Standard 12 Lead and 24 hour holter electrocardiographic observation in a biracial group of perinatally asphyxiated newborns

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Summary
Aim
To compare the cardiovascular responses to perinatal stress between two ethnic groups of newborns.

Study population
23 Nigerians and 14 Dutch perinatally asphyxiated newborns together with their respective control of 12 healthy Nigerian and 16 Dutch newborns.

Method
The study subjects and their controls were evaluated with standard 12-lead and 24-hour Holter electrocardiographic recordings within 36 hours of postnatal life.

Result
Rhythm, rate, P-wave, P-R interval and QRS duration were normal in study subjects and their respective controls on standard ECG. The prevalence of ST-segment depression in the mid-precordial lead V4 was significantly higher in the asphyxiated Nigerian infants (78%) than in the controls (42%); p < 0.05. Similarly, its prevalence in the Dutch infants (50%) was significantly different from the zero prevalence in the Dutch controls; p <0.05. Long duration ECG recordings showed a ‘fixed heart rate’ phenomenon in the asphyxiated infants but not in their controls.

Conclusion
ST-segment depression may imply myocardial ischaemia in asphyxiated Dutch newborns but such may not be inferred in asphyxiated Nigerian newborns. ‘Fixed heart rate’ phenomenon or loss of sinus arrhythmia appears to be causally related to hypoxaemia which results from perinatal asphyxia.

Keywords: Perinatal, Asphyxia, Biracial, Newborns, Electrocardiography, Holter-monitoring

Résumé
But
Comparer des réactions cardiovasculaire au stress périnatal entre des nouveau-nés de deux groupes ethniques.

Population d'étude
23 Nigerians et 14 Hollandais nouveau-nés périnatallement asphyxiers ainsi qu'avec leurs contrôles respectifs de 12 Nigerians et 16 Hollandais nouveau-nés en bonne santé.

Méthode
Les sujets d'étude ainsi que leurs contrôles ont été évalués avec le niveau 12 plomb 24 heures Holter electrocardiographique enregistrements en moins de 36 heures vie postnatale.

Résultats
La cadence, taux P-vague, intervalle P-R et la durée QRS étaient tous normaux chez les sujets d'étude et leur contrôles respectifs sur le mode ECG. La fréquence de ST-segment était dépressif dans le plomb mi-precordial V4 était sensiblement élevé chez les enfants nigérians asphyxiés 78% plus que dans le groupe de contrôle 42%. P<0,05. Pareillement, sa fréquence chez des enfants hollandais asphyxiés 50% était sensiblement différente du Zéro fréquence chez le groupe de contrôle d' hollandais P<0,05. Enregistrement de la longue durée de ECG a montré un phénomène de taux fixé de Coeur chez le groupe des enfants asphyxiés mais pas chez leurs contrôles.

Conclusion
État dépressif ST-segment pourrait laisser supposer une ischémie myocardique chez des nouveau-nés hollandais asphyxiés mais ça ne peut pas être la même chose chez les nouveau-nés nigériens asphyxiers. Le phénomène "taux fixé du Coeur" ou la perte du rythmisme sinus de l'hyperoxémie paraît être en passant lié à l'hypoxémie conséquences d'asphyxie périnatale.

Introduction
Several normal variations of the cardiac rhythm have been described in neonates.1-3 These include sinus arrhythmia, sinus bradycardia, sinus tachycardia, sino-atria block, wandering atrial pace maker and other atrial and ventricular extrasystoles. These have been linked to physiologic and morphologic postnatal changes, including postnatal development changes in the cardiac conducting system. Some of these observations may also be hypoxemia related as evidence exists4 showing that the conducting tissues of the heart have a differential susceptibility to hypoxemia or ischaemia. In baboons for instance, AV-block arrhythmias and a ‘fixed heart rate’ or a lack of sinus arrhythmia have been shown to follow diminished uteroplacental perfusion.5 These observations were apparently mediated via the parasympathetic pathway since they were blocked by atropine. A similar fixed heart rate has been noted to be frequently associated with low Apgar scores in human infants.1 Ventricular tachyarrhythmias, third degree A-V block (with increased risk of dying in the first few weeks of life) and other intraventricular conduction defects have all been noted in association with hypoxic heart failure and metabolic derangement in the newborn period.1

These observations have been borne out of animal experimentation and work carried out on Caucasian infants. The present study in which perinatally asphyxiated Nigerian newborns are compared with similarly asphyxiated Dutch newborns under the same study conditions is borne out of a necessity to establish any similarities and ethnic variations with respect to cardiac rhythms and conduction patterns.

