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Heart rate
newborn
polygraph
sleep state
sudden infant death syndrome

Polygraphic Studies of Normal Infants and Infants at Risk for the Sudden Infant Death Syndrome: Heart Rate and Variability as a Function of State

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Summary

Spontaneous heart rate and variability were examined as a function of age and state from birth to 6 months of age in 10 normal infants and 10 infants at risk for sudden infant death syndrome (SIDS). The risk group showed a higher heart rate at 3 months of age, particularly in the waking state. The risk infants' heart rate also increased more markedly during the first month after birth in both quiet and active sleep. Heart rate in both groups declined after 2 months in every state; however, the risk infants lagged in the 2- to 3-month decline seen with the normal infants. In the awake state, heart rate variability in the risk group did not follow the increase seen with the normals during the 1-week to 2-month age period.

Speculation

There are distinct developmental differences in mean heart rate, and to a lesser extent in mean heart rate variability, between groups of normal infants and infants at increased risk for sudden infant death. These differences may reflect delayed maturation or impaired functioning of the autonomic nervous system, and particularly of vagal control. However, some of these differences, for example, increased heart rate in the risk group at 3 months of age and a delayed decline in this group's heart rate values after 1 month of age, are also compatible with an interpretation of chronic hypoxia. The tendency of the differences to be most pronounced in the awake state adds support to the possibility of impaired oxygen regulation in the risk group. Thus, both chronic hypoxia and the dysfunction in autonomic control that might result from it could play a significant role in sudden infant death.

Waking and sleep states exert a significant modulatory effect on cardiac rate and variability in both infants and adults (2, 6, 14, 20). In fact, state-related changes in heart rate and variability can exceed those due to development during the first 6 months of life (9). State is also profoundly important in the etiology of the SIDS, since the timing (in the early morning hours) and location of death (often in a crib) indicate that most SIDS fatalities occur during sleep.

The available epidemiologic literature suggests that most deaths from SIDS occur in the first 6 months of life, and that the death rate peaks at 2–3 months of age (4, 23). Thus, development is also crucially linked to SIDS. Maturation of autonomic functions is particularly significant, and detailed information about such maturation is undoubtedly necessary to understand SIDS. For example, one would expect that the heart rate in the infant during the first 6 months of life would continuously decrease; on the contrary, we have previously reported increased heart rates, particularly in quiet sleep, at 1 month from the values observed in the newborn period, followed by a decline at 3 months in all states (9). Similarly, except for quiet sleep, heart variability was higher at 2 and 3 months of age than at 1 month and subsequent age periods.

Because there are marked changes in cardiac rate and variability that approximately coincide with the peak incidence of SIDS, and because sleep state plays an important role in the modulation of cardiac activity, we compared the development of heart rate and variability during sleep and wakefulness in infants at increased risk for this syndrome with that in normal infants.

MATERIAL AND MONITORING PROCEDURE

Ten subsequent siblings of SIDS victims of gestational ages between 39 and 41 weeks, and with 1-min Apgar scores of 8 or 9, participated in this study. Subsequent siblings of SIDS (SSIDS) were chosen as a risk group, since they have a 3- to 4-fold higher risk for SIDS than the general population (8). The SSIDS infants were matched with control infants studied earlier (9) according to the educational level of their parents. Educational level was used since it is highly correlated with socioeconomic level. The two groups were matched on socioeconomic level to minimize differences between the two samples. There is, in addition, some suggestive evidence that lower socioeconomic level may be related to a higher incidence of sudden infant death in some societies (4).

Four of the SSIDS infants were males and six were females. Birth weights ranged from 3090–4281 g. All the infants were full term and appropriate for gestational age, according to the intrauterine growth curve of Usher and McLean (22). Individual birth weights are listed, along with gender, for all 20 babies in Table 1. Neither the mothers of control infants nor the mothers of SSIDS infants had a prior history of diabetes or other illness. Each infant was admitted at 5 PM to the sleep laboratory for a 12-hr all-night monitoring session. These sessions were held during the first week of life and at 1, 2, 3, 4, and 6 months of age. The parents were informed of the nature and objectives of the study, and written permission was obtained prior to participation. The monitoring procedures have been described in detail earlier (9).

PHYSIOLOGIC RECORDING METHOD

Both sleep and cardiopulmonary variables were monitored. Sleep was monitored through two EEG derivations, together with chin EMG, eye movement, and gross somatic activity recordings.

The placement of EEG electrodes, the infrared transducer used to monitor eye movement, and the procedures for measuring somatic activity and respiration have all been described earlier (9). The ECG was recorded from two disposable electrodes placed symmetrically beneath the clavicles with a ground electrode above the umbilicus.

Table 1. *Birth weights (gram) of risk and control infants*

SSIDS		Controls	
Weight	Gender	Weight	Gender
3090	F	2878	M
3120	F	3040	F
3660	F	3118	M
3799	F	3300	F
3830	F	3420	M
3910	F	3515	F
3997	M	3785	M
4026	M	3855	F
4026	M	4096	F
4281	M	4550	M

Data were recorded on a 16-channel Grass model 78 polygraph (Grass Instruments Co., Quincy, MA) and simultaneously stored on a 14-channel analog tape recorder (Honeywell, Test Instruments Division, Denver, CO), together with a time code (Systron Donner Corp., Concord, CA). A detailed description of these methods have been published elsewhere (11).

