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Heart Rate Variability Analysis in Full-Term Infants: Spectral Indices for Study of Neonatal Cardiorespiratory Control

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ABSTRACT. The heart rate and respirations of twenty healthy full-term infants between 30 and 60 h postnatal age were studied during quiet sleep with the objective of defining spectral indices which represent normal neonatal heart rate variability (HRV) characteristics. Total HRV power and the distribution of power across different frequency bands varied considerably among infants. Cluster analysis on the measured variables indicated that the population divided into two groups that represented significantly different patterns of HRV behavior. In one group (11 subjects), infants had lower breathing rates and HRV power in a band about the respiration frequency [respiratory sinus arrhythmia (RSA) band] was more than 20% of the total power (TP). Additionally, the ratio of low frequency band power to RSA band power was <4. The other group of neonates (nine subjects) had relatively higher breathing rates, RSA power <20% of total power, and low frequency to RSA power ratio >4. Regression analysis of low frequency *versus* TP and RSA *versus* TP graphs gave strong support to the hypothesis that there were indeed two distinct patterns of HRV behavior. Separation of apparently normal neonates into two groups may be attributed partially to differences in respiratory rates and breathing patterns. However, it is possible that differences in the balance between sympathetic and parasympathetic nervous system control, perhaps related to autonomic maturation, also contribute to group separation. The indices developed from HRV spectral analysis in this investigation may be of value in the study of cardiorespiratory control in neonates. (*Pediatr Res* 26: 188–195, 1989)

Abbreviations

BASA, breath amplitude sinus arrhythmia
CR, cardiorespiratory
HF, high frequency
HR, heart rate
HRV, heart rate variability
LF, low frequency
RSA, respiratory sinus arrhythmia
TP, total power
VLF, very LF

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Recently, spectral analysis of HRV has been studied as a means of quantifying CR behavior in neonates (1–5). Before pathologic behavior and its underlying causes can be identified, normal HRV characteristics must be defined within the context of physiologic CR control.

Cardiorespiratory performance is governed by the autonomic nervous system. Beat-to-beat fluctuations in heart rate and blood pressure and their interaction with respiration are consequences of this complex autonomic control. Therefore, the behavior of these physiologic variables inherently contains measures of the ability of the autonomic nervous system to respond to disturbances and maintain homeostasis.

The rhythmic activity of autonomic neurons, both sympathetic and parasympathetic, influences systemic arterial pressure through peripheral resistance, HR, and stroke volume by acting on cardiac and vascular smooth muscles. The response of these effector muscles results in a behavior which is similar to that of a filter (6, 7), determining the final rhythmic output. Sensors, such as baroreceptors and chemoreceptors, provide measurements of CR system response and information feedback within closed loop operations. Respiration both influences the CR system with each respiratory cycle and also responds to sensors within it. Thus, the composite cardiorespiratory behavior can be viewed in terms of a set of oscillations, each arising from the combined action of neurons, effector muscles, and sensors. These oscillations are often reflected in the frequency spectrum of HRV and blood pressure, either as separate peaks or through spectral activity resulting from interactions among the oscillators. Interactions include partial or complete entrainment of one frequency peak into another and spectral components at harmonics, or integer multiples, of oscillator frequencies (8–10).

If the frequency content of the HRV spectrum is viewed in terms of control system behavior of the oscillations described above, information derived from various portions of the spectrum may be used to evaluate function. Kitney *et al.* (11) have proposed that HRV spectral characteristics may be employed to assess autonomic function in diabetic neuropathies of adults, and differences in power of various portions of the HRV spectrum have been used to evaluate autonomic control in renal failure (12). Porges (5) has used spectral analysis of heart period activity associated with the respiratory frequency band as a measure of RSA amplitude and used this to describe vagal tone. Haddad *et al.* (13) studied RSA in 4-wk-old puppies and in adult dogs and concluded that the physiologic mechanisms involved mature postnatally, whereas studies in a lamb model (14) imply that the sympathetic and parasympathetic nervous systems mature at different rates with the sympathetic system more prominent in fetal life. Dykes *et al.* (1) and Giddens and Kitney (7)

described an interaction between heart rate and respiration in which the modulation frequency in breath amplitude of full-term neonates appeared in the HR spectrum in addition to RSA. These investigators termed this phenomenon breath amplitude sinus arrhythmia (BASA).

Despite these and other studies, however, little data are available that quantify normal values of HRV characteristics in healthy neonates, either term or preterm. Before HRV behavior can be used to describe maturation or pathologies of CR control, it is essential to establish normal characteristics and their variability. Toward this end, we have studied a series of healthy, full-term neonates during their first few days of postnatal life with the objective of defining indices that represent apparently normal HRV characteristics.

MATERIALS AND METHODS

Data were collected on a group of 20 full-term infants at Grady Memorial Hospital in Atlanta between April 1986 and May 1988. These infants were 38 to 42 wk gestational age, with birth wt ranging from 2800 to 3800 g, and were within a narrow window of 30 to 60 h postnatal age. Only infants with Apgar scores >7 at both 1 and 5 min and without congenital anomalies were considered for study. Any maternal drug exposure or evidence of acute or chronic fetal distress excluded an infant. Informed consent was obtained to record the ECG and respiration signals of these infants according to a protocol approved by the Human Investigation Committee of Emory University.

