To Elliot,

with many thanks for all your help over the last 4 years

David Southall

HEART RATE AND RHYTHM PATTERNS IN THE FETUS, NEONATE AND CHILD

A Thesis submitted for the degree of
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BY

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ABSTRACT

To determine normal values recordings of heart rate and rhythm were made on healthy fetal subjects, infants and school children.

Five-minute recordings of the fetal heart beat from 934 expectant mothers showed episodes of bradycardia <100/minute, from 5 to 30 seconds in duration, in 12, tachycardia >180/minute, from 30 to 90 seconds in duration, in 5 and premature beats in 12.

Thirty-three of 3383 (1%) unselected newborn infants showed disorders of cardiac rhythm or conduction on a standard electrocardiogram. Twenty-six showed premature beats, 5 supraventricular tachycardia, and 2 the Wolff-Parkinson-White syndrome. Twenty-four-hour recordings of the electrocardiogram on 15 of 26 infants with premature beats showed in five cases additional supraventricular or ventricular tachycardia. Disorders of cardiac rhythm and conduction could not be detected after 12 weeks of age in 23 of 27 subjects followed into early childhood.

Twenty-four-hour recordings were also made on 134 newborn infants without cardiac arrhythmias on a standard electrocardiogram. The mean highest heart rate was 175±19 (S.D.) and lowest 93±12 (S.D.). Twenty-five had junctional escape rhythms. Premature beats were present in 19 infants; in six >1/hour and in one >12/hour. Sinus pauses were found in 51

of a randomly selected subgroup of 71 of these infants.

Twenty-four-hour recordings on 104 seven to eleven year old children, showed a mean highest heart rate of 164±17 (S.D.) and mean lowest rate of 56±6 (S.D.). The maximum duration of heart rates <55/minute was 40 minutes. Forty-one children had junctional escape rhythms with a maximum duration of 25 minutes. Nine had PR intervals ≥0.20 seconds including 3 with Mobitz Type I second degree A-V block. Nineteen had isolated premature beats (<1/hour). Sixty had sinus pauses with a maximum duration of 1.36±0.23 (S.D.) seconds.

Variations in heart rate and rhythm, hitherto considered abnormal, are present in healthy children.

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INTRODUCTION

The advent of techniques for long term recording of the electrocardiogram (ECG), have enabled clinicians to assess with more accuracy, the characteristics of cardiac arrhythmias in children. The diagnosis of abnormality, however, requires that the range of normal is precisely known. This thesis describes and discusses the frequency and characteristics of heart rate and rhythm variations found in children of different ages.

The first section deals with a study of fetal heart rate and rhythm in a population of expectant mothers without complications of pregnancy. The second section examines disorders of cardiac rhythm and conduction found by taking standard ECG recordings on an unselected population of newborn infants and investigates in detail, using 24-hour ECG recordings, heart rate and rhythm patterns in infants with cardiac arrhythmias and also those with normal rhythm and conduction on the standard ECG. The third section examines the findings of 24-hour ECG recordings in a randomly selected population of 104 healthy primary school children.

The thesis concludes with a critical comparison of heart rate and rhythm patterns found in children of different age groups.

CHAPTER 1

DETAILS OF THE 24-HOUR RECORDING SYSTEM, CRITERIA FOR ARRHYTHMIA ANALYSIS AND STATISTICAL METHODS

1. Twenty-four hour tape recordings of the electrocardiogram

Recordings were made using a 4-channel miniature analogue tape recorder (Oxford Medical Systems - Medilog 1 series). Electrocardiogram was recorded onto channel 1 from two prejelled electrodes placed on the chest wall in a modified lead II position. On channel 4 of the recorder a crystal controlled clock transferred a 60 Hertz signal onto the tape at the same time as the electrocardiogram. Using a C120 cassette running at 2mm/second the system was designed to have $<0.5\%$ variation in speed during the recording. The signal to noise ratio of the recorder was reported to be >30dB. A type AD-20 direct recording amplifier with a frequency response from 0.15 to 100 Hertz was used to detect the electrocardiogram.

Recordings were analysed using a play back system (Oxford Medical Systems) which processed the tapes at 60 times recording speed. The electrocardiogram was transferred into an arrhythmia detector (Oxford Medical Systems) which by using R-R interval discriminants, allowed detection of heart rates or rhythms outside preset limits. The clock signal was also received by a device ("Synclock") which constantly adjusted the replay speed of the playback system to keep the reference signal within preset limits. When an arrhythmia was detected a hard copy of 40 seconds of electrocardiogram encompassing the event was produced on standard ECG paper at 25 mm/second.

The playback system also incorporated a device for producing histograms of successive R-R intervals. This

system was designed to trigger on each QRS complex. However from analysis of the trigger output it was apparent that in some infants large T waves and movement artefact were frequently and incorrectly interpreted as QRS complexes. Though this automatic counter was used (Chapter 3, page 57) the majority of heart rate analysis reported in this thesis was obtained from direct measurement of R-R intervals from hard copy printouts.

Many problems were encountered in obtaining good quality 24-hour ECG recordings:

1. Lead breakages occurred frequently, particularly at the junction of the lead wire and clip on terminal. A "checking" device was constructed and used to test the leads before each recording for short circuits and lead fractures.

2. Certain brands of cassette tapes were found to deposit an oxide coating over the head of the tape recorder preventing signal reception. These brands were discontinued. Using a cotton bud and methylated spirits the recording head and pinch wheel were cleaned after each recording.

3. The tape was easily placed to run the wrong side of the drive spindle producing a blank recording; a problem that was avoided by careful training of the technicians.

4. Tape speed variation was found to occur if recorders were not regularly serviced. Within limits, the "Synclock" system in the analyser overcame this problem by

adjusting replay speed to lock into the reference time signal. Nevertheless, a module designed to produce an alarm on replay whenever the reference clock signal varied by >10% of preset limits (Reynolds Medical) was used to identify areas of tape speed variation.

5. Poor quality ECG recordings, resulting from electrode failure, were a major problem and were found to arise in three ways.

   i) Poor contact between the ECG electrodes and the skin. To reduce the frequency of this problem the skin at the proposed site of the electrode was first cleaned using a disposable alcohol swab (steret). A drop of electrode paste was then rubbed into the area using cotton wool, the skin was dried, and the electrode applied.

   ii) Many electrodes were used before a suitable and reliable product was found. In general, micropore rather than foam electrodes retained better contact with the skin over the 24 hours of recording. The "red dot" (3M Company) adult sized electrodes consistently gave good readings but the electrode paste was prone to drying out during storage. "Medicotest" (Cambac Instruments) electrodes have an offset tab which was found to be useful when applying leads and electrodes to infants who usually have a compliant chest wall. However a fault was found in approximately 10% of these electrodes and was traced to the metal strip inside the offset tab.

This fault has since been corrected by the manufacturers and these electrodes have provided good recordings. Small electrodes, specially made for children, were tested but were found to be unsuitable even in infants. In many cases the small electrodes were found to become dislodged before the 24 hours of recording had been completed. Adult electrodes were therefore used for the majority of the 24-hour ECG recordings.

iii) Dislodging of electrodes by young children and older infants was an expected problem which was partly overcome by taping the ECG leads to the skin.

The adequacy of ECG signals being received by the tape recorder was tested by using a portable monitoring device (XM2 - Oxford Medical Systems).

6. A high proportion (approximately 10%) of the mercury/ silver oxide batteries used to power the tape recorder were found to be faulty. This generally resulted in the tape recording stopping before the end of the 24 hours. The XM2 monitor used to test the ECG signals going on to the recording head (see 5 above) has a facility to test the battery function before the recording. However owing to the discharge characteristic of the batteries this part of the XM2 monitor was unreliable in detecting faults. To overcome the problem all batteries were tested "under load" before being used in the tape recorder.

7. A number of tape recordings were found to show loss of the clock signal particularly at the beginning of the recordings. By experimenting with cassettes this problem was found to be related to faulty tracking of the tape across the recording head, resulting in attenuation of the clock signal on the fourth (lowest on the tape) channel. This problem was eliminated by redesigning the pinch wheel.

2. Criteria on the surface electrocardiogram used for the analysis of cardiac arrhythmias

The criteria adopted for the diagnosis of arrhythmias on the surface electrocardiogram (from both standard and 24-hour recordings) are shown in Tables 1-4. [1-16] Most of these criteria come from adult studies and therefore may not be appropriate for diagnosing arrhythmias in infants and children. In particular heart rate discriminants which are useful in differentiating arrhythmias in adults, may not be applicable in the young child.

The quality of atrial activity that can be measured from 24-hour recordings also varies considerably and for this reason it may not always have been possible to apply many of the criteria described in these tables.

3. Statistical Methods

Histograms of frequency distribution were shown to approximate to curves known to have a normal distribution using the Kolmogorov-Smirnov test.[17]

Chi-squared (X^2) calculations were used to test for association between random samples of qualitative data where expected values were > 5.

TABLE 1

CRITERIA FOR THE ANALYSIS OF CARDIAC ARRHYTHMIAS

1. Premature beats (non-parasystolic) [1-3]

Atrial	Junctional	Ventricular
Premature	Premature	Premature
Atrial activity precedes premature beat.	P wave may precede, be within or follow the QRS. When present the P wave is usually negative in leads II, III and AVF indicating retrograde atrial activation. When P wave precedes QRS the PR interval is short.	Atrial activity usually occurs on the ST segment or during the QRS of the premature beat. Rarely a P wave may be seen to precede the premature beat when the latter is end-diastolic in position.
QRS duration and configuration, ST segment and T wave are usually similar to sinus beats though aberration may occur.		QRS duration and configuration, ST segment and T wave are usually abnormal when compared with sinus beats.
		The coupling interval is usually constant (variation ≤ 0.08 seconds)[4] but multifocal premature beats may have variable coupling intervals and morphology.[5]
Premature beats may be conducted in 3 ways (1) normally (2) with delay (3) with aberration. They may also be completely blocked at the A-V node.		

TABLE 2

CRITERIA FOR THE ANALYSIS OF CARDIAC ARRHYTHMIAS

2. Parasystolic premature beats[6-9]

Fixed arithmetical relationship between premature beats with some missing intervals.

Coupling intervals usually vary ($>$ 0.08 seconds)

In those of ventricular origin fusion beats are strongly suggestive, though not diagnostic, of a parasystolic mechanism.

TABLE 3

CRITERIA FOR THE ANALYSIS OF CARDIAC ARRHYTHMIAS

3. Supraventricular and ventricular tachycardia[1-3,8]

Paroxysmal supraventricular tachycardia	Paroxysmal ventricular tachycardia
	Fusion beats diagnostic.
QRS duration usually similar to sinus beats.	QRS is usually prolonged with an abnormal configuration.
A-V dissociation is unusual.	A-V dissociation is frequently present (but when present does not exclude a junctional origin for the arrhythmia). 1:1 V-A conduction may occur, especially with A-V junctional tachycardia originating below the His-bundle where the P wave is often closer to the preceding rather than the following QRS complex.
Position of the P wave in relation to the QRS is variable and depends on the mechanism of the tachycardia.	P wave usually closer to preceding rather than following QRS complex.
Accompanying premature beats of supraventricular characteristics may be helpful in making a diagnosis.	Accompanying premature beats of ventricular characteristics may be helpful in making a diagnosis.
At the onset of the arrhythmia the P wave usually precedes or is superimposed on the QRS complex.	At the onset of the arrhythmia the P wave usually follows the first QRS complex.
	Occasional narrow QRS complexes indicating supraventricular capture beats may be present.

TABLE 4

CRITERIA FOR THE ANALYSIS OF CARDIAC ARRHYTHMIAS

4. Special forms of supraventricular tachycardia

1. Paroxysmal A-V junctional tachycardia[1,3,10,11]

The paroxysm is often, but not always,[12] initiated and/or terminated by a premature beat.

A prolonged PR interval at the onset of the tachycardia, though suggesting a re-entry mechanism, may not always be present.[12]

1:1 A-V association is usually present when a re-entry mechanism is responsible.

The relationship between the P waves and QRS complex may give a clue to the origin of the re-entry circuit. P waves close to the preceding QRS complex (within the first 25% of the R-R interval) suggest A-V nodal re-entry; P waves close to the preceding QRS complex (within 25-50% of the R-R interval) suggest re-entry using an accessory pathway.[13]

A single blocked P wave (as in atrial tachycardia) or missing P wave (as in ventricular tachycardia) eliminates the possibility of a re-entry tachycardia involving the A-V junctional tissue.

The detection of pre-excitation during sinus rhythm gives a clue to the mechanism of the tachycardia.

2. Multifocal atrial tachycardia[14-16]

Varying P wave morphology.

Varying PR intervals.

Irregular P-P and R-R intervals.

Isoelectric baseline between P waves.

3. Atrial flutter[1-3]

P waves have an inferior vector.

Regular, undulating pattern. No isoelectric baseline.

2:1 A-V conduction is usually present though A-V conduction may vary (usually by even numbers 1, 2 or 4:1).

Usually the heart rate is higher than in paroxysmal SVT (in adults 250-350/minute).

CHAPTER 2

HEART RATE AND RHYTHM PATTERNS IN THE FETUS

INTRODUCTION

Although frequent case histories have been reported, the incidence of cardiac arrhythmias in a healthy fetal population is unknown. Tables 5-8 describe previous case reports of fetal arrhythmias, their clinical effects, if any, on the developing baby, and their treatment. Table 5 describes premature beats, Table 6 supraventricular and ventricular tachycardia, Table 7 atrio-ventricular block and Table 8 disorders of cardiac impulse generation or conduction (the long QT and Wolff-Parkinson-White syndromes).

The principal problem encountered in diagnosing and studying a cardiac arrhythmia in the fetus before labour is the difficulty of obtaining a fetal electrocardiogram (ECG) of high quality, non-invasively from the surface of the maternal abdomen. Artefact in the form of electromyograph (EMG) signals from uterine and skeletal muscle and the presence of the maternal electrocardiogram have presented the main difficulties. The indirect diagnosis of a cardiac arrhythmia has usually been accomplished by the detection of associated heart rate or rhythm changes from phono- or echo-cardiograms. The detection of disorders of conduction or repolarisation is only possible before labour if there are accompanying disturbances of heart rate or rhythm.

PATIENTS AND METHODS

Between June 1977 and July 1978 five-minute recordings of the fetal heart beat were taken by a single technician from 934 successively presenting expectant mothers attending two antenatal clinics in West Dorset. No mother refused to participate in the study. Two hundred and sixty six were monitored at 30-35 weeks gestation and 718 were monitored at 36-41 weeks of gestation. Fifty of the latter group were also monitored at the earlier period of pregnancy. Mothers with complications of pregnancy, requiring admission to hospital, were excluded from the study. The study population included six mothers with uncomplicated twin pregnancies.

Recordings made before the onset of labour

The fetal heart rate and rhythm were measured using a unidirectional ultrasound transducer (Sonicaid). The doppler signal from the fetal heart and major blood vessels was recorded beat by beat onto an audio cassette recorder (Sony TC 207).

The technician was taught to alter the direction of the transducer to obtain the highest quality signal. To avoid possible maternal anxiety when disorders of heart rate or rhythm were encountered, a stethoscope connected to the ultrasound probe was used to listen to the signals from the transducer. Recordings were taken from mothers in the supine position. Uterine contractions were not monitored. The maternal pulse rate was measured during each recording to ensure that the signal received was not from maternal vessels.

TABLE 5

PREMATURE BEATS IN THE FETUS

Comments from previous reports in the literature

1. This arrhythmia was first detected using phonocardiography in 1930.[18]
2. The earliest diagnosis was made at 18 weeks of pregnancy.[19]
3. Usually, but not always,[20] there was no association with structural congenital heart disease.
4. Additional supraventricular tachycardia was recorded in one case.[21]
5. In one report of 61 cases, 14 were present only before labour.[22]
6. In one report, of 47 that persisted after birth, 13 were atrial, 25 junctional and 9 ventricular in origin.[22] In a second publication 23 infants showed supraventricular and 2 ventricular premature beats.[23]
7. The majority that persisted after birth disappeared within the first week of life.[18,23] Occasionally they continued into later infancy.[24]
8. High "Apgar" scores are recorded in the majority of cases.[22,23,25]
9. Premature beats have been misdiagnosed as "fetal distress" and resulted in Caesarian section.[26]
10. Blocked atrial premature beats have falsely shown the appearance of fetal bradycardia when recorded on standard fetal heart rate monitors.[27]
11. Bigeminal rhythm has also been misinterpreted by electronic fetal heart rate monitors as bradycardia.[28]
12. In one case report propranolol given to the mother abolished ventricular premature beats in the fetus.[29] Adrenaline given to the mother has been shown to increase the frequency of premature beats.[30]

TABLE 6

SUPRAVENTRICULAR AND VENTRICULAR TACHYCARDIA IN THE FETUS:

Comments from previous reports in the literature

1. Supraventricular tachycardia (SVT) in the fetus was first reported in 1933.[31]

2. A male preponderance (1.5:1.0) has been reported.[32,33]

3. Atrial flutter[34], continuous atrial tachycardia[35], paroxysmal atrial tachycardia[36], atrial fibrillation[22] and ventricular tachycardia[37,38] have all been described. Continuous atrial tachycardia and atrial flutter were most frequently reported.

4. Sometimes associated structural congenital heart disease (1 of 10 cases[33], 4 of 37 cases[32]) was present.

5. Fetal heart failure in utero (hydrops fetalis) has been described[32,33,34,39-44] with abdominal ascites[32] a prominent clinical feature.

6. Neonatal hypoglycaemia[33], placental oedema[35], high birth weight[33], low "Apgar" scores[32], birth asphyxia[42] and diuresis after birth[35] were commonly reported clinical findings.

7. Post-natal heart failure was more frequently reported than intrauterine heart failure[34], probably because of the changes in fetal circulation that occur with birth.

8. Caesarian section has been inappropriately performed in some cases of fetal SVT, without heart failure, misinterpreted as fetal distress.[33,34]

9. Associated preexcitation has been reported (see Table 8).

10. Digoxin was successful in treating neonates with congenital SVT.[32] Two mothers with fetal SVT were themselves treated with digoxin.[32] The arrhythmias failed to respond, but digoxin given to the infant after birth also failed to correct the SVT.

11. Cytomegalovirus infection[45] and amphetamine administration to the mother[46,47] were reported in association with fetal SVT.

12. Two unexplained and unexpected intrauterine deaths with preceding grossly irregular heart rhythms have been reported.[18]

TABLE 7

ATRIO-VENTRICULAR (A-V) BLOCK IN THE FETUS:
Comments from previous reports in the literature

1. The first antepartum diagnosis of this condition was reported in 1945.[45]
2. A-V block has been reported in 3 of 67,000[49] and 1 of 6,891[50] pregnancies. In a study of 68 cases of fetal arrhythmias, 6 were found to have A-V block.[22]
3. In one report, 181 of 599 cases[51] were associated with structural congenital heart disease. In the presence of heart disease the prognosis is less favourable.[22,51]
4. The lowest fetal heart rates in A-V block have been reported to vary between 27 and 80/minute.[49]
5. Fetal heart failure associated and probably directly due to A-V block has been reported.[40,52]
6. Birth asphyxia[53,54] and death in the neonatal period have been reported (53 of 599).[51]
7. A-V block has been frequently reported in association with collagen diseases in the mother.[55-58]
8. Pacemaker therapy has been required to control heart failure in the neonatal period.[53,54]

TABLE 8

PRE-EXCITATION AND THE LONG QT SYNDROME IN THE FETUS:
Comments from previous reports in the literature

1. All cases reported had associated changes in heart rate or rhythm.
2. Three of 6 cases[32], 2 of 10 cases[33], and 1 of 3 cases[52] of fetal SVT had associated Wolff-Parkinson-White Syndrome on the neonatal ECG.
3. Three cases of the Long QT Syndrome were diagnosed in the neonate following persistent bradycardia in the fetus.[59,60]
4. Subsequent sudden infant death occurred in a neonate with the Long QT Syndrome and fetal bradycardia.[60]

All recordings were analysed by producing a hard copy printout of the tape recordings using a Medilec fibre-optic writer. The heart rate was then measured over a nine beat period and the presence of premature beats noted and counted. All episodes of bradycardia <100/minute or tachycardia >180/minute were notified to the obstetrician. (Tables 9-12)

Recordings made during labour (Tables 10-12)

The electrocardiogram on three subjects with premature heart beat signals, one with tachycardia >180/minute and 4 with episodes of bradycardia <100/minute on the antepartum recordings were monitored continuously during labour using a scalp electrode and fetal ECG monitoring system (Hewlett Packard). A connection allowed the fetal ECG to be recorded continuously onto a 24-hour ECG recorder throughout labour.

Recordings made after birth (Tables 10-12)

A single 24-hour tape recording of the ECG was made at between 1 and 7 days of age from 8 subjects who had demonstrated premature beats and 7 who had shown episodes of bradycardia <100/minute before labour. When premature beats were detected after birth the number in each of 4 randomly selected hours of recording was counted and a mean number per hour calculated. None of 4 subjects with tachycardia >180/minute before labour were studied after birth.

Assessment of infants at 1 minute after delivery

The "Apgar"[61] score was measured in each case by the midwife in charge of the delivery of the infant. Unfortunately it was not always recorded. The relationship between mode of

delivery, presence of bradycardia <100/minute before labour and "Apgar" score at 1 minute was determined for 828 subjects in whom the "Apgar" scores had been recorded in the notes (Table 13).

Assessment of standard fetal heart rate monitor

Using a suitably attenuated interface, tape recordings of the doppler signals on all subjects with premature beats and heart rates outside the range 100-180/minute were replayed through a standard fetal heart rate monitoring system (Ormed Fetograph).

RESULTS

Doppler ultrasound recordings of the fetal heart beat taken before labour

Twelve subjects (1.7%) showed multiple premature beats (Table 9) ranging from 3 to 102 per 5 minutes of recording (Table 10). This arrhythmia was not detected on recordings taken between 30 and 35 weeks of gestational age.

Four subjects showed episodes of tachycardia >180/minute (Table 9) ranging from 40 to 90 seconds in duration (Table 11). One subject showed tachycardia at both 33 and 37 weeks of gestational age.

Twelve subjects showed episodes of bradycardia <100/minute (Table 9) ranging from 5 to 30 seconds in duration (Table 12). The lowest heart rate varied from 35 to 90/minute and in one case, first detected at 30 weeks of gestational age, bradycardia was also found at 36 weeks gestation (subject 17 Table 12).

Recordings of the fetal electrocardiogram during labour (Figures 1 & 2)

Premature beats were detected in two of the three fetal subjects studied (Table 10). In one case (subject 4) the premature beats were probably of supraventricular origin (177/hour) and in the second case (subject 7) of ventricular origin (17/hour). Only one of 4 subjects with tachycardia >180/minute was studied (Table 11). The highest heart rate detected during labour was 185/minute.

Four of 12 subjects with bradycardia <100/minute were studied (Table 12). The lowest heart rates detected were 84, 57, 65 and 64/minute for subjects 20, 23, 25 and 27 respectively.

During labour 7 subjects showed Type I or Type II dips in heart rate.

Twenty-four hour recordings of the electrocardiogram taken after birth (Figures 1 and 2)

Only 5 of 8 subjects studied continued to show premature beats on recordings taken during the first 7 days of life (Table 10). Three infants showed supraventricular premature beats with a frequency of 13, 116, and 290/hour for subjects 2, 4 and 9 respectively. Two infants showed ventricular premature beats with a frequency of 20 and 2/hour for subjects 7 and 8 respectively.

All 7 subjects who had shown bradycardia <100/minute before labour showed similar episodes after birth (Table 12). The lowest heart rates detected ranged from 66-97/minute.