Patients, Methods and Materials
Fourteen perinatally asphyxiated Dutch newborns and 23 perinatally asphyxiated Nigerian newborns were recruited to the study from the Neonatal Intensive Care Unit (NICU) of the Academic Medical Centre (AMC), Amsterdam and the Special Care Baby Unit (SCBU) of the University College Hospital (UCH), Ibadan respectively. Perinatal asphyxia was defined simply by a 1 minute Apgar score of ≤ 6. Sixteen healthy Dutch newborn controls and 12 healthy Nigerian newborn controls were recruited from the lying - in wards of the two centres.

Cord blood pH, glucose and bicarbonate in Amsterdam and cord blood bicarbonate and glucose in Ibadan were estimated to corroborate the selection criteria of the study subjects and controls. The profiles of the study subjects and their respective controls are shown in table 1.

Each infant had a standard 12-lead ECG at between 6 - 12 hours of age. This period was chosen to correspond with the best 'window' for the detection of hypoxaemia - related myocardial dysfunction, which results from perinatal stress, and also to precede the 24 hours Holter ECG recordings. At the AMC, the standard ECG recordings were made with the commercially available

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WAJM VOL 22 NO 3, SEPTEMBER 2003
Siemens 6-channel recorder. Rhythm, rate and conduction disorders were sought in addition to hypertrophy patterns and ST-T wave changes. Findings were compared with the standards established by Davignon et al and nomenclature followed the definition of the World Health Organisation (WHO) and International Society of Cardiology (ISC) Task force reports of 1978 and 1979. The 24-hour ECG recording was commenced immediately after the standard ECG recording in each infant using a commercially available Oxford instrument company Holter recorder on TDK AD 120 brand of cassette magnetic tapes. The recordings were carried out under normal neonatal ward environmental conditions (figure 1). The babies could be handled at will without interfering with the recording process. The electrode positions used in all the babies were the modified V_{1}, like system to facilitate identification of ectopic beats and a V_{5}, like system to detect myocardial ischaemia. The ground or negative electrode was positioned in the left flank. During recordings, the ward staff and mothers handled the babies freely, but all activities were charted by the ward nursing staff on log-sheets specially provided for the purpose. All the 24-hour cassette tape recordings were then played back at the standard sixty times the recording speed on the Oxford Medilog ECG analysis system (figure 2) by a trained technician at the AMC who was blinded to the clinical information on the study subjects and the same criteria defined by the WHO/ISC takes force were followed in interpreting the findings. The Medilog system was programmed to identify among others, sinus arrhythmias, sino-atrial block, sinus bradycardia with or without junctional escape rhythm and fixed heart rate phenomenon. Others included premature atrial complexes (PACs) and premature ventricular complexes (PVCs). A close reference to the log-sheets of activities on each baby was kept in interpreting the findings. The playback procedure was programmed to stop automatically on detection of any anomaly. Paper tracings of the anomaly were then automatically generated for closer scrutiny.

Heart-rate variation during activity and rest was measured by histogram module in the analyzer and plotted for the 24-hour period of the recording (figure 3). Thus it was possible to recognize the highest and the lowest heart rates attained during a 24-hour period as well as the more constant heart rate (mean heart rate).

Findings were compared between both ethnic groups and their respective ethnic controls. Where appropriate tests of significance were applied using the students 't' test or chi squared with Yates correction for the small numbers of subjects. The level of significance was p<0.05.

Results

Standard ECG

Rhythm

All the infants studied were in sinus rhythm except for one Dutch control who had numerous atrial extrasystoles. Statistically this did not constitute a significant variation.

Rate

The mean heart rate in the asphyxiated Nigerian infants (range 105 - 150; mean 131 ± 13 beats/minute) was significantly lower than that in the Nigerian controls (range 120 - 170; mean 144 ± 17 beats/minute) (t = 2.31; p < 0.01).

The rates in the asphyxiated Dutch infants (range 77 - 180; mean 133 ± 29) did not differ significantly from the rates in the Dutch controls (range 105 - 170; mean 138 ± 14) (t = 0.52; p > 0.05). The rates in the asphyxiated Dutch and Nigerian infants were similar (t = 0.62; p > 0.05) The rates in their respective controls were similar as well (t = 0.56; p > 0.05).

P-Wave

The P-wave was normal in all the asphyxiated infants as well as their controls.