Infants were fed before testing and then placed on a bed in the prone position. The bed was situated in an environment free from excessive noise and light stimuli. Two observers monitored the infant for sleep state, determined by the method of Prechtl (15). In addition to sleep state, all movements, twitches and sighs were noted on the strip chart tracing as data were being recorded. When the infant was observed to be in a state of quiet sleep, a continuous recording for a duration of at least 4 min was obtained.

The ECG and respiration signal were detected with a Hewlett Packard model no. 78834A neonatal heart rate and respiration monitor (Hewlett-Packard Co., Palo Alto, CA). The analog signals of ECG and respiration were recorded on a four channel RACAL Store-4 FM tape recorder (RASCAL Recorders, Inc., Sarasota, FL) at a tape speed of 15/16 in/s. Determination of the HR from the recorded ECG signal followed the procedures described in the appendix of Dykes *et al.* (1), except that a simple difference filter accentuated the QRS portions of the ECG and attenuated the P and T waves. A small interval was then easily identified around the QRS complex and the original unfiltered signal was searched for a peak within this interval. This modification improved R-wave detection, particularly in situations where there were P-waves with large amplitude. The resulting HR data and the digitized respiration signal for 100-s records were spectrally analyzed using Fast Fourier Transform methods to determine HRV power in various frequency bands and using autoregressive methods to identify oscillations at discrete frequencies, as previously described (1). All data digitization and analysis were performed on a MASSCOMP 5500 computer system (Massachusetts Computer Co. Westford, MA).

The gestational age, postnatal age, and birth wt were examined for their influence on mean HR, respiratory frequency, and total power of the HRV spectrum. The power spectrum was separated into three regions: very low frequency (VLF), 0 to 0.04 Hz (Hz = cycles/s); LF, 0.04 to 0.20 Hz; and HF, more than 0.2 Hz (Fig. 1). These divisions correspond to basic autonomic oscillations, *i.e.* cellular or humoral, vasomotor, and respiratory rhythms, respectively (6, 16–18). Spectral content of HRV power was calculated for the following bands: VLF, LF, and RSA (defined as a band within ± 0.2 Hz of the respiratory frequency obtained from peak amplitude in the respiratory power spectrum). The LF band was also subdivided into band 1 (0.04 to 0.08 Hz) and band 2 (0.08 to 0.16 Hz) because these frequencies may be of particular interest with regard to BASA and baroreceptor activity. In addition, the ratio of LF to RSA power was computed. The units of total power and the power associated with bands in the spectrum are Hz^2. Values on tables and graphs are given in units of 10^{-2} Hz^2.

Only segments recorded during quiet sleep, which were free of movements, twitches, and respiratory sighs, were used for analysis. Initial screening of the 100-s data segments to be used was performed by visual inspection of the time series and autoregressive power spectra of the HR and respiration. HR data segments that had upward or downward trends in the HR spectrum were not used if the power in the frequency band between 0.0 and 0.04 Hz was >20% of the total power. The reason for this was 2-fold: *1*) a trend could increase the power in this region without evidence of any well-defined peak, and *2*) if there were well-

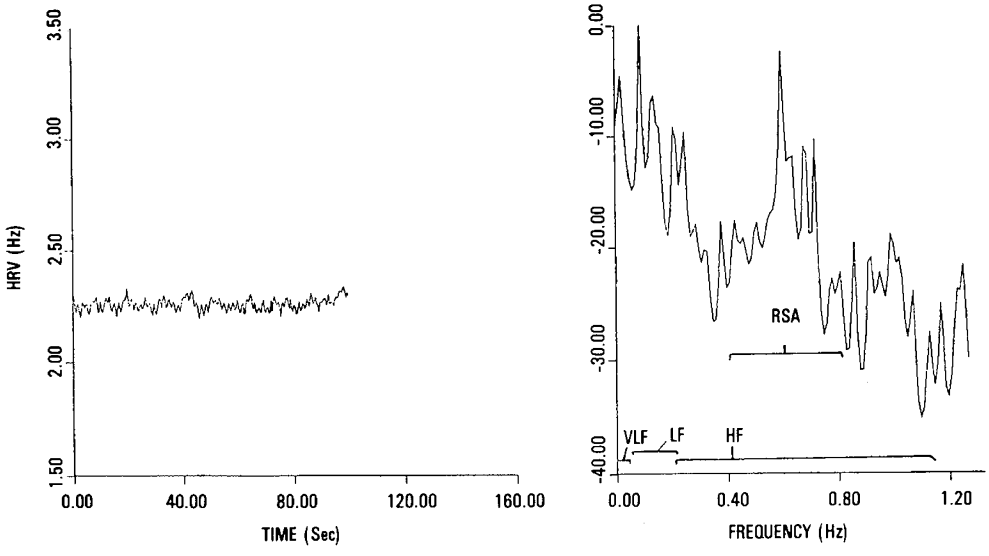

Fig. 1. HRV time series and logarithmic power spectrum from a full-term infant at 2 d of age. Spectral activity was divided into VLF (0 to 0.04 Hz), LF (0.04 to 0.20 Hz), and HF (>0.20 Hz) regions. RSA is the major component of the HF region. These divisions correspond to basic autonomic oscillations, *i.e.* cellular/humoral, vasomotor, and respiratory rhythms, respectively.

defined peaks in this region causing a power >20%, this was not considered a "stable" data record.