Mode of delivery and "Apgar" scores of infants studied

Of 12 subjects with premature beats 9 underwent spontaneous vaginal delivery, two required forceps for delay in the second stage and one underwent a Caesarian section for delay in the first stage associated with an irregular fetal heart beat (Table 10). At 1 minute after birth 9 had "Apgar" scores of ≥ 7 and 3 of 6.

Of 4 subjects with tachycardia >180/minute 3 underwent spontaneous vaginal delivery, and one required forceps for delay in the second stage. In all cases the "Apgar" score at 1 minute was ≥ 8.

Of 12 subjects with bradycardia <100/minute (Table 12) 7 underwent spontaneous vaginal delivery. Four received forceps; in one case for delay in the second stage but in 3 for Type II dips

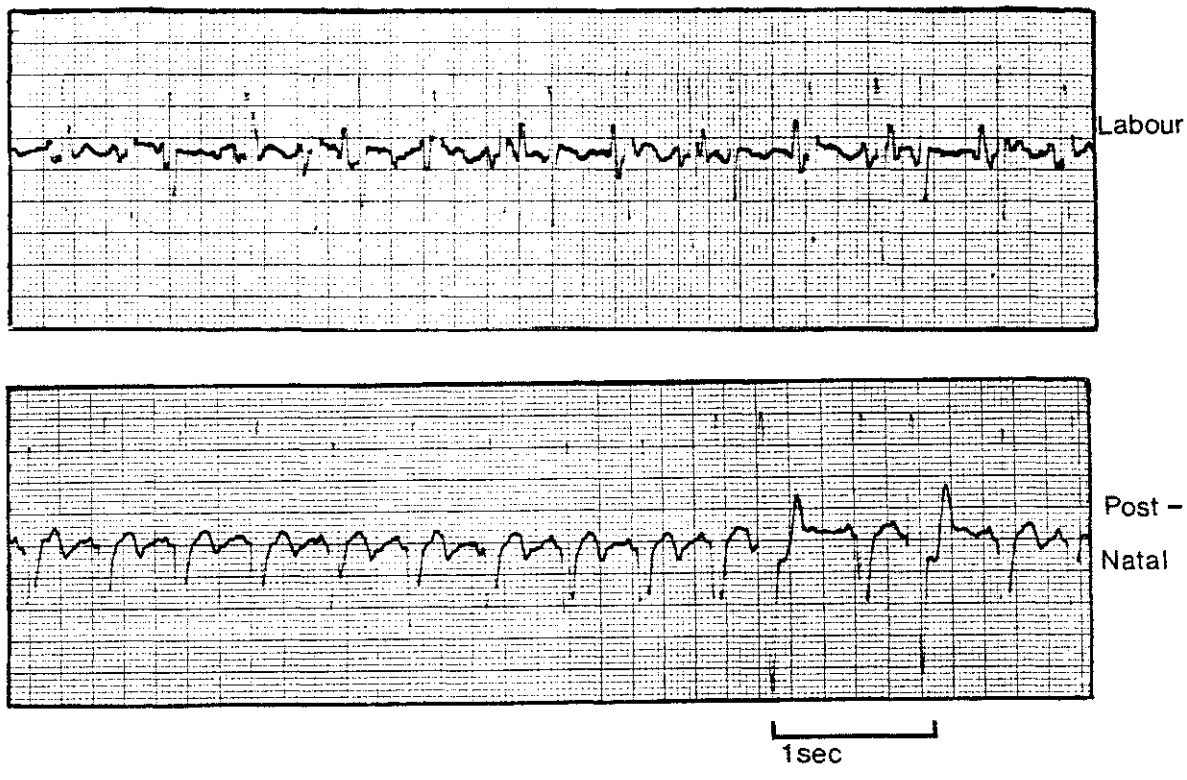
in fetal heart rate.[62,63] One subject (number 24) underwent an elective Caesarian section as a direct consequence of the detection of episodes of bradycardia <100/minute by the screening procedure undertaken before labour as part of this research study. At 1 minute after birth 10 had "Apgar" scores of ≥7, one of 6, and 1 of 5.

The relationship between mode of delivery, presence of bradycardia <100/minute before labour and "Apgar" scores at one minute are shown in Table 13. Eight hundred and twenty eight of a total of 934 subjects had "Apgar" scores at one minute recorded in their notes. Eight hundred and sixteen had heart rates above 100/minute on the antenatal recording and of these 119 had "Apgar" scores of <7. Two of 12 subjects with bradycardia <100/minute before labour subsequently showed "Apgar" scores of <7. There was no significant relationship between episodes of bradycardia <100/minute on the antenatal recordings and subsequent "Apgar" scores <7. (X^2 = 0.026 P is NS)

Doppler recordings of the fetal heart before labour replayed through a fetal heart rate monitor

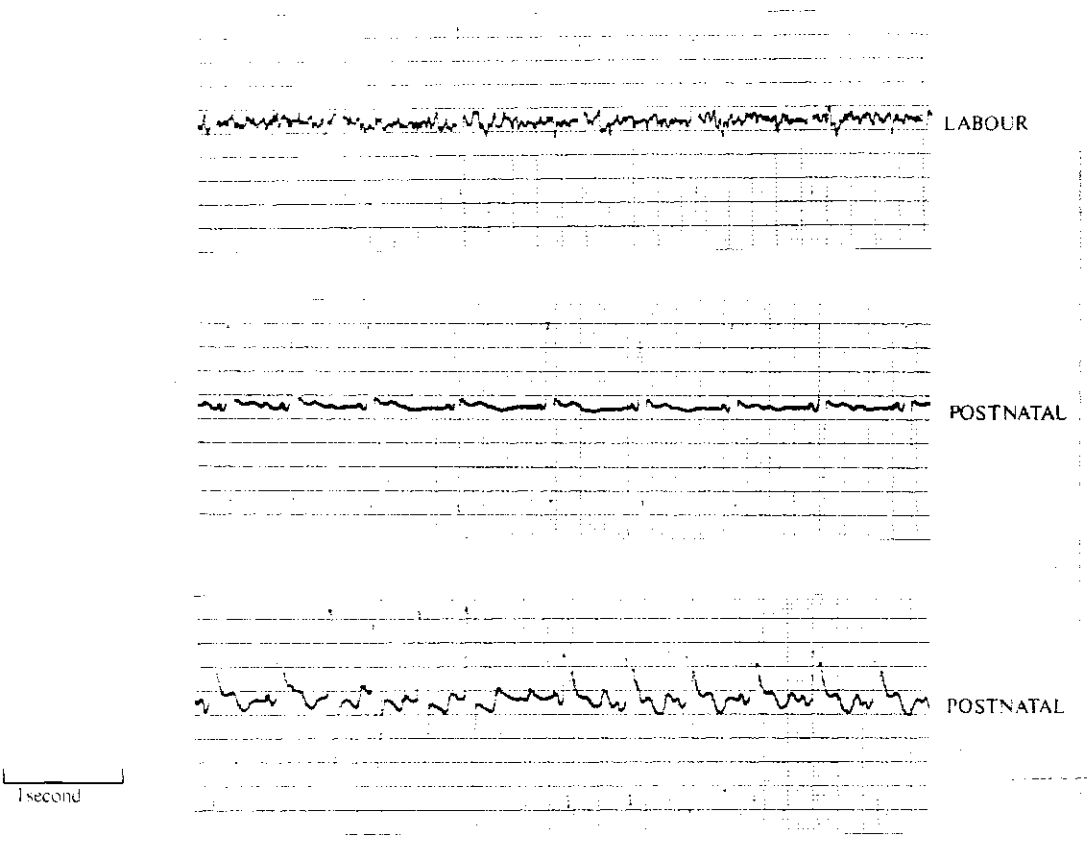
The fetal heart rate monitor failed to detect all premature beats, 2 of 4 episodes of tachycardia >180/minute, 4 of 6 episodes of bradycardia of between 70-100/minute and 5 of 6 episodes of bradycardia below 70/minute (Figure 3).

Figure 1



The upper panel shows a continuous ECG recording taken during labour from a scalp electrode on a fetus who had demonstrated premature beats at 37 weeks gestation (subject 7). Multiple premature beats (17/hour) are present and are also shown in the lower panel (20/hour); a 24-hour ECG recording taken post-natally from the same subject. A ventricular origin for the arrhythmia is suggested by an abnormal QRST configuration and complete compensatory pause.

Figure 2



The top panel is a continuous ECG recording taken during labour from a scalp electrode on a fetus who had demonstrated episodes of bradycardia down to 53/minute at 37 weeks gestation (subject 23). Type II dips were noted on the cardiotocograph at the time of this recording and though artefact may have hidden P waves the ECG demonstrates a probable junctional escape rhythm of 57/minute. The lower two panels show sections of post-natal 24-hour ECG recordings from the same subject, illustrating a sinus bradycardia of 78/minute. The short PR interval suggests that the QRS complexes do not result from conduction of sinus P waves. In the lower panel is a short episode of possible ventricular tachycardia (170/minute).

TABLE 9

NUMBER OF SUBJECTS WITH RHYTHM DISTURBANCES OR RATES OUTSIDE THE RANGE 100 - 180 PER MINUTE ON A FIVE MINUTE DOPPLER RECORDING OF THE FETAL HEART

Number of weeks gestation	Number of subjects recorded	Number of subjects with episodes of bradycardia < 100/minute	Number of subjects with tachycardia >180/minute	Number of subjects with premature beats
30 - 35	269	1 (0.3%)	1 (0.4%)	0 (0%)
36 - 41	720	11 (1.5%)	4 (0.6%)	12 (1.7%)

TABLE 10

DETAILS ON THE 12 SUBJECTS WITH PREMATURE BEATS BEFORE LABOUR

Subject No.	Gestation at recording (weeks)	Number of premature beats in 5 minutes of recording	Continuous ECG in labour	24 hour ECG after birth	Mode of delivery	"Apgar" score at 1 minute
1	36	8	NR	No arrhythmia	SVD	9
2	36	16	NR	APB 13/hour	SVD	8
3	32, 36	0, 20	No arrhythmia	No arrhythmia	SVD	8
4	36	20	APB 177/hour	APB 116/hour	SVD	6
5	36	56	NR	No arrhythmia	SVD	8
6	36	67	NR	NR	SVD	7
7	37	3	VPB 17/hour (Figure 1)	VPB 20/hour (Figure 1)	Forceps for delay	9
8	37	13	NR	VPB 2/hour	SVD	8
9	37	26	NR	APB 290/hour	SVD	7
10	37	44	NR	NR	SVD	9
11	39	3	NR	NR	Forceps for delay	6
12	39	102	NR	NR	LSCS for delay and irregular fetal heart rate	6

NR - No recording made
APB - Atrial premature beat
VPB - Ventricular premature beat

SVD - Spontaneous vertex delivery
LSCS - Lower segment Caesarian section

TABLE 11

DETAILS ON 4 SUBJECTS WITH EPISODES OF TACHYCARDIA >180/MINUTE BEFORE LABOUR

Subject Number	Gestation at time of recording (weeks)	Rate/Minute	Duration of Episodes (seconds)	ECG in labour (highest and lowest rates)	Mode of Delivery	Apgar Scores at 1 minute
13	36	200	65	NR	SVD	8
14	33; 37	200; 190	46; 60	185; 88	SVD	8
15	37	200	40	NR	Forceps (Delay)	8
16	41	200	90	NR	SVD	9

NR - No recording made
SVD - Spontaneous vertex delivery

TABLE 12

DETAILS ON 12 SUBJECTS WITH EPISODES OF BRADYCARDIA <100/MINUTE BEFORE LABOUR

Subject Number	Gestation at Recording (weeks)	Lowest Rate/ Minute	Duration of Bradycardia (seconds)	Lowest heart rate detected on continuous ECG during labour	Lowest heart rate detected on postnatal 24-hour ECG	Complications of labour	Method of Delivery	"Apgar" score at 1 minute
17	30, 36	70 , 70	5	NR	NR	Type I & II dips at 8 cms dilation	Forceps	9
18	32	73	10	NR	75	None	SVD	9
19	34	80	12	NR	NR	None	SVD	8
20	36	67	30	84	66	Type I dips at 5 cms dilation	Forceps	6
21	32, 36	90 , 78	5	NR	NR	Delay	Forceps	9
22	37	35	25	NR	90	Type II dips at 4 cms dilation	SVD	9
23	37	53	5	57 (Figure 2)	78 (Figure 2)	Type II dips at 4 cms dilation	SVD	8
24	37	58	11	LSCS	97	-	LSCS+	9

continued ....

TABLE 12 continued Details on 12 Subjects with Episodes of Bradycardia <100/Minute Before Labour

Subject Number	Gestation at Recording (weeks)	Lowest Rate/ Minute	Duration of Bradycardia (seconds)	Lowest heart rate detected on continuous ECG during labour	Lowest heart rate detected on postnatal 24-hour ECG	Complications of labour	Method of Delivery	"Apgar" score at 1 minute
25	37	70	5	65	77	Type I dips at 6 cms dilation	SVD	5
9	37	80*	15	NR	67*	Type I dips at 7 cms dilation	SVD	7
26	34, 38	100, 90	5	NR	NR	None	SVD	9
27	39	82	20	64	NR	Type II dips at 7 cms dilation	Forceps	8

NR - No recording made
* - Premature beats also detected (290/hour on post natal ECG)
\+ - Performed because of episodes of bradycardia detected by the "research recording"
SVD - Spontaneous vertex delivery
LSCS - Lower segment Caesarian section

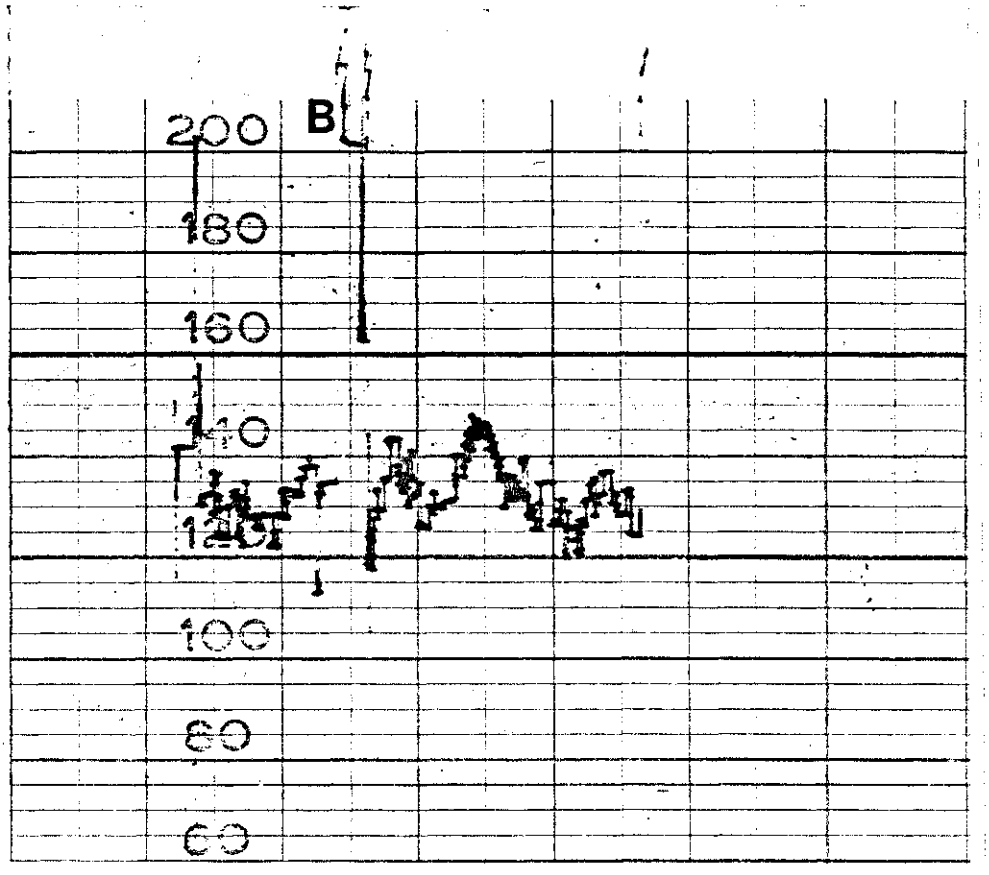
TABLE 13

RELATIONSHIP BETWEEN MODE OF DELIVERY, EPISODES OF BRADYCARDIA <100/MINUTE AND "APGAR" SCORES *

Mode of Delivery	Number of Deliveries with Recorded "Apgar" Scores	Number with Episodes of Bradycardia <100/minute Antenatally		Number without Episodes of Bradycardia <100/minute Antenatally	
		Apgar Score < 7	Apgar Score ⩾ 7	Apgar Score < 7	Apgar Score ⩾ 7
Spontaneous	661	2	6	75	578
Forceps or vacuum	106	0	3	25	78
LSCS	61	0	1	19	41
TOTALS	828	2	10	119	697

* Recorded by midwife at 1 minute after birth

Figure 3



A print out from a standard antepartum fetal heart rate monitor obtained by replaying a tape recording of an episode of bradycardia recorded before labour (subject 22). Heart rate/minute is shown on the vertical axis and paper speed was 1 cm./minute. B marks the position of an episode of bradycardia of lowest rate 35 beats/minute. At this point the pen has lifted and an artefactual signal is seen. This artefact may have resulted from an automatic increase in trigger level during the time between heart beats.

DISCUSSION

This study was performed to establish the incidence and, where possible, the natural history of fetal arrhythmias in an unselected population of expectant mothers. Disorders of cardiac rhythm and conduction in the neonate have been reported to result in heart failure[42], brain damage[64], episodes of so-called 'near-miss cot death'[64] and in one case sudden infant death.[60] Such disorders in the fetus may have similar clinical effects and may even be one cause of unexplained still births.

There are two main problems with this present study. Firstly only a relatively short recording time of 5 minutes was obtained from each subject and thus short lived and intermittent changes in heart rate or rhythm may well have escaped detection. Secondly any subjects showing abnormal rhythms or heart rates outside the range 100-180/minute were identified to the obstetrician. This policy inevitably led to a different management of the remaining part of pregnancy and delivery in those subjects with abnormalities. In many cases the fetal electrocardiogram was monitored during labour and in one subject with frequent episodes of bradycardia <100/minute before labour a Caesarian section was performed at 37 weeks gestation (subject 24). The natural outcome of fetal subjects with these unusual heart rate or rhythm patterns therefore continues to remain unknown.

The frequency of premature beats found in the fetus (1.2%) is similar to that reported in healthy newborn infants (0.8%) (see Chapter 3) where 26 of 3383 infants showed this disorder of rhythm on a standard electrocardiogram. Twenty-four hour ECG recordings on 15 of the 26 neonates with premature beats revealed two with

episodes of supraventricular tachycardia and three with ventricular tachycardia. Arrhythmias other than premature beats, have been previously reported in the fetus (Tables [5] - [8]) and both supraventricular tachycardia and atrioventricular block have been associated with fetal heart failure. The true incidence of arrhythmias, other than premature beats, in the fetus probably requires the study of a larger population using longer recording times. Ultrasound recordings will, of course, also fail to detect the long QT syndrome or pre-excitation unless these abnormalities are accompanied by a change in heart rate or rhythm.

All subjects with premature beats had "Apgar" scores exceeding 5 (Table 10) conforming with previous reports [22,23,25] and suggesting that this arrhythmia is not usually associated with disordered cardio-respiratory adjustments to birth.

Five-minute recordings of the fetal heart beat have shown episodes of bradycardia <100/minute or tachycardia >180/minute in 1.5% of apparently healthy subjects studied. Bolte and Berendes [50] using 3 to 5 minute recordings of the fetal electrocardiogram showed a higher frequency of such heart rate changes (2.8%) but their study predominantly included mothers with complications of pregnancy. Decelerations of the fetal heart rate in labour, late in response to a contraction (Type II dips), [62,63] have been suggested as evidence of fetal hypoxaemia [65] and have been correlated with a poor fetal outcome [66] and fetal acid base disturbance. [67] Similar decelerations of the fetal heart rate before labour have also been correlated with intrauterine death or with a poor fetal outcome. [68] This present study however suggests that some decelerations before, and possibly during, labour may be physiological and care must be taken NOT to

imply abnormality and possibly harmful connotations to normal variability. It was interesting to notice that neither episodes of bradycardia or tachycardia before labour in this study were significantly related to "Apgar" scores at one minute of <7. This may suggest that some fetal subjects have an inherent tendency to manifest frequent episodes of bradycardia which are not associated with fetal hypoxia. Such episodes when they also occur subsequently during labour may lead to unnecessary intervention.

Antepartum fetal heart rate monitoring systems, using either ultrasound or electrocardiogram from the surface of the maternal abdomen have problems in avoiding artefact, particularly where long-term recordings are made. Artefact may be due to loss of signal strength during fetal movement or excessive background noise. To overcome these problems most currently available monitors have an inbuilt electronic device to prevent the registration of rapidly changing heart rates on the assumption that such changes are <u>always</u> due to artefact. This was probably the reason why the monitor used in this study failed to detect premature beats and a large proportion of true episodes of bradycardia. Such monitors in a clinical setting will therefore fail to detect abrupt changes of heart rate and/or rhythm. Furthermore the upper and lower limits of heart rate that can be printed out by these monitors are 200 and 60 beats per minute respectively (Figure 3). Supraventricular and ventricular tachycardias in the neonate often occur with heart rates in excess of 200/minute (see Chapter 3). Sinus tachycardia in this age group may also exceed 200/minute for short periods of time (also see Chapter 3). The design of presently available fetal heart

rate monitors is thus inappropriate for detecting short episodes of sinus tachycardia and for documenting abnormal tachy-arrhythmias.

In summary cardiac arrhythmias and episodes of marked bradycardia <100/minute or tachycardia >180/minute occur in the developing baby during normal pregnancy. Assessment of these patterns in the antenatal period should take a knowledge of these normal findings into consideration and the present problems with long-term recording techniques must be overcome.

CHAPTER 3

HEART RATE AND RHYTHM PATTERNS IN THE NEWBORN INFANT

INTRODUCTION

The variation in heart rate and the nature of cardiac rhythm and conduction patterns are poorly documented in normal healthy infants.

Disorders of cardiac rhythm and conduction have usually been reported in the newborn because of their important clinical effects such as heart failure,[69] so called 'near miss' sudden infant death[64] and more recently, in one case, the Sudden Infant Death Syndrome.[60] Ferrer et al[70] have suggested from a selected population of infants that arrhythmias occur frequently at this age and Jones et al[71] have described arrhythmias in 4.8% of standard ECGs performed on over 1,000 neonates prior to discharge from a Brighton maternity hospital.

To estimate the incidence of disorders of cardiac rhythm and conduction in the population, a standard electrocardiogram was made on 3,383 newborn infants born in Weymouth and Dorchester maternity hospitals. The results of these recordings will be described together with 24-hour ECG recordings on 33 subjects from this group who showed disorders of cardiac rhythm or conduction on their standard electrocardiogram. Whenever possible the natural histories of these unusual ECG patterns were also studied by repeated standard and 24-hour ECG recordings at follow up during infancy and early childhood.

To allow accurate interpretation of 24-hour ECG recordings on infants with disorders of cardiac rhythm or conduction the range of heart rate and rhythm in normal healthy

infants must be known. A single 24-hour ECG recording was therefore taken on 140 well full term infants who did not show abnormalities of rhythm or conduction on a standard electrocardiogram.

PATIENTS AND METHODS

1. Standard ECG study on an unselected population of newborn infants

Between April 1974 and January 1978 a standard 11-lead ECG (I, II, III, aV_R, aV_L, aV_F, V_{4R}, V_1, V_2, V_4, and V_6) with a ten second rhythm strip was performed, using a Hewlett-Packard portable ECG machine (model 1504B), on 3,383 newly born infants about to be discharged from Weymouth and Dorchester maternity units. Their ages ranged from 1 to 56 days with a mean age of 3 days. All infants who had been admitted to the neonatal intensive care unit were included in the study and in these cases recordings were taken when infants were well and about to be discharged from hospital. Recordings were also performed on infants with congenital anomalies, including structural heart disease. Between April 1, 1975 and September 1, 1976 babies who were discharged at 48 hours were excluded from the study. From September 1, 1976 until March 1 , 1978 all infants (including early discharge patients) were screened. To avoid some of the possible effects of haemodynamic changes that occur during the transistion from fetal to infant circulatory patterns, recordings were, when possible, not taken during the first 24 hours of life.

