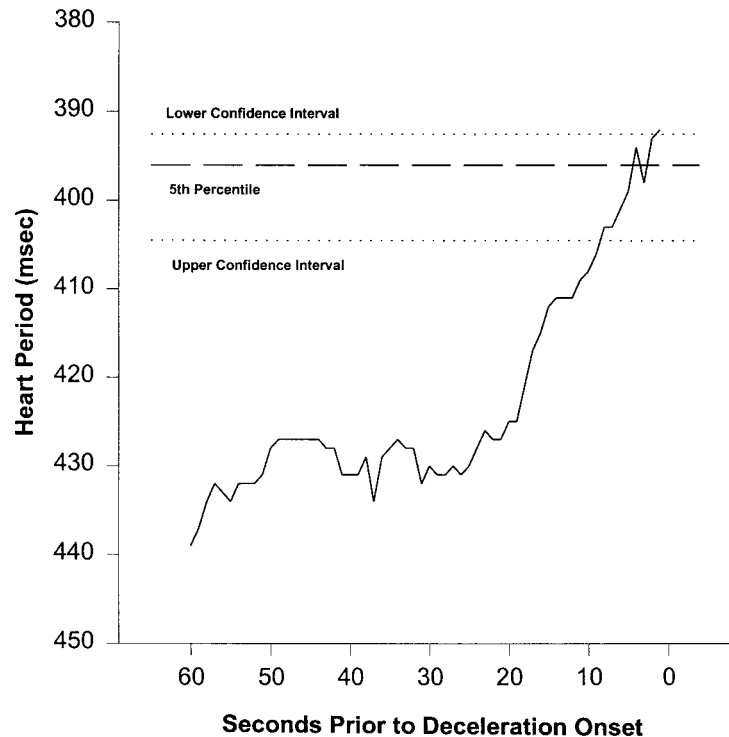In cases characterized by frequent decelerations,

FIGURE 1   Example of a significant acceleration prior to the onset of a deceleration.

such as during fetal distress, it may be impossible to accurately quantify the amplitude of RSA. When the fetal heart rate pattern is characterized by frequent decelerations, the slopes of the decelerations are similar to the slope of RSA, thus creating a situation in which it is statistically impossible to accurately quantify and interpret RSA. To deal with the limitations in method, the analyses required periods in which frequent decelerations did not occur.

To demonstrate the effectiveness of the methodology relative to other applications of spectral analyses (e.g., Davidson, Rankin, Martin, & Reid, 1992), a relatively stable segment of fetal heart period was analyzed. In Figure 3a, the time-sampled fetal heart period is plotted. Note in the figure that there are several observable oscillations. In Figure 4a, the power spectrum is illustrated for the data in Figure 3a. The spectrum reflects a dominant periodicity at approximately 0.04 Hz with no clearly identifiable periodicity at any higher frequency within the normal frequencies of spontaneous breathing. Because physiological periodicities are not perfect sine waves, all slow heart rate oscillations have higher-frequency components that appear in the Fourier transform at integer harmonics of the peak frequency. When the higher-frequency peak is of low amplitude relative to the amplitude of the very-slow oscillation, there is the possibility that the slow periodicity is contributing variance to the

spectral representation of the higher-frequency oscillation. Thus, a high-frequency peak in the spectrum might represent the sum of both the amplitude of the true physiological oscillation (e.g., RSA) and a harmonic component from a slow oscillation. The moving polynomial approach, by removing the variance associated with low-frequency activity, optimizes the quantification of the heart rate oscillation at the respiratory frequency. Two statistical factors contribute to this enhanced ability to detect the frequency and to quantify the amplitude of RSA. First, the variance associated with the harmonics of slow-frequency activity is removed, thus allowing the quantification of the variance at the respiratory rhythm to be statistically independent of the harmonic influences from high-amplitude, slow-frequency components. Second, if the low-frequency components are removed prior to spectral analysis, statistical windowing procedures in the frequency domain do not include the low-frequency activity, which if included would distort the spectral representation by increasing the amplitude and shifting the peak to slower frequencies.