Statistical analyses were performed on data points representing averaged values for all stable 100-s segments of recording available for each infant. K-means cluster analysis was performed on the data using the BMDP statistical software package. A Wilcoxon, two sample test for independent samples was used to compare means of the two groups. In conducting the analysis of residuals from the linear regression of RSA on TP and LF on TP for the two groups, the residuals behaved well in normal probability plots and in displaying independence; however, the residual variances as estimated by mean square error were found to be significantly different for RSA on TP using an F test. For this reason, the slopes of the regressions were compared using a Behrens-Fisher approximate t statistic (19).

RESULTS

Mean HR and respiratory frequency. There was no relationship between mean HR or respiratory rate *versus* gestational age, postnatal age, or birth wt. Additionally, there was no relationship found between mean HR and respiratory rate.

Total HR variability power. The total power in HRV comprises all variations about the mean HR regardless of frequency of variability. Total power varied considerably among infants, but there were no significant trends in total power as a function of either gestational age, postnatal age, or birth wt, respectively. Although not statistically significant, there was a tendency for total HRV power to decrease as the mean HR decreased (r = 0.34).

HRV and respiration signal spectra. Two examples of HRV time series and power spectra with their corresponding respiration signal time series and power spectra are presented in Figures 2 and 3. Although the HRV spectral details vary considerably, not only among individual subjects but also among records taken from the same subject, these figures illustrate two notably different types of behavior. In Figure 2, oscillations in HR corresponding to the respiratory rate (*i.e.* RSA) are prominent in the time series and contain significant power as seen in the HR power spectrum. Additionally, LF oscillations are observable in the HR time series, and the LF region of the HR spectrum also contains power comparable in magnitude to that of RSA. In contrast, Figure 3 shows results from an infant who had very little RSA, as seen clearly in both HR time series and HR spectral presentations. However, there is prominent LF HRV power in this infant. Examining the spectra of the respiration records corresponding to the two examples above, the width of the respiratory peak in Figure 3 was greater than that of Figure 2. This behavior was typical for infants that had low RSA power in the HR spectrum.

HRV spectral indices. Table 1 presents the percent RSA, TP, and the ratio of LF to RSA power obtained from the HRV spectral analysis of each infant in the study. The entries for percent RSA and total power are averages for all stable data records available on that infant, whereas the LF/RSA ratio is the average for the LF to RSA power ratio for these same data records. Observation of these data and of the time series and spectral records for each infant (cf. Figs. 2 and 3) suggested that there might be two types of HRV behavior within the overall group of 20 infants: one subgroup having relatively large RSA power and small LF/RSA ratio and the other having relatively small RSA power and large LF/RSA ratio. To test this observation a K-means cluster analysis was performed. The pooled data were provided as input to the clustering algorithm with variables