P - R Intervals

Atiro-ventricular conduction was normal in all asphyxiated as well as control infants. The ranges were (0.08 - 0.13; mean 0.10 ± 0.02 second) and (0.08 - 0.12; mean 0.10 ± 0.02 second) in the asphyxiated Nigerian infants and controls respectively; and (0.08 - 0.12; mean 0.10 ± 0.01 second) and 0.08 - 0.12; mean 0.10 ± 0.01 second) in the asphyxiated Dutch infants and controls respectively. The P-R intervals were thus similar in all cases.

QRS Complex

Intra-ventricular conduction as recorded on standard 12-lead ECG was normal in all asphyxiated infants and controls. The ranges and means of the QRS durations were similar in all groups (0.04 - 0.08; mean 0.05 ± 0.01 second).

Right ventricular hypertrophy (RVH) was observed in 2 (9%) of the asphyxiated Nigerian infants and in none of the Nigerian controls; but the difference was not statistically significant, p > 0.05. Similarly, its prevalence in the asphyxiated Dutch infants (21%) did not differ significantly from the zero prevalence in the Dutch controls (p > 0.05). All the infant in whom RVH was observed had Apgar scores of less than 3 at 1 minute and they all had cardiogenic shock. One Nigerian and 3 Dutch infants who had cardiogenic shock did not show ECG evidence of RVH. To better relate ECG findings with clinical conditions, the clinical categorizations of the study subjects are detailed in an earlier communication.8-10

ST-Segment

Significant ST-segment depression in V_{1} - V_{6} (>1mm below the isoelectric line) was observed in 18 (78%) asphyxiated Nigerian and 50% of the Dutch infants. The corresponding values for their respective controls; 42% and 0% were significantly different, p being <0.02 and 0.01 respectively. The prevalence in both groups of Nigerian infants was higher than the corresponding values in the Dutch babies.

T-Wave

Flat or inverted T-waves were observed in 9 (39%) of the asphyxiated Nigerian infants and 4 (29%) of the asphyxiated Dutch subjects. The corresponding prevalence in the respective controls were 25% and 6%. The differences were not statistically significant (p > 0.05).

24 - Hour ECG

Rhythm

Although phasic sinus arrhythmia was observed in 19 (83%) of the asphyxiated Nigerian infants and in all the 12 controls, the difference was not statistically significant (p > 0.05). Similarly the prevalence of phasic sinus arrhythmia in the asphyxiated Dutch infants (79%) did not differ significantly from the 100% prevalence in the Dutch controls.

Brief duration (10 - 30 seconds at a time) of
Table 2  Prevalence of aberrant findings from 12-lead ECG in asphyxiated Nigerian infants (AN) and controls (CN) asphyxiated Dutch infants (AD) and control (CD)

<table>
<thead>
<tr>
<th>ECG FINDINGS</th>
<th>AN (n = 23)</th>
<th>CN (n = 12)</th>
<th>AD (n = 14)</th>
<th>CN (n = 16)</th>
<th>AN vs CN</th>
<th>AD vs CD</th>
<th>AN vs AD</th>
<th>CN vs CD</th>
</tr>
</thead>
<tbody>
<tr>
<td>RVH</td>
<td>2(9)</td>
<td>0</td>
<td>3(21)</td>
<td>0</td>
<td>0.36 &gt;0.05</td>
<td>1.8 0.05</td>
<td>0.36 0.546</td>
<td>- -</td>
</tr>
<tr>
<td>ST-segment</td>
<td>Depression</td>
<td>18(78)</td>
<td>5(42)</td>
<td>7(50)</td>
<td>0</td>
<td>3.20 &lt;0.02*</td>
<td>7.83 0.01*</td>
<td>2.01 0.155</td>
</tr>
<tr>
<td>Flattened T wave in V5/ V6</td>
<td>9(39)</td>
<td>3(25)</td>
<td>4(29)</td>
<td>1(6)</td>
<td>0.21 &gt;0.05</td>
<td>1.31 0.05</td>
<td>0.09 0.766</td>
<td>0.74 0.301</td>
</tr>
</tbody>
</table>

* = statistically significant difference

Table 3  Prevalence of dysrhythmias observed from 24-hour ECG recordings: in asphyxiated Nigerian (AN) and Dutch (AD) infants and their respective controls (CN) and (CD)