ANALYTIC METHODS

ACQUISITION AND STORAGE OF SIGNALS

The R wave of the ECG signal was identified by an electronic peak detector. Its time of occurrence was determined by a crystal clock, with an accuracy of 2.0 msec. Intervals between successive R waves (R-R intervals) were calculated on a PDP-12 computer (Digital Equipment Corp., Maynard, MA) and stored on digital tape, together with digitized values of the other physiologic measures (*cf.* Ref. 9).

Each minute of the record was coded by trained personnel into quiet sleep (QS), active sleep (AS), waking (AW), or transitional state (Tr) (this latter category represents an indeterminate collection of states), using state criteria and decision-making rules described earlier (9). The state scorers were trained to achieve a percentage agreement on state selection of 80%. The criteria for state selection were similar to those described by Anders *et al.* (1), but, in addition, applied quantifiable values to each sleep parameter across an age range of 1 week to 6 months. The state codes were stored in a file on a digital tape for correlation with heart rate and variability data.

ASSESSMENT OF HEART RATE AND VARIABILITY

For each minute, the heart rate value was calculated by measuring the median R-R interval during that minute, and converting this value to beats/min. The interquartile range of the intervals was used to measure the cardiac variability. For each minute, the interquartile range was obtained by computing the absolute value of the difference between the first and third quartile limits of R-R interval lengths, and dividing this value into 60 to give the variability within that minute. Minute-by-minute values for median cardiac rate and variability over the entire 12 hr (720 successive data values) were plotted on an incremental plotter (Houston Instruments, Bellaire, TX). The median and interquartile range were chosen as appropriate measures of heart rate and variability since these two statistical procedures are relatively insensitive to occasional deviant values caused by missed beats or interfering noise.

ARTIFACT REMOVAL

The ECG signal was occasionally contaminated with artifactual noise which, if included in the calculations, would give erroneous results. Artifacts manifested themselves principally as excessively long or short intervals. To deal with this problem, we employed artifact removal procedures that have been previously described (9). The percentage of minutes of data removed due to artifact contamination ranged from 0–8%. Most contaminated data occurred in the waking state, whereas QS and AS state data were relatively unaffected. Except for those minutes of data removed because of contamination with artifacts, R-R intervals over the entire 12-hr record were used to derive results.

QUANTITATIVE ANALYSIS

Statistical assessment of the influence of age, state, and group (*i.e.,* risk or normal) on heart rate and heart rate variability was achieved by subjecting heart rate and variability values to analysis of variance procedures (partially nested and partially crossed design, ages and states fixed, subjects random and nested within group) and Duncan's multiple range tests.

RESULTS

HEART RATE

Individual median heart rate values for the risk and normal infants are listed in Table 2 for each age and state; averaged values are shown in Figure 1 as a function of age. The normal group includes eight infants considered previously (9), and two infants whose data have recently been compiled. The left graph in Figure 1, showing SSIDS and normal heart rates plotted as a function of age over all states, demonstrates that the heart rates for the two groups were very similar up to 3 months of age. A mixed model analysis of variance (ANOVA) indicated that the SIDS group had a significantly higher overall heart rate pattern at this age ($P < 0.05$). However, further analysis pointed to a clearly significant group difference in the waking state ($P < 0.025$), while the group difference in quiet sleep alone, and in the combined sleep-related states, reached only a 10% level of significance. Thus, waking heart rate made the primary contribution to the differing heart rate pattern at 3 months of age. No significant group differences were found at either of the later ages.

Although the group difference in the heart rate at 3 months of age in the combined sleep-related states (QS + AS + TR) was not great, distinct developmental differences between the SIDS and normal infants did emerge over all states, and in sleep alone. The left graph in Figure 1 suggests, and statistical tests confirmed, that overall the SIDS infants evidenced a more abrupt increase in heart rate at 1 month than the normal subjects, and the major component of their decline in heart rate was spread out between 1 and 4 months, whereas the normal infants' heart rate decline was restricted mainly to the 1- to 3-month period, with the 2- to 3-month drop being particularly rapid.

The right graph in Figure 1 shows SSIDS and normal heart rates in sleep (QS + AS + TR) alone. It is apparent that the same general profile of developmental differences was preserved with the deletion of wakefulness. Specifically, the mixed model ANOVA, followed by Duncan's tests, revealed a significant increase in normal heart rates in sleep between 1 week and 1 month ($P < 0.05$), while the decrease in sleeping heart rate between 1 and 2 months was not significant ($P > 0.05$). The most salient finding was a precipitous drop in the normal heart rates between 2 and 3 months ($P < 0.001$), with only small decrements occurring between 3 and 6 months. In contrast, the heart rates during sleep in the SIDS group showed a sharp increase between 1 week and 1 month ($P < 0.005$), an equally sharp decrease between 1 and 2 months and, finally, a significant decrease between 3 and 4 months ($P < 0.05$).