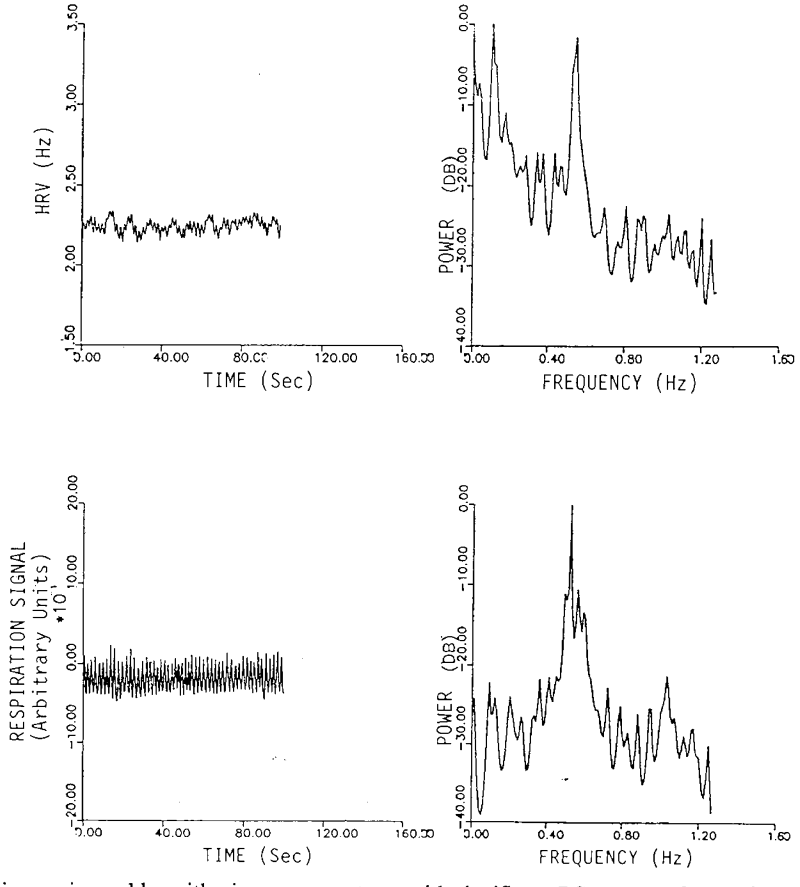

Fig. 2. *Upper panel*, HRV time series and logarithmic power spectrum with significant RSA power, from a full-term infant during quiet sleep at 2 d of age. *Lower panel*, the corresponding respiration signal time series and logarithmic power spectrum from this infant.

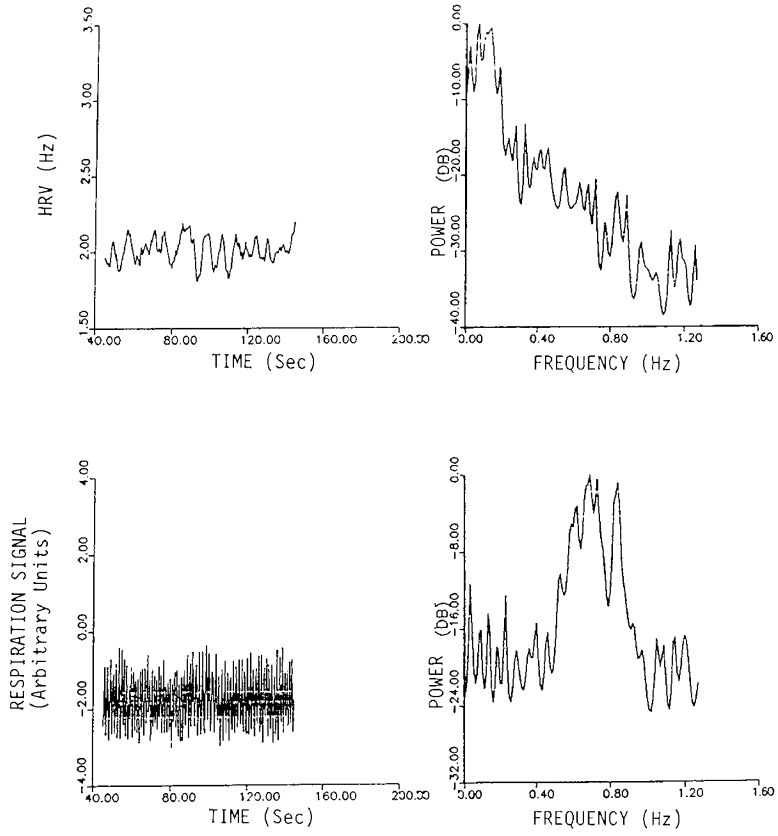

Fig. 3. *Upper panel*, HRV time series and logarithmic power spectrum with very little RSA power, from a full-term infant during quiet sleep at 2 d of age. *Lower panel*, the corresponding respiration signal time series and logarithmic power spectrum.

Table 1. *Percent RSA and LF/RSA ratios for 20 healthy full-term infants during quiet sleep at 2 d of age*

% RSA	Total power ($\times 10^{-2}$ Hz^2)	LF/RSA ratio	Infant
73	0.0818	0.20	19
63	0.1168	0.31	17
55	0.1899	0.48	11
51	0.1031	0.42	13
46	0.0959	0.44	4
40	0.1807	1.00	14
39	0.1473	1.19	5
38	0.2520	0.85	12
35	0.0484	1.08	2
29	0.0727	1.59	3
25	0.1918	2.04	18
12	0.0541	4.63	16
11	0.0714	4.67	20
11	0.0566	4.78	6
11	0.0467	5.77	1
7	0.2193	10.1	9
5	0.3553	14.5	8
4	0.1335	16.6	7
4	0.6314	18.8	15
3	0.1695	30.1	10

standardized and two clusters specified as group A and group B. The clustering variables, F ratios related to associated analysis of variance, and significance levels were: RSA ($F = 56.6$, $p < 0.0005$), LF/RSA ($F = 18.9$, $p < 0.0005$), TP ($F = 0.0169$, $p = 0.422$), respiratory frequency ($F = 22.8$, $p < 0.0005$), HR ($F = 0.0058$, $p = 0.849$).

After the two groups were defined, the powers in the LF band and RSA band were plotted *versus* total power for the infants in the two groups. Figures 4 and 5 present data for group A and group B neonates, respectively. The linear regression equation and correlation coefficients are included in each figure. Because the data are plotted as a function of total power, the slopes of the regression lines reflect the percent power in the frequency band in each case. The Behrens-Fisher test showed that slopes differed significantly between the two groups for LF *versus* total power ($p < 0.001$). For RSA *versus* total power, slopes were also significantly different ($p < 0.02$).