Our approach starts by attempting to describe the amplitude of oscillations in a frequency band that would reflect the spontaneous breathing rhythm in a human newborn infant. In human newborns, this frequency would be in the range of 20 to 80 breaths per min or approximately 0.3 Hz to 1.3 Hz. We approach
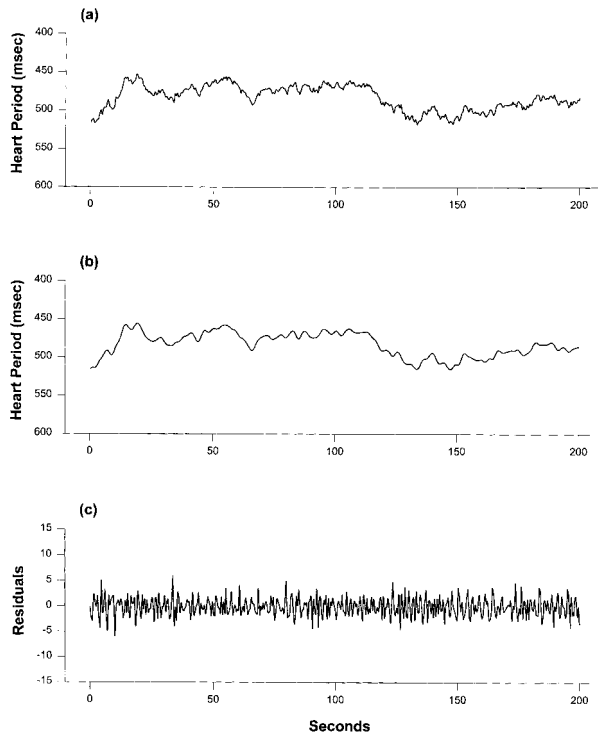
FIGURE 2 Sequential steps in calculating RSA: (a) time-sampled fetal heart period (i.e., 200-ms intervals); (b) data illustrated in Figure 2a smoothed with a 3rd order 21-point moving polynomial; (c) residual time series generated by subtracting data in Figure 2b from data in Figure 2a.

FIGURE 3 (a) Time-sampled fetal heart period data; (b) data illustrated in Figure 3a smoothed with moving polynomial procedure.

this problem by applying the moving polynomial procedure (Porges & Bohrer, 1990) to statistically remove virtually all of the variance below .3 Hz from the fetal heart period time series. In Figure 3b, the fetal heart rate data illustrated in Figure 3a are processed by the moving polynomial procedure described above. Figure 4b illustrates the power spectrum of the residual time series illustrated in Figure 3b. The power spectrum has a prominent peak at approximately .40 Hz, a frequency similar to the spontaneous breathing frequency of healthy newborn infants.

RESULTS

Transitory Acceleration Precedes Decelerations

Table 1 contains the number and percent of decelerations which have been preceded by significant accelerations for each of the 6 subjects. The data illustrate that for all subjects the probability of the heart period value immediately preceding the onset of the decel-
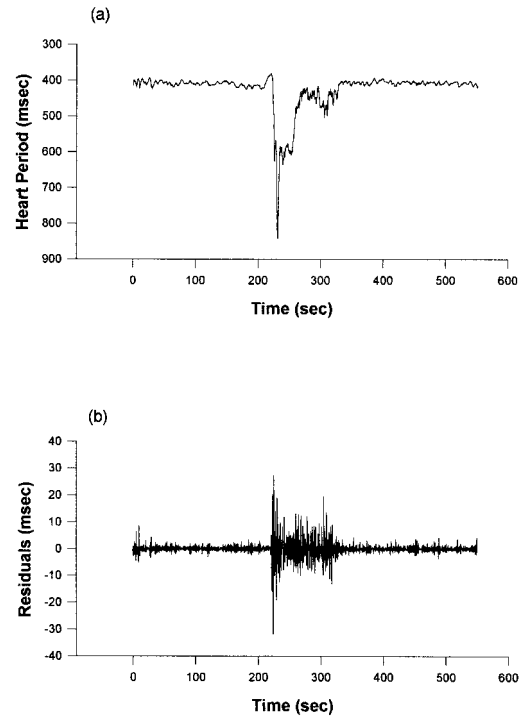
eration being outside the 5th percentile was greater than the statistical expectation (i.e., 5%). All subjects exhibited accelerations outside the range for at least one deceleration and in 4 of the 6 fetuses, virtually every deceleration (10/11) was preceded by a transitory acceleration.