Figure 2 shows graphs of heart rates for the normal and risk infants plotted as a function of age and state. Inspection of these graphs indicates that the different group pattern of heart rate decline in the 2- to 3-month age period was also present within each state. Statistically, the normal infants showed a significant drop in heart rate between 2 and 3 months in each state ($P < 0.005$), while the SIDS infants showed no significant drop during this period in any state ($P > 0.05$).

There was greater state differentiation between the two groups

Table 2. *Individual heart rate values*

	SSIDS						Controls					
Subjects	1 wk	1 mo	2 mo	3 mo	4 mo	6 mo	1 wk	1 mo	2 mo	3 mo	4 mo	6 mo
QS												
A	127.5	133.3	125.7	117.8	117.8	116.2	114.6	141.0	132.0	124.4	121.1	102.6
B	134.0	133.3	127.6	121.6	111.9	103.1	126.0	145.9	124.9	125.9	112.7	109.4
C	128.7	138.0	129.3	123.4	116.6	114.0	131.8	135.2	129.0	116.1	110.5	112.8
D	125.3	127.8	124.9	117.4	107.5	104.2	139.8	138.2	125.1	115.1	116.5	109.5
E	145.2	159.0	141.0	149.4	134.5	121.2	136.3	137.4	138.5	122.7	122.0	114.7
F	124.9	142.0	142.6	127.3	126.9	119.5	114.5	137.3	136.4	114.5	134.5	121.1
G	143.3	147.0	130.5	121.7	122.5	130.3	121.5	134.7	123.5	120.4	124.3	118.8
H	129.0	140.0	119.1	122.9	117.6	118.6	139.8	137.9	128.2	111.3	105.9	118.9
I	116.3	139.0	125.6	137.4	128.0	117.5	120.8	127.5	125.2	104.3	104.5	116.7
J	121.1	131.8	131.8	110.2	103.1	121.2	138.9	129.8	130.1	118.7	113.6	112.3
Mean	129.5	139.1	129.8	124.9	118.6	116.6	128.4	136.5	129.3	117.3	116.6	113.7
SD	9.1	9.0	7.2	11.1	9.7	8.1	10.2	5.2	5.1	6.5	9.1	5.6
AS												
A	125.2	133.6	128.9	122.9	121.0	121.1	117.8	144.0	139.5	127.8	125.1	107.7
B	138.0	135.5	130.8	125.9	115.2	111.1	131.3	145.7	126.1	132.8	117.9	115.7
C	130.4	139.7	131.6	125.4	122.5	120.1	133.4	138.1	132.0	120.2	116.9	116.0
D	132.7	126.7	125.4	121.1	108.5	111.6	143.1	140.9	127.2	119.4	119.8	118.3
E	147.0	157.9	139.3	148.5	134.7	124.7	143.3	138.7	137.3	126.6	126.1	120.7
F	124.2	142.3	140.7	125.1	125.8	117.4	131.2	136.5	138.9	119.0	138.7	128.8
G	145.4	150.0	131.9	124.6	127.7	132.8	128.4	132.2	126.3	122.0	125.9	121.7
H	132.4	141.8	119.3	122.4	117.7	119.4	147.7	139.5	132.3	116.6	111.0	122.4
I	129.6	142.2	131.4	141.1	133.0	125.6	125.3	128.0	127.2	112.9	111.9	122.6
J	129.8	138.2	131.6	120.5	111.9	127.1	142.5	132.8	130.8	122.2	118.1	117.5
Mean	133.5	140.8	131.1	127.8	121.8	121.1	134.4	137.6	131.8	122.0	121.1	119.1
SD	7.7	8.6	6.1	9.3	8.7	6.8	9.5	5.5	5.2	5.8	8.2	5.6
AW												
A	170.6	157.0	168.5	169.0	152.7	147.2	159.8	184.2	173.5	152.6	151.7	134.8
B	164.2	161.2	160.0	144.9	128.0	128.6	164.2	168.2	179.8	161.3	153.2	160.3
C	155.7	167.9	155.7	171.2	152.0	135.8	151.3	159.8	162.6	147.3	143.4	122.6
D	162.2	170.0	163.4	164.0	161.9	163.5	174.4	179.5	172.3	146.4	138.7	146.0
E	169.7	183.5	166.8	174.8	151.6	143.9	156.3	159.1	166.4	149.7	145.6	162.8
F	154.5	144.6	178.1	157.1	160.7	144.7	149.7	151.6	162.7	149.8	159.8	158.7
G	176.4	184.4	172.1	150.9	195.7	161.2	157.5	173.1	150.7	146.8	161.9	140.0
H	171.4	161.8	156.5	170.6	148.9	143.0	191.0	170.6	169.1	162.4	162.7	167.5
I	151.3	171.5	168.9	177.0	149.1	154.4	162.3	154.4	158.9	137.0	150.0	139.8
J	159.3	157.0	133.5	146.6	146.8	152.9	155.5	169.0	160.5	154.3	146.9	150.1
Mean	163.5	165.9	162.4	162.6	154.7	147.5	162.2	166.9	165.7	150.8	151.4	148.3
SD	8.4	12.3	12.3	11.9	17.1	10.8	12.3	10.7	8.4	7.5	8.1	14.2
TR												
A	130.1	135.8	133.4	127.3	130.5	126.7	137.9	156.6	142.3	129.0	127.0	106.1
B	134.4	133.9	132.3	126.5	115.0	111.8	131.6	149.4	142.2	131.3	116.4	116.5
C	127.9	144.8	132.2	136.4	123.6	116.7	137.5	141.7	142.0	121.0	117.6	117.8
D	131.1	129.6	125.7	120.2	115.3	109.8	137.9	144.6	149.0	117.0	124.6	122.6
E	147.9	158.5	145.6	158.9	136.4	127.2	135.4	138.0	136.1	125.4	125.3	118.0
F	127.5	143.2	149.2	127.6	132.2	123.5	127.2	142.7	139.0	116.7	138.3	125.5
G	143.7	153.2	128.0	123.8	124.8	132.0	123.7	150.6	127.1	126.2	125.2	121.0
H	137.3	141.8	122.2	121.6	120.3	125.2	161.1	144.3	135.2	113.9	109.9	126.2
I	125.9	143.6	128.3	142.6	140.2	122.3	121.4	127.9	129.8	111.1	112.4	125.8
J	128.6	135.7	132.5	113.7	111.9	127.7	142.7	135.9	130.6	123.6	122.0	123.8
Mean	133.4	142.0	132.8	129.9	125.0	122.3	135.6	143.2	137.3	121.5	121.9	120.3
SD	7.4	8.9	8.5	13.0	9.6	7.3	11.3	8.1	6.9	6.7	8.2	6.1