Group comparisons. Table 2 is a compilation of power calculated for the various spectral bands representing averages of 100-s data records for each infant. Column 1 gives the number of data records used for each infant. Means and SD are given for each group. There was no statistical difference in mean HR between the two groups, but there was a significant difference with respect to respiratory frequency ($p < 0.001$). Power in all bands below 0.2 Hz was increased for group B *versus* group A. There were statistically significant differences in percent LF power ($p < 0.001$) and in percent RSA power ($p < 0.001$).

The averaged percent RSA was graphed *versus* respiratory frequency for each infant (Fig. 6). Group B infants generally had a higher respiratory rate than group A infants, but there was overlap near 0.7 Hz.

Inasmuch as variability in total HRV power can be large among infants and over time for a given infant, two of the group A subjects were examined on three separate occasions during the second and third postnatal days: *1*) morning of d 2; *2*) afternoon of d 2; and *3*) morning of d 3. Table 3 presents the results of these studies and Figure 7 illustrates the variation of percent RSA power with respiratory frequency. Analysis did not show a significant difference between the slopes, although one neonate

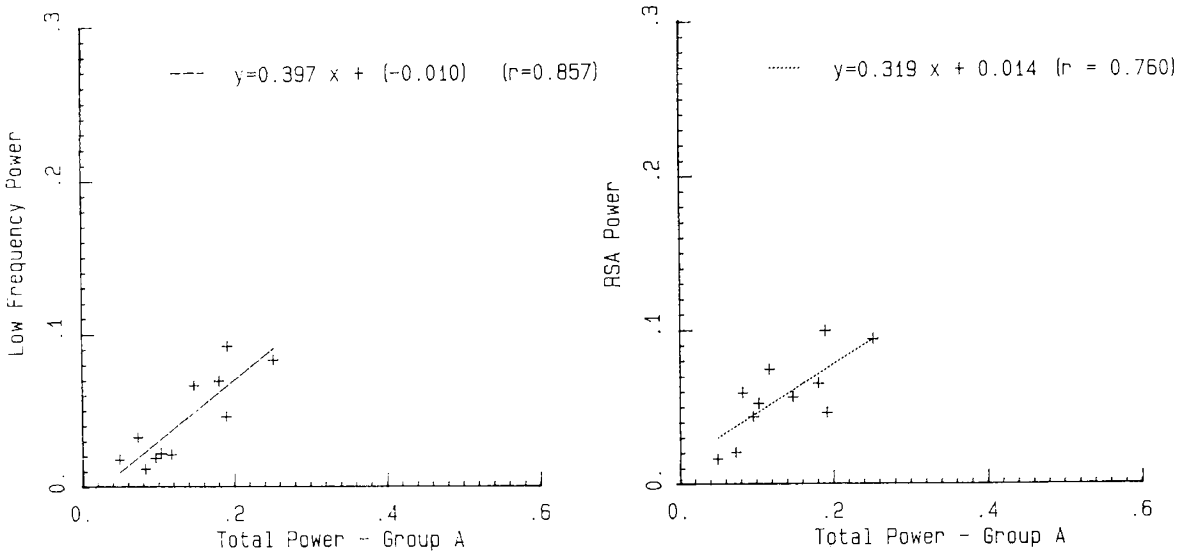

Fig. 4. The low frequency power and RSA power are plotted *versus* total power for infants in group A. The regression line equation and the correlation coefficient are shown with each plot.

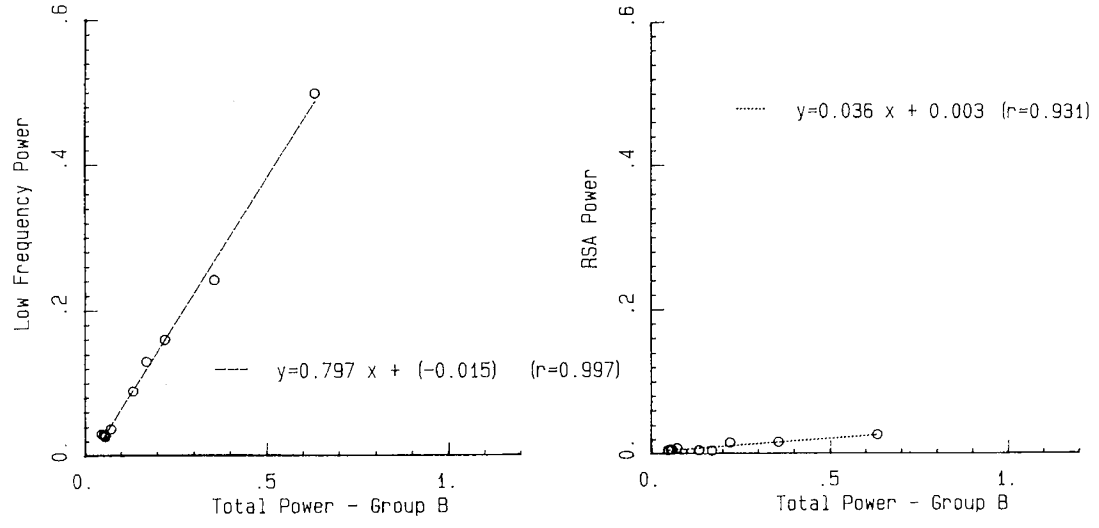

Fig. 5. The low frequency power and RSA power are plotted *versus* total power for infants in group B. The regression line equation and the correlation coefficient are shown with each plot.

had a higher percent RSA than the other at a given respiratory frequency.