An Increase in RSA Is a Component of the Recovery From a Deceleration

Postdeceleration levels of RSA ($M = 2.49$, $SD = .45$) were consistently higher than predeceleration levels ($M = 1.34$, $SD = 1.05$). All 6 subjects displayed an average increase in RSA. These differences are statistically significant, binomial test, $p < .05$. Figure 5 illustrates this characteristic pattern. Note the obvious increase in beat-to-beat variability and the quantified index of the amplitude of RSA. Convergent with the increase in RSA, the stabilized postdeceleration baseline was characterized by longer heart periods (i.e., slower heart rate). All 6 subjects displayed an average increase in heart period from predeceleration levels ($M = 432$ ms, $SD = 25$) to postdeceleration levels ($M = 452$ ms, $SD = 26$). These differences are statistically significant, binomial test, $p < .05$. Although the
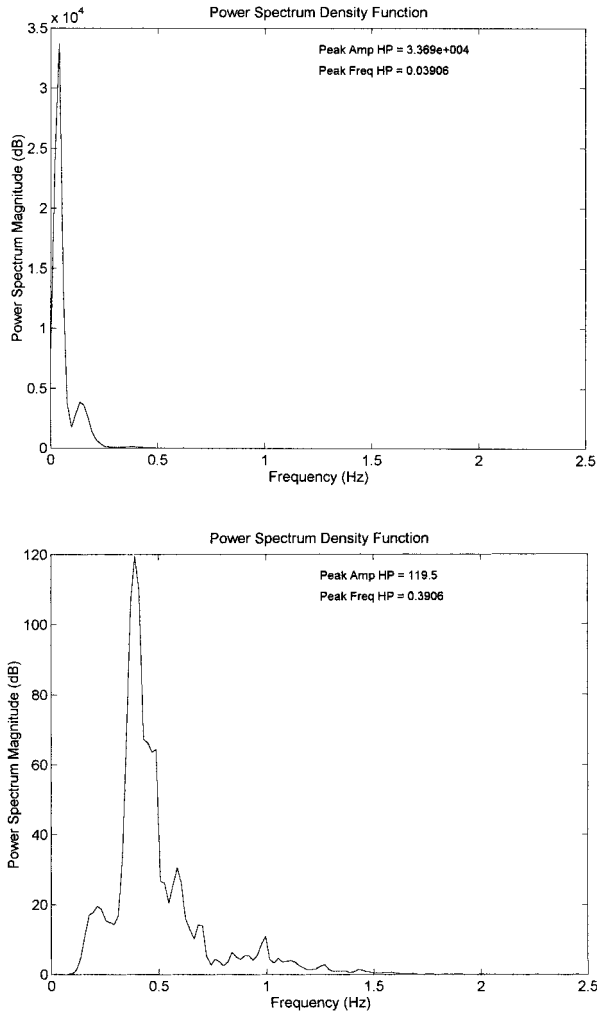
FIGURE 4 (a) Power spectrum of fetal heart period data plotted in Figure 3a; (b) Power spectrum of fetal heart period data plotted in Figure 3b.

number of decelerations provided by each of the fetuses varied, of the 19 decelerations observed across the 6 fetuses, 18 (95%) of the decelerations were associated with increased RSA and 12 (63%) episodes with increased heart period (or heart rate slowing) during the postdeceleration recovery phase.

Fetuses With Chronic Low-Amplitude RSA Have More-Frequent and Severe Bradycardia

The heart period pattern observed in the intrauterine fetal death case provides the only illustration in our restricted database of frequent and clinically relevant bradycardia occurring in a heart rate pattern devoid of RSA. As illustrated in Figure 6a, the heart period pattern is characterized by frequent bradycardia. In Figure 6b, one bradycardic event denoted by the two dotted lines in 6a is plotted. Note the flat beat-to-beat pattern that precedes and follows the bradycardia. Figure 6c further amplifies the pattern that precedes the bradycardia. Additionally, transitory tachycardia did not precede any of the bradycardia, suggesting an inability to further withdraw nucleus ambiguus vagal tone, which was already chronically depressed. However, as with the other fetuses, no significant correlation was found between RSA and the magnitude of the bradycardia or tachycardia.

