FOETAL ACID-BASE STATUS
and
FOETAL ELECTROCARDIOGRAPHY

by

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CONTENTS

SUMMARY

A study has been made of foetal and maternal acid-base balance and of the foetal electrocardiogram in 108 women in labour. 102 subjects were selected where the foetus was considered to be clinically at risk from foetal asphyxia and six apparently normal subjects were included.

Foetal blood samples were obtained from the foetal scalp during labour and from the umbilical cord at delivery. The foetal electrocardiogram was recorded throughout labour from scalp electrodes attached directly to the foetal scalp, and the neonatal electrocardiogram was recorded within 24 hours of delivery. The clinical condition of the infant was assessed immediately following delivery, at 24 hours and at six weeks following delivery. Foetal and maternal venous blood samples were analysed for pH, pCO_2, standard bicarbonate, plasma electrolytes and glucose where sufficient sample was available. Samples of the foetal electrocardiogram were analysed for configuration and time constants and were subsequently studied in relationship to foetal biochemical parameters and to the neonatal electrocardiogram.

A comprehensive analysis of the interrelationship between maternal and foetal biochemical measurement has been included. This section of the study has confirmed the work of previous authors in establishing that a significant

relationship occurs between maternal and foetal acid-base status, and between foetal acid-base status during labour and the clinical condition of the infant at birth and 24 hours after delivery. Electrolyte values in foetal and maternal plasma were studied in relation tofoetal and maternal acid-base values and a significant negative linear relationship was established between foetal and maternal blood pH values and plasma potassium levels. The mean values and standard errors of all variants have been recorded where appropriate. Time constants for the foetal electrocardiogram have been considered as whole group mean data in relationship to the time of sampling and in data grouped according to the acid-base and electrolyte values of the foetus. Mean values for electrocardiographic data were defined in relation to known normal acid-base values and it is considered that these values represent normal foetal E.C.G. time constants. Significant prolongation of electrical systole (Q-T interval) was seen to occur at the time of delivery in relation to low pH values in cord venous blood, provided the Q-T values were corrected for heart rate. Q-T prolongation was related to hyperkalaemia in cord venous blood with a higher degree of significance.

Hyperkalaemia was also shown to be related to T wave depression and inversion, changes which resemble those of hypokalaemia in the adult. On the evidence available, it is

suggested that the changes seen in the foetal electrocardiogram resemble those of hypokalaemia because, despite the apparent hyperkalaemia, a condition of cellular potassium depletion exists in states of chronic foetal acidosis.

Foetal acidosis, as demonstrated by low scalp blood pH values, was also shown to be associated with increased right axis deviation of the heart following delivery.

Foetal cardiac axis has been estimated using the technique of Larks for estimating foetal cardiac axis from abdominal electrodes. The mean values obtained using this method are similar to those reported from abdominal lead recordings although the relationship between the cardiac axes of foetal E.C.G. complexes obtained early and late in labour showed differing relations with the subsequent post-delivery leads.

An attempt has been made to define the critical limits of the time constants of the foetal electrocardiogram. The overlap of values between normal and abnormal groups suggests there are limitations on the application of these measurements for clinical usage.

REGULATIONS OF THE DEGREE OF DOCTOR OF MEDICINE AT THE UNIVERSITY OF ADELAIDE

Regulation 4, Paragraph (a)

In accordance with Regulation 4, Paragraph (a) of the Regulations of the Degree of Doctor of Medicine at the University of Adelaide, I hereby declare that this thesis contains no material which has been accepted for the award of any other degree of diploma in any University and that, to the best of my knowledge and belief, the thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

E. M. Symonds, M.B.B.S.

REGULATIONS OF THE DEGREE OF DOCTOR OF MEDICINE

Regulation 4, Paragraph (b)

"The candidate shall indicate wherein he considers the thesis as work to advance medical knowledge or practice".

A review of the extensive literature of foetal electrocardiography has revealed inadequacies in the description and definition of the configuration of the normal foetal electrocardiogram as assessed against known biochemical measurements of the foetus.