Standard electrocardiograms were made with the infants supine, asleep or sucking a sterile teat in the nurseries of the maternity departments or the neonatal intensive care unit after informed parental consent was obtained. To minimize parental anxiety, the technician explained that the baby would feel no discomfort from the recording and that the procedure was completely safe and designed to supplement the routine clinical

examination of the heart. Parents were told the results on the same day and only unequivocal abnormalities were mentioned. Only one parent refused to permit the recording.

From September 1, 1976, all infants with cardiac arrhythmias or preexcitation on the standard ECG were further studied by 24-hour tape recordings of the ECG.

Clinical details of the antenatal, birth, and post-natal history of all infants with disorders of cardiac rhythm or conduction were collected (Table 14).

Subjects with arrhythmias or conduction disorders were then seen in a special outpatient clinic (usually between two and six weekly intervals) where a larger number of randomly selected control infants without arrhythmias were also seen (65 subjects). A clinical examination was made and standard ECG recordings were performed on both infants with disorders and controls. From September 1976, 24-hour recordings were also used at the follow up clinics to study infants with arrhythmias. Tape recorders were attached in the hospital and recordings were made at the baby's home. The appropriate family practitioner was informed about all infants in whom an arrhythmia or conduction disorder was detected.

2. Twenty-four hour ECG recordings on a randomly selected population of infants without disorders of cardiac rhythm or conduction on a standard ECG

To establish normal values a single 24-hour recording of the electrocardiogram was taken on 140 randomly selected full-term infants who did not show disorders of cardiac rhythm or

conduction on a standard ECG. These infants were also born in the Dorchester and Weymouth maternity hospitals between January 1977 and June 1978 and recordings were made in the post-natal wards of these hospitals at between 12 hours and 10 days of age. All infants were well at the time of these recordings and were studied under the environmental conditions standard to the post-natal wards. During the recording period infants were handled and fed in the normal way. Of 140 infants studied recordings on 6 contained <12 hours of adequate data (2 resulted from failure of electrode contact and 4 from lead breakage).

To determine the frequency distribution of heart rates during activity and rest sections of the recording were analysed using an automatic counter of QRS complexes. Histograms were made in this way on a randomly selected subgroup of 79 of the total 134 infants during two hours of activity and two hours of rest (both periods of time assessed by reference to the log of infant activity kept by the mother). (Figures 4 and 5)

Maximum heart rates were then measured by direct ECG analysis (over 8 R-R intervals) on the whole group of 134 infants (Figure 6). To obtain the information a search was made through the whole of each 24-hour recording for the most rapid heart rate; a section of which was replayed onto ECG paper at 25 mm/second. The minimum heart rates (over 2, 4 and 8 consecutive R-R intervals were also measured in this way (Figures 7-9) and the heart rhythm at this time (whether sinus or junctional was noted).

The frequency of premature beats over the 24 hours of recording was computed from counts made in 4 randomly selected hours of recording. No attempt was made to account for infant activity.

Values for highest and lowest heart rates were analysed and compared for 2 age groups; the first 1-3 days and the second 4-10 days.

Seventy-one of the 134 (53%) recordings were randomly selected and analysed for the presence of P-P intervals which exceeded the previous P-P interval by more than 50%. If the QRS complex by which this interval was terminated was not preceded by a P-wave the P-Q interval was measured. The following patterns were seen and counted.

(1) A sudden increase in the P-P or P-Q interval exceeding the previous P-P interval by more than 110% (an ECG pattern described as sinus arrest (Figure 10).

(2) A sudden increase in P-P or P-Q interval exceeding the immediately preceding interval by between 90 and 110% (an ECG pattern described as representing sinus arrest or second degree sino atrial exit block) (Figure 11).

(3) A sudden increase in P-P or P-Q interval exceeding the immediately preceding P-P interval by between 50 and 90%. (Figure 12) This group was also evaluated with respect to the pattern of P-P intervals (a minimum of 3) by which the pause was preceded:

(i) a progressive increase in P-P intervals

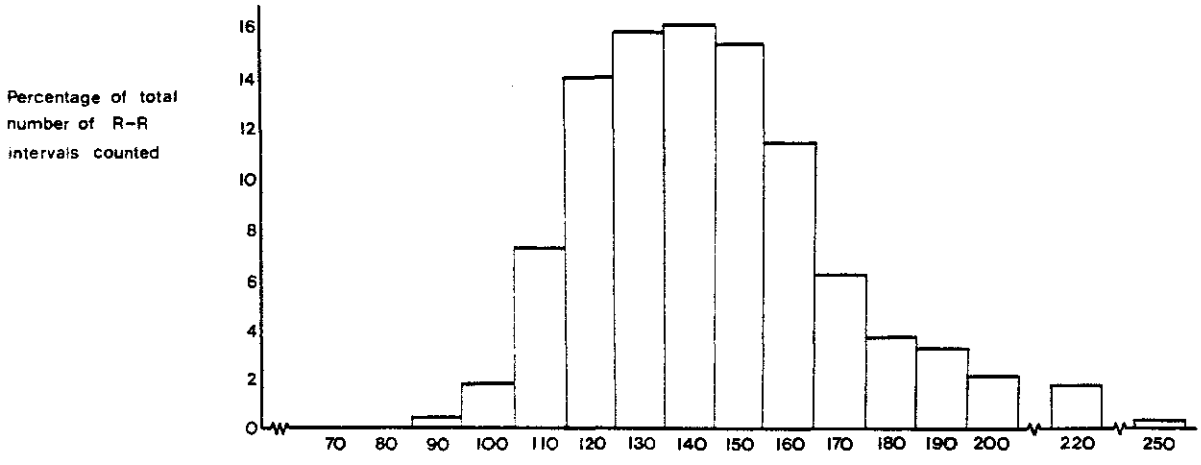
(ii) regular P-P intervals

(iii) irregular P-P intervals

(iv) a progressive decrease in P-P intervals, (an ECG pattern termed Wenckebach sino-atrial exit block) (Figures 12c and 13)

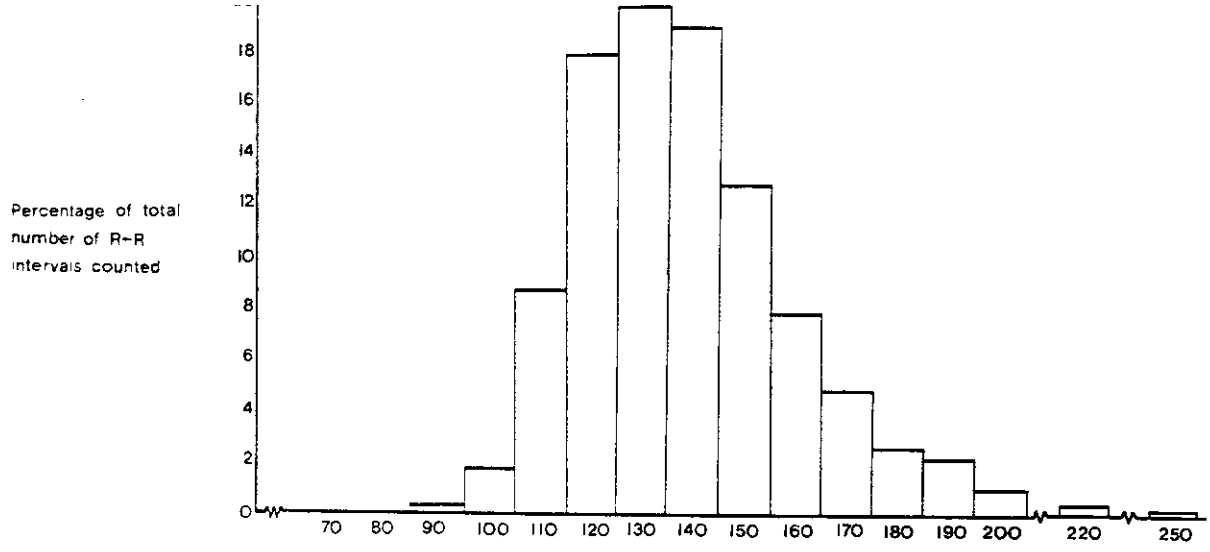
To differentiate Wenckebach sino-atrial exit block pattern from sinus arrhythmia the calculations of Schamroth and Dove[72] were used and successive P-P intervals were plotted (Figure 14).

Figure 4



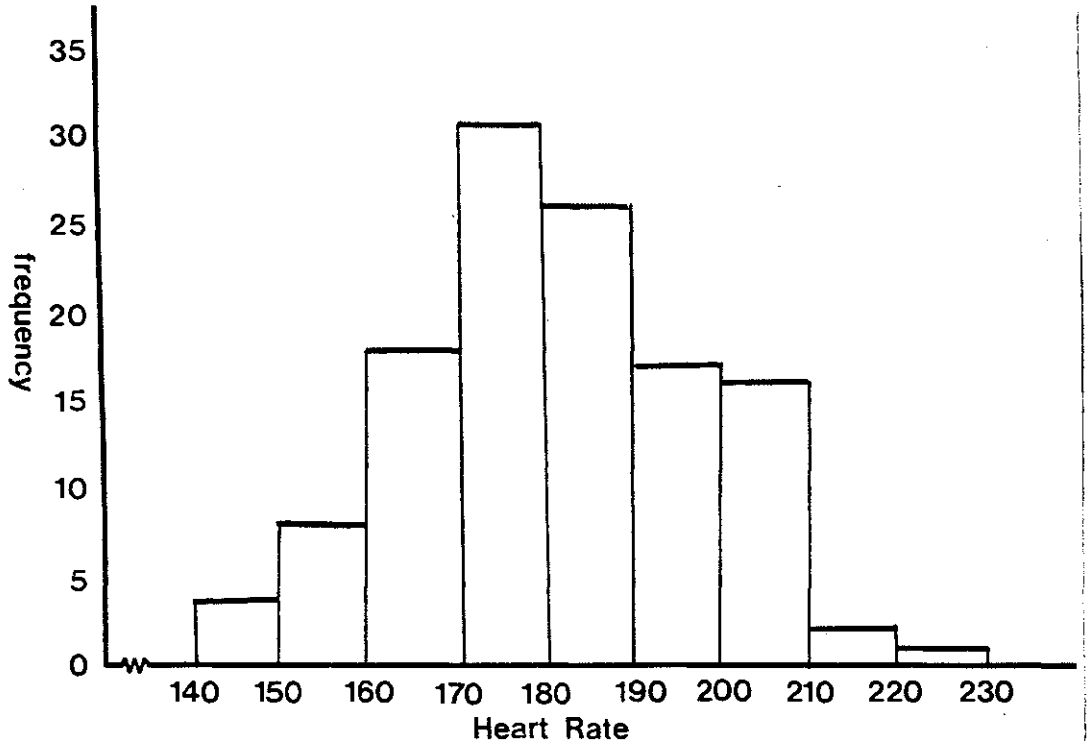
Histogram from automatic R-R interval counter showing the frequency distribution of heart rates on 79 infants during 2 hours of activity. Heart rates over 220/minute are probably artefactual and represent the inclusion of movement potentials and high T waves. Because of these latter problems the whole histogram is probably biased to show higher heart rates.

Figure 5



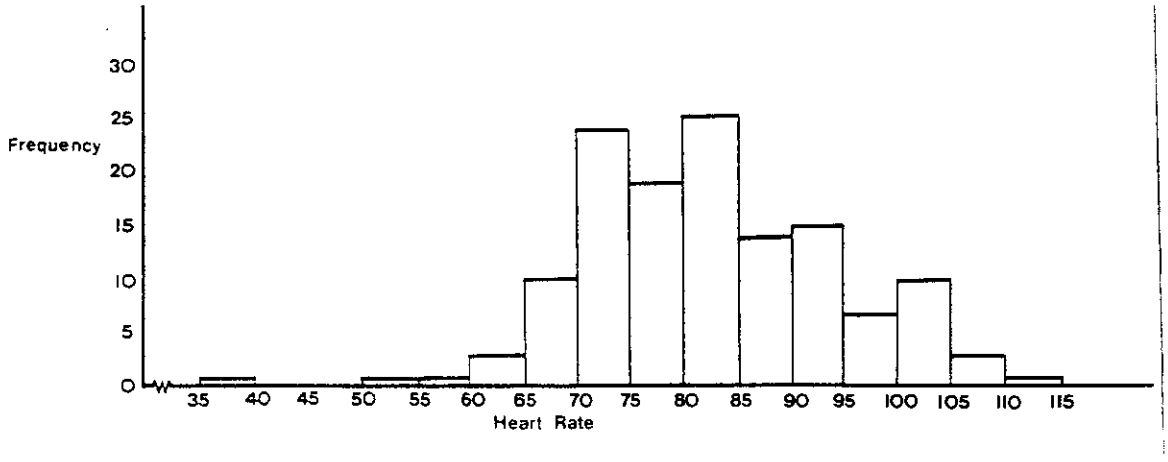
Histogram from automatic R-R interval counter showing the frequency distribution of heart rates on 79 infants during 2 hours of rest. Heart rates over 220/minute are probably artefactual and represent the inclusion of movement potentials and high T waves. Because of these latter problems the whole histogram is probably biased to show higher heart rates.

Figure 6



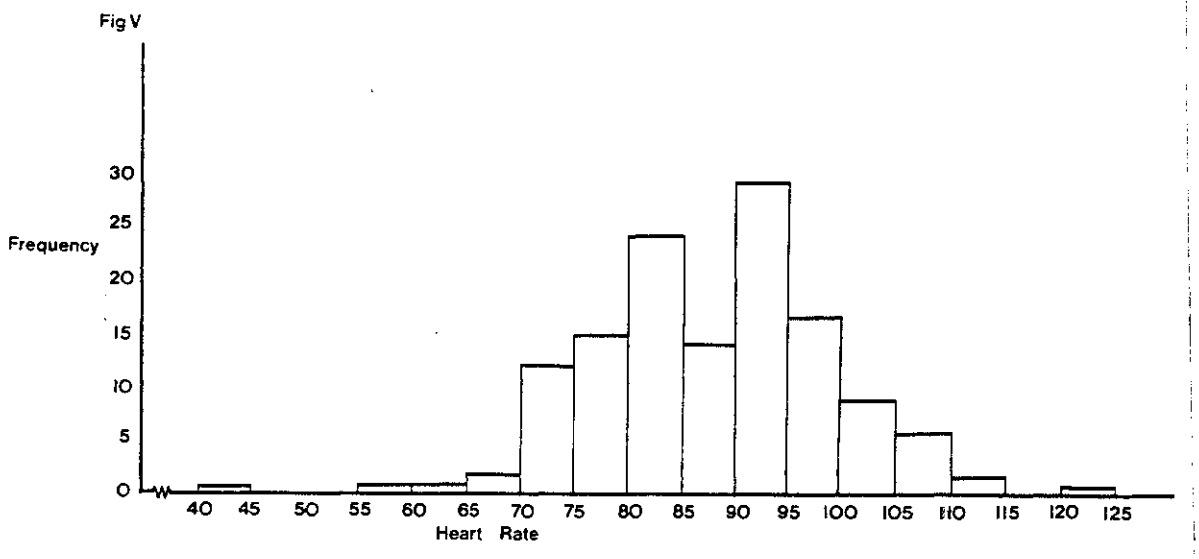
Histogram from direct ECG analysis of the highest heart rates on each of the 134 infants measured over 8 consecutive R-R intervals. (The histogram was shown to approximate to a curve of normal distribution by the Kolmogorov-Smirnov test).[17]

Figure 7



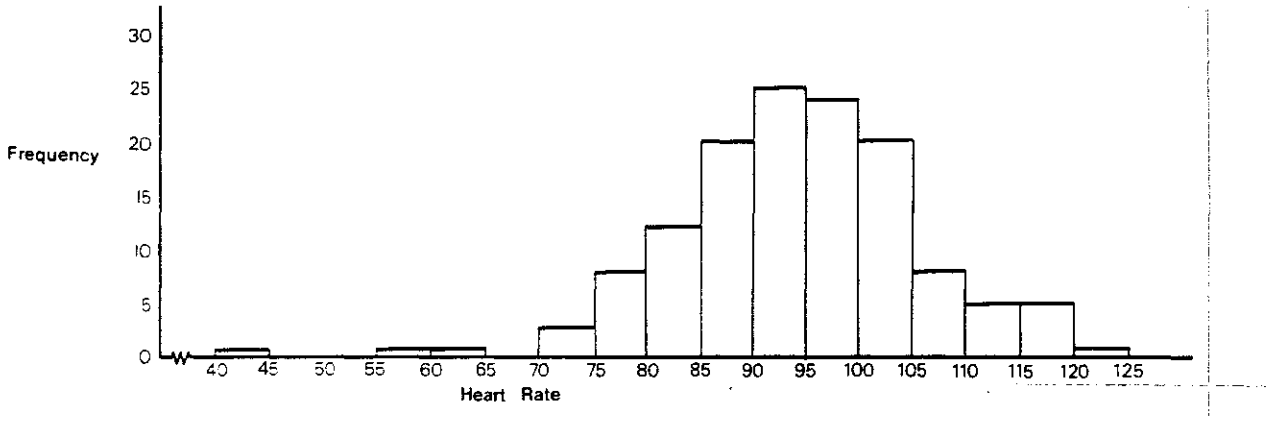
Histogram from direct ECG analysis of the lowest heart rates measured on each of the 134 infants over 2 consecutive R-R intervals. (The histogram was shown to approximate to a curve of normal distribution by the Kolmogorov-Smirnov test)[17]

Figure 8



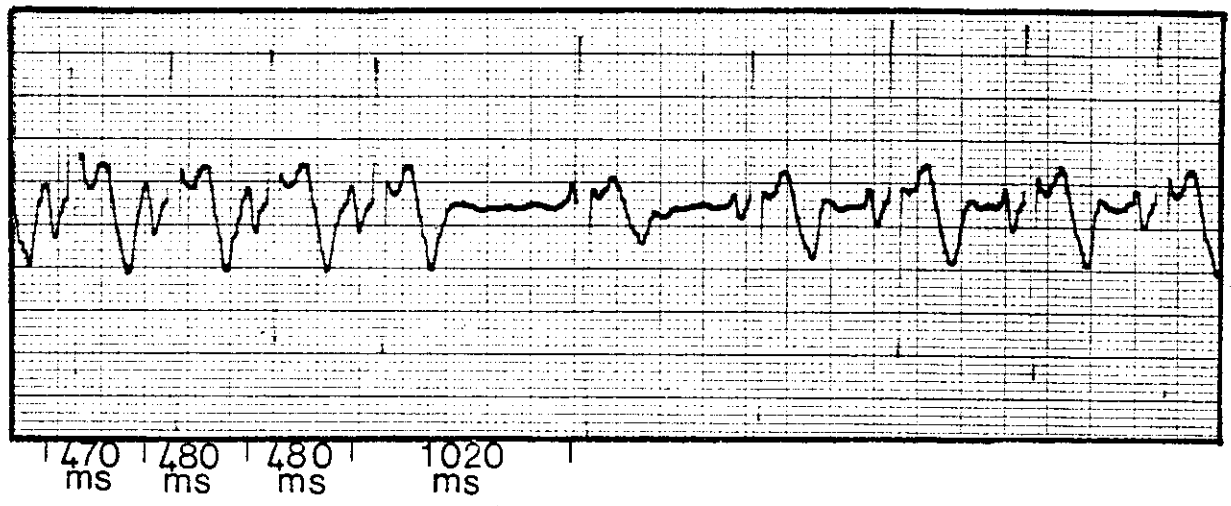
Histogram from direct ECG analysis of lowest heart rates on each of the 134 infants measured over 4 consecutive R-R intervals. (The histogram was shown to approximate to a curve of normal distribution by the Kolmogorov-Smirnov test[17])

Figure 9



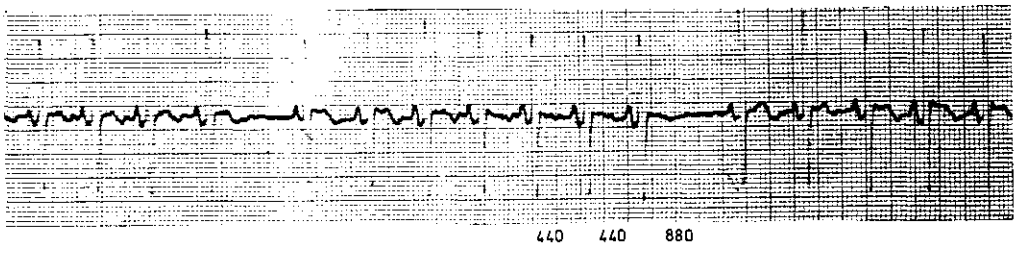
Histogram from direct ECG analysis of lowest heart rates on each of the 134 infants measured over 8 consecutive R-R intervals. (The histogram was shown to approximate to a curve of normal distribution by the Kolmogorov-Smirnov test)[17]

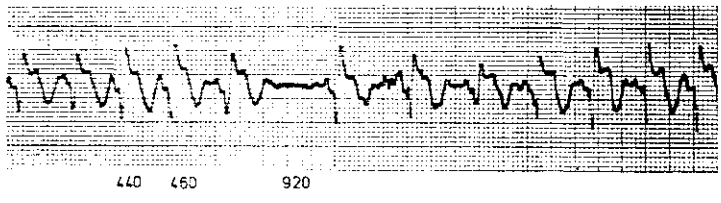
Figure 10



An extract from a 24-hour ECG recording showing sudden lengthening of P-P interval by more than 110% of the immediately preceding P-P interval. This ECG pattern and rhythm disturbance cannot be distinguished from sinus arrest.