DISCUSSION

The Polyvagal Theory provides a useful model for explaining fetal heart rate patterns and for predicting the likelihood of decelerations during normal delivery. According to this theory, fetal heart rate decelerations may be mediated via a primitive unmyelinated vagal system (i.e., vagal pathways originating in the dorsal motor nucleus of the vagus) in the absence of influences from a more phylogenetically advanced vagal

Table 1.   Number and Percentage of Decelerations Showing Transitory Accelerations

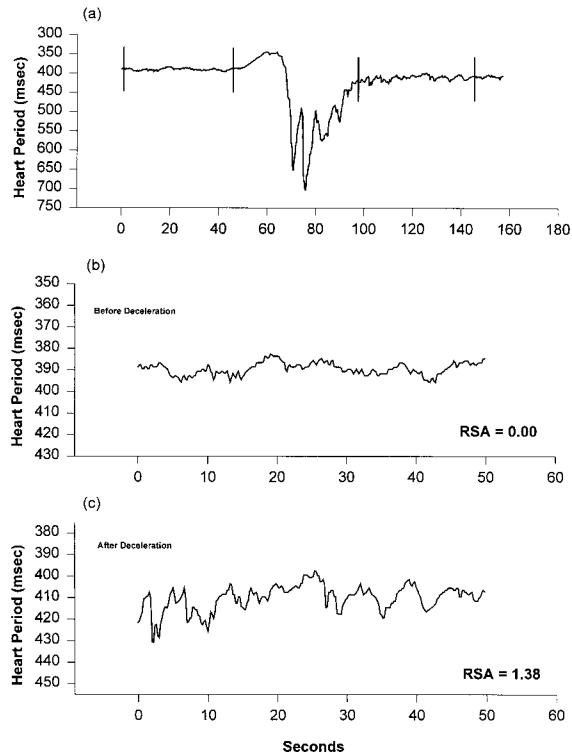| Subject	Total Number of Decelerations	Number with Significant Accelerations	Percent with Significant Accelerations
4478	6	1	17
5972	3	3	100
6851	11	6	55
6919	3	2	67
7374	4	4	100
8372	1	1	100
Totals	28	17	

FIGURE 5 (a) Heart period pattern demonstrating the increase in RSA following a deceleration; (b) The 30-s segment prior to the deceleration shows low-amplitude RSA; (c) The 30-s segment following a deceleration shows increased-amplitude RSA.

FIGURE 6 Segment of heart period data, shown at increasing magnification from (a) to (c), collected from a hydrocephalic fetus demonstrating the frequent and severe bradycardic activity that can occur in the absence of RSA.

system (i.e., vagal pathways originating in the nucleus ambiguus). Tonic depression of nucleus ambiguus vagal tone may be due to either physiological compromise or developmental immaturity. The current study provides preliminary support for three hypotheses derived from the Polyvagal Theory. First, the onset of a heart rate deceleration is reliably preceded by a transitory tachycardia (i.e., decrease in heart period) consistent with the predicted transitory effect on the pacemaker due to the withdrawal of nucleus ambiguus vagal tone. Second, recovery from a deceleration is regularly characterized by an increase in RSA amplitude demonstrating a reestablishment of nucleus ambiguus vagal tone. And finally, the absence of RSA has been shown to be associated with frequent and massive bradycardia in at least 1 fetal distress subject, consistent with the proposed vulnerability of the sinoatrial node to vagal influence from the dorsal motor nucleus of the vagus during physiological states characterized by low nucleus ambiguus tone.