This thesis was undertaken to study the configuration and time constants of the foetal electrocardiogram and to quantitate these parameters against measurements of foetal acid-base status, foetal plasma electrolytes and blood glucose levels.

Whilst the studies of Enhorning and Westin in 1954 on previable foetuses under conditions of increasing asphyxia showed specific change in the foetal electrocardiogram, these changes could not be equated with those produced by conditions of asphyxia "in utero" in the presence of an intact foeto-placental circulation. Hon and Lee in 1963 described electrocardiographic changes in 9 foetuses preceding death in utero. These findings showed the changes that occur in the electrocardiogram in extreme conditions

but gave no indication of whether changes in the foetal electrocardiogram occurred in relationship to milder degrees of asphyxia.

This thesis is considered to advance medical knowledge by describing the configuration and time constants of the foetal electrocardiogram both during labour and at the time of delivery in normal and acidotic subjects. Significant prolongation of electrical systole (Q-T interval) has been demonstrated under conditions of foetal acidosis. Furthermore, the same findings in association with T wave inversion have been demonstrated in the presence of foetal hyperkalaemia at birth. The changes described in the foetal electrocardiogram under conditions of asphyxia are those described in the adult under conditions of hypokalaemia. The hypothesis is advanced that these changes are caused by actual intracellular potassium depletion resulting from a potassium gradient being established between the maternal and foetal circulation under conditions of foetal asphyxia. Calcium and magnesium values were not included in this study.

The foetal cardiac axis has been studied during labour and considered in relationship to foetal acid-base status and the clinical condition of the infant after delivery. In addition, the foetal electrocardiogram has been examined in relationship to the neonatal electrocardiogram and to the clinical progress of the infant for six weeks following

delivery.

Examination of the biochemical data has confirmed previous findings that foetal acidosis during labour is associated with acidosis at the time of delivery and with clinical depression of the newborn infant.

Foetal acidosis has been shown to be associated with foetal hyperkalaemia and the potential importance of secondary electrolyte changes in their effect on cardiac activity has been demonstrated.

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E. M. Symonds

CHAPTER 1

HISTORICAL REVIEW

FOETAL ELECTROCARDIOGRAPHY

SECTION 1. The Use of Abdominal Electrodes

In 1887, Waller showed that if a pair of electrodes are strapped to the front and back of the chest and connected with a Lippmann's capillary electrometer, the mercury in this instrument could be seen to move slightly but sharply at each beat of the heart. Thus, each beat of the heart was seen to be accompanied by an electrical variation. The electrodes used in this study consisted of zinc covered by chamois leather soaked in brine.

A similar movement was detected if the two hands or one hand and one foot were plunged into two dishes of salt solution connected with the two sides of the electrometer, but the movement of the mercury was reduced. Whilst the importance of this observation was obvious, the capillary electrometer was a cumbersome instrument and it was not until Einthoven introduced the string galvanometer (1903), with its greatly enhanced sensitivity, that a true indication of the current variations as related to the potential difference alterations could be recorded.

In 1906, Cremer, in an article entitled "Direct Conduction of Impulse Current of the Human Heart from the Oesophagus and the Electrocardiograph of the Foetus", described the first record of the foetal electrocardiograph.

Cremer used a variety of electrodes, including pure silver, silver plated metals and nickel plated metal. In this paper, he describes the first use of the oesophageal lead and for these experiments he used a man who worked as a sword-swallower. This was probably advisable, in view of the fact that the silver oesophageal lead was 10 centimetres long and 1.5 centimetres in diameter. Spurred on by these successes, Cremer attempted to find a pregnant female to volunteer to having oesophageal and rectal leads or stomach and bladder leads, without success. He was "forced to combine one electrode on the abdomen with one in the vagina"and, with this method, he produced the first record of foetal heart electrical activity. The impact of this discovery was hardly tumultuous. Over the next 24 years, only four further papers were published on this subject. Maekawa and Toyoshima summarised this literature in 1930 and, at the same time, pointed out that " it is very difficult to record the electrocardiogram of the fetus by leading off from its mother's body ... because the electrical current of the fetal heart is almost all shunted by the

amniotic fluid in which the fetus floats."