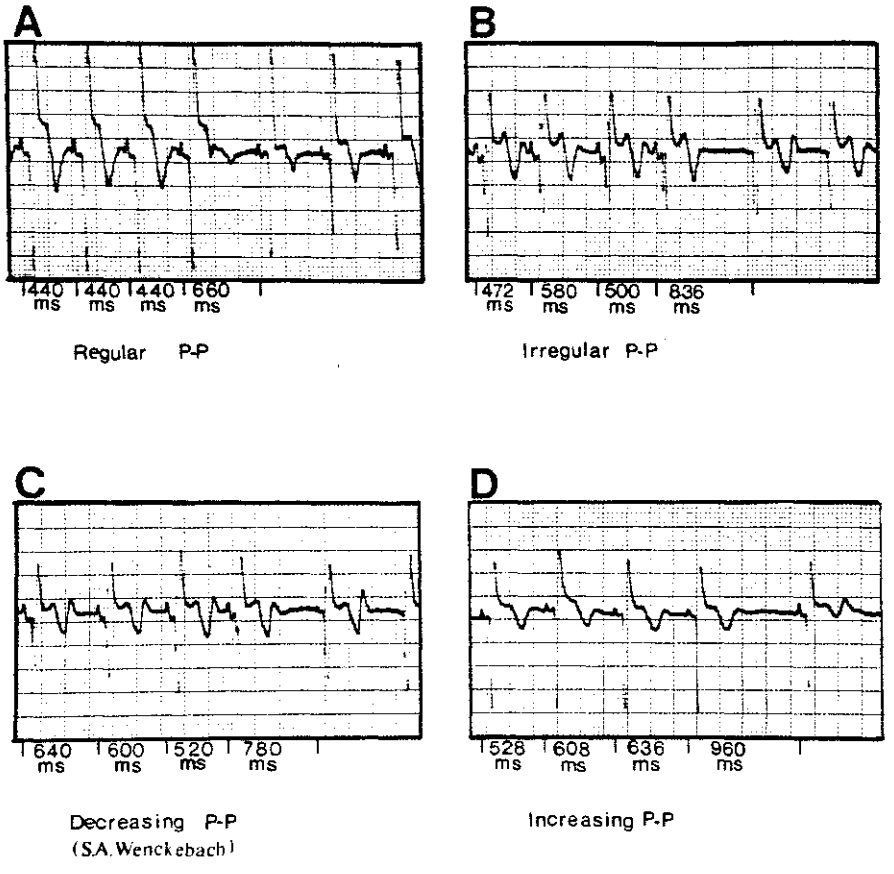
Figure 11





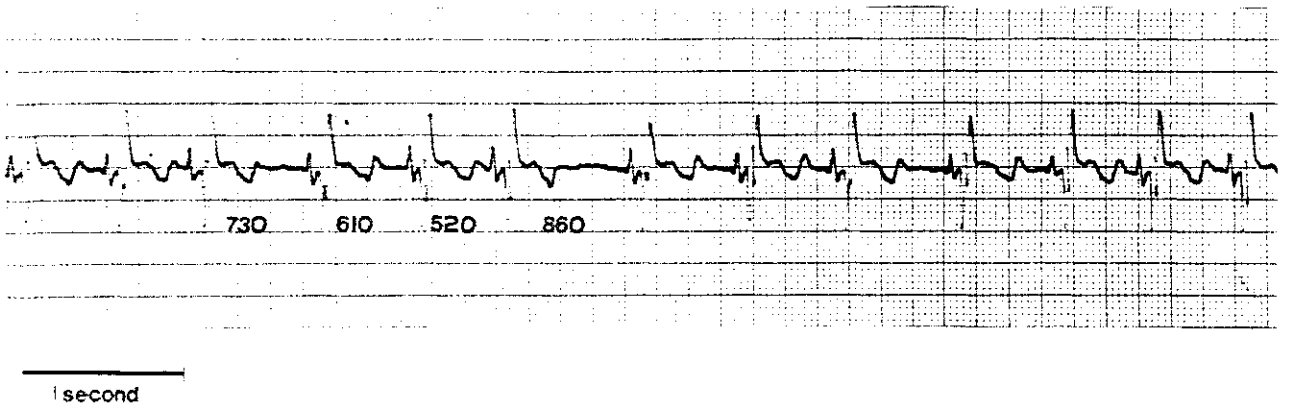
Extracts from 24-hour recordings showing sudden lengthening of P-P interval by between 90 and 110% of the immediately preceding P-P interval. These surface ECG patterns cannot be distinguished from sinus arrest or second degree sino-atrial exit block.

Figure 12



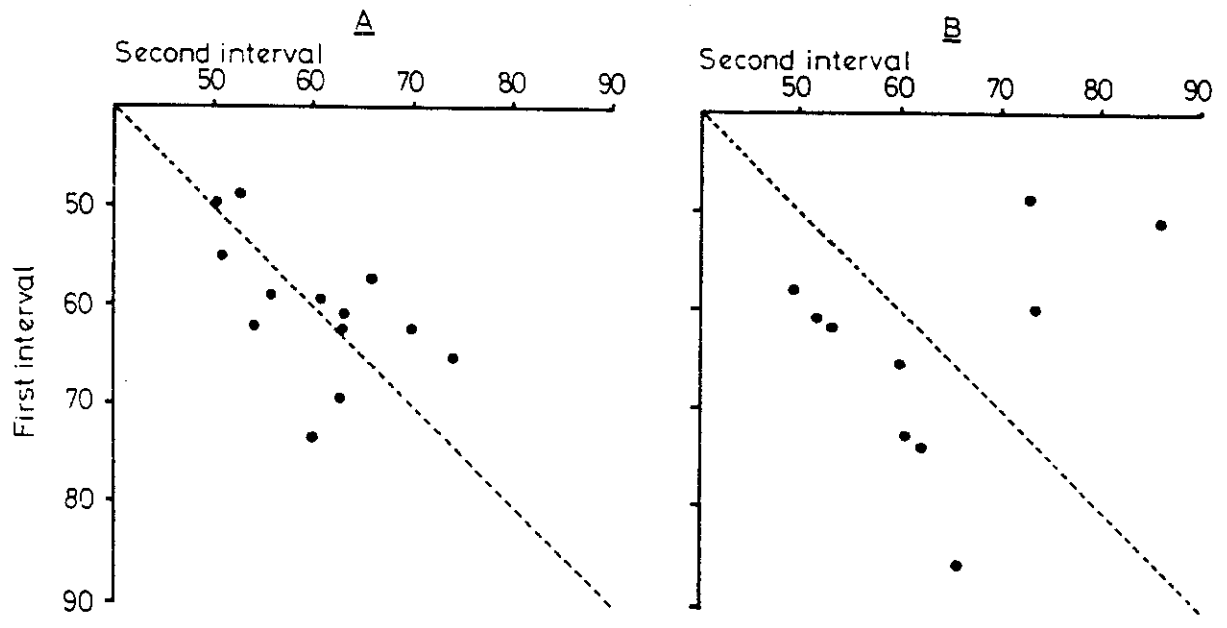
Four extracts from 24-hour ECG recordings on different infants showing pauses. In each case there is sudden lengthening of P-P or P-Q interval exceeding the immediately preceding P-P interval by between 50 and 90%. In (A) the preceding P-P intervals are regular; in (B) they are irregular and in (D) they are increasing before the pause. In (C) there is progressive decrease in P-P intervals before the pause and this ECG pattern cannot be distinguished from Wenckebach sino-atrial exit block.

Figure 13



Twenty-four-hour recording showing a pause which is preceded by a progressive reduction in P-P intervals. This pattern cannot be distinguished from Wenckebach sino-atrial exit block.

Figure 14



A graph showing a plot of P-P intervals on the Y axis against succeeding P-P intervals on the X axis. The line shows the position of P-P intervals of equal duration.

Graph A shows the pattern of P-P intervals descriptive of sinus arrhythmia.

Graph B shows the pattern of P-P intervals descriptive of Wenckebach sino-atrial exit block.[72]

RESULTS

1. Standard ECG study

Thirty-three infants showed cardiac arrhythmias or preexcitation on the standard ECG (Tables 14-17).

Eighteen were girls and 15 boys; no infant was preterm, but two (infants 4 and 19) were small for gestational age. Three were noticed to have an irregular or rapid fetal heart rate during pregnancy or labour (infants 3, 27 and 31). Three infants had "Apgar" scores at one minute of <7, including one (infant 20) who required intubation and ventilation for birth asphyxia. Infant 29, with supraventricular tachycardia, had one cyanotic attack before treatment with digoxin was started.

In 23 of 27 infants followed up by standard and/or 24-hour recordings, cardiac arrhythmias or preexcitation could not be detected after 12 weeks of age. Infant 14, who had multiple atrial premature beats, ceased to demonstrate an arrhythmia at 20 weeks of age and multiple ventricular premature beats in infant 16 could not be detected at 66 weeks of age. Two infants 4 and 31, continued to demonstrate arrhythmias at the time of their last follow-up appointments at 55 weeks and 170 weeks, respectively.

Table 16 describes the results of 24-hour ECG recordings in subjects with premature beats. Three of 8 infants had ventricular premature beats and demonstrated episodes of ventricular tachycardia (Figure 15ab), but the one infant studied by a 24-hour recording who had ventricular premature beats with features suggesting a parasystolic origin (Figure 16) did not

show ventricular tachycardia. Two of 15 infants with multiple atrial premature beats demonstrated episodes of supraventricular tachycardia (SVT).

Table 17 shows the results of 24-hour recordings and, where appropriate, describes the treatment in subjects with supraventricular tachyarrhythmias on the standard ECG (Figures 17ab, 18, 19). Within 48 hours of the start of treatment with digoxin, three infants who had shown cardiac arrhythmias on their screening ECG demonstrated continuous sinus rhythm on subsequent standard ECG recordings. One infant with multifocal atrial tachycardia, and episodes of SVT at 250 beats/minute for five minute periods on 24-hour tape, was treated with propranolol. This reduced the frequency and duration of SVT, but did not remove the multifocal atrial rhythm. At 18 months of age therapy with propranolol was discontinued. Multifocal atrial tachycardia (without long periods of SVT on 24-hour recordings) has continued and the child is now 3 years old (Figure 17ab).

The Wolff-Parkinson-White syndrome was found in 2 subjects during the first week of life. On the initial recordings preexcitation was continuous but at six weeks of age it was intermittent and by 3 months could not be detected on either standard or 24-hour recordings (Figures 20ab). Similar recordings at 6 months, and 1 year of age have failed to demonstrate preexcitation.

Follow-up standard ECG recordings on 65 control subjects who did not show cardiac arrhythmias in the neonatal period failed to demonstrate in any case arrhythmias during later infancy.

TABLE 14

ANTENATAL AND POSTNATAL HISTORY OF INFANTS WITH ARRHYTHMIAS OR PREEXCITATION

Infant Number	Sex	Arrhythmias	Gestation at Birth (weeks)	Birth Weight (gm)	Complications of Pregnancy	Apgar Score 1,5 minutes	Neonatal Complications
1	F	SPBs	40	3,100	0	9, 10	0
2	F	SPBs	40	3,490	0	9, 10	0
3	M	SPBs	42	4,370	Irregular FH in labour	8, 9	0
4	F	SPBs	40	2,500	Poor weight gain, low oestriols	9, 10	0
5	M	SPBs	39	3,810	Slow FH in labour	9, 10	0
6	F	SPBs	40	3,250	0	9, 10	0
7	F	SPBs	39	3,960	0	9, 9	0
8	F	SPBs	39	3,510	PET	7, 9	0
9	M	SPBs	40	3,600	PET	9, 10	0
10	M	SPBs	40	3,580	0	9, 10	0
11	F	SPBs	40	3,300	0	9, 10	0
12	F	SPBs	40	3,350	0	9, 10	0
13	F	SPBs	40	3,425	0	9, 9	0
14	F	SPBs	39	2,895	0	9, 10	0
15	M	VPBs	39	3,920	0	9, 10	0
16	M	VPBs	40	3,250	0	9, 10	0
17	M	VPBs	39	3,690	PET	9, 10	0
18	F	VPBs	39	2,860	PET	9, 10	Jaundice
19	F	VPBs	40	2,500	PET	7, 10	0
20	M	VPBs	40	3,680	0	4, 10	Birth asphyxia
21	M	VPBs	40	3,290	APH, slow FH in labour	5, 9	0
22	F	VPBs	40	3,270	0	8, 10	0
23	F	VPBs	40	2,500	PET	7, 10	0
24	F	VPBs	41	3,860	0	7, 9	0
25	M	VPBs	40	3,870	0	7, 9	0

Contd. ....

Table 14 Antenatal and Postnatal History of Infants with Arrhythmias or Preexcitation continued

Infant Number	Sex	Arrhythmias	Gestation at Birth (weeks)	Birth Weight (gm)	Complications of Pregnancy	Apgar Score 1, 5 minutes	Neonatal Complications
26	M	APBs and VPBs	39	3,340	0	8, 10	0
27	F	Atrial flutter	40	3,330	PET, persistent fetal tachycardia	9, 10	0
28	F	Paroxysmal SVT	40	3,600	0	9, 10	0
29	F	Continuous SVT	38	3,980	Hydramnios, PET	8, 9	Cyanotic attack
30	M	Multifocal atrial tachycardia	40	3,570	0	9, 10	0
31	M	Multifocal atrial tachycardia	38	3,140	PET, irregular FH	9, 10	0
32	M	Preexcitation	40	3,200	0	6, 10	0
33	M	Preexcitation	40	3,000	0	9, 10	0

Abbreviations:

SPBs	Supraventricular premature beats
FH	Fetal heart beat
PET	Preeclamptic toxaemia
VPBs	Ventricular premature beats
SVT	Supraventricular tachycardia

TABLE 15

FOLLOW-UP DATA ON INFANTS WITH CARDIAC ARRHYTHMIAS OR PREEXCITATION ON STANDARD ECG

Infant Number	ECG Finding	A Age at detection (days)	B First age at which ECG pattern could not be detected (weeks)	Number of Recordings between A and B	Total duration of follow-up (weeks)
1	SPBs	8	4	0	4
2	SPBs	8	4	0	4
3	SPBs	1	4	0	4
4	SPBs	1	-*	6	55++
5	SPBs	7	6	0	6
6	SPBs	8	8	1	8
7	SPBs	1	4	1	84++
8	SPBs	2	-**		None
9	SPBs	2	-**		None
10	SPBs	1	2	0	12++
11	SPBs	1	-**		None
12	SPBs	1	5	1	5
13	SPBs	2	-**		None
14	SPBs with parasystolic features	2	20	2	20++
15	VPBs	2	4	1	26++
16	VPBs	1	66	16	140++
17	VPBs	3	9	2	22++
18	VPBs	7	3	2	8++
19	VPBs	2	2	0	8++
20	VPBs	1	12	0	56++
21	VPBs	2	-**		None
22	VPBs	1	-**		None

continued .....

TABLE 15 continued Follow-up Data on Infants with Cardiac Arrhythmias on Preexcitation on Standard ECG

Infant Number	ECG Finding	A Age at detection (days)	B First age at which ECG pattern could not be detected (weeks)	Number of Recordings between A and B	Total duration of follow-up (weeks)
23	VPBs with parasystolic features	8	6	0	4
24	VPBs with parasystolic features	7	12	1	16
25	VPBs with parasystolic features	7	8	2	16++
26	SPBs and VPBs	5	12	2	12++
27	Atrial flutter	5	2***	3	60
28	Paroxysmal SVT	8	6***	1	54
29	Continuous SVT	1	4***	0	142++
30	Multifocal atrial T	8	12ø	4	180++
31	Multifocal atrial T	3	-ø	12øø	170++
32	WPW (type A)	8	12	2øø	42++
33	WPW (type A)	7	12	2øø	105++

Abbreviations:

SPBs Supraventricular premature beats
VPBs Ventricular premature beats
SVT Supraventricular tachycardia
T Tachycardia
WPW Wolff-Parkinson-White Syndrome

* Arrhythmia had not disappeared by last follow-up examination
** No follow-up was possible
++ Duration also determined by 24-hour ECG
*** Treated with antiarrhythmic drugs
ø Treated with antiarrhythmic drugs, but arrhythmia still present at last follow-up examination
øø Preexcitation intermittent at this time

TABLE 16

FINDINGS OF 24-HOUR ECG RECORDINGS IN INFANTS WITH PREMATURE BEATS ON STANDARD ECG

Infant Number	Standard ECG Finding	Infants Studied by 24-hour ECG	New Information from 24-hour ECG
1-13	Multiple SPBs	4, 7-10, 13	-
14	Multiple SPBs with parasystolic features	14	SVT with maximum heart rate of 210 beats /minute
15-22	Multiple VPBs	15-20	VT 200 beats/minute (Infant 15), VT 220 beats/minute (Figure 15ab, Infant 16) VT 220 beats/minute (Infant 10)
23-25	Multiple VPBs with parasystolic features (Figure 16)	25	-
26	Multiple SPBs and VPBs	26	SVT (210 beats/minute)

Abbreviations:

SPBs	Supraventricular premature beats
SVT	Supraventricular tachycardia
VT	Ventricular tachycardia
VPBs	Ventricular premature beats

TABLE 17

FINDINGS OF 24-HOUR ECG RECORDINGS

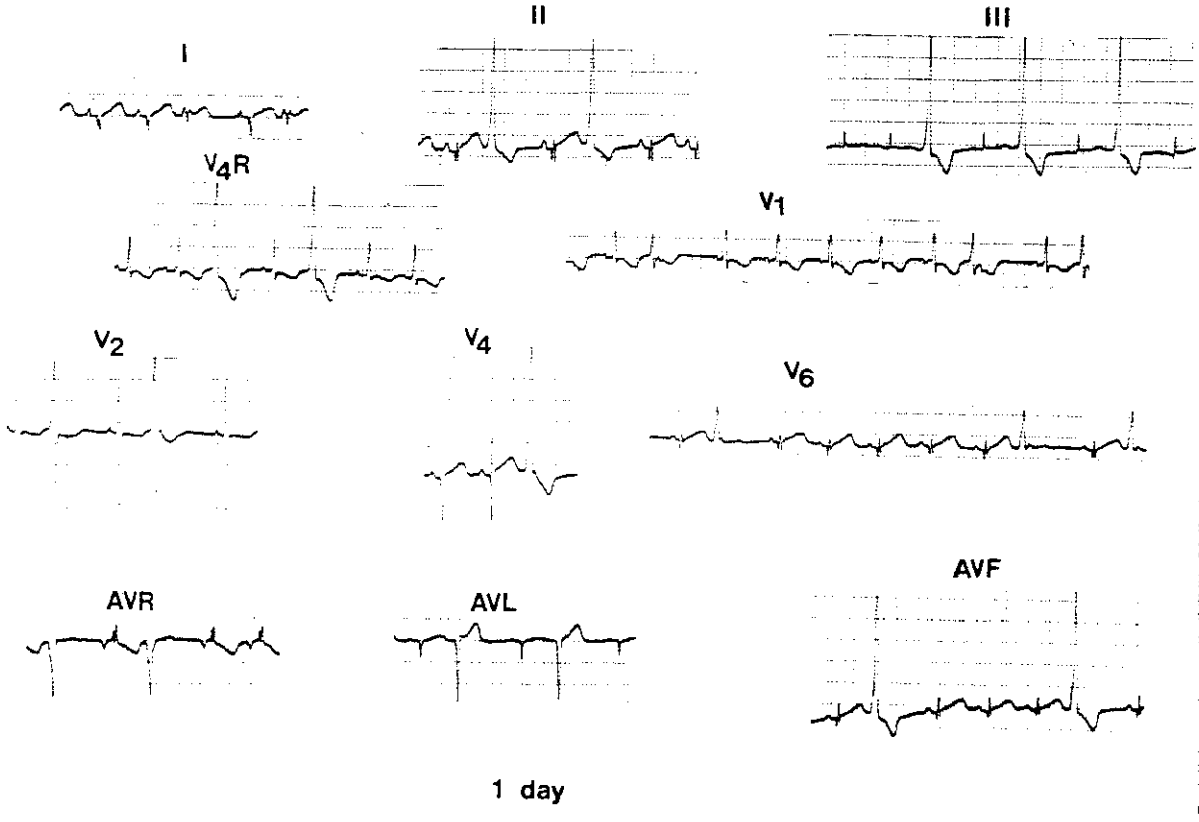
AND TREATMENT OF SUBJECTS WITH SUPRAVENTRICULAR TACHYARRHYTHMIAS ON STANDARD ECG

Infant Number	Standard ECG Finding	No. 24-hour ECGs	Information from 24-hour ECGs	Treatment
27	Atrial flutter (Fig. 18)	-	-	Digoxin
28	Paroxysmal reentry AV nodal SVT (Figure 19)	-	-	Digoxin
29	Continuous SVT	1	-	Digoxin
30	Multifocal AT	-	-	None
31	Multifocal AT with possible VPBs (Figure 17ab)	1	SVT 250 beats/minute	Propranolol

Abbreviations:

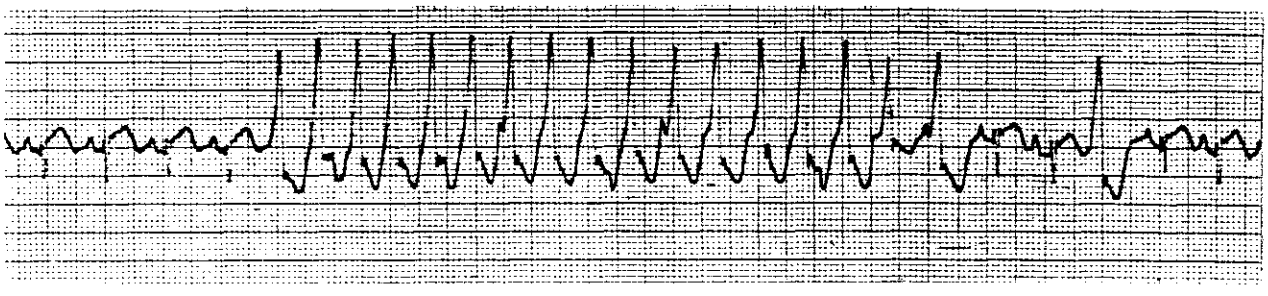
AV	atrioventricular
SVT	supraventricular tachycardia
AT	atrial tachycardia
VPBs	ventricular premature beats

Figure 15a



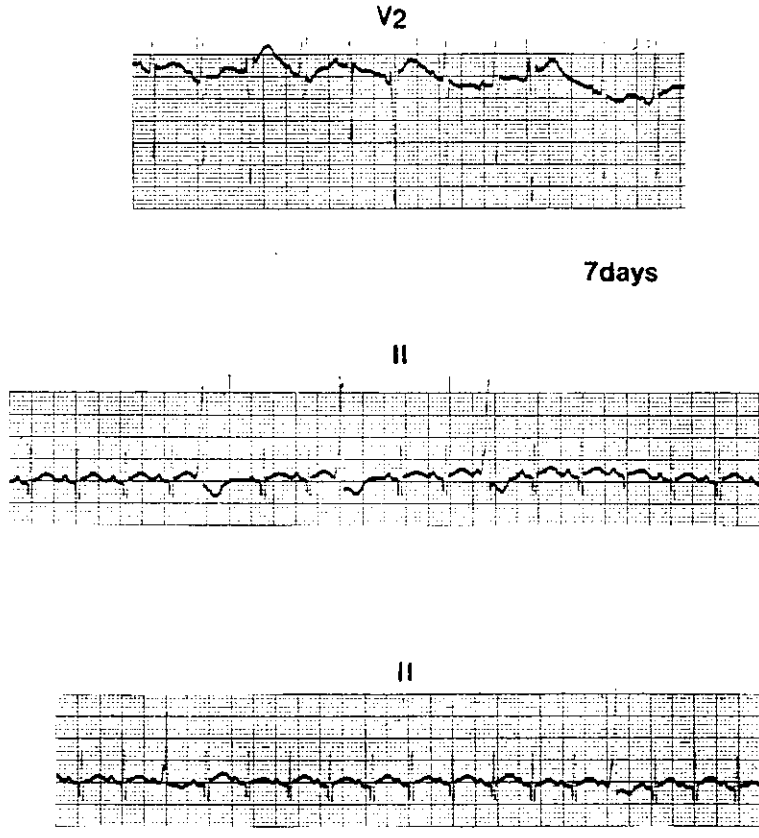
Standard ECG recording on an apparently healthy infant at 1 day of age (subject 16) showing multiple non-parasystolic ventricular premature beats. The QRS complex is prolonged and abnormal in configuration. The majority but not all premature beats show a constant coupling interval and a complete compensatory pause.