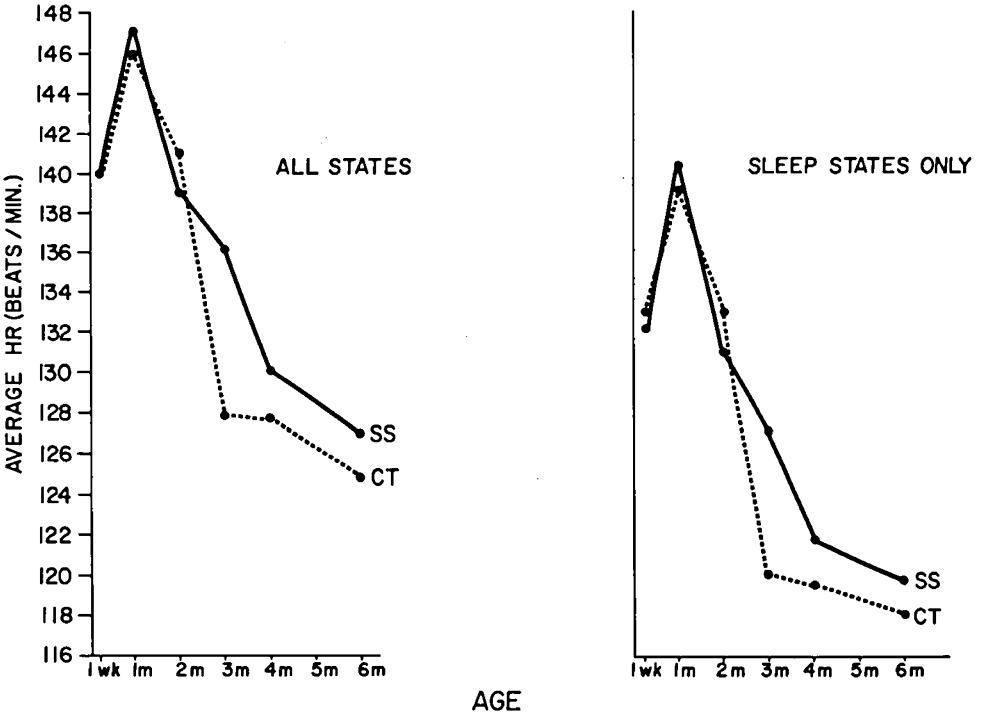

Fig. 1. Heart rates of control (CT) and SSIDS(SS) infants over time for all states combined (left) and sleep states only (right). Note the more rapid decrease in heart rate between 2 and 3 months of age for the control infants.

with respect to the initial 1 month rise in heart rate. For risk infants, there were significant heart rate increases between 1 week and 1 month in QS, AS, and TR ($P < 0.005$, $P < 0.01$, and $P < 0.05$, respectively); for normal subjects, the only significant increase during this period occurred in QS ($P < 0.05$). Similarly, the risk infants showed significant heart rate decreases between 1 and 2 months in QS, AS, and TR ($P < 0.005$, $P < 0.001$, and $P < 0.01$, respectively), while the normal infants showed a more moderate 1- to 2-month heart rate drop in QS and AS ($P < 0.05$).