DISCUSSION

Conceptually, HRV characterization can be considered to include several levels of description, *e.g. 1*) mean HR, *2*) total power of HRV, and *3*) spectral content of HRV power. Each level provides the potential for yielding additional information concerning CR control. Although data for mean HR are available for neonates, there have been no studies that establish normal values for total HRV power or for the distribution of HRV power as a function of frequency. In addition, for these data to be useful the variations within a truly normal population must be acceptably small.

The data from our investigation of 20 full-term healthy neonates contained a large degree of CR variability. Although the mean HR and respiratory rates of our infants would be classified as "normal," attempts to define population normal values for total HRV power and for the distribution of power within various frequency bands did not yield distinctive relationships. It was not until the infants were divided into two groups according to RSA power and LF/RSA ratios (*i.e.* a division based upon the third level of characterization), that useful indices were obtained.

The mean percent RSA power was the major factor that led to the definition of two groups, with group A having significantly larger percent RSA power than group B ($p < 0.001$). Because RSA power is very small in group B, the linear relationship found between LF and total power in this group likely is due to the fact that most of the power is in the LF band. However, there is no *a priori* reason that the LF and RSA power in group A should also show a strong linear relationship with total power.

Group B infants, who had relatively low percent RSA power, exhibited large values of LS/RSA ratios. In fact, no group B infant had either an individual or averaged ratio value less than 4. Although this ratio is not identical to the one used by Kitney (3) in a study related to infants at risk for sudden infant death syndrome, it is similar in nature. In that study of infants ranging from 3 to 90 d postnatal age, Kitney (3) proposed that a high ratio of LF to HF spectral power indicated increased risk of sudden infant death syndrome. The fact that six of 20 clinically normal neonates in our study had ratios of LF to HF greater

Table 2. *Quantitative results obtained from spectral analysis on 20 full-term infants during quiet sleep at 2 d of age*

No. of records	Mean HR	Total power	(power in 10^{-2} Hz^2) 0.00–0.04	0.04–0.08	0.08–0.16	0.04–0.20	RSA	Respiration frequency
Group A								
2	2.416	0.0818	0.0045	0.0026	0.0073	0.0118	0.0595	0.54
3	2.086	0.1168	0.0088	0.0070	0.0117	0.0212	0.0746	0.71
2	2.315	0.1899	0.0121	0.0088	0.0216	0.0461	0.0996	0.66
4	2.448	0.1031	0.0155	0.0058	0.0115	0.0222	0.0525	0.61
3	2.333	0.0959	0.0155	0.0046	0.0116	0.0190	0.0437	0.67
2	1.821	0.1807	0.0254	0.0123	0.0491	0.0694	0.0656	0.56
2	2.235	0.1473	0.0127	0.0132	0.0426	0.0666	0.0566	0.52
4	2.149	0.2520	0.0263	0.0130	0.0456	0.0829	0.0944	0.63
2	2.065	0.0484	0.0056	0.0052	0.0109	0.0180	0.0163	0.62
2	2.260	0.0727	0.0051	0.0066	0.0193	0.0326	0.0206	0.65
3	1.949	0.1918	0.0250	0.0127	0.0637	0.0921	0.0464	0.63
Mean	2.189	0.1346	0.0142	0.0083	0.0268	0.0438	0.0573	0.62
SD	0.196	0.063	0.008	0.004	0.020	0.029	0.026	0.06
Group B								
1	2.253	0.0541	0.0076	0.0089	0.0173	0.0301	0.0065	0.68
2	2.169	0.0714	0.0199	0.0058	0.0306	0.0375	0.0082	0.90
3	2.067	0.0566	0.0107	0.0070	0.0143	0.0271	0.0057	0.80
1	2.620	0.0467	0.0074	0.0046	0.0252	0.0306	0.0053	0.70
2	2.255	0.2193	0.0259	0.0573	0.0981	0.1602	0.0158	0.72
3	2.170	0.3553	0.0490	0.0844	0.1329	0.2422	0.0166	0.85
1	2.120	0.1335	0.0271	0.0310	0.0530	0.0895	0.0054	0.85
2	2.028	0.6314	0.0516	0.1070	0.3410	0.4994	0.0265	0.70
2	2.325	0.1695	0.0255	0.0301	0.0959	0.1303	0.0044	1.0
Mean	2.223	0.1931	0.0250	0.0373	0.0898	0.1385	0.0105	0.80
SD	0.176	0.193	0.016	0.038	0.103	0.154	0.008	0.11
Statistical significance:								
	$p > 0.9$	$p > 0.9$	$p > 0.1$	$p < 0.08$	$p < 0.04$	$p > 0.05$	$p < 0.001$	$p < 0.001$