Figure 15b



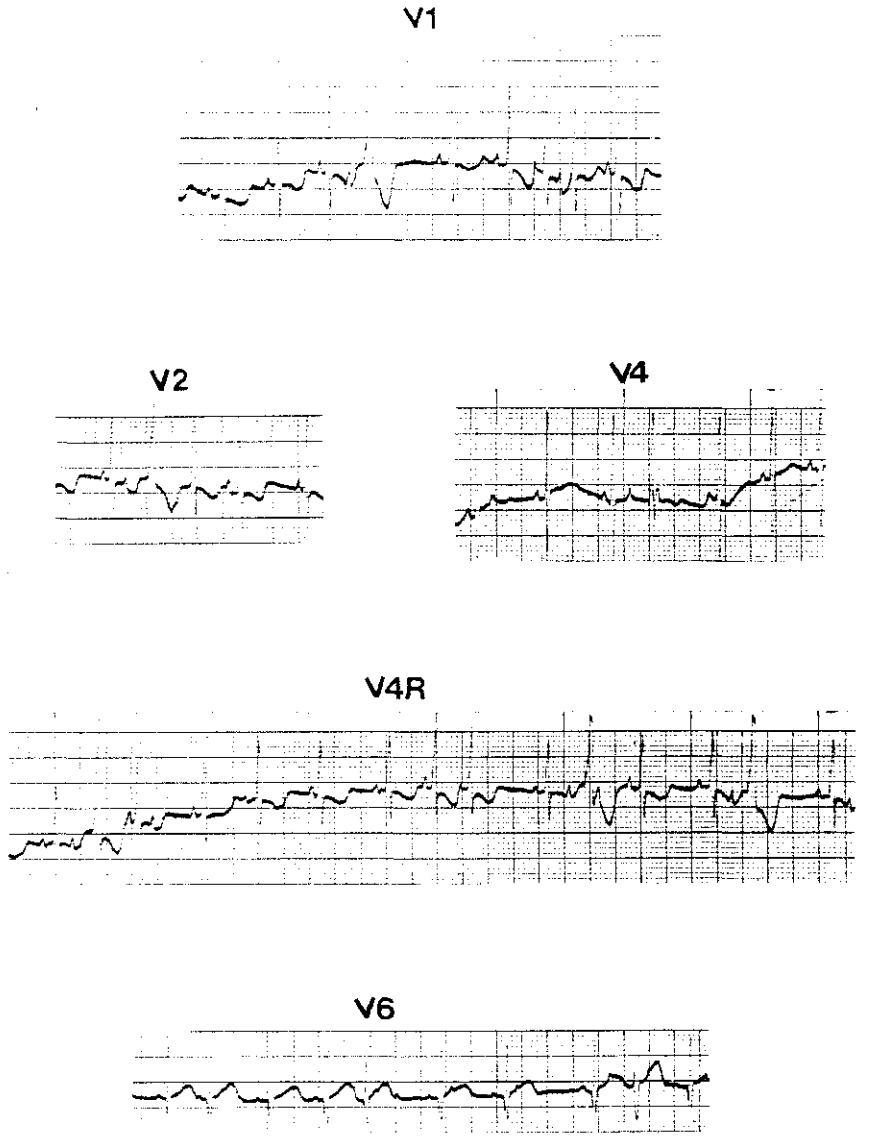
Twenty-four hour ECG recording at 6 days of age (also subject 16) showing a 3 second episode of ventricular tachycardia. During the tachycardia the QRS is prolonged and abnormal in configuration, there is A-V dissociation, and at the onset of the tachycardia atrial depolarisation follows the first QRS complex. The penultimate beat of the tachycardia represents fusion, confirming a ventricular origin for the arrhythmia. Similar episodes with a maximum duration of 10 minutes continued until 15 months of age.

Figure 16



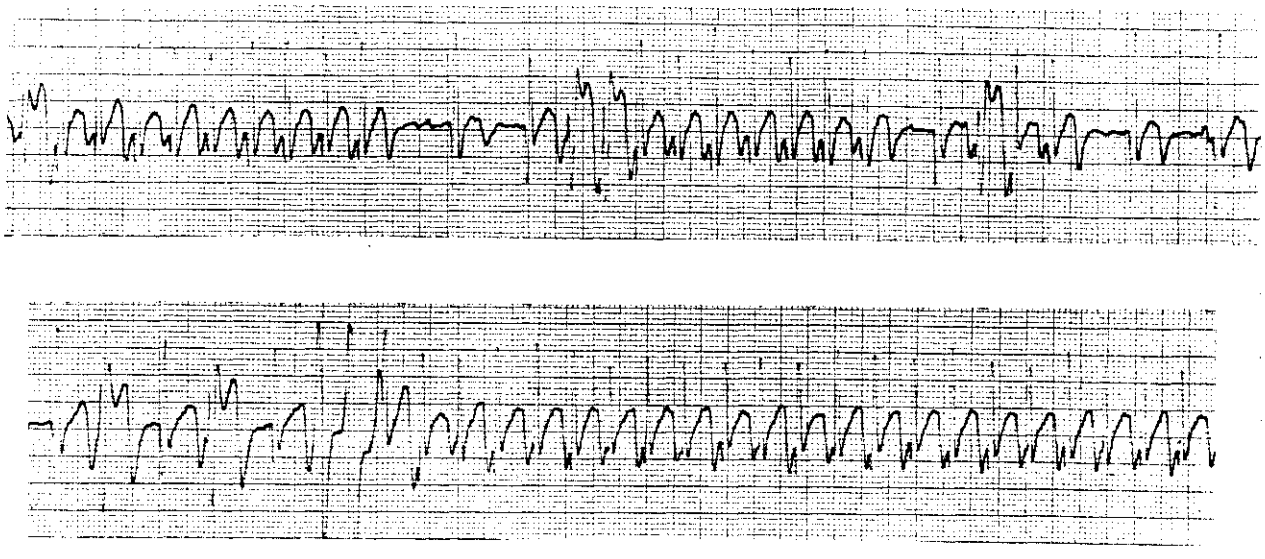
Standard ECG recording on a healthy infant aged 7 days (subject 23). There are multiple ventricular premature beats. There is no constant coupling interval, a mathematical relationship exists between the premature beats but fusion beats were not detected. Some of these features suggest a parasystolic origin for the arrhythmia. Ventricular tachycardia was not shown on 24-hour ECG and this arrhythmia had disappeared by 9 weeks of age.

Figure 17a



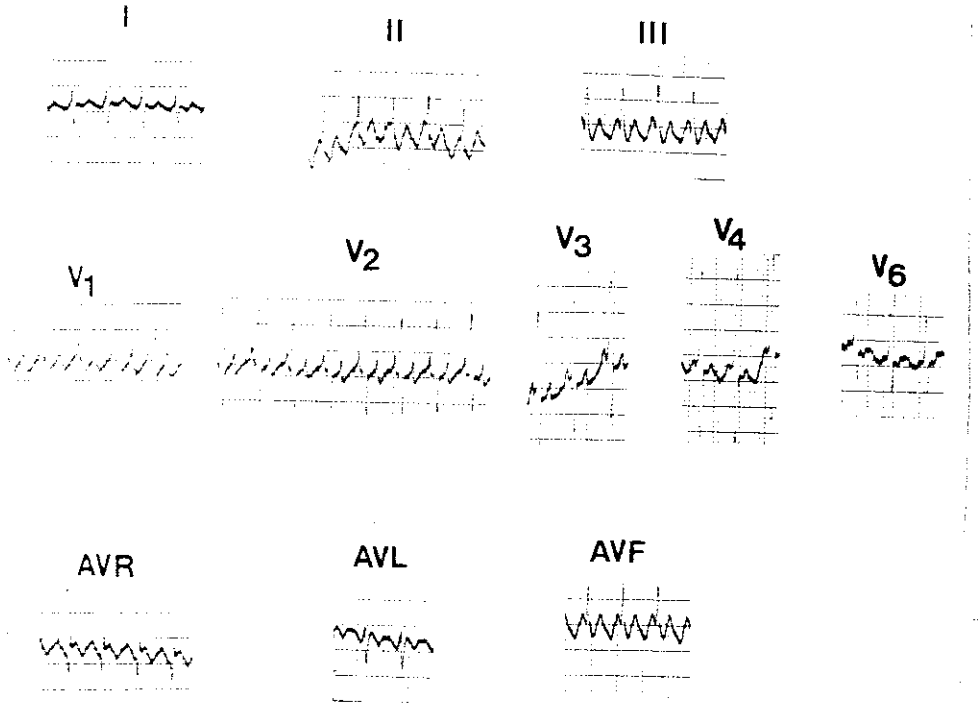
A standard ECG recording on a 3 day old male infant (subject 31). There is a chaotic atrial rhythm or multifocal atrial tachycardia. Frequent atrial premature beats are present with different P wave morphologies. There are varying PR and P-P intervals. Premature beats conducted with aberration are also present and usually follow long PR intervals suggesting they are supraventricular rather than ventricular in origin.

Figure 17b



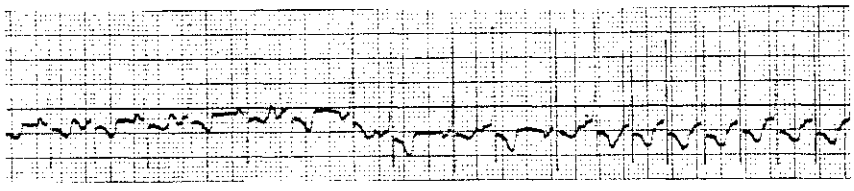
A section of 24-hour ECG recording at 6 weeks of age (also subject 31) showing episodes of SVT (250/minute). The third episode of SVT shown at the end of the lower panel continued for 10 minutes. This episode begins with 2 premature beats conducted with aberration. Following the administration of propranolol the multifocal atrial tachycardia continued but episodes of SVT were abolished. Chaotic atrial rhythm has continued and this child is now 3 years of age.

Figure 18



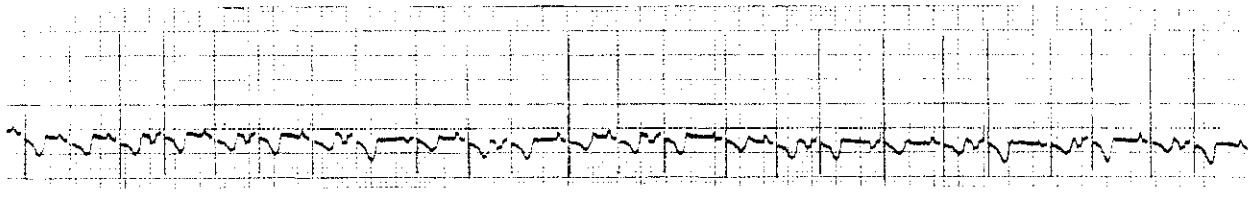
Standard ECG recording on a 5 day old healthy infant (subject 27) without heart failure showing atrial flutter with 2:1 atrio-ventricular conduction. Atrial activity is rapid (380/minute) and shows a regular undulating pattern without an isoelectric baseline. This infant was given digoxin and within 48 hours sinus rhythm had been established. Digoxin was withdrawn at 9 months of age without recurrence of the arrhythmia.

Figure 19



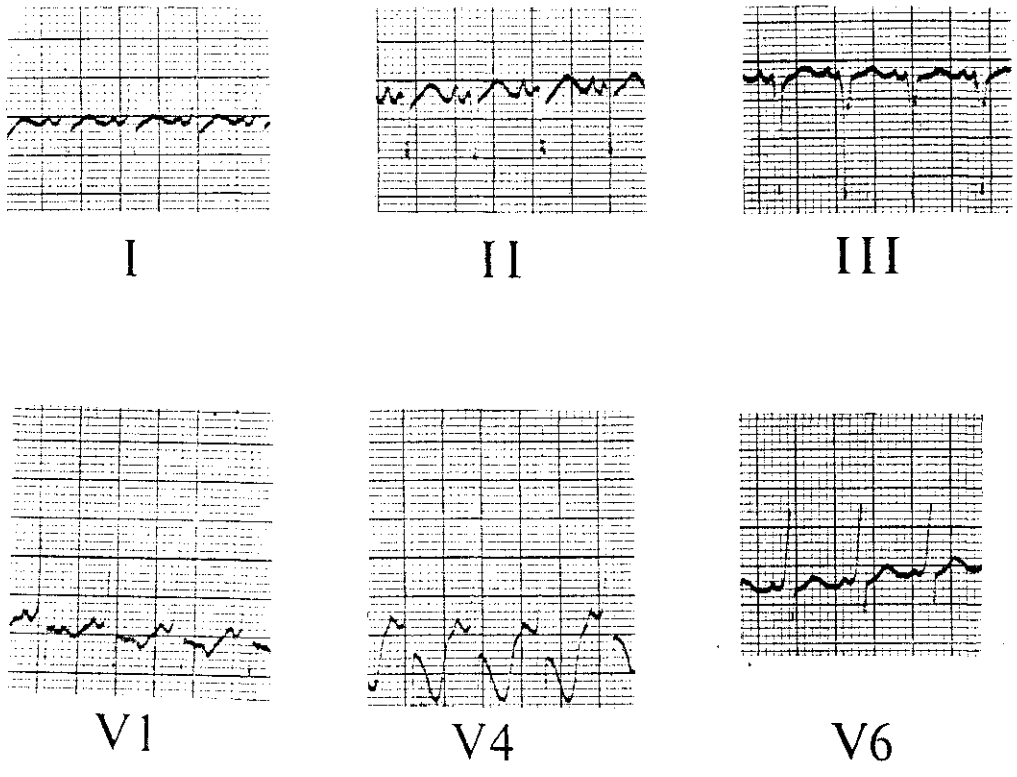
8 days

V_1



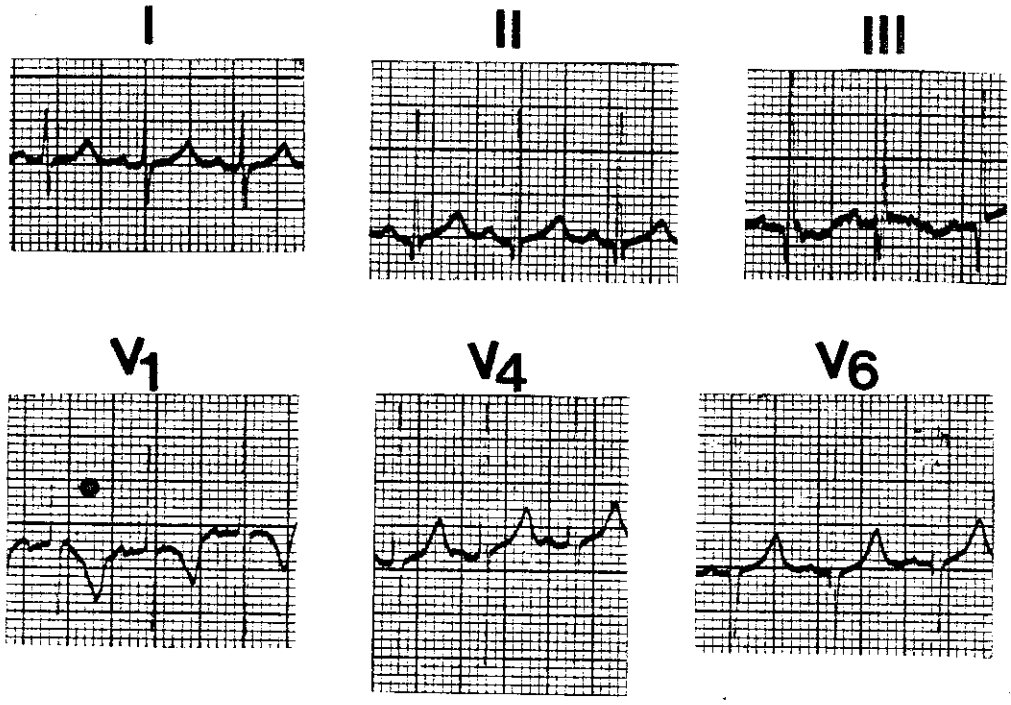
A standard ECG recording on an 8 day old infant (subject 28) showing frequent supraventricular premature beats. The upper panel shows a short episode of SVT. The paroxysm is initiated by a premature beat, with a possibly prolonged PR interval. There is a variation in cycle length at the onset of the paroxysm but this soon becomes fixed and regular. 1:1 atrio-ventricular association is present. The features of this arrhythmia suggest a paroxysmal A-V nodal tachycardia. During sinus rhythm there was no evidence of pre-excitation.

Figure 20a



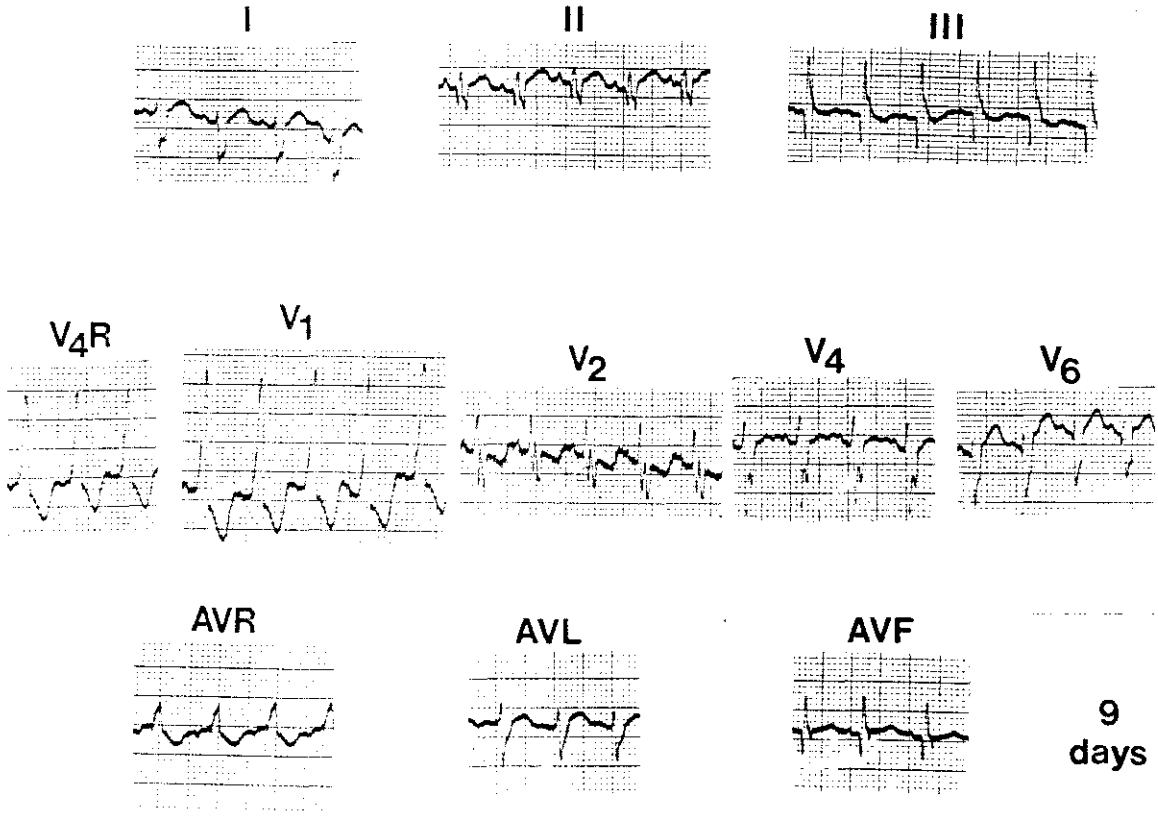
Standard ECG recording on an apparently healthy infant (subject 33) at 6 days of age showing type A Wolff-Parkinson-White Syndrome.

Figure 20b



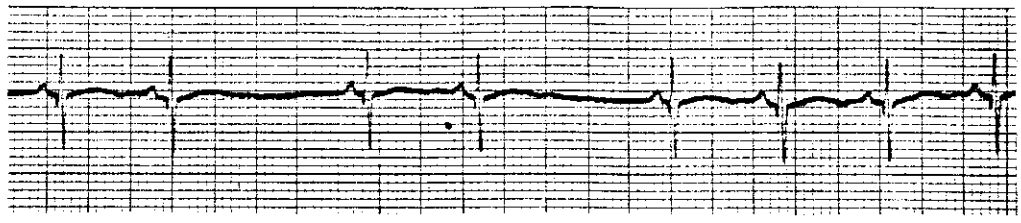
Standard ECG recording at 6 weeks of age (also on subject 33) showing no evidence of preexcitation.

Figure 21



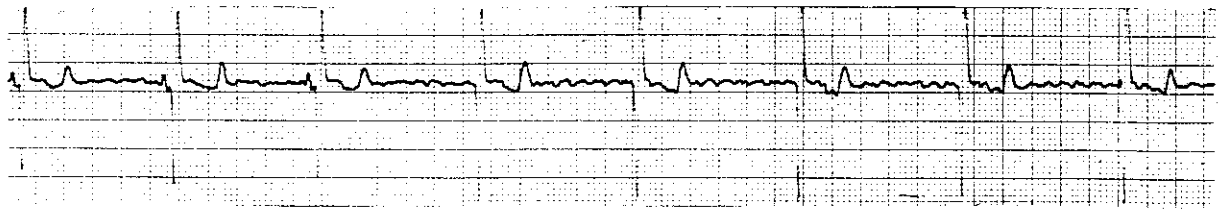
Standard ECG recording on a nine day old infant (subject 34) without structural heart disease showing complete right bundle branch block with a QRS duration of 0.10 seconds. The frontal QRS axis is +165°.

Figure 22a



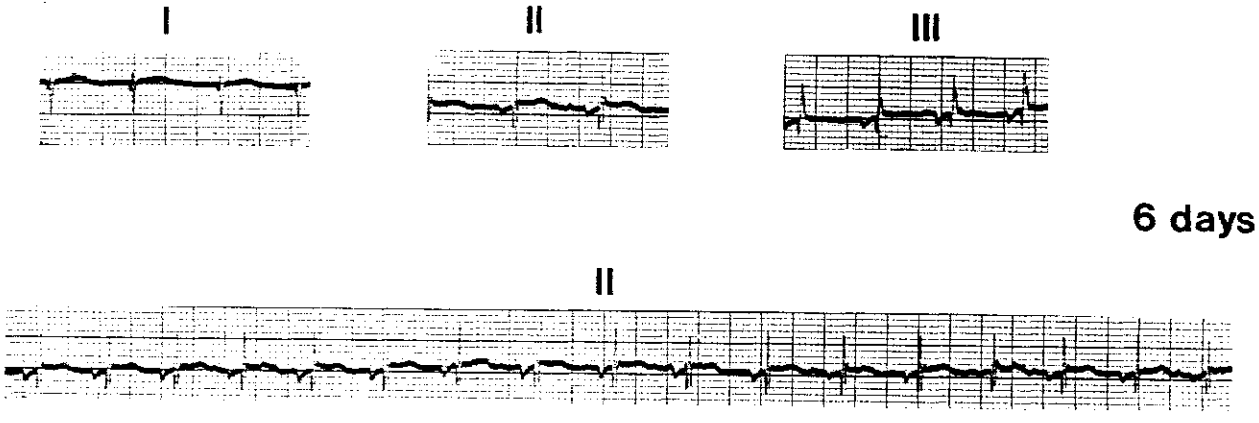
Standard ECG recording on a 1 day old infant (subject 35) showing two short pauses which result in an overall bradycardia of <100/ minute (as measured over 8 R-R intervals).

Figure 22b



Twenty-four-hour ECG recording at 6 days of age showing (on subject 35) a 20 second episode of bradycardia including a junctional escape rhythm. Measured over 8 R-R intervals the heart rate falls to 52 beats/minute.

Figure 23



A standard ECG recording on an apparently healthy 6 day old infant showing a superior P wave axis with a normal PR interval. This pattern of atrial conduction suggests that the pacemaker originates from an ectopic atrial origin probably in the region of the coronary sinus.

One infant (subject 34) showed complete right bundle branch block (QRS duration 0.10 seconds) but had no clinical evidence of structural congenital heart disease (Figure 21). This conduction pattern has continued. The child has remained well and is now 5 years of age. Twelve infants showed a superior mean frontal QRS axis (-45 to 135°). Structural congenital heart disease was present in three of these subjects (atrioventricular canal defect in one and ostium primum atrial septal defects in two). Twelve infants had short episodes of bradycardia <100 beats/minute on the standard ECG recordings (measured over a minimum of 4 R-R intervals) (Figure 22a). When investigated by 24-hour ECG recordings three of this latter group showed more marked slowing of heart rate to <60 beats/minute (Figure 22b). Finally, one subject without structural congenital heart disease showed a persistently superior P-wave axis (Figure 23).

2. Twenty-four hour ECG recordings on 134 neonates without disorders of cardiac rhythm or conduction on a standard ECG

Sinus rhythm was predominant, accompanied in all cases by a minimal, continuous irregularity of heart rate.