HEART RATE VARIABILITY

Summary individual heart variability values for the two groups of 10 risk and 10 normal infants are listed by age and state in Table 3; values averaged over individuals are depicted in Figure 3. As in the case of heart rate, the values for the two groups are relatively similar for all but one age. In this instance, however, the apparent group difference is at 1 month, rather than at 3 months, and it is not statistically significant ($P > 0.05$).

AS was the only state in which both the SIDS and normal groups manifested significant differences between ages in heart rate variability. Specifically, the normal infants' variability dropped significantly between 1 week and 4 months ($P < 0.005$), while the risk infants' variability decreased significantly between 1 week and 1 month ($P < 0.05$), increased between 1 and 3 months ($P < 0.05$), and declined between 4 and 6 months ($P < 0.005$).

The developmental patterns in heart rate variability also differed between the two groups in the waking state. In particular, cardiac variability in the SIDS infants did not undergo any significant changes with age ($P > 0.05$), whereas variability in the normal infants increased significantly between 1 week and 2 months ($P < 0.05$), leveled off between 2 and 3 months, and then decreased significantly between 3 and 6 months of age ($P < 0.05$).

DISCUSSION

The principal finding in this study is that the development of heart rate differs in infants at risk for sudden infant death from that in normal infants. This difference is manifested as higher heart rates for SSIDS at 3 months of age, particularly during the waking state, and as a more abrupt increase in heart rate during the months after birth, with a more gradual decrease in rate from 1–4 months of age. Moreover, heart rate variability in SSIDS does not follow the increase seen in normals during the 1-week to 2-month age period in the awake state.

Although the SIDS and normal groups have very similar overall heart rate values at birth, the SSIDS infants have a more abrupt heart rate increase during the month after birth, followed by a slower decrease in heart rate after that point. It is important to note that the delay in heart rate decrease was greatest at the peak risk period for SIDS, which lies between 2 and 3 months of age. The delay in declining heart rate during this period thus suggests that infants at risk for SIDS may have delayed maturation of some crucial aspect of cardiac control.

The heart rate difference seen between the normal and risk groups at 3 months of age is paralleled by differences in other autonomic functions. For instance, Hoppenbrouwers *et al.* (13), have found that respiratory rate is higher in these infants at 3 months, and in the first week of life. In addition to this difference in respiratory activity, the degree of coupling between cardiac variability and respiration (a measure of sinus arrhythmia) has been shown to be lower at specific periods in SSIDS infants (10). All of these measures, respiratory rate, sinus arrhythmia, and heart rate, are dependent on, or related to, the degree of vagal tone.

The abnormal behavior in this cluster of variables suggests that some dysfunction has occurred in the development of parasympathetic control. We may gain some insight into the nature of the possible deficit by noting deviations from the normal development of these parameters.

We recall that heart rate in the normal infants rises significantly from the newborn period to 1 month in QS. An increase in heart rate after the newborn period is also seen in some animals. Assali *et al.* (3) and Woods *et al.* (25) have demonstrated that heart rate in the lamb rises sharply from stable fetal values during the first few days after birth, and subsequently declines until it approaches adult values at 6–8 weeks. Similar postnatal heart rate increases have also been found in rats (24). Although the time course of elevated heart rate differs in humans, and the mechanisms respon-