<table>
<thead>
<tr>
<th>Dysrhythmias</th>
<th>AN (n = 23)</th>
<th>CN (n = 12)</th>
<th>AD (n = 14)</th>
<th>CN (n = 16)</th>
<th>AN vs CN X²</th>
<th>AD vs CD X²</th>
<th>AN vs AD X²</th>
<th>CN vs CD X²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinus arrhythmia</td>
<td>19(83)</td>
<td>12(100)</td>
<td>8(57)</td>
<td>16(100)</td>
<td>0.95 &gt;0.05</td>
<td>1.80 &gt;0.05</td>
<td>1.72 0.190</td>
<td>- -</td>
</tr>
<tr>
<td>Sinoatrial block</td>
<td>1(4)</td>
<td>2(17)</td>
<td>2(14)</td>
<td>2(13)</td>
<td>0.21 &gt;0.05</td>
<td>&gt;0.02 &gt;0.05</td>
<td>0.21 0.650</td>
<td>0.05 0.815</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
<td>2(9)</td>
<td>0</td>
<td>2(14)</td>
<td>0</td>
<td>0.08 &gt;0.05</td>
<td>0.004 &gt;0.05</td>
<td>0.00 0.988</td>
<td>- -</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>4(17)</td>
<td>2(17)</td>
<td>-</td>
<td>-</td>
<td>0.35 &gt;0.05</td>
<td>-</td>
<td>0.15 0.696</td>
<td>- -</td>
</tr>
<tr>
<td>Sinus bradycardia+junctional escape rhythm</td>
<td>0</td>
<td>0</td>
<td>3(21)</td>
<td>4(25)</td>
<td>-</td>
<td>-</td>
<td>0.04 &gt;0.05</td>
<td>3.37 &lt;0.05*</td>
</tr>
<tr>
<td>WAP</td>
<td>0</td>
<td>0</td>
<td>3(21)</td>
<td>1(6)</td>
<td>-</td>
<td>-</td>
<td>0.21 &gt;0.05</td>
<td>2.87 0.900</td>
</tr>
<tr>
<td>Fixed rate PAC with normal ventricular conduction</td>
<td>4(17)</td>
<td>0</td>
<td>3(21)</td>
<td>0</td>
<td>0.95 &gt;0.05</td>
<td>-</td>
<td>1.8 0.05</td>
<td>0.02 0.897</td>
</tr>
<tr>
<td>Ventricular conduction PVC (&lt;5 in 24 hrs.)</td>
<td>3(13)</td>
<td>0</td>
<td>1(7)</td>
<td>1(6)</td>
<td>0.45 &gt;0.05</td>
<td>-</td>
<td>-</td>
<td>0.00 0.988</td>
</tr>
<tr>
<td>PVC (≥5 in 24 hrs.)</td>
<td>2(9)</td>
<td>0</td>
<td>4(29)</td>
<td>0</td>
<td>0.08 &gt;0.05*</td>
<td>3.09 &lt;0.05*</td>
<td>1.28 0.258</td>
<td>- -</td>
</tr>
</tbody>
</table>

WAP = wandering atrial pace maker; PAC = premature atrial complex; PVC = premature ventricular complex; X² = Chi square (with Yates correction); P = probability * = Statistically significant difference.

Table 4  Heart rate variations as seen from 24 hour ECG recordings in asphyxiated Nigerian and Dutch infants and their controls

<table>
<thead>
<tr>
<th>Heart rates</th>
<th>AN (n = 23)</th>
<th>CN (n = 12)</th>
<th>AD (n = 14)</th>
<th>CD (n = 16)</th>
<th>AN vs CN t</th>
<th>AD vs CD t</th>
<th>AN vs AD t</th>
<th>CN vs CD t</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximum</td>
<td>95 - 180</td>
<td>150 - 180</td>
<td>130 - 200</td>
<td>150 - 205</td>
<td>-0.283 0.778</td>
<td>-0.99 0.326</td>
<td>-1.41 0.016*</td>
<td>-3.114 0.004*</td>
</tr>
<tr>
<td>Mean (± SD)</td>
<td>158 (± 23)</td>
<td>160 (± 11)</td>
<td>169 (± 23)</td>
<td>176 (± 15)</td>
<td>-</td>
<td>-</td>
<td>-0.00 0.988</td>
<td>- -</td>
</tr>
<tr>
<td>Minimum</td>
<td>20 - 150</td>
<td>70 - 140</td>
<td>5 - 130</td>
<td>32 - 130</td>
<td>-1.108 0.027*</td>
<td>-0.617 0.542</td>
<td>-1.230 0.027*</td>
<td>-1.892 0.016*</td>
</tr>
<tr>
<td>Mean (± SD)</td>
<td>105 (±35)</td>
<td>117 (±18)</td>
<td>91 (±31)</td>
<td>98 (±31)</td>
<td>-0.508 0.012*</td>
<td>-0.603 0.550</td>
<td>-0.859 0.396</td>
<td>2.011 0.054</td>
</tr>
</tbody>
</table>

* = Statistically significant difference

The prevalence of atrioventricular conduction was similar in asphyxiated Nigerian infants and controls. The same was true of the Dutch subjects, (Table 3).