The frequency distribution of heart rates (from the automatic analysis of R-R intervals) at rest and during activity are illustrated in Figures 4 and 5. The maximum heart rate measured in this way was 250/minute and the minimum 85/minute. Direct ECG analysis of all heart rates measured in this way showed them to be spuriously high because of the incorrect interpretation of movement artefact and large T waves as QRS complexes.

The histogram of the highest heart rate attained over 24 hours measured by direct ECG analysis over 8 R-R intervals on each of the 134 infants forms a curve of normal distribution with a mean heart rate of 175 ± 19 (S.D.) (Figure 5). The highest heart rate found was 225/minute. The histograms of the lowest heart rates also measured by direct ECG analysis over 24 hours form curves of normal distribution and are shown in Figures 7-9 for 2, 4 and 8 R-R interval measurements respectively. The mean lowest heart rate when measured over 2 R-R intervals was 82 ± 12 (S.D.), over 4 R-R intervals was 87 ± 12 (S.D.) and over 8 R-R intervals was 93 ± 12 (S.D.).

All infants showed sudden short episodes of bradycardia (Figure 24).

At their lowest heart rates 109 infants (81%) had sinus bradycardia and 25 (19%) had junctional escape rhythm.

Three infants showed lowest heart rates of <65/minute.

(1) a female infant aged 8 days who showed 1 episode during the 24-hour recording (9 R-R intervals in duration) in which the heart rate fell to 42 beats/minute. This episode began with 2 supraventricular premature beats, was followed by a sinus pause of 1.92 seconds and was completed by a slow junctional escape rhythm (Figure 25).

(2) A male infant aged 2 days who showed 1 episode (13 R-R intervals in duration) in which the heart rate fell to 59 beats/minute. His only previous sibling had

died suddenly and unexpectedly during infancy (SIDS).

(3) A female infant aged 4 hours old when the 24-hour recording was started who showed 7 episodes of bradycardia (between 11 and 17 R-R intervals in duration) in which the heart rates fell to between 59 and 53 beats/minute. The first 3 episodes of bradycardia coincided with cyanotic attacks, observed by the nursing staff and treated by stimulation and oronasal suction. Although a further 4 episodes of bradycardia occurred between the hours of 12 midnight and 6 a.m. no further cyanotic attacks were detected.

Changes in P wave configuration and variation in P-R intervals occurred in 33 (25%) infants. (Figure 26).

Nineteen infants (14%) had supraventricular premature beats but only one had more than 12/hour (Table 18).

There was no significant difference in highest and lowest heart rates between the two age groups (1-3 days and 4-10 days) (Table 19).

Of the sub-group of 71 infants examined for pauses

(1) 5 infants (7%) had pauses with P-P or P-Q intervals exceeding the preceding P-P intervals by >110% (Figure 10).

(2) 8 infants (11%) had pauses with P-P or P-Q intervals exceeding the preceding P-P intervals by between 90 and 110% (Figure 11).

(3) 51 infants (72%) had pauses with P-P or P-Q intervals exceeding the preceding P-P intervals by between 50 and 90%. The P-P interval patterns preceding these pauses were as follows (Figures 12 a-d).

(i) A progressive increase in P-P intervals in 23 infants (32%)

(ii) Regular P-P intervals in 14 infants (20%)

(iii) Irregular P-P intervals in 35 infants (50%)

(iv) A progressive decrease in P-P intervals in 23 infants (32%)

(Figures 12c and 13) In all 23 infants with this pattern graphs of succeeding P-P interval measurements (Figure 14) supported the presence of so called Wenckebach sino atrial exit block rather than sinus arrhythmia.[72]

Some subjects exhibited more than one pattern.

The longest sinus pause detected in the total population of 134 infants was 1.92 seconds (Figure 25).

TABLE 18

ANALYSIS OF PREMATURE BEATS FROM A SINGLE 24 HOUR ECG RECORDING ON 134 INFANTS

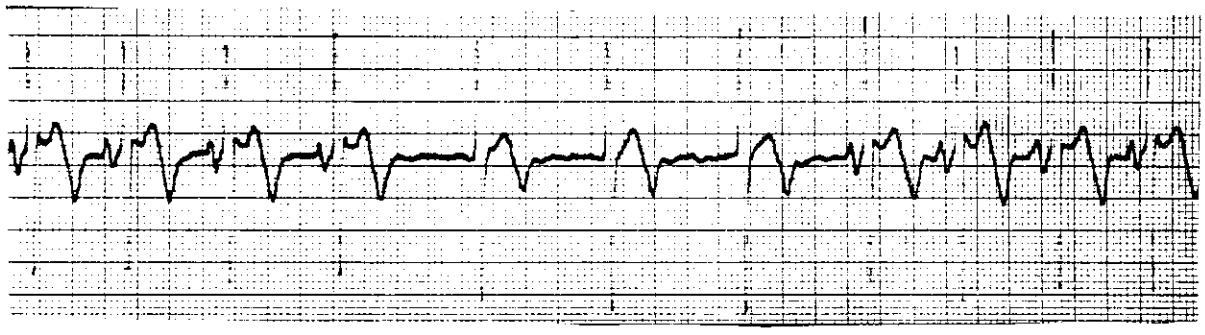
Number of Premature Beats in 24 hours	Number of Infants
<12	12
12 - 24	1
24 - 48	2
96 - 120	2
144 - 168	1
480 - 600	1

TABLE 19

COMPARISON OF LOWEST AND HIGHEST RATES OVER 24 HOURS IN AGE GROUP 1 - 3 DAYS AND AGE GROUP 4 - 10 DAYS

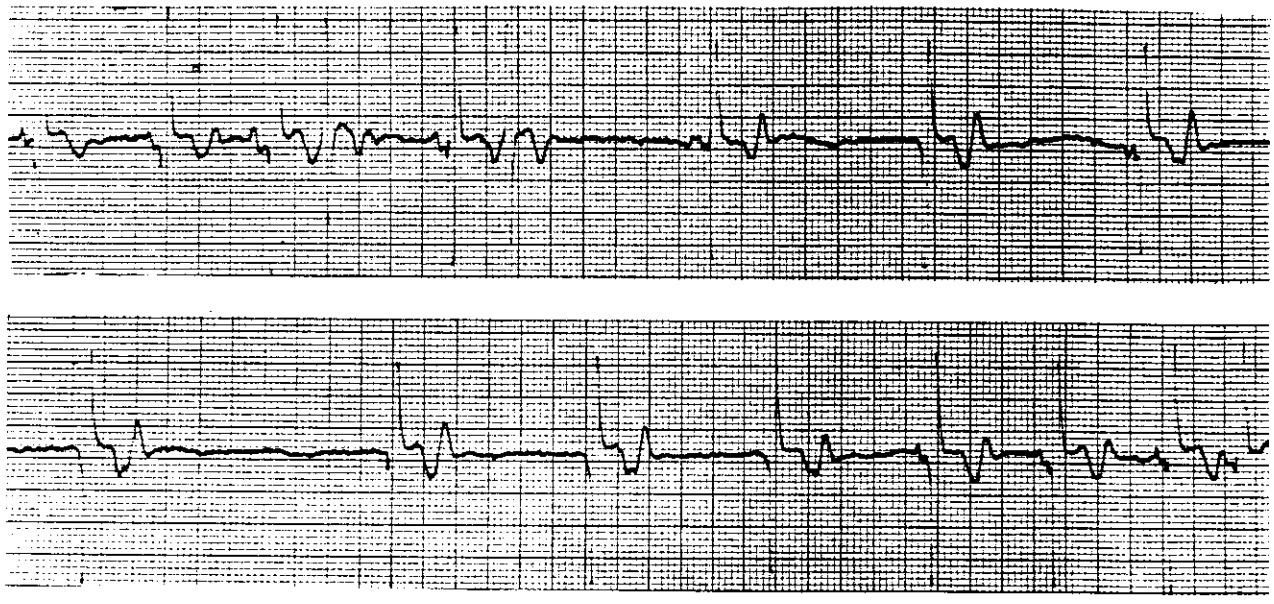
	Highest Rate/Minute (measured over 9 beats)				Lowest Rate/Minute (measured over 3 beats)				Lowest Rate/Minute (measured over 9 beats)			
Age (days)	Min.	Max.	Mean	S.D.	Min.	Max.	Mean	S.D.	Min.	Max.	Mean	S.D.
1 - 3	120	220	173	19	53	105	81	11	59	119	93	12
4 - 10	140	205	179	16	36	110	82	14	42	120	94	13
p value	$p < 0.10$				p not significant				p not significant			

Figure 24



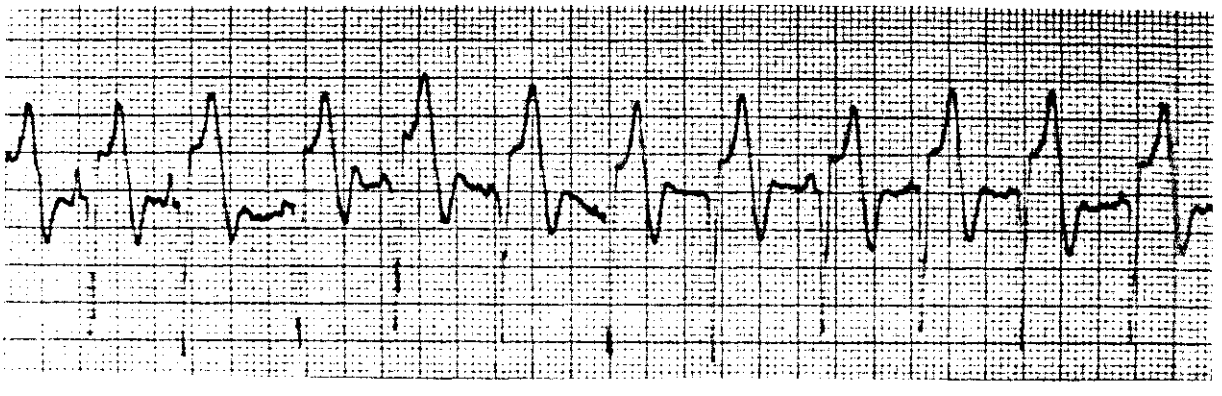
Twenty-four-hour ECG recording showing a sudden, short-lived episode of sinus bradycardia with a junctional escape rhythm. Measured over 4 R-R intervals the heart rate falls to 72 beats/minute.

Figure 25



Twenty-four-hour ECG recording showing a sudden episode of bradycardia of approximately 12 seconds duration where the heart rate falls to 42/minute (as measured over 8 R-R intervals). This episode of bradycardia begins with 2 supraventricular premature beats and is followed by a pause and a slow junctional escape rhythm. The first escape beat is preceded by a P wave but is probably junctional in origin. This episode of bradycardia includes a pause of 1.92 seconds.

Figure 26



Twenty-four-hour ECG recording showing variation in both P wave configuration and the durations of PR intervals.

DISCUSSION

Disorders of cardiac rhythm and conduction occur with an incidence of approximately 1% in the newborn infant. Sometimes, these disorders result in heart failure, but more often, as demonstrated in this study, they remain unnoticed, having apparently no harmful effect on the health of the infant. Similar findings of Jones et al[71] in Brighton confirm that these ECG patterns are not related to a local environmental factor.

Conduction and rhythm patterns of all types are present with premature beats being the most frequent finding. Atrioventricular block has been previously reported at this age,[69] but was not detected in this study.

One of the most interesting features of these ECG patterns is their time scale. In 23 of the 27 (85%) subjects followed up into later infancy or early childhood disorders of cardiac rhythm or conduction could not be detected after 12 weeks of age. Anatomical studies have shown that the conducting system undergoes maturation throughout intrauterine life.[73, 74] It is possible that late development of the conducting tissue is responsible for the transient disorders of cardiac rhythm and conduction detected in a small proportion of normal infants.

The natural history of arrhythmias detected in 4 infants in this study may have been modified by treatment with antiarrhythmic drugs. These rhythm patterns were treated because they are known to produce heart failure and because of the suspicion that they may have predisposed to sudden death.

It may have been incorrect to administer potentially harmful drugs to these well infants.

A total of 26 infants showed either supraventricular or ventricular premature beats or both. Their transient nature in the majority of subjects studied suggests that an underlying immaturity of the conducting system may have provided the necessary anatomical substrate for the arrhythmias.[73,74] Four of this group of 26 infants showed premature beats with characteristics suggesting a parasystolic[6-9] origin in which both increased automaticity and abnormal exit or entrance block may have been present.[75]

Three of 7 subjects with ventricular premature beats when studied by 24-hour recordings also demonstrated ventricular tachycardia. Though this latter arrhythmia has been associated in some children with sudden death[76] a decision was taken not to treat these otherwise well infants. This decision was based partly on a paucity of knowledge concerning the dosage and side effects of appropriate antiarrhythmic drugs in this age group, and partly because of a suggestion from the literature that the outcome in infancy is good when ventricular arrhythmias are not associated with other pathology.[69,77] All 3 subjects with ventricular tachycardia in this study are now more than 2 years of age and have remained well. In fact, all infants with ventricular premature beats suffered no apparent ill effects from their arrhythmias.

Ventricular arrhythmias in infancy and childhood have been shown to be dangerous, that is, to result in syncope, heart

failure, or sudden death in the presence of structural congenital heart disease,[77,78] metabolic disturbance[79] or myocarditis,[80,81] and a long QT interval.[60,82] When ventricular arrhythmias are present in well newborn infants who do not have additional pathology, the evidence from this study would also suggest that they are benign. Nevertheless prospective studies should perhaps be performed before ventricular arrhythmias in apparently healthy infants are considered to be harmless.

Multifocal atrial tachycardia has been rarely reported in childhood and until recently was described only in adults or children with severe pulmonary disease.[14,15] In 1977 Farooki and Green[16] described two neonates with this ECG pattern; in neither case was there evidence of heart failure and response to treatment with digoxin was reported to be incomplete. The mechanism for this arrhythmia is unknown but multiple ectopic pacemaker foci within the atria have been suggested. One of the two infants with this condition was not treated; he remained well and normal sinus rhythm was established at 3 months of age.

Atrial flutter has been frequently reported in infancy.[69] It is usually associated with heart failure and has been said to comprise between 9% and 14% of all neonatal tachyarrhythmias.[34,83] In a review of this condition, 11 of 36 subjects in whom it was diagnosed before birth or in the early neonatal period were reported to have died.[84] The underlying mechanism for atrial flutter cannot be known without electrophysiological studies but both re-entry within the atria[85] and a rapidly discharging unifocal ectopic focus[86] have been described. Serious haemodynamic consequences may occur with atrial flutter because of the

inherent possibility of 1:1 atrioventricular conduction and this was the principal reason for treating the infant found to have this ECG pattern in this study.

Paroxysmal A-V junctional tachycardia was seen in one infant in this study. No evidence of pre-excitation was detected during sinus rhythm and the characteristics of the arrhythmia on the surface ECG (Figure 19) were suggestive of re-entry within the A-V junctional area. Nevertheless the presence of an extranodal accessory pathway may be concealed (that is there is no evidence of pre-excitation during sinus rhythm) and sometimes, although pre-excitation is present, intranodal pathways are used for sustaining the arrhythmia.[87] Unfortunately electrophysiological studies were not performed in this case and the precise mechanism of this arrhythmia remains unknown. A series of post mortem studies on infants dying suddenly from a variety of causes has shown changes in the conducting tissue structure of the A-V node which are evident predominantly during infancy.[88,89] These histological patterns, which have been termed 'moulding of the A-V node', may provide the anatomical substrate for this type of arrhythmia. Despite a predominant heart rate of 220 beats/minute, this baby did not develop heart failure but, nevertheless, was treated with digoxin (70% of such cases in the neonatal period are reported to develop cardiac failure). A permanent re-entry A-V nodal tachycardia was not detected in the infants studied here but was described in one case from the Brighton study.[71]

Two infants demonstrated the Wolff-Parkinson-White syndrome. This incidence of 0.07% is similar to that reported

from other studies on unselected populations of children.[90-93] A male preponderance has also been reported[92,94] and both cases found in this study occurred in male infants. In the neonate without congenital heart disease the type A Wolff-Parkinson-White pattern is most frequently reported,[83,95] and again both cases in this series showed this configuration. Both infants were followed into the second year of life and in neither case was evidence of SVT found on standard or 24-hour ECG recordings. These findings concur with a previous report from Walsh[96] but are at variance with the majority of the published literature which describes a high frequency of SVT (in one case 100%)[97] in association with neonatal pre-excitation. Though standard and 24-hour ECG recordings from 3 months of age have failed to demonstrate pre-excitation the extranodal accessory pathway may not have disappeared but may have failed to conduct the sinus impulse at the time of recording. Engle,[98] in a review of 3,400 older children, did not demonstrate an example of pre-excitation. His findings could suggest that pre-excitation when not associated with tachyarrhythmias or structural congenital heart disease, is predominantly a conduction disorder of early infancy. As suggested by many authors,[73,74] it is possible that the accessory pathway, in some cases either ceases to exist or fails to provide a conducting pathway as the infant matures.

Complete right bundle branch block was present in one subject. This conduction pattern (which may reflect the absence of portions of the intraventricular conducting system occasionally seen in postmortem studies[101]) has not been previously reported in

healthy children without associated structural congenital heart disease. Nevertheless right bundle branch block is frequently seen in healthy adults.[100] Subjects with delay in intraventricular conduction are prone to re-entrant arrhythmias such as ventricular premature beats or ventricular tachycardia.[101,102] None were seen in this infant on 24-hour or standard ECG recordings. This conduction pattern has continued into later childhood and may therefore persist into adult life.

The variations in heart rate found in the population of 134 normal infants studied by 24-hour recordings were, not surprisingly, wider than previously reported from data on the standard electrocardiogram.[103] The mean lowest heart rate measured by direct ECG analysis over 8 R-R intervals was 93/minute with a standard deviation of 12. Heart rates of as low as 70/minute for short periods of time are therefore to be expected in the normal newborn infant. Although 25% of infants at their lowest rates had short episodes of junctional escape rhythm, prolonged periods of junctional rhythm were not found. A persistent ectopic atrial rhythm was however seen in one subject studied only by a standard ECG recording. Unfortunately follow-up recordings could not be made on this particular infant and we do not know the natural history of this finding.

Sudden slowing of heart rate occurred in all newborn infants studied over the 24-hour period. Such episodes of bradycardia have previously been reported in association with sudden deep breaths, hiccoughs, bowel movements and vomiting.[104-106] Twelve of the 3,383 infants studied by standard ECG recordings also showed episodes of bradycardia <100/minute and when this group was

studied over 24 hours more marked slowing of heart rate was detected in all cases. Combined 24-hour recordings of electrocardiogram and breathing movement have shown in some cases that episodes of bradycardia are associated with cessation of breathing movement (apnoea).[107] In other instances no obvious changes in breathing movement were detected in association with bradycardia and it is possible that, as previously described by Guilleminault,[108] these heart rate changes may be related to upper airway obstruction.

Sudden pauses were found in the majority of infants studied and may represent the surface ECG manifestations of sinus arrest or sino-atrial exit block. In some reports their presence has led to a diagnosis of sinus node dysfunction.[109,110]

Studies of cardiac rhythm patterns after the surgical treatment for congenital heart disease have also described junctional rhythm and pauses similar to those described in normal infants.[111-113] However these "normal" findings have then been assumed to result from disorders of sinus node function assumed to be acquired as a result of the operation. In a study of pre and post-operative 24-hour ECG recordings in infants and children with transposition of the great arteries it was thus not surprising to find these patterns as frequently pre-operatively as post-operatively.[114]

Since this study has now shown that junctional escape rhythms and ECG patterns indistinguishable from those previously described as sinus arrest or sino atrial exit block occur frequently in healthy infants other criteria, on standard

or 24-hour ECG recordings, are necessary to determine if there is an abnormality of sinus node function. Perhaps the most valuable of these is a measurement of the time taken for an escape rhythm to be initiated following a pause. The longest escape interval found in the healthy newborn infants studied here was 1.92 seconds. In a recent report of an infant with the "sick sinus syndrome" (that is, a disorder of impulse generation in the sinus node and atrioventricular junctional escape pacemaker tissue) episodes of asystole exceeding 4 seconds were described.[115]

In Table 20 the findings of the present study have been compared with three earlier reports describing long term monitoring of the electrocardiogram in the newborn infant. The maximum and minimum heart rates found by Valimaki[116,117] are higher than those detected by the method of direct ECG analysis used in this study. The measurement of heart rate variability using an automatic analysis of R-R intervals has shown that both movement artefact and large T waves may be interpreted as extra QRS complexes thus producing spuriously high heart rates. This may explain the difference between the findings of Valimaki and those of this present project. The shorter time of recording used by Morgan and Guntheroth[118] may explain the failure to detect junctional escape rhythms or variations in P wave and P-R intervals.

In conclusion, disorders of cardiac rhythm and conduction occur with an incidence of approximately 1% in the newborn infant. Most have disappeared by 3 months and virtually all by one year of age. In all cases detected by standard ECG

TABLE 20

COMPARISON OF REPORTS ON LONG-TERM ECG MONITORING IN THE NEONATE

	Southall et al 1978	Valimaki 1969 [101]	Valimaki and Tarlo 1971 [102]	Morgan and Guntheroth 1965 [103]
Duration of recording (hours)	24	36	30	5
Number studied	134	50	68	50
Age at recording (days)	1-10	1-2	1-2	1-7
Maximum heart rate per minute	220	260	260	170
Minimum heart rate per minute	42	86	80	75
Junctional escape rhythms (%)	19	10	13	-
P wave and P-R Interval variation (%)	25	20	15	-
Premature beats (%)	14	8	9	2+

recordings no apparently harmful effects appeared to result from these disorders but in some cases the natural outcome may have been obscured by treatment with antiarrhythmic drugs. Twenty-four-hour ECG recordings on a population of newly born infants without cardiac arrhythmias on a standard ECG showed 19 additional subjects with premature beats but only one infant had more than 12/hour. These long term recordings, however, showed that the highest and lowest heart rates detected were considerably different from those previously reported from other long term ECG recordings at this age. Analysis of the recordings also demonstrated that sinus pauses and junctional escape rhythms occur frequently in the "normal" healthy neonate.

CHAPTER 4

HEART RATE AND RHYTHM PATTERNS IN 7 TO 11 YEAR OLD SCHOOL CHILDREN

INTRODUCTION

Frequent case histories describing cardiac arrhythmias and their clinical effects have been documented in children. In addition a number of reports have attributed some faints and attacks of dizziness in childhood to abnormalities of sinus node function, a diagnosis often made from the interpretation of surface ECG recordings alone.[108,109,119-121] The supposition, however, that these ECG patterns were abnormal has been made in the absence of adequate long-term ECG data on the incidence of such patterns in healthy, symptom-free children without heart disease. This study of 24-hour recordings of the electrocardiogram in a randomly selected population of school children provides normal data with which ECG patterns on subjects with symptoms can be compared.

PATIENTS AND METHODS

One hundred and four healthy school children between the ages of 7 and 11 years (mean 9.4 years) were randomly selected from the register of a primary school in Dorchester. The school served both an urban and a rural community and included children of all social classes. None of the parents of the children selected refused to co-operate with the study.

A single 24-hour recording was made on each child. The tape recorder was attached at school, usually during the morning and remained in place until the following day. Normal activities were encouraged. Attempts were made to document a diary of activity during the recordings of electrocardiogram but notes kept by the children were often inaccurate and therefore not used in the analysis.

Twelve recordings were rejected because of poor quality. Five developed lead fractures during recording, 2 had electrode displacement, in 4 a fault in the cassette resulted in jamming of the tape, and in 1 oxide coating of the recording head prevented signal reception. Forty-nine recordings of good quality came from boys and 43 from girls; a total of 92 recordings. Eleven of the girls, but none of the boys, were undergoing puberty.