Whilst the latter part of this statement was incorrect, these authors did emphasise for the first time the need for "some system of amplifying the action current", and using a direct current amplifier designed by Maekawa, they demonstrated the foetal electrocardiogram using only abdominal leads. The difficulties up to this time were largely technical in getting at the foetus, and over this same time some quite sophisticated work was produced on neonates. For example, Krumbhaar, in 1916, gave an excellent description of the changes that occurred in the electrocardiogram in the first twelve years of life. He showed that, at birth, there is a right ventricular preponderance and that this disappears by two to three months. By six months, the infant's ECG is practically the same as that of the adult, except that Q_2 and Q_3 are apt to be unduly prominent, and that the P-R interval is shorter, and sinus arrhythmia is practically absent in the first year of life.

In 1933, Steffan and Strassmann described further improvement in the recording of the foetal electrocardiogram by using a new tension electrocardiograph. The improvement in these recordings was considerable, so much so that in 1938, Bell claimed that these authors were the

first people to demonstrate a fetal ECG. Careful persual of the original article of Cremer shows that Bell was wrong, and that Cremer's recordings are correct.

In 1936, Strassmann presented another series of recordings using standard maternal limb leads, and demonstrated the foetal ECG from a variety of electrode positions. He also demonstrated action potentials from maternal abdominal muscles and at the same time, stated that foetal ECGs could only be demonstrated in the last two months of pregnancy.

A further paper by Strassmann and Mussey, in 1938, reported a series of recordings of the foetal electrocardiogram using basically the same technique as described in 1936. Thigh and upper arm electrodes were again used and, for the first time, some attempt was made to produce a more systematic approach to the clinical application of foetal electrocardiography. The authors stressed that the foetal electrocardiogram must only be regarded as positive if the R waves of the foetus could be followed through the entire tracing, and the foetal heart rate determined. In a series of recordings from 52 patients, it was found that the recordings were positive in 87% of recordings during the last 70 days of pregnancy, and 91% of recordings during the last 20 days.

Later in 1938, a significant advance in technique was reported by Bell, with the introduction of the valve electrocardiograph. This instrument had three times the sensitivity of previous machines and gave as much amplification as could be obtained at that time without undue oscillation. Base line movement from muscular movement was reduced by using a coupling condenser, and this allowed the galvanometer to return quickly to zero. Despite these precautions, fairly rapid random oscillations due partly to electrical causes, but more significantly to action potentials arising from the muscles of the mother's abdomen, gave rise to irregular disturbances of the base line.

Metal electrodes covered with gauze soaked in saline were applied to the maternal abdomen, the best results being obtained with one electrode on the fundus of the uterus and the other over the pubic symphysis. Foetal deflections of 25 to 50 microvolts were demonstrated. Despite this improvement in technique, the results were, in fact, worse than those obtained by Strassmann, and this demonstrated that amplification alone did not provide an answer to the problems of foetal electrocardiography, unless suitable suppression of background noise was available. Of 33 women examined near term, only 10 gave positive results, 11 were doubtful and 12 were negative. Bell concluded that:

1. Negative results were not due to deficiency of R waves of the foetus;
2. The size and sex of the child were not factors affecting the foetal electrocardiogram;
3. The amount of liquor was not important as results were no better after the liquor was drained;
4. There was some relationship between abdominal girth and the foetal electrocardiogram.

Apart from isolated case reports such as that of Johnson in 1938, these papers constituted the main work on foetal electrocardiography as obtained by electrodes applied to the mother up to 1940. Other work was also done up to this time on direct application of electrodes to the pre-viable foetus. Easby (1934) reported the electrocardiogram from a four and a half month old foetus, and Heard, Burkley and Schaefer (1936) described the electrocardiogram of 11 foetuses from 9½ to 25 weeks gestation obtained by hysterotomy or following spontaneous abortion. Their findings with the three standard leads were described in some detail and, in particular, the presence of right heart predominance was emphasised.

Work by Hoff et al. in 1939 on the chick embryo advanced this type of study further by studying the genesis of the electrocardiograph wave form from the simple tubular

ventricle to the complete adult stage.