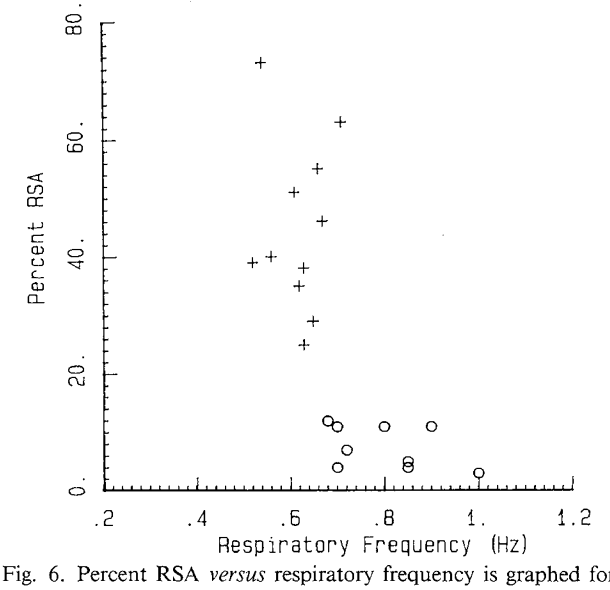

Fig. 6. Percent RSA *versus* respiratory frequency is graphed for all infants in the study. Points represent the averaged data from 100-s records available on each infant (group A = +, group B = o).

than 10 underscores the need to consider postnatal age in CR control when defining risk factors. The LF/RSA ratio is a reflection of both sympathetic and vagal response. Higher values of the ratio correspond to greater sympathetic and/or less vagal response and lower values are indicative of less sympathetic and/or greater vagal response.

The appearance of two clearly different types of HRV behavior, virtually equally divided in number among a group of 20 apparently normal, full-term infants is thought provoking. In fact, the foremost questions are whether there are two different CR control patterns or whether alternative explanations for the data exist. At least three possibilities might explain differences in the two patterns of behavior in this clinically normal population: *1*) sleep state; *2*) respiratory characteristics; and *3*) maturation.

The first of these relates to classification of sleep state. Low frequency power increases in active sleep that leads to both a decrease in percent RSA power and an increase in the LF/RSA ratio. Thus, if data were collected during active sleep, a group A infant might erroneously be placed in group B. However, two observers classified our subjects as being in quiet sleep, using the criteria of Prechtl for state 1 and state 2 sleep. Additionally, the records selected for analysis were taken from sections where respiration and HR were quite stable. Upon analysis, whenever an observer noted a change to active sleep, it was found that this was accompanied by departures from stability in the recorded data. Furthermore, the classification of infants who had multiple records during quiet sleep was consistent in each case.

Interactions between respiration and HRV are well documented (1, 4, 10, 20) and were considered as possible explanations for the observed behavior. Examination of the respiratory rates for groups A and B (see Table 2), showed that group B infants breathed faster. Inasmuch as RSA decreases as respiratory rate increases (8, 9, 21), the emergence of two different groups could be simply a reflection of different breathing frequencies. However, we have examples of infants whose respiratory rates are at times within the range found for group B but whose percent RSA and LF/RSA ratios are typical for infants in group A. As a consequence, there are HRV spectral differences between the two groups that cannot be accounted for by respiratory rate alone.

Similarly, data obtained from two group A infants studied at three different times showed that although percent RSA power

Table 3. *Quantitative results obtained on two group A infants during quiet sleep at three different times*

Infant/ postnatal age	Mean HR	Total power ($\times 10^{-2}$ Hz^2)	LF power ($\times 10^{-2}$ Hz^2)	RSA power ($\times 10^{-2}$ Hz^2)	Respiration Frequency	% LF	% RSA	LF/RSA ratio
No. 13	2.412	0.1200	0.0248	0.0597	0.58	21	50	0.415
46 h PNA	2.452	0.0728	0.0161	0.0396	0.64	22	54	0.407
	2.469	0.0981	0.0206	0.0475	0.61	21	48	0.434
	2.46	0.1213	0.0273	0.0633	0.61	23	52	0.431
No. 13	2.236	0.0717	0.0242	0.0269	0.64	34	38	0.900
48 h PNA	2.269	0.0751	0.0288	0.0288	0.67	38	38	1.00
	2.295	0.0674	0.0265	0.0250	0.72	39	37	1.06
	2.301	0.0817	0.0289	0.0362	0.70	35	44	0.798
No. 13	2.163	0.2914	0.0676	0.1769	0.57	23	61	0.382
70 h PNA	2.167	0.3070	0.0808	0.1889	0.55	26	62	0.428
	2.188	0.3185	0.0858	0.1907	0.56	27	60	0.450
	2.168	0.2901	0.0845	0.1659	0.54	29	57	0.509
	2.156	0.3392	0.1064	0.1855	0.56	31	55	0.574
	2.156	0.4444	0.1115	0.2649	0.55	25	60	0.421
	2.175	0.3469	0.0963	0.1988	0.56	28	57	0.484
No. 14	1.842	0.1147	0.0324	0.0579	0.57	28	51	0.560
49 h PNA	1.799	0.2467	0.1065	0.0734	0.55	43	30	1.45
No. 14	2.031	0.1951	0.0798	0.0370	0.70	41	19	2.16
51 h PNA	2.038	0.1321	0.0653	0.0225	0.73	49	17	2.90
No. 14	1.868	0.2360	0.0936	0.0866	0.60	40	37	1.08
71 h PNA	1.869	0.3261	0.1166	0.1284	0.55	36	39	0.908
	1.892	0.2244	0.0783	0.0985	0.53	35	44	0.795
	1.849	0.3360	0.1097	0.1142	0.54	33	34	0.961