WAIM VOL 22 NO 3 SEPTEMBER 2003

255
Fig. 1 24 Hour (Holter monitor) ECG recording in progress on a baby in a cot

Fig. 2 The Oxford Medilog ECG analyzer with the chart recorder

Fig. 3 Section of a 24-hour heart rate histogram

Fig. 4 Section of the 24-hour ECG strip chart recording in one of the asphyxiated Nigerian infants showing maintenance of sinus rhythm even with rates at 20bpm.

Fig. 5 Section of the 24-hour ECG strip chart recording in one of the asphyxiated Nigerian infants showing uniform PVCs.

Intra-ventricular conduction

Intra-ventricular conduction defects observed were in the form of uniform premature ventricular complexes (PVC) figure 5. They were observed in 5 asphyxiated Nigerian infants and in one of the controls, (p > 0.05). They were also observed in 36% of the asphyxiated Dutch infants and in 6% of the Dutch controls (p < 0.05). Table 3 shows other observations that were made including statistical comparisons of observations.

Rate

Sinus bradycardia (<90 beats/minute), which tended to occur during sleep and defecation, was observed in 2 (9%) asphyxiated Nigerian infants and 2 (14%) asphyxiated Dutch infants but in none of the controls. However the differences were not statistically significant (p > 0.05). Sinus tachycardia on the other hand (>180 beats/minute) which tended to occur during crying episodes did not occur in any of the asphyxiated Nigerian infants but was observed in 4 (29%) asphyxiated Dutch infants and 2 (13%) Dutch controls. The difference was not statistically significant (p > 0.05). Four asphyxiated Nigerian infants and 3 asphyxiated Dutch infants
who did not show sinus arrhythmia had a ‘fixed rate’ phenomenon. The minimum and mean heart rates were significantly lower in the asphyxiated Nigerian infants than their controls but not in the Dutch infants; the heart rate comparisons in all the groups of infants are shown in (Table 4). The comparisons of heart rates between all four groups of infants studied are shown in table 4.

Discussion

The prevalence and spectrum of clinical hypoxaemia-related cardiovascular disorders among the present study subjects have previously been reported in a separate communication and several other reports have also pointed attention to similar clinical problems causally related to asphyxia-montorium or perinatal asphyxia.

The relatively lower 1-minute Apgar score in the AD infants (mean 2.2) compared with the corresponding mean score in the AN infants (5.5) probably reflects the ready availability and application of sophisticated obstetric monitoring devices and neonatal resuscitative measures in Holland allowing successful delivery of distressed fetuses that would have succumbed in utero. This is borne out by the fresh stillbirth records from the two hospital sites of the study. There were 30 fresh stillbirth records in the 708 deliveries in the obstetric departments at the U. C. H. during the study period, which contrasts sharply with 8 fresh stillbirths at the A. M. C. in the 823 deliveries recorded during the corresponding study period. Surprisingly however, the prevalence of hypoxaemia-related cardiovascular disorders was similar in both the AD infants (64%) and AN infants (61%). We may deduce therefore, that prompt and appropriate intervention not only enhances survival in severely asphyxiated infants but also reduces the prevalence of hypoxaemia-related disorders at birth.

The advantages of long duration electrocardiographic recordings over standard 12-lead ECG in the study of cardiac dysrhythmias and heart rate variations are known and have been demonstrated in the present study. Whereas standard ECG indicated that all the infants were in sinus rhythm, various arrhythmias were in fact present in quite a number of the study infants as well as their controls. These included supraventricular and ventricular extrasystoles observed in 50% and 36% in the asphyxiated Nigerian and Dutch infants respectively. The finding of a significant difference in the occurrence of <5 PVC in 24-hour between the asphyxiated Dutch infants and their controls may not be regarded in itself as important. It is an acceptable finding in most newborn infants. The accuracy of the scanning method used in detecting these arrhythmias might be questioned. It is recognised that the scanning method might not be sufficiently sensitive to detect all arrhythmias, which underestimates the true incidence. Nevertheless, since the same observer scanned all recordings, such underestimation would be expected to be evenly distributed among the infants. We can therefore reasonably assume a true representation of the prevalence of dysrhythmias among the study infants as reported in the present study.