Further progress in the next decade was limited, and surprisingly few advances occurred in the field of foetal electrocardiography, as more emphasis was placed on foetal phonocardiography. Some effort was also made to rationalise clinical studies of the foetal heart rate. For example, Sontag and Newley (1940) recorded a series of half-minute observations of foetal heart rate during the last five calendar months of pregnancy in 63 normal foetuses. Foetal heart rates in excess of 160 beats per minute occurred in 83% of all cases, and heart rates less than 100 beats per minute in 28% of cases. This paper emphasised for the first time the difficulty of using random clinical measurement of foetal heart rate as a guide to foetal welfare.

In 1941, Mann and Bernstein reported a comparative study of various lead systems using a standard amplifier electrocardiograph. Electrodes were applied to the arms and legs, and at the same time, a variety of abdominal lead positions were tried. The abdominal leads were clearly superior and a foetal electrocardiogram was demonstrated at 16 weeks gestation. Thirty-six of the 40 patients showed positive recordings.

In the same year, Dressler and Moskowitz reported the first combined study of the human foetal electrocardiogram and the foetal phonocardiogram. The purpose of this study was to develop a positive method using a simple technique for determining the presence of foetal life. Forty patients were studied in the last two months of pregnancy. The foetal stethogram was positive in 100% of cases, but the foetal electrocardiogram was positive in only 80% of cases.

A significant technical advance was made by Ward and Kennedy (1942), when they used a three channel balanced amplifier developed to record electroencephalograms. Whilst the percentage of positive tracings from abdominal electrodes was no better than those obtained in earlier work, the recordings obtained were clearly the best foetal electrocardiograms obtained up to this time. The authors suggested that the problems of detection were purely those of amplification, but it is apparent from subsequent studies that amplification alone has limitations, as it also leads to amplification of background noise and obscures the foetal signal.

In an extensive study of abdominal lead recordings, Goodyer, Geiger and Monroe (1942) used a standard amplifier type electrocardiograph with a single stage resistance-

coupled pre-amplifier of simple design. The pre-amplifier circuit increased the overall sensitivity 20 fold. A 0.01 microfarad series condenser was placed in the output of the pre-amplifier to eliminate undulation of the base-line. Abdominal leads were applied in a series of positions and vaginal leads were used but were not satisfactory because of baseline tremor. In this study, 154 pregnant women were examined. Positive results were obtained in 87% of all patients, the earliest recording being obtained at 17 weeks' gestation. The R wave only was seen with a spike voltage of up to 70 microvolts, the direction of the R wave depending on foetal presentation. Foetal heart rate was studied in relation to duration of pregnancy, maternal heart rate and foetal sex, but no correlation was established. The average amplitude of the foetal R wave increased near term, but no consistent progression in amplitude was found during pregnancy.

Further similar studies by Paley and Krell appeared in 1944, but apart from some isolated case reports, no further significant contributions to this subject occurred in the 1940s.

In a further attempt to improve the quality of recordings obtained from abdominal electrodes, Vara and Halminen (1952) placed their patients in an earthed cage

of metal wire. Ninety-two patients were examined and in 10 patients, recordings of the foetal electrocardiogram were obtained as early as the third month of pregnancy. These workers also used a lead placed in the vagina, but not attached to the foetal presenting part.

The next significant technical advance in foetal electrocardiography occurred in 1953, when Smyth described a technique which gave the best recordings up to this time. Using a direct writing electrocardiograph of conventional design, a balanced preamplifier operated by batteries was introduced into the circuit. Energy-frequency analysis of the QRS complex (foetal and neonatal) showed that the energy was widely distributed, but was maximal between 20 to 40 cycles per second, and the frequency response was therefore adjusted to cut off below 30 cycles per second and above 100 cycles per second. One and a quarter inch silver discs were used for electrodes, with saline used as a conducting solution on the skin. One electrode was placed at the uterine fundus and one over the suprapubic area in the midline. Undesirable background electrical noise was reduced by using a pre-amplifier which was of a balanced input type sensitive to differences in potential between two electrodes, but much less sensitive to potentials between either electrode and earth. Only two recordings in 100 patients were negative, and these foetuses

were subsequently delivered stillborn. Smyth also recorded the foetal electrocardiogram from a direct foetal lead, a subject that is reviewed in the next section.