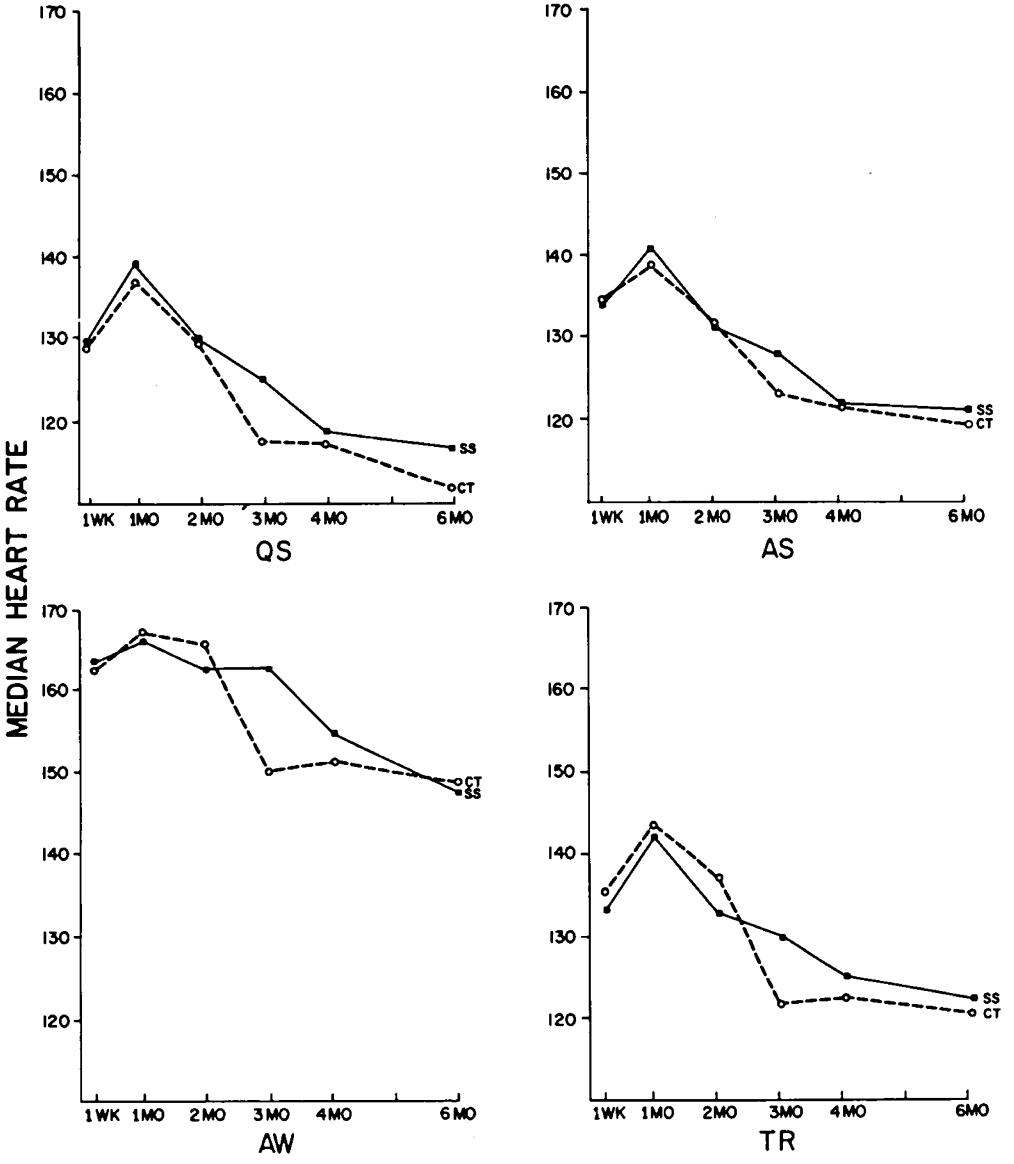

Fig. 2. Average median rates for normal and SSIDS infants by sleep state over the first 6 months of life.

sible for the increased rate have not yet been precisely determined, it seems reasonable to suggest that a variety of newborn animals respond to the stress of immediate postnatal life by increased rate, and that this high rate then falls as parasympathetic control develops, and as intrinsic cardiac conducting mechanisms mature (7, 12). However, infants at risk for SIDS demonstrate a far more marked pattern of increased postnatal heart rate with sharp increases between 1 week and 1 month in both QS and AS; moreover, there is a delay in their heart rate decline in all states.

Developmental differences in cardiac variability are also apparent between the two groups in the waking state. Specifically, cardiac variability in the normal group increases from the first week of life to the second month, and then falls from the third to the fourth month period. This quadratic component of development is not observed in the risk group, suggesting that the cardiac response to a variety of inputs is disturbed in these infants.

It is possible that the mechanisms of parasympathetic control are inadequately developed in the risk group, and that, for example, the retarded decline in heart rate after the first month results from immature development of that system. We have found in an accompanying study of these same infants (10) that the proportion of cardiac variability that is attributable to respiration develops differently in SSIDS infants than in normal infants during the first 6 months of life. A variety of causal mechanisms for this different development can be postulated. However, impairment of autonomic activity in the vagus provides a particularly plausible explanation. Salk *et al.* (21), in reporting a single case study of cardiac response to stimulation, describe an extreme lack of control in the response of an infant who subsequently died of SIDS, and suggest immaturity of autonomic control in this subject.

It is possible that immaturity of autonomic control, and in particular of parasympathetic control, may characterize these risk infants. However, whether these deficits result from receptor, integrative, or motor dysfunction is still not known.

There is evidence that victims of SIDS exhibit a variety of pathologic signs indicating that the infants have been suffering from long standing hypoxia (15–19). SIDS victims at autopsy showed signs of chronic hypoventilation; many exhibited greater heart weights, increased muscle tissue in pulmonary arteries, increased volume of chromaffin cells in the adrenal medulla, and abnormal retention of brown fat cells about the adrenals. Moreover, SIDS victims showed abnormal volumes of cells in their carotid bodies.

A defect in the receptor mechanisms for oxygen and carbon dioxide, as implied by abnormalities in carotid body receptors, might lead to chronic hypoventilation. Increased cardiac rates