Additional evidence of the role of the dorsal motor nucleus of the vagus in bradycardic activity is provided by research demonstrating an association 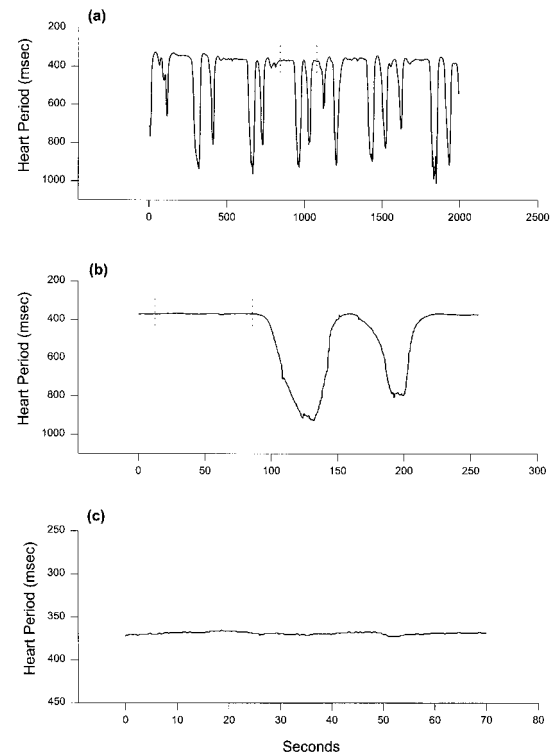between bradycardic activity and meconium staining (Goodlin & Haesslein, 1977). In addition to the heart, efferent fibers of the dorsal motor nucleus of the vagus innervate the muscles of the sphincter. Thus, it is theoretically consistent that if hypoxia stimulated vagally mediated relaxation of the sphincter muscles and the release of meconium, this same potent efferent system might produce bradycardia.

According to the Polyvagal Theory, transitory accelerations in fetal heart rate suggest that the nucleus ambiguus vagal pathways are being used as a "vagal brake" to regulate heart rate. A transitory removal of the vagal brake would produce a short latency acceleration and the reinstatement of the vagal brake would produce an immediate slowing of heart rate to a more-normal or optimal level. Thus, transitory accelerations would provide an indication that the nucleus ambiguus vagal system was controlling the fetal heart rate. From this perspective, the Polyvagal Theory accounts well for research demonstrating fetal heart rate accelerations to be indicators of fetal well-being (DiPietro, Hodgson, Costigan, Hilton, & Johnson, 1996; Emory & Noonan, 1984; Krebs, Petres, Dunn, & Smith, 1982). It should also be noted that the Polyvagal Theory offers an alternative explanation to that given by James et al. (1976) for the transient tachycardia ob-

served prior to bradycardia during hypoxic conditions. While James and colleagues attribute the tachycardia to sympathetic activation, the Polyvagal Theory presents a plausible alternative explanation in which these patterns could be the result of transient withdrawal of parasympathetic tone originating in the nucleus ambiguus and functional disinhibition of the pacemaker.

The current study offers only one example to support the hypothesis that reduced RSA is associated with increased likelihood of bradycardic activity. Although the fetus had neurological anomalies, the heart period pattern was consistent with the previously reported relationship between increased risk of fetal bradycardia and low beat-to-beat variability (Dawes et al., 1993). Therefore, the protective role of the nucleus ambiguus proposed by the Polyvagal Theory, and the high level of RSA associated with nucleus ambiguus tone, is also consistent with the view of heart rate variability as a positive indicator of fetal well-being (Kruse, 1982).

Because it has been questioned whether the unmyelinated vagal fibers originating in the dorsal motor nucleus of the vagus are capable of producing the massive bradycardia observed in the fetus (e.g., Hopkins et al., 1996; Jones et al., 1995), an alternative explanation might be considered. For example, it is possible that the myelinated vagal fibers originating in the nucleus ambiguus might contribute to clinical bradycardia. This explanation would suggest that the myelinated vagal pathways might, under different conditions, respond to two regulatory systems. During conditions when blood gases are within normal range the vagal fibers of the nucleus ambiguus, as a part of a "rhythmic" vagal system, may produce a coordinated respiratory rhythm in heart rate and bronchial activity to facilitate oxygen diffusion. However, during conditions of compromise when blood gas status threatens survival, not only would RSA be depressed but the respiratory drive involving the nucleus ambiguus also would be suppressed. In the absence of the rhythmic vagal system, a "tonic" vagal system may drop heart rate level to reduce metabolic demands. Perhaps during fetal distress, a tonic increase in the influence of the myelinated vagal fibers to the heart (i.e., producing bradycardia) is paralleled by the increase in the tonic influence of the unmyelinated vagal fibers to the gut (i.e., producing meconium).

Consistent with Jacksonian principles (Jackson, 1958) and the Polyvagal Theory (Porges, 1995, 1997), two alternative neurophysiological mechanisms might explain the clinical bradycardia observed in the human fetus. The first explanation is consistent with the phylogenetic distinctions between the vagal systems of mammals and reptiles. According to the first expla-

nation, fetal bradycardia would be due to the potentiation of unmyelinated vagal pathways originating in the dorsal motor nucleus of vagus by hypoxia, a state in which vagal tone from the myelinated vagal pathways originating in the nucleus ambiguus are depressed.