The application of foetal electrocardiography for the diagnosis of foetal life appeared to be the dominant interest in this subject at this time, and further publications by Southern (1954), Schomig (1954) and Bernstine (1955) emphasised the value of foetal electrocardiography as a diagnostic aid.

Davis and Meares (1954), using an electroencephalograph, claimed to demonstrate complete foetal complexes from maternal abdominal leads at 19 weeks gestation. However, it is doubtful from examination of the published recordings that the claim of these authors was valid. Despite significant improvement in instrumentation, the only consistent signal recorded from the foetus by using abdominal electrodes was the foetal R wave.

The question now arose as to whether the techniques of foetal electrocardiography could be used in studying foetal asphyxia. Enhorning and Westin (1954) studied the electrocardiograms of 37 previable foetuses under conditions of increasing asphyxia. A steady decline in heart rate and blood pressure was noted and positive T waves became negative in leads I and II with prolongation of the

P-R interval. The introduction of oxygenated heparinised blood into the umbilical vein caused a rapid and sharp rise in heart rate and arterial blood pressure.

In 1957, Southern reported the first extensive clinical study of the prenatal foetal electrocardiogram in relation to foetal anoxia. The study resulted from the development of specially designed equipment which made available continuous oscilloscopic monitoring and camera recording of the foetal prenatal electrocardiographic complexes. Two amplifier channels with a lead selector system were used and recordings obtained with abdominal electrodes. Ninety-six patients were studied and oxygen saturation studies were performed at birth on umbilical arterial and venous blood. The study included 46 normal subjects in late pregnancy, 22 patients with clinical signs of foetal distress, 12 patients with toxaemia or chronic maternal hypertension, and eight cases of postmaturity. This study suggested that, in cases of foetal distress, P wave amplitude was increased, the P-R interval was lengthened from a normal value of 0.06 second to 0.12 second, and the S-T segments was isoelectric or depressed. This paper is discussed in more detail later in this review.

In the latter part of the 1950s, it is apparent from the literature that a wider search was being made for other

applications of foetal electrocardiography. The difficulties of obtaining suitable recordings and of obtaining a satisfactory foetal parameter for comparison with these values were clear by this time. Hellman et al. (1958) stated that, up to this time, no characteristic "anoxic" electrocardiographic pattern had been defined. With the development of a variety of cardiotachometers, interest became centred on continuous electronic monitoring of the foetal heart rate. Because of the difficulty in obtaining a satisfactory electrocardiographic signal, foetal heart sounds were used. Hellman et al. (1958), Smith and Carey (1956) and Corner and Stran (1957) reported the use of phonocardiography and heart ratemeters in the study of foetal anoxia, but no systematic changes were described. Using abdominal foetal electrocardiography, Hon (1958) evaluated the foetal heart rate of 80 patients in labour. The foetal ECG was separated from the maternal ECG by in-phase cancelling of the maternal complex in a differential amplifier. In this technique, described in "Science"in 1957 by Hon, one input channel is connected to electrodes from the lower abdomen, where both maternal and foetal electrocardiograms are present. The other input channel is connected to two electrodes on the upper abdomen where the maternal electrocardiogram alone is present. This maternal complex is used for cancellation. Heart rate was

then determined by manual scanning over ten second intervals and, later, by the use of a cardiotachometer. In most patients, foetal heart rate did not drop significantly, but in some vertex presentations the heart rate dropped to 60 to 70 beats per minute, but returned to normal before the end of contractions. It was suggested that this bradycardia is related to raised intracranial pressure, but that if the cervix was less than four centimetres dilated, or more than eight centimetres dilated, it is abnormal. Persistent bradycardia between contractions was considered to indicate foetal anoxia.