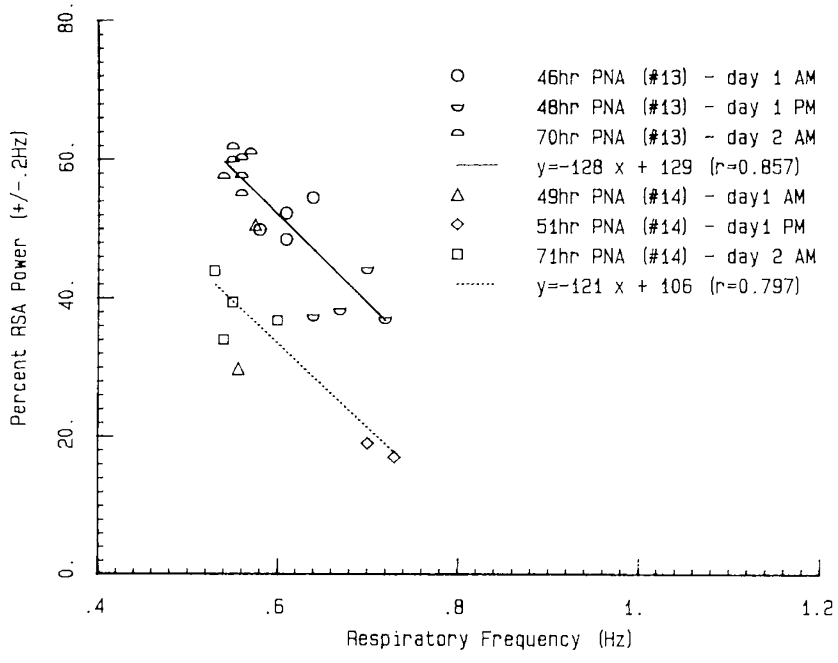

Fig. 7. Percent RSA power *versus* respiratory frequency is graphed for each of two group A infants. The regression line equation and the correlation coefficient are shown for each infant. This figure represents data taken at three different times for each infant.

decreased as respiratory frequency increased in a given subject (Fig. 7), the ratios of LF to RSA power still corresponded to group A values regardless of the breathing rate (compare LF/RSA ratios in Tables 1 and 3). The fact that the percent RSA was different for the two infants at similar respiratory frequencies may arise from differences in vagal tone.

In addition to respiratory rate, the bandwidth of the respiratory rate in the respiratory power spectrum for infants in group A was less than that for infants in group B (see Figs. 2 *versus* 3). The parasympathetic system dominates during quiet sleep and respiratory drive is under automatic (metabolic) control, resulting in slower and more regular respiration (22–24). Not all neonates may be able to control respiration in a consistently reproducible way to generate a narrow band breathing frequency. This irregularity would result in a broader respiratory band in the respiration spectrum. With the dispersion of the respiratory frequency, there may be a corresponding decrease in the stimulus effect upon the HR.

Previous investigations in neonates have shown that respiration interacts with HR at frequencies other than the respiratory rate (1, 4). BASA has been observed in the LF region of the HRV spectra. Also, LF peaks in the power spectrum of the respiration data from full-term and preterm infants, compatible with chemoreceptor respiratory feedback control, have been described by

Hathorn (25). Cross-spectral analysis of respiration and HR data obtained from neonates may reveal that group separation can be achieved using respiratory characteristics other than the rate of breathing. This is an area for future study that will require extensive analysis.

Finally, it is tempting to speculate that the two groups reflect different levels of autonomic maturity at birth, because group A infants behave more like adults (26). Sympathetic activity, such as standing after atropine administration, markedly reduces gain in adult HRV above 0.1 Hz with a phase delay, whereas the pure vagal response in the supine position after administration of propranolol yields a higher gain at all frequencies with no phase delay (27, 28). Consequently, one possible interpretation of the group differences may be that group A infants have greater parasympathetic activity. This is consistent with the premise that the sympathetic nervous system dominates in more immature infants and the parasympathetic system develops further postnatally. If this is the case, group B neonates should eventually develop group A behavior. Longitudinal studies are required to determine this possibility.

In summary, we have described spectral activity and indices in healthy full-term infants which can be calculated from data readily obtained from routine monitoring available in neonatal nurseries. It appears that two distinctively different types of behavior occur in clinically normal neonates during the first few days of postnatal life. Investigations in premature infants, as well as longitudinal studies for both full-term and preterm infants, are required to determine whether these differences may be related to maturation in CR control.

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