Table 3. *Individual heart rate variability values*

	SSIDS						Controls					
Subjects	1 wk	1 mo	2 mo	3 mo	4 mo	6 mo	1 wk	1 mo	2 mo	3 mo	4 mo	6 mo
QS												
A	4.2	3.9	5.5	8.5	6.6	6.6	3.1	3.8	3.0	4.9	2.2	5.5
B	7.2	7.2	6.4	6.3	10.5	9.5	9.0	4.7	6.3	6.8	7.0	6.3
C	3.3	4.1	4.5	6.3	4.5	4.4	4.9	4.6	3.3	3.9	5.6	4.8
D	3.9	6.4	5.6	5.8	7.5	8.3	6.1	4.6	5.5	7.1	6.5	6.2
E	3.3	3.3	5.6	5.1	4.5	4.9	5.6	5.3	2.5	3.1	3.4	5.8
F	8.2	12.5	4.8	6.1	8.9	7.1	5.3	6.6	6.5	7.9	7.3	8.0
G	4.2	3.7	5.0	4.6	5.3	3.8	7.3	5.8	6.8	6.3	6.5	5.4
H	3.6	4.1	7.8	6.6	7.1	5.6	10.0	7.3	9.8	8.0	6.1	5.7
I	6.0	4.5	4.7	8.6	6.0	5.7	6.4	6.5	11.1	7.8	7.6	7.4
J	8.2	5.7	11.0	8.2	9.1	6.5	6.3	4.5	4.1	5.6	5.5	5.7
Mean	5.2	5.5	6.1	6.6	7.0	6.2	6.4	5.4	5.9	6.1	5.8	6.1
SD	2.0	2.8	2.0	1.4	2.0	1.8	2.0	1.1	2.9	1.7	1.7	1.0
AS												
A	7.2	7.1	7.0	9.1	7.6	4.8	8.7	9.3	7.3	9.1	4.7	8.0
B	12.6	10.9	9.8	12.1	12.6	10.5	13.7	8.3	12.3	11.3	10.9	8.5
C	8.4	8.1	8.5	10.6	8.8	6.4	8.8	9.4	10.5	8.3	9.0	7.8
D	11.1	10.4	8.6	8.7	9.8	8.7	12.7	11.4	12.5	11.4	8.7	9.2
E	12.1	10.6	12.4	11.0	7.4	7.1	10.5	9.6	9.3	8.6	6.0	7.1
F	12.5	11.4	11.4	10.2	11.7	9.3	9.8	11.5	12.0	12.0	10.4	9.4
G	9.9	11.3	12.3	10.0	9.2	8.3	12.9	13.8	11.9	13.2	12.8	9.6
H	11.7	9.2	12.5	12.5	11.5	9.4	16.0	12.9	15.7	12.5	10.4	8.1
I	11.5	8.0	10.9	11.4	9.7	9.6	15.4	15.9	12.4	13.1	12.3	10.4
J	19.2	14.1	14.2	18.8	16.0	11.2	8.3	10.9	8.5	7.8	10.1	6.4
Mean	11.6	10.1	10.8	11.4	10.4	8.5	11.7	11.3	11.2	10.7	9.5	8.5
SD	3.2	2.1	2.2	2.9	2.6	2.0	2.8	2.4	2.4	2.1	2.6	1.2
AW												
A	8.7	8.0	6.5	9.3	8.4	7.3	11.9	9.5	9.3	11.9	8.9	12.9
B	13.5	12.3	14.0	17.5	12.3	17.1	11.8	13.3	15.2	15.6	17.2	12.9
C	10.5	10.2	11.9	15.4	12.1	14.7	7.8	12.4	14.1	12.6	11.4	11.8
D	9.9	12.0	11.6	10.2	7.7	7.5	11.1	12.8	14.6	14.7	12.9	15.4
E	8.7	9.4	9.1	12.7	7.6	8.1	10.6	7.6	8.4	9.0	5.8	8.2
F	15.8	10.7	13.5	9.7	10.8	13.4	11.8	13.4	17.2	13.7	13.2	8.9
G	7.9	10.3	13.3	11.6	14.9	12.7	11.4	16.6	21.1	26.4	14.1	11.3
H	11.3	12.4	17.0	12.9	14.7	14.8	13.1	14.0	14.6	14.4	12.0	11.8
I	11.5	10.9	14.8	12.0	15.7	11.7	12.6	10.7	13.2	10.0	10.9	9.8
J	12.4	13.7	11.8	14.9	23.2	16.2	10.7	13.7	12.6	12.5	11.8	12.9
Mean	11.0	11.0	12.4	12.6	12.7	12.4	11.3	12.4	14.0	14.1	11.8	11.6
SD	2.4	1.7	3.0	2.7	4.7	3.6	1.5	2.5	3.6	4.8	3.0	2.2
TR												
A	6.8	6.4	9.1	14.5	12.1	6.3	11.1	9.0	7.6	7.9	5.0	8.4
B	12.8	8.9	12.9	14.1	14.2	14.4	11.8	8.9	15.7	12.4	10.7	8.9
C	6.6	7.7	7.8	14.7	11.6	9.2	8.0	11.5	12.7	13.1	10.1	11.1
D	6.9	11.2	8.6	12.0	10.2	11.3	9.9	11.0	13.0	12.6	11.9	13.8
E	9.6	5.7	10.3	9.8	9.0	10.7	9.8	9.4	10.0	5.7	9.2	7.5
F	12.7	11.4	10.4	8.8	13.1	12.0	8.5	10.8	13.9	12.4	12.7	12.1
G	6.8	5.8	8.1	9.1	10.9	8.5	13.0	16.4	11.7	20.9	10.0	8.9
H	7.1	7.6	13.7	11.6	14.9	11.2	15.4	14.3	18.1	11.5	9.1	10.5
I	7.9	5.6	9.2	14.2	13.1	15.0	11.4	13.4	16.7	15.8	20.0	18.2
J	17.0	12.3	13.1	17.3	21.1	13.6	10.0	12.2	7.1	9.5	13.0	8.6
Mean	9.4	8.3	10.3	12.6	13.0	11.2	10.9	11.7	12.7	12.2	11.2	10.8
SD	3.6	2.6	2.2	2.8	3.4	2.7	2.2	2.4	3.7	4.2	3.8	3.2