Questionnaires were sent to all parents requesting historical information of previous fits, faints, attacks of dizziness or other serious illness and were completed in all cases. A full clinical examination was made on each child.

Standard ECG recordings were not made.

The highest and lowest heart rates (from 2, 4 and 8 R-R intervals) occurring in each child over a 24-hour period were measured from the ECG print-out after direct analysis of the whole of the recording (Table 21 Figure 27).

The frequency of bradycardia at each of 3 levels: <55/minute, <50/minute and <45/minute (as measured over 8 or more R-R intervals) was measured over 3 randomly selected hours of day time (10.00 to 18.00 hours) and night time (00.00 to 06.00 hours) recordings in 26 subjects with heart rates below 55/minute. The maximum duration of bradycardia <55/minute was also measured in these 26 subjects (Tables 22-23).

The presence of junctional escape rhythm, when present over 3 or more beats, was documented in all children studied (Table 24 Figure 29). The frequency and maximum duration of this rhythm over the 24 hours of recording was also measured in a randomly selected subgroup of 24 children who showed junctional rhythm (Tables 22-23).

In each child the 24-hour recording was examined for the following P-P interval patterns (Table 24).

(1) A gradual increase and decrease in P-P intervals (sinus arrhythmia)[122-124]

(2) A sudden increase in P-P interval exceeding the previous P-P interval by more than 110% (sinus arrest) (Figure 30)[122-124]

(3) A sudden increase in P-P or P-Q interval exceeding the immediately preceding P-P interval by between 90 and 110% (sinus arrest)[122-124] This ECG pattern could be termed Second degree sino-atrial block but in this instance the sinus interval immediately following the pause should be equal in length to the pre-pause interval (Figure 31).[122-124]

(4) A progressive decrease in P-P interval ending with a sinus pause exceeding the previous P-P interval by between 50 and 90% (Wenckebach second degree sino-atrial exit block).[72] This pattern was only counted when a graphical differentiation from sinus arrhythmia could be made (Figure 24). Figure 32 on subject A shows the P-P interval pattern of sinus arrhythmia and Figure 33 on a different subject B shows the P-P interval pattern of Wenckebach second degree sino-atrial block.

The frequency of patterns (2), (3) and (4) in the total population of 92 children is shown in Table 24. The number of episodes of patterns (2), (3) and (4) in a sub-group of 30 children with these patterns was also measured (Table 22).

The maximum pause (the longest P-P or P-Q interval) was measured from the analysis of the whole 24-hour recording of each child.

Finally, all recordings were analysed for the presence of

premature beats (number per hour) computed by a direct count from 6 randomly selected hours of recording.

TABLE 21

RANGE OF HEART RATES FROM DIRECT ANALYSIS OF 24-HOUR ECG RECORDINGS ON 92 HEALTHY CHILDREN AGED 7 - 11 YEARS

	Highest Rate	Lowest Rate		
	9 BEATS	3 BEATS	5 BEATS	9 BEATS
Mean ± S.D.	164 ± 17	49 ± 6	52 ± 6	56 ± 6
Maximum	195	64	68	69
Minimum	130	37	38	42

TABLE 22

NUMBER OF EPISODES OF JUNCTIONAL RHYTHM, NUMBER OF SINUS PAUSES AND NUMBER OF EPISODES OF BRADYCARDIA ON SUB-GROUPS WITH THESE FINDINGS ON THE 24-HOUR RECORDING

Number of Episodes	Number of Children Showing Junctional Rhythm	Number of Children Showing Sinus Pauses			Number of Children Showing Heart Rates Below 55/Minute		Number of Children Showing Heart Rates Below 50/Minute		Number of Children Showing Heart Rates Below 45/Minute	
		A*	B*	C*	Day	Night	Day	Night	Day	Night
1-5	4	1	3	7	2	8	1	11		3
6-10	5	4	6		1	5		2		
11-15	1	3	7			2		2		
16-20		1	1			3		1		1
21-25		3	1			3				
26-30		2	1			2				
31-35		4	1			1				
36-40	1	2				1				
41-45										
46-50		1	1							
>50	13**	3	1	1		1		1		
Total No. of Children studied in each sub-group	24	30			26					

** Present at all rates below 60-65/minute
A* P-P or P-Q exceeding previous P-P by 50-90% with incrementally decreasing P-P prior to pause
B* P-P or P-Q exceeding previous P-P by 90-110%
C* P-P or P-Q exceeding previous P-P by >110%

TABLE 23

MAXIMUM DURATION OF JUNCTIONAL ESCAPE RHYTHM AND EPISODES OF BRADYCARDIA < 55/MINUTE ON SUB-GROUPS WITH THESE FINDINGS ON THE 24-HOUR RECORDING

Maximum Duration	Number of Children Showing Junctional Rhythm	Heart Rate < 55/Minute
1 - 10 seconds	12	
11 - 20 seconds		13
21 - 30 seconds	4	4
31 - 40 seconds	1	
41 - 50 seconds		1
51 - 60 seconds	2	
1 - 2 minutes		6
3 - 5 minutes	1	1
6 - 10 minutes	1	
10 - 20 minutes	2	
20 - 30 minutes	1**	1*
Total Number of Children Studied	24	26

** 25 minutes

* 40 minutes

TABLE 24

RHYTHM PATTERNS ON 24-HOUR ECG RECORDINGS

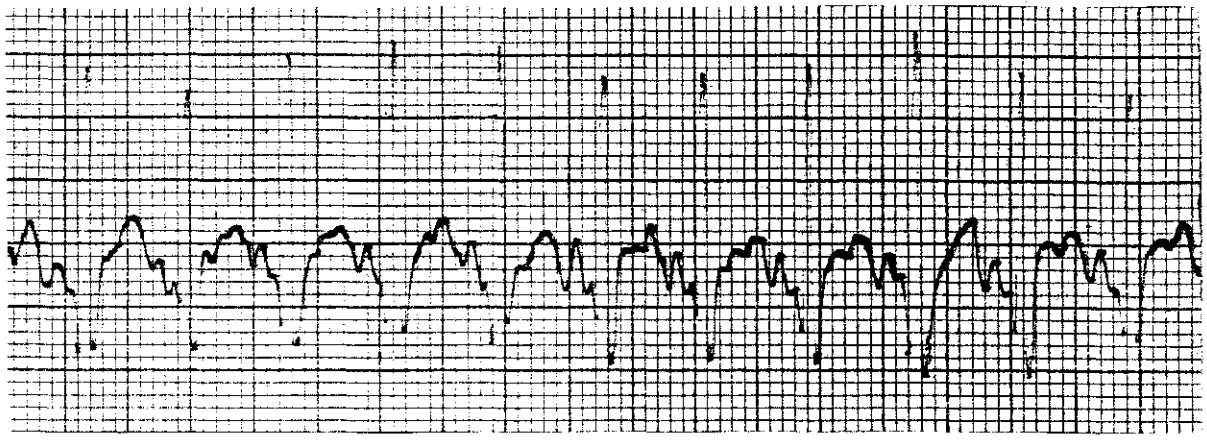
	Rhythm at Lowest Heart Rate		Sinus Pauses: Total No: Subjects = 60			Premature Beats		1ST Degree A-V Block (PR ≥ 0.20s)	2ND Degree A-V Block
	Junctional	Sinus	A*	B*	C*	0	<1/hr.		
Number of Subjects (Total n = 92)	41	51	43	34	8	73	19	9	3

A* = P-P or P-Q exceeding previous P-P by 50-90% with incrementally decreasing P-P prior to pause

B* = P-P or P-Q exceeding previous P-P by 90-110%

C* = P-P or P-Q exceeding previous P-P by ≥110%

Figure 27



Section of 24-hour recording showing the highest heart rate detected in a 9 year old boy. (A sinus tachycardia of 188/minute).

RESULTS

In all cases a full clinical examination failed to detect evidence of structural heart disease or of any other serious illness. No child studied had symptoms of any kind during the course of the recording.

Ten children gave a history of previous fainting episodes but none had regular faints. There was no difference in heart rate values or rhythm pattern findings between children who had a history of fainting and those who did not (Table 25).

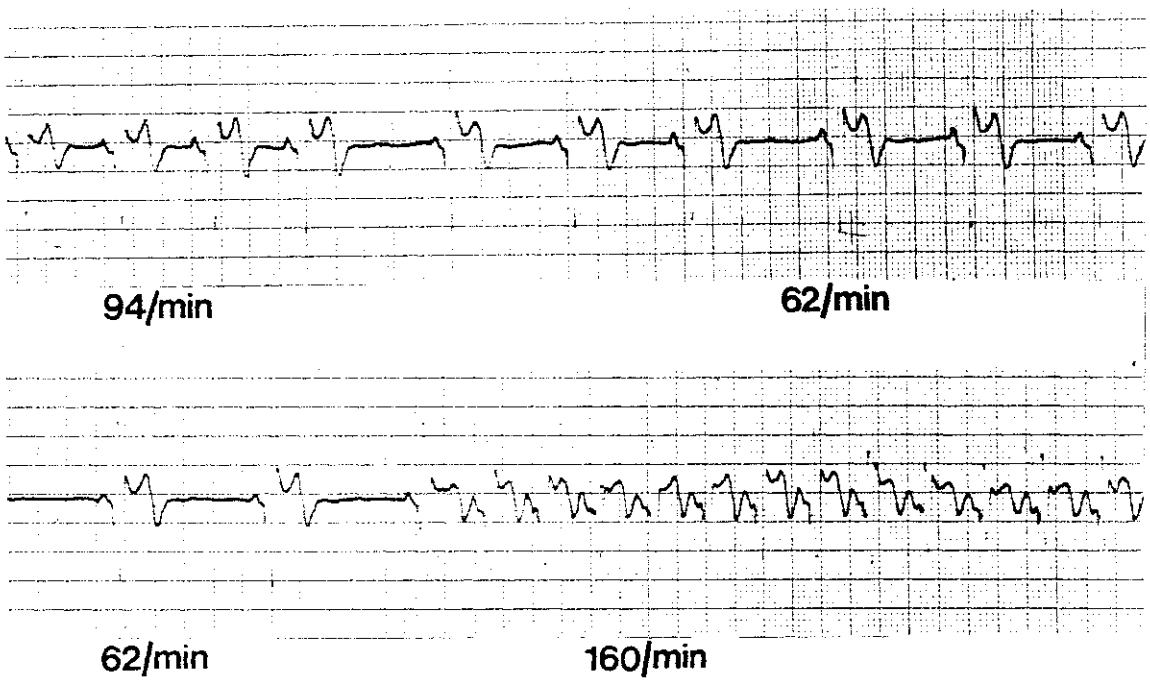
Mean values for the highest and lowest heart rates over 24 hours in each subject are shown in Table 21. Lowest and highest heart rate histograms did not significantly differ from those of normal distribution.

All children showed at times a phasic variation in heart rate, normally termed sinus arrhythmia.[104,122] Abrupt changes in heart rate were also frequently detected (Figure 28).

Episodes of bradycardia <55/minute (Tables 22 and 23)

In a sub-group of 26 subjects studied during day time and night time epochs the majority occurred during the night where heart rates <50/minute were present in most children. Four children had episodes of bradycardia <45/minute during the night. In the majority of children episodes of bradycardia were <30 seconds in duration. In one child one episode of bradycardia continued for 40 minutes.

Figure 28



Section of 24-hour recording of the electrocardiogram on a healthy boy aged 8 years showing abrupt changes in heart rate. Following sinus rhythm of 94/minute there is an irregular slowing of heart rate to 62/minute. The first two beats in the second panel have an abnormal P wave and shortened PR interval; they may represent junctional escape beats. The subsequent tachycardia of 160/minute is probably sinus in origin.

Junctional escape rhythm (Tables 22 and 23) (Figure 29)

Forty-one children (45%) had episodes of junctional rhythm (Table 24). In a sub-group of 24 children with this escape rhythm the majority had >50 episodes in 24 hours (Table 22). This rhythm was <10 seconds in duration in most children studied. In 5 children episodes continued for >1 minute, including one child where junctional rhythm was present for 25 minutes (Table 23).

P-P interval patterns (Tables 22 and 24) (Figures 30-33)

A total of 60 children (65%) showed pauses in P-P or P-Q intervals. In a random sub-group of 30 children the majority showed <30 episodes of type A, <15 episodes of type B, and <6 episodes of type C.

Duration of pauses

The mean longest pause occurring over 24 hours for each child was 1.36±0.23 (S.D.) seconds. The overall longest pause seen was 1.88 seconds.

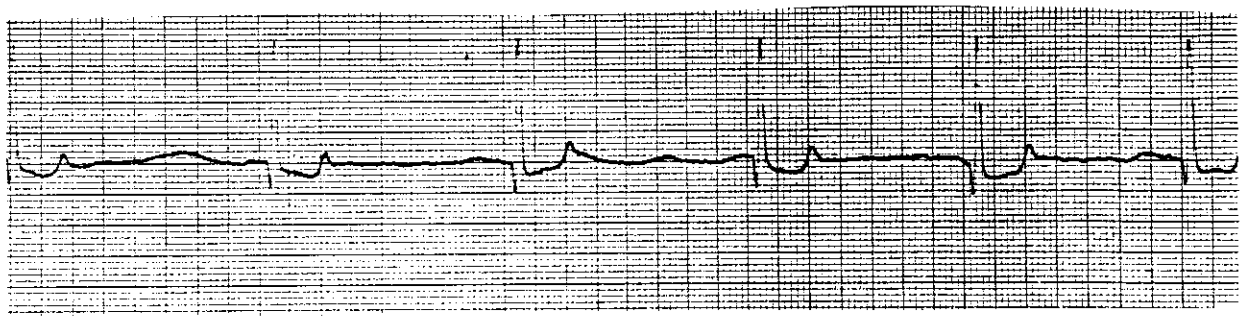
Atrio-ventricular block (Tables 24 and 26)

Nine children showed intermittent P-R prolongation ⩾0.20 seconds including 3 with Mobitz type I second degree A-V block (Figures 35-36).

Premature Beats

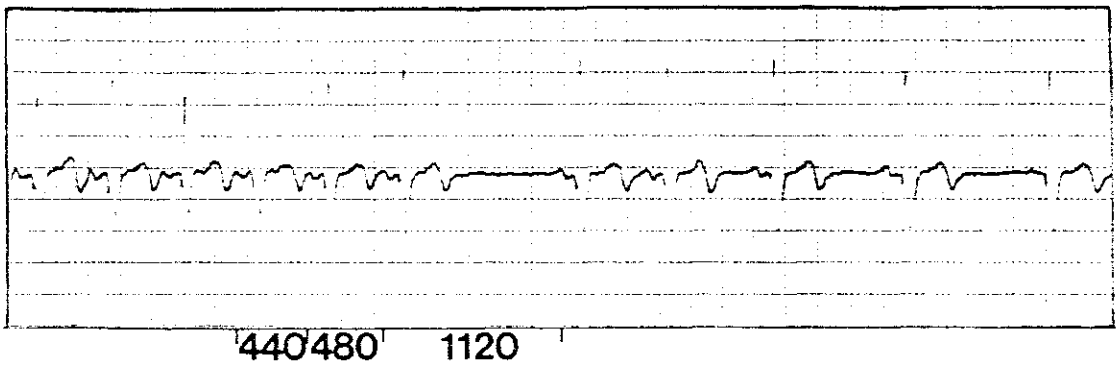
Supraventricular premature beats (<1/hour) were seen in 19 (21%) children. One additional child had ventricular premature beats (<1/hour).

Figure 29



Section of 24-hour recording of the electrocardiogram on a healthy girl aged 9 years showing an episode of junctional rhythm at a heart rate of 37/minute (as measured over 2 R-R intervals)

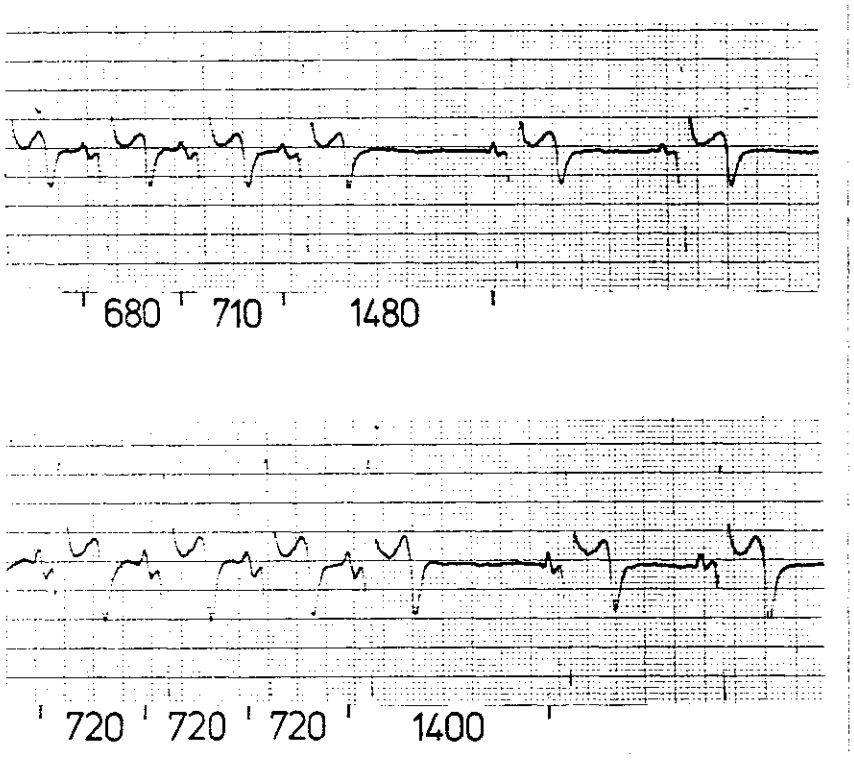
Figure 30



Section of 24-hour recording on a healthy girl aged 9 years showing a sudden prolongation of P-P interval (1120 ms.) exceeding the immediately preceding P-P interval (480 ms.) by >110% (sinus arrest).

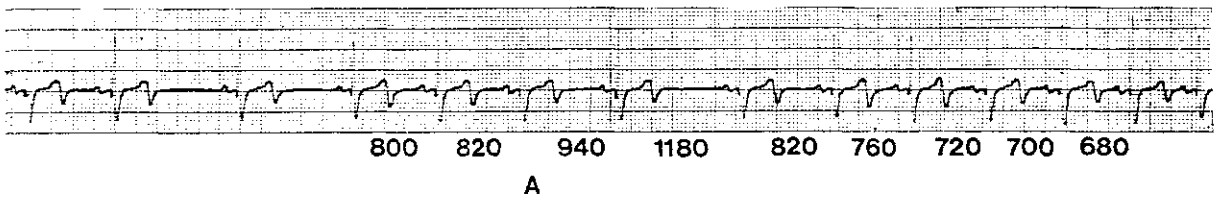
This pause could also be attributed to complete sino-atrial exit block (the arithmetical relationship between the P wave before and immediately after the pause could have been disturbed by autonomic activity).

Figure 31



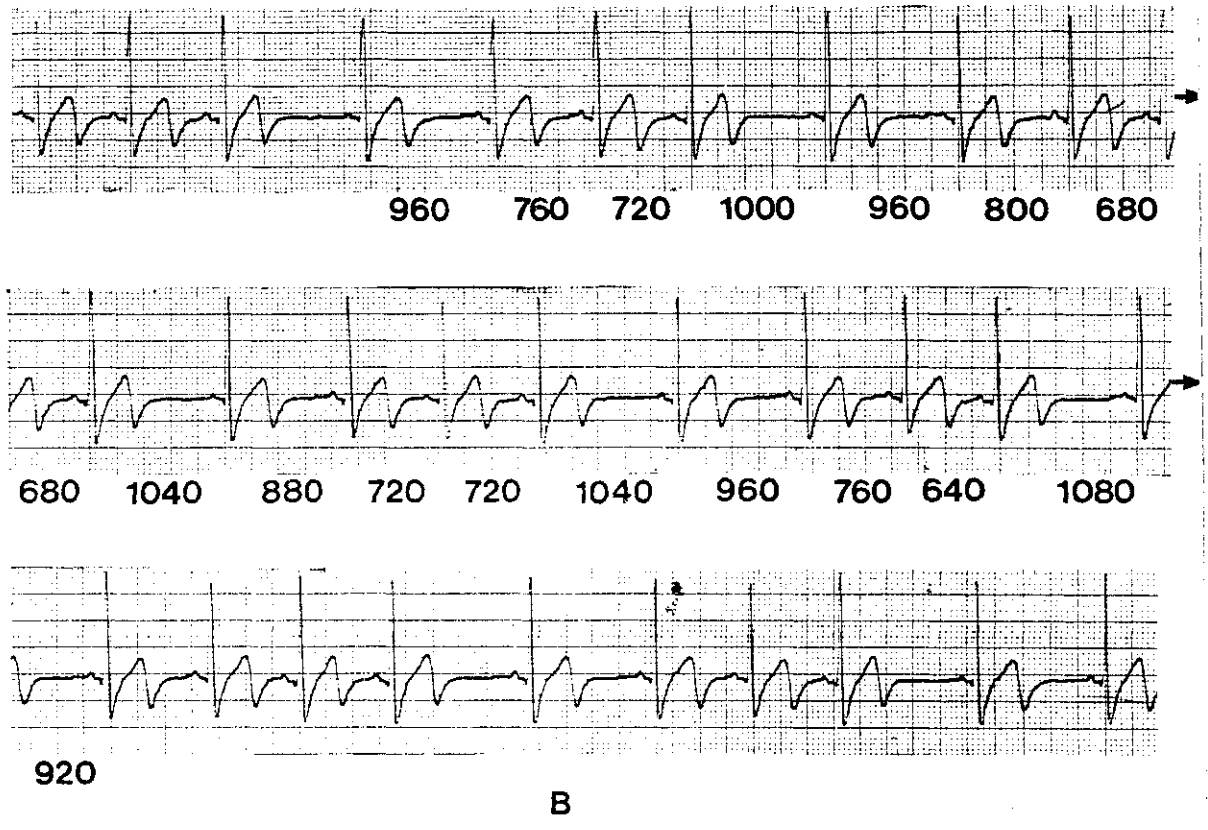
Sections of 24-hour recordings from a healthy boy aged 11 years showing pauses with sudden prolongation of P-P intervals exceeding the immediately preceding P-P intervals by between 90 and 110%. This sudden doubling of P-P interval on the surface ECG may have resulted from sinus arrest. Second degree sino-atrial exit block could also produce such a pause but the sinus interval following the pause should be equal in length to the pre-pause interval. This latter situation may have been modified by autonomic activity and it is impossible from the surface electrocardiogram alone to know the underlying mechanism for this event.

