Shape #3

If $A^r_1 \sim B^r_3$ and the turbulent portion of the signal is of relatively low frequency, i.e., $r_3 \sim r_4$, the autocorrelation appears as in figure 2b. If the two time scales are not readily apparent, as in figure 2b, $r_3$ is the correct turbulent scale and the function's width is determined on the axis at point P. The pattern in figure 2b is differentiated from that of figure 1b, which has no information about constriction, because figure 2b displays a sharp, triangular peak with straight sides. Figure 2b is observed in tortuous arteries with minimal occlusion and was produced by the kinked artery previously illustrated in text (fig. 5).

Shapes #4 and #5

Additional cases when turbulence and pulse wave amplitudes are the same order of magnitude are shown in figures 2c and 2d. In both cases, $r_3$ must be differentiated from $r_4$ and determined at the point P where the autocorrelation curvature is at a maximum.

Shape #6

Finally, if the turbulence is minimal and hence $A^r_1 >> B^r_{3}$, there is no flow information available. In this case $r_3$ merely reflects the properties of the pulse wave and the data must be discarded. This case would appear as in figure 1b.

It is evident from the above that if a kink is sufficiently turbulent, the autocorrelation can be interpreted correctly to yield the appropriate turbulent time scale. Visual observation of the oscilloscope signal trace is necessary initially in deciding whether there is excessive ambient noise in the signal. Ambient noise is not distinguished from turbulence in the autocorrelation, but is readily distinguished in oscilloscope form. When ambient noise is excessive, especially if the turbulent signal is of very low amplitude, the data must be discarded (see fig. 2, text).

References


The Role of the QT Interval in the Sudden Infant Death Syndrome

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AND RICHARD R. LIBERTHSON, M.D.

SUMMARY To evaluate the role of QT interval prolongation in the genesis of the sudden infant death syndrome (SIDS), the postresuscitation electrocardiograms of 21 aborted SIDS infants were reviewed. The infants had been found apneic, cyanotic, limp and unresponsive during sleep and required vigorous physical stimulation and mouth-to-mouth resuscitation. Three subsequently experienced repeat similar episodes from which they could not be resuscitated.

THE SUDDEN INFANT DEATH SYNDROME (SIDS) is the leading cause of infant death between the ages of 1 and 12 months, and claims approximately 10,000 lives in the United States each year.1-4 Because these deaths are sudden, usually occur outside of the hospital, are observed only rarely by physicians, and have few associated autopsy findings,4-6 we still know very little about their etiology. Explanations for these deaths include asphyxia, laryngeal or bronchospasm, infection, occult endocrine, neurologic or renal disease, apnea, and autonomic dysfunction.4-11 In addition, QT interval prolongation with fatal ventricular arrhythmia has been proposed as a cause for SIDS.12-17 However, because SIDS victims themselves rarely have electrocardiographic evaluation before death, recent workers have examined relatives of SIDS infants because it is known that QT prolongation in some families is genetically transmitted.18-20 Based on this indirect approach, these studies suggest a causal relationship between QT prolongation and the SIDS.21,22 In this report, we examine the postresuscitation QT intervals of aborted SIDS infants, including three who subsequently died from actual SIDS.

Methods

Between 1974 and 1976, 21 survivors of the aborted SIDS were referred to the Massachusetts General Hospital...
(MGH) for evaluation and treatment. These infants were found apneic, cyanotic, limp, and unresponsive during sleep, and required vigorous physical stimulation and mouth-to-mouth resuscitation. No infant required electrical defibrillation. All infants had complete history and physical examination as well as laboratory evaluation including complete blood count, urinalysis, measurement of serum calcium, phosphate, magnesium, glucose, and amino acids, evaluation of urinary amino acids, chest and skull radiography, electroencephalograms, and continuous twelve-hour pneumogram recordings to evaluate respirations and heart rate. All known causes of sudden death were ruled out.