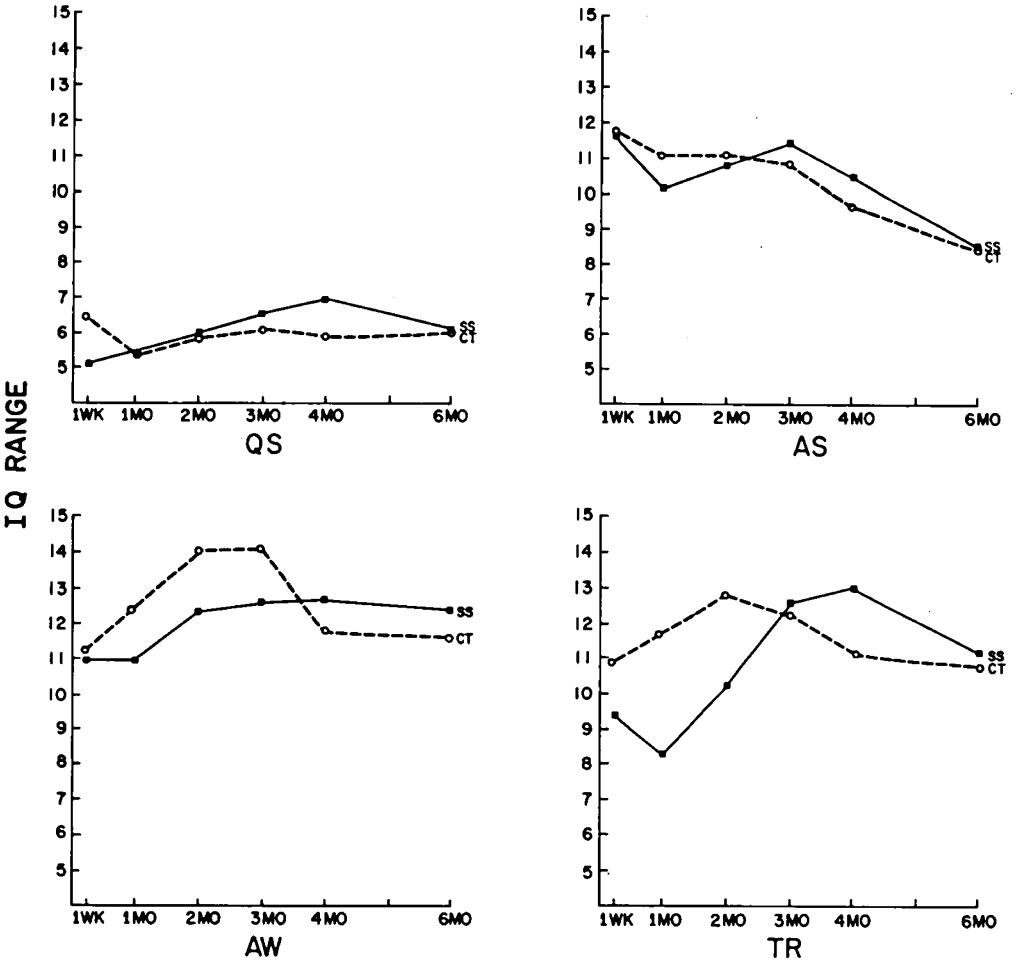

Fig. 3. Average interquartile range of R-R intervals for normal and SSIDS infants by state over the first 6 months of life.

would be consistent with an interpretation of hypoxia. These increased cardiac rates are pronounced in the awake state at 3 months of age, perhaps reflecting the increased demand for oxygen in waking relative to sleeping states. Increased respiratory rates would also be expected from such hypoxia.

The developmental differences in heart rate that appear between the SIDS and normal groups as early as 1 week of age, along with differences in other autonomic functions, do suggest that chronic hypoxia, and the autonomic control impairment that might result from such hypoxia, may play a significant role in sudden infant death.

CONCLUSION

Postnatal development of heart rate and variability during sleep and waking periods was examined in 10 normal infants and 10 infants at risk for sudden infant death. The infants were recorded for 12 hours at 1 week, and again at 1, 2, 3, 4, and 6 months of age. Heart rate values were higher in the risk group at 3 months of age, particularly in the awake state. The risk group's heart rates also increased more markedly during the first month after birth in both quiet and active sleep. Heart rates in both groups declined after the age of 1 month in every state; however, the normal group's heart rate values fell sharply between 2 and 3 months, whereas the risk group's values declined more gradually between 1 and 4 months. In the awake state, heart rate variability in the risk group did not follow the increase seen with the normals during the 1-week to 2-month age period.

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