Further publications by Larks et al. (1960), Wong and Cassels (1960) and Hon and Hess (1960) continued to stress the use of abdominal foetal electrocardiography for the diagnosis of foetal life and foetal presentation. Bergman and Hall (1958), in a study of 449 foetal electrocardiograms, showed that the amplitude of the QRS complex increases during the last two months of pregnancy and decreases in amplitude beyond term. These authors suggested that prenatal foetal electrocardiography might be useful in the detection of true postmaturity if serial examinations were made.

Experimental studies at this time by Jackson et al. (1960) drew attention again to the potential value of the

ECG configuration of the foetus as a reflection of the foetal homeostasis. ECG leads were inserted through the uterus in rabbits into the limbs of the foetal rabbit, and recordings were made using standard limb leads. The ECG of the foetus showed a marked right axis deviation. Handling of the uterus produced marked bradycardia and T wave flattening. Anoxia induced by occlusion of ovarian and uterine vessles produced bradycardia and T wave "flutters".

In 1961, Stern, Lind and Kaplan recorded the electrocardiogram directly from the foetus removed at hysterotomy in 20 patients from eight to 20 weeks gestation. Electrocardiograms were obtained with four limb leads and one chest lead and the recordings were obtained after the foetus had been removed from the uterus, but was still attached to the placenta in the uterus. The average electrical axis was $+119^{o}$, supporting the concept of right ventricular preponderance in foetal life. The P wave tended to be peaked and the average PQ interval was 0.085 second. The average QRS interval was 0.03 second and the QT interval was 0.29 second. Terminal changes in the ECG following removal of the foetus from the uterus showed a decline in heart rate, dissociation of the ST segment and bizarre forms of the QRS complex.

Further technical advances were described in 1961.

Marshall and Shubeck described a method of time lapse electrocardiograms for use in "at risk" cases. The abdominal foetal electrocardiogram was sampled electronically for four seconds every ten minutes, using an automatic timing device. Sureau and Trocellier (1961) described a method of eliminating the maternal electrocardiogram by using multiple abdominal leads and a potentiometer to record only a foetal QRS complex. The inadequacy of amplification alone is obtaining a clear foetal signal had been recognised by this time and, in an effort to improve signal-to-noise ratio, Hon (1961a) described a technique using a series of three preamplifiers fed into a final operational amplifier.

The use of foetal electrocardiography for clinical "office" work was described by Shubeck (1961). In the same year, Smartout, Campbell and Williams, using abdominal leads from the maternal abdomen in 36 patients, demonstrated that the foetal heart rate varies constantly whilst the mother is at rest. A mixture of oxygen and 5% carbon dioxide did not alter these patterns. Using similar methods, Hon and Wohlgemuth(1961c) recorded the changes in the foetal heart rate in response to a standard exercise test. Minor fluctuations in the pre-exercise baseline of the foetal heart rate disappeared

after exercise. However, in six of the 26 patients examined, gross changes in foetal heart rate occurred. Foetal bradycardia was followed by tachycardia, and the authors suggested that maternal exercise might decrease uterine blood flow and hence put a temporary additional load on oxygen transfer, producing an abnormal foetal heart rate pattern. The evidence for the clinical value of this test is not convincing.

In 1962, earlier findings on the value of foetal electrocardiography in the detection of foetal life were confirmed in two extensive studies by Schmidt, Cruikshank and Saunders, and by Wells and Swartout. Lamkee, Huntington and de Alvarez (1962) showed that the average foetal heart rate declines from an average of 165 beats per minute at 12 weeks gestation to 130 beats per minute at term. They also emphasised that foetal position can be roughly determined by spatial vector cardiography. In a study of abdominal ECGs in 84 patients during delivery, Larks and Longo (1962) showed that late and persistent bradycardia is seen frequently near delivery. Occasionally, slight or pronounced morphologic changes were seen in the QRS complexes, including widening and notching, and ST segment depression was also seen in association with foetal depression.

In an isolated case report, Freistedt (1962) described an abnormal foetal electrocardiogram with the presence of a foetal bigeminal rhythm.

Sureau (1962) introduced a note of caution in the interpretation of the foetal electrocardiogram. He presented a series of 39 cases with spurious foetal tachycardia recorded by abdominal foetal electrocardiography. The importance of using a time constant of at least 0.3 second if artifacts in the displacement of the ST segment were to be avoided was also stressed.