Figure 32



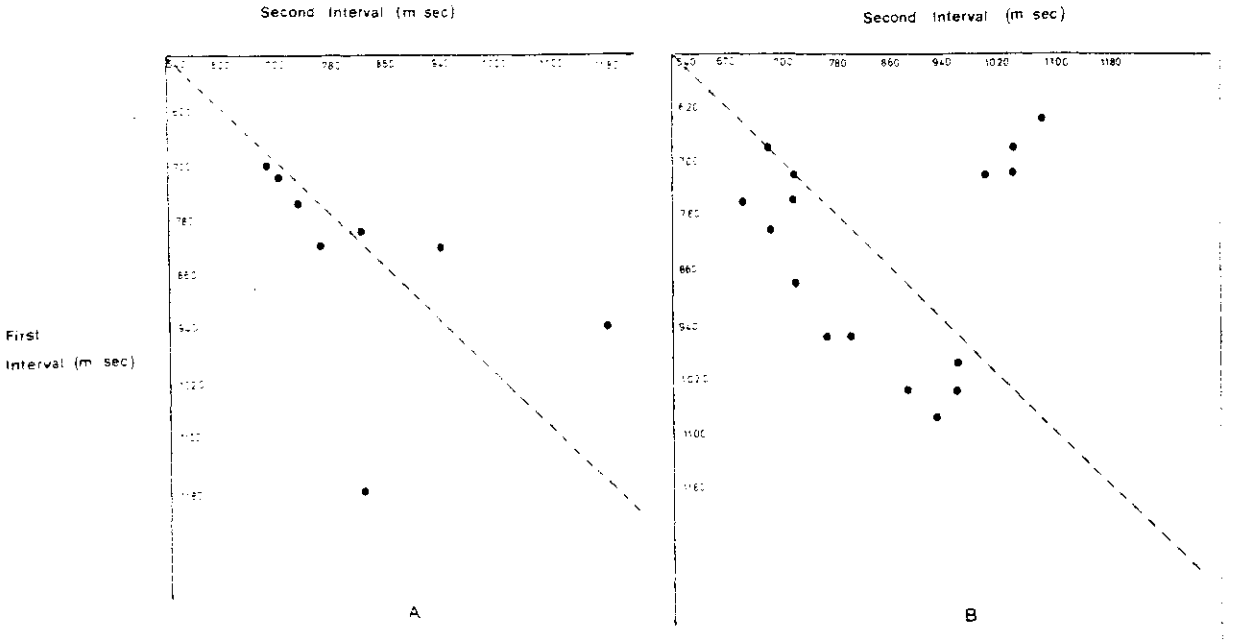
Section of 24-hour recording on subject 'A', a healthy boy aged 9 years, showing a phasic variation in heart rate termed sinus arrhythmia.

Figure 33



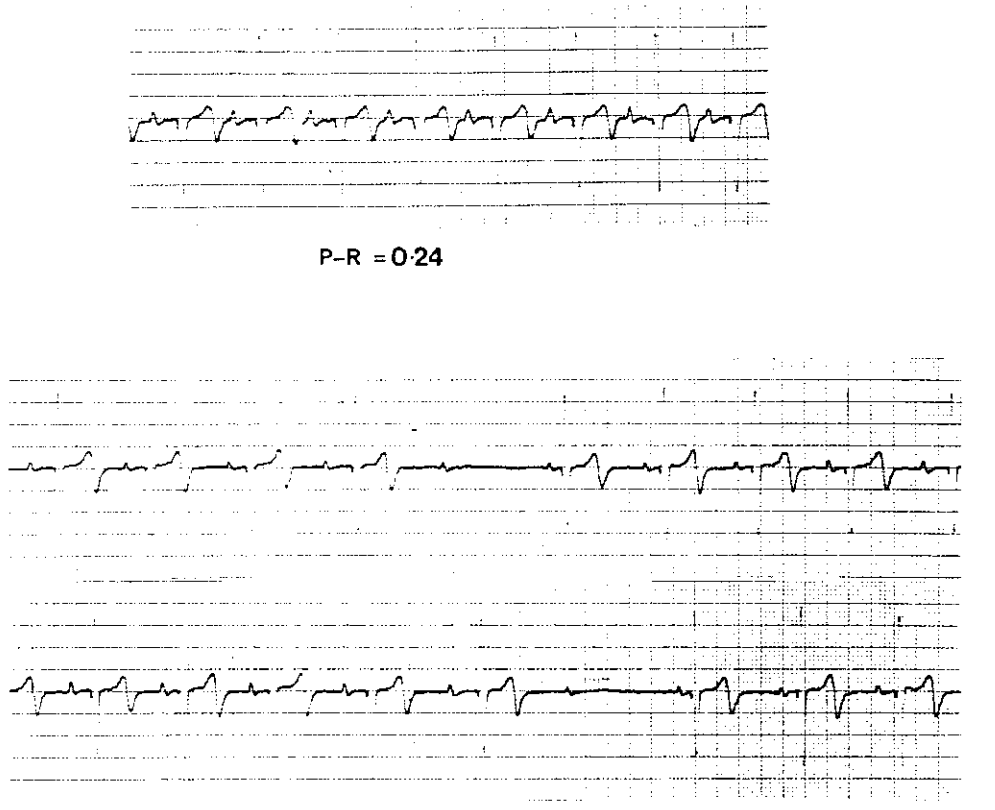
Section of 24-hour ECG recording on subject B, a healthy boy aged 8 years, showing episodes where progressive reduction in P-P intervals are followed by pauses exceeding the immediately preceding P-P interval by between 50 and 90%. The calculations of P-P interval increment describe the pattern termed Wenckebach sino-atrial exit block rather than sinus arrhythmia.

Figure 34



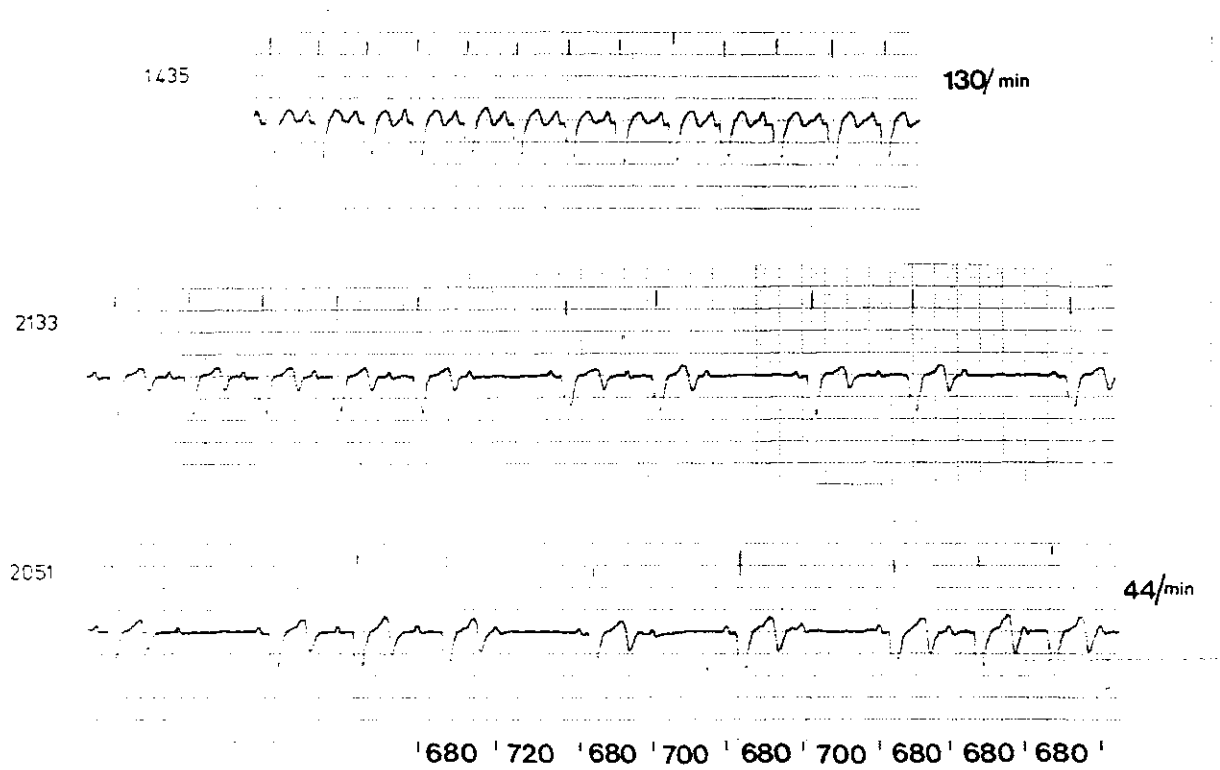
Two graphs showing a plot of P-P intervals on the Y axis against succeeding P-P intervals on the X axis. The line at 45° shows the position of P-P intervals of equal duration. Graph A from subject A (Figure 32) demonstrates the pattern termed sinus arrhythmia. Graph B from subject B (Figure 33) shows the pattern termed Wenckebach sino-atrial block.

Figure 35



Sections of 24-hour recording from a 9 year old girl. The top panel shows an episode of sinus rhythm with a P-R interval of 0.24 seconds. The lower two panels demonstrate Mobitz type I (Wenckebach) A-V block.

Figure 36



Sections of 24-hour recording from a 9 year old girl. The top panel at 1435 hours shows a sinus tachycardia of 130/minute with a normal PR interval. At other times during the recording the PR interval was 0.24 seconds. At 2133 hours and 2051 hours there are episodes of Mobitz type I (Wenckebach) A-V block.

TABLE 26

DETAILS ON CHILDREN WITH FIRST OR SECOND DEGREE A-V BLOCK ON 24-HOUR RECORDINGS

	First Degree A-V Block	Second Degree A-V Block
Subject	PR Interval(s)	Number of Episodes
1	0.24	53 **
2	0.24	5 *
3	0.24	9
4	0.20	
5	0.24	
6	0.24	
7	0.24	
8	0.20	
9	0.28	

** 49 occurred during the day

* History of 3 faints

Heart rate and rhythm variations in children with a history of fainting (Table 25)

There were no significant differences between the heart rate and rhythm pattern of 82 children who had not fainted and 10 who had.

TABLE 25

HEART RATE AND RHYTHM VARIATIONS IN CHILDREN WITH A HISTORY OF FAINTING

Subject	Age	Sex	Lowest heart rate over 24 hours (over 9 beats	Premature Beats	Sinus Pauses			Junctional Rhythm	Longest Sinus Pause (secs.)	A-V Block	Number of Faints
					A*	B*	C*				
1	9	M	51	0	+	+	+	+	1.40	-	1
2	9	F	50	0	+	+	-	+	1.44	-	1
3	9	M	62	0	+	-	-	-	1.20	-	2
4	8	M	50	0	+	+	-	+	1.70	-	4
5	11	M	52	0	+	-	-	+	1.52	-	1
6	11	F	63	0	-	-	-	-	1.28	-	1
7	11	M	56	+	-	-	-	-	1.72	-	1
8	11	M	56	+	-	-	-	-	1.50	-	1
9	11	M	47	0	-	-	-	+	1.64	PR = 0.24S	1
10	9	F	55	+	+	+	-	+	1.32	PR = 0.24S Mobitz 1 2^0 Block	3

Significance tests against 82 children who have not fainted	(a)	(b)	(c)	(d)	(e)	(f)	(g)	(h)

(a) Mann Whitney U test = 343 P is NS
(b) $X^2 = 0.549$ P is NS
(c) $X^2 = 0.762$ P is NS
(d) $X^2 = 0.043$ P is NS
(e) $X^2 = 0.150$ P is NS
(f) $X^2 = 1.02$ P is NS
(g) Mann Whitney U test = 275 P is NS
(h) $X^2 = 1.250$ P is NS

A* = P-P or P-Q exceeding previous P-P by 50-90% with incrementally decreasing P-P prior to pause
B* = P-P or P-Q exceeding previous P-P by 90-110%
C* = P-P or P-Q exceeding previous P-P by $<110\%$

DISCUSSION

Twenty-four hour recordings have shown considerable variation in heart rate and rhythm in apparently healthy children.[121] Such recordings, although recommended, have not previously been performed in children of this age. Nevertheless Holter monitoring of 50 medical students aged 23-27 years has shown similar, and unexpected, findings.[126]

Abrupt changes in heart rate were frequently present over the 24-hour recording period and may reflect the activity of the autonomic nervous system. The larger number of episodes of bradycardia documented during nocturnal recordings probably represent the influence of variations in autonomic tone created by changes in sleep state.[127] Sinus node re-entry may also have been responsible for some of the sudden episodes of sinus tachycardia.[128-129]

Irregularities of sinus rhythm have been termed sinus arrhythmia[104,122] (a 'normal' finding related to breathing movements)[122,124] or sinus arrest and sino-atrial exit block (a presumed manifestation of abnormal sinus node function). A diagnosis of sino-atrial exit block or sinus arrest can, however, only be determined with accuracy by recording potentials directly from the sinus node. Though it has been stated that measurement of atrial potentials, that is P-P intervals, on the surface ECG, may be used to diagnose exit block or arrest such measurements may be complicated by other factors. For example precise arithmetic multiples of P-P intervals in sino-atrial exit block may be disturbed by autonomic effects or by escape pacemakers whose inherent

rates are only slightly slower than the sinus pacemaker. Previously published definitions of exit block or sinus arrest have therefore not been used. Instead pauses have been categorised into groups on the basis of P-P or P-Q interval measurements. Pauses indistinguishable from sinus arrest and sino-atrial exit block were found in 65% of healthy children. Whether the mechanism of the pauses was primarily sinus arrest or sino-atrial exit block, or the secondary effects of activity of or hypersensitivity to variations in autonomic tone, remains unknown. The high frequency of these pauses in healthy children suggests that the underlying mechanism may not be of clinical importance.

Prolonged pauses associated with episodes of unconsciousness have been reported in infants and children.[115] [130] In this study the longest pauses detected were 1.36 ± 0.23 (S.D.) This would suggest that pauses at this age in excess of 1.82 seconds should be regarded as possible evidence of abnormalities in impulse generation in the sinus node and escape pacemaker tissue.

"Sinus arrhythmia" has been renamed "irregular sinus rhythm" by the World Health Organisation Working Party on terms relating to cardiac rhythm.[131] Perhaps it would also be appropriate to include short pauses, termed sinus arrest or sino atrial exit block, in this way as components of an irregular sinus rhythm.

Junctional rhythm may occur in two ways. Firstly the intrinsic pacemaker rate of the sinus node may fall below that

of the A-V node, the latter then initiating further impulse generation. This situation is termed a "junctional escape rhythm" and may result from physiological sinus bradycardia or from abnormal sinus node function.[132] Secondly, the inherent pacemaker rate of the A-V node may be accelerated.[133] Junctional rhythm was seen frequently in this population of normal children and the high frequency of these episodes may be due to a small, age related, difference in intrinsic pacemaker rate between the sinus node and the A-V junctional tissue. The 95th centile for maximum duration of junctional rhythm seen in this study was 20 minutes. More prolonged episodes, when slower than the predominant sinus rate, may indicate abnormal sinus node function or, when faster than the predominant sinus rate, may indicate an accelerated junctional focus.

Junctional escape rhythms and short pauses (<1.82 seconds) were found in a high proportion of healthy children. A knowledge of these normal findings must be taken into consideration when surface ECG recordings are used to diagnose sinus node dysfunction. Short pauses (<1.82 seconds), episodes of bradycardia below 50/minute and junctional escape rhythms in children with syncope or episodes of dizzyness have already been taken by some workers as sufficient evidence to diagnose abnormal sinus node function.[109,119-121] In some cases such findings have resulted in a decision to insert a permanent pacemaker system.

Patients with disorders of impulse generation have in some cases also been reported to have abnormal tachycardias

in addition to their episodes of bradycardia (the "sick sinus" or "tachycardia-bradycardia" syndromes).[134,135] The degree and nature of both tachycardia (where P wave discrimination is often difficult) and bradycardia regarded as abnormal must also take a knowledge of normal heart rate values into consideration.

Analysis of heart rate and rhythm after surgery for congenital heart disease has also, perhaps incorrectly, attributed some postoperative findings to sinus node injury.[111-113,136,137] In a recent report on heart rate and rhythm associated with Mustard's operation for transposition of the great arteries short sinus pauses and junctional escape rhythms were found as frequently before as after the operation.[114]

Intermittent first-degree A-V block (a P-R interval $\geq$ 0.20 seconds) was found in 9 children.[103] In 3 cases Mobitz type I second degree A-V block was also present. The latter arrhythmia has been reported in healthy young athletes and in older men undergoing strenuous exercise.[138,139,140,141] In two large studies on asymptomatic adults it was also found in 3 of 67,375[142] and in 2 of 19,000 respectively.[143] In the latter study A-V block though present in the supine position was abolished by standing up. Electrophysiological studies and autonomic function tests were performed on two male adolescents with second degree A-V block detected at a routine medical examination and showed an area of block proximal to the His bundle which was abolished by exercise and atropine.[138] Other workers from studies of otherwise healthy subjects with A-V block have also suggested an autonomic origin for this arrhythmia.

One group were able to demonstrate that second degree A-V block in two adolescents was due to a differential effect of vagal tone on the sinus and A-V pacemaker function.[144] Intracardiac electrophysiological and autonomic function tests may have been of value in the 3 children found to have this arrhythmia in this present study. A recent report by Young et al[145] has shown that, at follow up, 7 of 16 children with Mobitz Type I A-V block subsequently developed complete A-V block and 2 received a permanent pacemaker system for episdoes of unconsciousness. One of the three children in this present study had suffered from 3 previous fainting episodes but none had occurred for three years. There is a significant incidence of sudden unexpected death in adolescents, particularly those undergoing competitive sport,[146] and it is possible that children with an inbuilt tendency to atrio-ventricular conduction disturbances are more at risk. It is extremely important that the children found to have A-V block in this study are followed up into adult life.

Premature beats were seen in 22% of cases but were infrequent; none had more than 1/hour. Supraventricular and ventricular premature beats were more frequently found in newborn infants (see Chapter 3).

When making the diagnosis of an abnormal heart rate or rhythm pattern it is important to differentiate between standard and long term recordings of the electrocardiogram. A range of heart rate from 37 to 195/minute was found on 24-hour recordings compared with a range of 55-115/minute for children of similar age from standard ECG studies alone.[103]

Thus a constant heart rate of 40/minute on a standard electrocardiogram is probably abnormal at this age since it implies that bradycardia is present for long periods of time. A short episode of bradycardia of 40/minute on a 24-hour recording is within the normal range. The assessment of both the frequency and the duration of heart rate or rhythm changes is therefore important. By chance alone it is of course possible that any arrhythmia detected by long term recordings may appear on a standard ECG. The findings of this study would suggest that the range of normal for standard ECG recordings in children may require reappraisal.

Ten per cent of children in this study had a history of fainting but their frequencies of heart rate or rhythm patterns were not significantly different from those of children who did not faint (Table 25). Though cardiac arrhythmias may cause a fall or cessation of cardiac output sufficient to produce cerebral hypoperfusion and hence syncope, autonomic changes may also produce similar disturbances of cardiac conduction together with direct effects on venous or arterial tone.[147] Moreover, isolated failure of impulse generation in the sinus node should not result in asystole unless there is also a failure of the A-V junctional or ventricular escape pacemaker tissue. Further studies are required to determine how the autonomic nervous system and the intrinsic pacemaker and conducting system of the heart interact. Such studies may throw some light onto the mechanism of fainting.

How then should children who present with faints or transient disturbances of consciousness be investigated? Causes outside the cardiovascular system, such as epilepsy or hypoglycaemia should, of course, always be excluded. In this connection the advent of ambulatory long term recording of the electroencephalogram may be of considerable diagnostic value. Carotid sinus massage or autonomic function tests may help to establish a cardiovascular cause but do not differentiate between hypersensitivity to autonomic tone and a primary abnormality of cardiac conduction or impulse generation.[147] A standard electrocardiogram is easy to perform and non-invasive and perhaps should be used to investigate all children presenting with these symptoms. Such recordings may demonstrate a prolonged QT interval, atrio-ventricular block, pre-excitation or a ventricular or supraventricular cardiac arrhythmia. Twenty-four-hour ECG recordings are also non-invasive but further studies are perhaps needed to determine the underlying mechanisms of fainting before this technique is widely adopted. Nevertheless a small number of children with these symptoms may have intermittent arrhythmias and now that the "normal" range of heart rate and rhythm variations in healthy children is known interpretation of a 24-hour ECG recording may be of diagnostic value.

Where a child who has syncope is shown to have abnormalities of cardiac impulse generation or conduction on standard and/or 24-hour ECG recordings further information may be obtained by intracardiac electrophysiological studies. Abnormalities of sinus node

function may not however, always be elicited by this latter technique.[148]

In conclusion, ECG patterns previously reported to indicate abnormality of sinus node function occur frequently in the healthy child. Similarly, the range of heart rates is wider than previously reported at this age. It is essential that findings in normal, healthy children are taken into consideration when interpreting heart rate and rhythm patterns in clinical situations.

CHAPTER 5

A COMPARISON OF HEART RATE AND RHYTHM PATTERNS IN THE FETUS, NEWBORN INFANT AND OLDER CHILD

A difference in the size of populations studied and variation in methods used make a comparison of heart rate and rhythm between age groups difficult. Nevertheless it may be relevant to compare information on 5 minute recordings of the fetal heart beat with standard ECG recordings in the newborn infant. Premature beats were not detected in fetal subjects studied between 30 and 35 weeks gestation; a finding perhaps resulting from chance (with a relatively small sample of the population) rather than because arrhythmias do not actually occur at this stage of pregnancy. The frequency of premature beats between 36 and 40 weeks gestation was 1.7% which is higher than their frequency in the neonate (0.8%). Premature beats were the only type of arrhythmia detected in the fetus, a discrepancy which may result from the different recording technique used in the two groups. Recordings in the fetus were able to measure only changes in heart rate and would thus fail to detect conduction disorders with sinus rhythm such as the Wolff-Parkinson-White Syndrome or the long QT syndrome and arrhythmias, such as atrial flutter, where the heart rate (although persistently rapid) may not exceed 180/minute.

Twenty-four hour tape recordings on infants with cardiac arrhythmias on a standard electrocardiogram demonstrated more marked rhythm patterns such as supraventricular or ventricular tachycardia in 5 of 26 cases. Long term tape recordings performed on 8 of 12 subjects with premature beats before birth did not show abnormal tachycardias, a discrepancy

perhaps again resulting from the smaller number of subjects studied.

Suddenly developing, short-lived, episodes of bradycardia were found in the fetus and may be related to similar episodes seen on 24-hour recordings in the neonate. In the 134 infants studied in the neonatal period all showed short episodes of bradycardia but in some cases episodes were frequent and associated with very slow heart rates. It is possible that the detection of episodes of bradycardia in the fetus, from 5 minutes recordings, may reflect those subjects who will subsequently show frequent slowing of heart rate after birth. It is equally possible that episodes of bradycardia in the fetus are unrelated to those in the neonate and result from different mechanisms.

Twenty-four hour recordings on newborn infants without arrhythmias on a standard electrocardiogram showed 6 with premature beats >1/hour. Taking standard and 24-hour ECG findings together premature beats are more frequently present in the neonate than in the older child. Atrioventricular block was found in the older child but not seen on standard or 24-hour recordings in the neonate. The mechanism for this latter arrhythmia is uncertain but its absence in the neonate may reflect a difference in response to autonomic activity.

The range of heart rate will obviously differ from neonate to older child. However it would seem logical for maximum and minimum heart rate values to be similar in the neonate to those in the fetus late in pregnancy. Five antenatal subjects had heart

rates over 180/minute on 5 minute recordings and heart rates up to 220/minute were seen during activity in healthy neonates. It is possible that the range of heart rates occurring in the healthy fetus is wider than the range generally accepted by obstetricians (that is, 100-180/minute).

FINAL CONCLUSIONS

1. Multiple premature beats were detected in 1.2% of fetal subjects and 0.8% of newborn infants. By 12 weeks of post natal age the majority could no longer be detected. This arrhythmia did not appear, in the short term, to have harmful effects. Nevertheless longer follow up studies must and will be performed on these children.

2. More marked arrhythmias and conduction disorders such as supraventricular or ventricular tachycardia, atrial flutter and the Wolff-Parkinson-White Syndrome were also detected in a small proportion of apparently healthy newborn infants. The natural histories of some of these arrhythmias were probably obscured by antiarrhythmic therapy. Only further prospective studies without intervention will determine if such patterns are harmful.

3. Twenty-four-hour tape recordings of the electrocardiogram have shown that there is a wide variation in heart rates in normal infants and children. Junctional escape rhythms and sinus pauses also occur frequently. A knowledge of these findings may be of value when interpreting heart rate and rhythm patterns in children with symptoms that could be secondary to disordered cardiac impulse generation.

4. Intermittent second degree atrio-ventricular block was detected in 3% of healthy school children. This arrhythmia has previously been reported to have harmful connotations. In the short term there were no symptoms referable to this arrhythmia in the population studied but a longer follow up on a larger population should, perhaps, be performed.

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