The mean maximum and minimum heart rates obtained from 24-hours ECG recordings in the asphyxiated Nigerian infants were lower than those in their controls and the same observation is true of the Dutch infants. However, when the overall mean heart rate is considered, while the difference was statistically different between the asphyxiated Nigerian infants and their controls, there was no such difference between the asphyxiated Dutch infants and their controls (table 4). This is in keeping with the previously reported depressant effect of perinatal asphyxia on heart rate as it relates to the Nigerian infant in the present study. The comparison between the asphyxiated Nigerian and Dutch infants and their controls show no statistical difference. The apparent differences therefore between these groups may be a reflection of the severity of asphyxiation rather than that of ethnic difference. The occurrence of a ‘fixed heart rate’ or lack of sinus arrhythmia in the asphyxiated infants and its absence in the controls is also in keeping with previous reports of such a phenomenon in asphyxiated infants. The asphyxiated Nigerian and Dutch infants were similar when compared (p = 0.897) and so were their controls. The mechanism of this phenomenon is unknown. One suggestion is that it results from the disappearance of neural control of the sinus node, possibly as a result of cerebral depression.

The lower heart rates and ‘fixed heart rate’ phenomenon were thus the only disorder of rate and rhythm that may be attributed to perinatal asphyxia since other dysrhythmias were similarly prevalent in both the subjects and controls.

It is remarkable that sinus bradycardia with junctional escape did not occur in any of the Nigerian infants but was found in a relatively high proportion of the asphyxiated Dutch infants. The reason for this is uncertain, but it is tempting to relate it to the relatively advanced neural development. By the fifth to eighth week of foetal development, the human heart has attained most of its adult characteristics and the sinus node is readily discernible. At birth primitive (P) and transitional (T) cells are readily distinguishable even on light microscopy, but there continues to be differentiation within the first few years of life. The P cells are believed to make up the cardiac pacemaker and are quite predominant in early life but decreases in prevalence with age. The T cells on the other hand are less common in the newborn period and increase in proportion with age. It is postulated that the T cell probably serve to organise or filter the impulses that are generated by the P cells and transmit them to the cells of the intermedial tracts. The relative paucity of T cells early in life (and the concomitant P cell predominance) might allow irregular or asynchronous transmission of P cell impulses to the atria, which later in life, might be organised by the more dense network of T cells. Thus the maintenance of sinus rhythm even at low rates may indicate that the sinus node of the African (Nigerian) infant is more advanced in development that that in the Caucasian in terms of prevalence of T cells and neutral control. In this regard, it is worth noting that several studies have suggested that the African infant is more mature neurologically at birth and during the first year of life than his Caucasian counterpart.

The ST segment depression demonstrated in asphyxiated Dutch and Nigerian infants in mid-precordial leads probably have different implication. ST segment depression in the mid-precordial leads has been reported as normal in young Africans but is seen in Caucasians of the same age group. This is borne out in the present study. Whereas it was observed in 42% of the healthy Nigerian controls, it was not observed at all in the healthy Dutch controls and the difference was statistically significant (table 2). The implications are obvious; whereas ST segment depression may be taken as a good indicator of myocardial ischaemia in Dutch infants, it may not have such a significance in the Nigerian infant. Although the phenomenon of flat or inverted T wave in V2 and V3 is recognised as a sign of left ventricular overwork, it may occur normally during the first 3 days of life. The ECGs in the present series were recorded within the first 24 hours of life. Therefore, not much significant may be attached to flat/inverted waves in V2 or V3 except of course, where such changes coexist with other clinical signs as frank cardiogenic shock.

In conclusion, the ‘fixed heart rate’ phenomenon and generally slower heart rates were the forms of dysrhythmias for which asphyxia was found to play a causal role. None of the other forms of dysrhythmias documented could be said to be specific to asphyxiated infants in the present study. Racial differences were found only with respect to the ability of the Nigerian infants to remain in sinus rhythm even at very slow heart rates. In addition, whereas ST segments depression in the mid-precordial lead may signify myocardial ischaemia in the Dutch infants, such a
significance may not be readily attached to it in Nigerian infants.

Acknowledgement
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References