The second explanation assumes that the "new" myelinated vagal system originating in the nucleus ambiguus might have a phylogenetically ordered response hierarchy. According to the second explanation, under normal conditions, the vagal fibers from the nucleus ambiguus convey a respiratory rhythm to the heart and produce RSA. However, once blood gas status is compromised, the respiratory rhythm would be depressed (i.e., similar to neurophysiological processes that would produce apnea postpartum) and the more-tonic influences to the bronchi and heart would be expressed through the myelinated vagal fibers originating in the nucleus ambiguus. Thus, the bradycardia in the term fetus might be dependent upon the myelinated vagal fibers to the heart in mammals, which during conditions of compromise would function like the reptilian unmyelinated vagal fibers that originate in the dorsal motor nucleus of the vagus. This latter model might explain both the lack of evidence demonstrating potent bradycardia in mammals via the unmyelinated pathways and the occurrence of bradycardia during states characterized by depressed RSA.

Consistent with the points addressed by Paul et al. (1975), there is a relation between the ability to interpret data and the technology available to collect, store, and display fetal heart rate data. This point is critical in the clinical interpretation of fetal heart rate patterns, where the neurally mediated oscillations (i.e., RSA) in fetal heart rate are of low amplitude and relatively high frequency. Therefore, the accuracy of R-wave detection, the quality of the ECG signal, the digitizing rate, the resolution of fetal heart rate monitors (e.g., often fetal heart rate monitors provide moving averages of heart rate rather than true beat-to-beat values), and even the limitations of the statistical models used to analyze data may change the characteristics of the data and limit or bias the interpretation. Because the important information regarding neural tone to the heart via the nucleus ambiguus is conveyed in slight variations measured in changes of only a few milliseconds, it has been recommended that heart period data be quantified with an accuracy of 1 ms (Berntson et al., 1997; Riniolo & Porges, 1997).

The current study has limitations. First, uterine contraction activity and clinical judgment of decelerations (i.e., early, late, or variable decelerations) were not temporally indexed with the archived heart rate data. Thus, it is not possible to relate the current data to the more-traditional clinical indices of heart rate deceler-

ation. Most likely, because 6 fetuses were normally delivered, the decelerations quantified were of the variable type. Second, interpretations of the current results, as with any such research, are dependent on the particular definitions of heart rate decelerations and accelerations described herein. Third, the small sample of subjects may have precluded an ability to evaluate the relation between amplitude of RSA and magnitude of deceleration. However, the amplitude of RSA and the magnitude of deceleration might be related, but not in a manner that would be detected with linear correlation. Perhaps hypoxia or an amplitude of RSA below some absolute threshold is required to potentiate the magnitude of the deceleration. If these points are supported in future research, then the hypothesis could be supported by group differences between distressed and normal fetuses, even in the situation in which no significant correlations are observed within either the normal group, as reported in this study, or a distressed group. The data set did not provide an adequate test of the hypothesis within a distressed group because there was only 1 compromised fetus. Fourth, because the observations are limited by the sample (i.e., primarily healthy fetuses) and by the temporal constraints of the analyses (at least 60 s between decelerations), the study may have been biased to study only variable decelerations. Future research is needed to ascertain whether the Polyvagal Theory provides a plausible explanation for the physiological mechanisms determining early and late decelerations.

In summary, the Polyvagal Theory provides a new and unique perspective on the interpretation of fetal heart rate patterns. The theory provides an explanation for the paradoxical observation of vagal bradycardia in the absence of tonic vagal tone manifested in beat-to-beat variability. By increasing our understanding of the mechanisms responsible for the onset of and recovery from bradycardia, methods might be implemented to alert clinicians to states in which potentially life-threatening bradycardia are most likely to occur. With this knowledge, the probability of timely intervention, and thus, the survival prospects during delivery of the fetus at risk, might be improved. Further research evaluating RSA level as a predictor of the frequency of bradycardia and the time course of vagal responses relative to changing environmental variables may bring us closer to this goal.

NOTES

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