The 21 aborted SIDS infants included three infants who subsequently experienced similar episodes from which they could not be resuscitated. These three became actual SIDS babies. At autopsy, no cause of death was found in them. In six of the 21 aborted SIDS infants, the aborted episode was so severe that transient hypoxic damage following resuscitation was present, and 10 resuscitated infants had subsequent prolonged monitored apneic episodes lasting longer than 20 seconds which required bag and mask resuscitation and vigorous physical stimulation. Forty-six normal control infants matched for sex and age were selected from our ambulatory clinic population, and normal values for the QT interval were selected from the literature. All infants had standard 12 lead electrocardiograms taken within 48 hours of the aborted SIDS episode. These electrocardiograms were recorded while the infants were resting quietly using Marquette series 2,000 electrocardiographic recorders at 25 mm/sec paper speed. QT intervals were determined by averaging six complexes in lead II. Care was taken to avoid including U or P waves, and the QT intervals were measured from the onset of the Q wave (or the R wave if no Q wave was present) to the end of the T wave (junction with the isoelectric baseline). Only complexes with normal QRS and T waves were evaluated and no patient was taking medication which is known to alter the QT interval at the time of recording. Heart rate was calculated from the same complexes in which QT intervals were measured by averaging six R-R intervals in lead II. All QT intervals were corrected for heart rate (QTc) by dividing the QT by the square root of the R-R interval. Measurements were made on four separate occasions by two observers without knowledge of whether the patient was an aborted SIDS or a normal control. The QTc intervals of the aborted patients were plotted on the nomogram of QTc intervals derived by Alimurung et al.

Results

The general characteristics of these infants are shown in table 1. Physical and neurologic examinations were normal except in the six patients with transient postresuscitation hypoxia, and laboratory data and chest and skull radiographs were also normal. The mean serum calcium level was 9.9 mm/100 ml (range 9.0 to 10.9). Electroencephalograms were normal except in two of the infants who had transient hypoxic changes. Pneumogram recordings in ten infants demonstrated episodes of prolonged sleep apnea (greater than 20 seconds).

The QT intervals in aborted SIDS ranged from 0.20 to 0.33 seconds with a mean of 0.26 seconds compared to our normal infants in whom QT intervals ranged from 0.20 to 0.31 seconds with a mean of 0.25 seconds. The QTc for the aborted SIDS ranged from 0.35 to 0.42 seconds with a mean of 0.39 seconds compared to the QTc for normal controls which ranged from 0.34 to 0.43 seconds with a mean of 0.39 seconds. The QT intervals of our three infants who subsequently died from actual SIDS were 0.27, 0.28 and 0.28 and their calculated mean QTc was 0.39 seconds. Reports in the literature of 861 normal infants reveal a mean QTc of 0.39 seconds. The differences among the QTc in the aborted SIDS patients, the actual SIDS, our normal control infants, and reported normals in the literature were not statistically significant; and the QTc intervals in the aborted and actual patients were within the normal range when plotted on Alimurung's nomogram. To rule out possible age related QT variations, we compared our study and control populations by age as shown in table 2 and did not find significant differences in QTc intervals between these groups.

Discussion

While the etiology for SIDS is still unclear, review of our infants with aborted SIDS as well as three infants who subsequently were actual SIDS, reveals that QT interval prolongation is not present in these infants following resuscitation. Thus, unlike previous reports, which demonstrated QT prolongation in relatives of SIDS and thus suggested that QT prolongation is related to SIDS, we could not demonstrate QT prolongation in the aborted or actual SIDS infants themselves. In addition, as shown in table 2, we could find no age subgroup in which the QTc intervals differed from controls. Further strong evidence against involvement of QT prolongation in SIDS derives from the fact that the presumed mechanism for death in QT prolongation is ventricular fibrillation which in nearly all patients requires defibrillation; in none of our resuscitations was defibrillation needed. It has been suggested that QT prolongation may be related to sleep or activity, and it is therefore possible that our single ECG samples did not detect intermittent QT prolongation; however, evaluation of this requires continuous long-term ECG recordings.

While it is conceivable that aborted SIDS is not the same
entity as actual SIDS and therefore that our study infants are not representative of the actual SIDS population, we believe this is not so because three of our study infants subsequently died of actual SIDS, six had significant clinical hypoxia demonstrating the severity of their aborted episodes, and ten infants had pneumogram documentation of prolonged, severe, and clinically significant apnea, which we believe could have precipitated actual SIDS.

We conclude from our data that demonstrable QT prolongation is not present in resuscitated aborted SIDS, and therefore is unlikely to be a major cause for the sudden infant death syndrome.

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References


Table 2. QTc Intervals in Aborted SIDS and Normal Control Infants by Age

<table>
<thead>
<tr>
<th>Age</th>
<th>Aborted SIDS</th>
<th>Normal control infants (MGH)</th>
<th>Normal controls literature</th>
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<tr>
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<td>.35-.42</td>
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<td>Total</td>
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<td>.39</td>
<td>.35-.42</td>
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</table>
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