Two significant new approaches were introduced in 1962. Using maternal electrocardiography and foetal phonocardiography, Morton, Tolles and Hellman sampled rate analogs. These samples were then converted by analog-to-digital conversion system to digital computer words. Samples were taken at a rate of 40 samples per minute and maternal and foetal heart rate baselines were established during a 20 minute run-in period and in response to atropine administration. This paper presented a new approach to data processing.

Kendall, Farell and Kane introduced a new technique of foetal radioelectrocardiography. Using a small frequency modulated radio transmitter, the electrocardiogram

was transmitted from abdominal electrodes to a desk radio receiver. The advantages of this system are good quality recordings with very much reduced interference. These authors suggested that the most significant part of the foetal complex is the ST segment, and suggested that ST segment depression should indicate foetal anoxia.

The use of computer techniques in handling ECG data was introduced by Hon and Lee in 1963(f). Abdominal and scalp leads were used and after suitable amplification, the foetal electrocardiograph was recorded on magnetic tape with an Ampex FR1100 recorder. The filtered and amplified signal was then used as the input to a pulse generator which provided a pulse to act as a trigger to instruct the computer to begin averaging. A marked difference in signal-to-noise ratio was produced and good ECGs were obtained by averaging 10 to 50 complexes. As the R to R peak is variable, the preceding R wave could not be used to trigger the averaging computer and so the tape was reversed and a fixed signal placed on the tape in front of the R wave at a set interval. By playing the tape in the correct manner, the trigger signal preceded the actual complex which could then be averaged.

Further work in 1963 by Buxton, Hsu and Barter, and Mayes, Bradfield and Smyth recorded experiences with the

clinical applications of foetal electrocardiography in the diagnosis of foetal life. Caughey and Krohn (1963) described the variations in signal strength recorded from abdominal foetal ECGs and confirmed the findings of earlier workers in describing the presence of a diminished signal strength from 26 to 30 weeks gestation. These authors also described the difference in conductivity in foetal fat, vernix, amniotic fluid and uterine vascularity that would be necessary to produce these changes.

Rozkowski, Kretowicz and Wichrzychi (1963) described the use of abdominal foetal electrocardiography for routine monitoring of the foetal heart rate. Similar studies using phonocardiography for heart rate monitoring in 1000 labours were described by Ginsburg and Gerstley (1963).

A wider use of qualitative and quantitative aspects of the foetal electrocardiogram was introduced in 1963. Larks described a series of 10 abnormal abdominal ECGs from a group of 1500 recordings. The changes described in the QRS complex included widening and flattening of the complex, pulsus bigeminus and other cardiac arrhythmias. In seven of these patients, complications of the cord were described. Quantitation of the ECG voltage showed that ECG voltage increases from 30 weeks gestation to term.

Mattingly and Larks (1963) described abnormal ECG complexes in conditions of "abnormal uterine environment". These changes included W shaped QRS complexes and ST segment depression.

In a further study of ECG voltage, Larks and Larks (1964) showed that the mean amplitude of the foetal electrocardiogram from abdominal leads was 25.2 μV and that, where the ECG was of low voltage, voltage of the neonatal ECG in lead II was also low.

In 1965(a), Larks introduced a new parameter of assessment by introducing a method of estimating the axis of the foetal heart from the sum of positive and negative deflections from the abdominal ECGs. By this method, the electrical axis of the foetal heart fell between $+90^{\circ}$ to $+180^{\circ}$, with a mean of $+134^{\circ}$. Breech presentation showed a negative value. Instances of foetal difficulty, foetal distress and congenital heart disease seem to be associated with values from 0 to 180°. Larks showed that the abdominal foetal electrocardiogram is similar to lead II of the neonatal ECG. The estimation of the foetal heart axis is based on the algebraic sum of the R wave and Q or S wave. This is related to the neonatal axis which is derived from the fact that the projection upon lead II is mathematically related to the electrical axis vector X the

sine of the angle which the electrical axis forms with the 150^{o} line. In a further study of 11 complicated cases selected from a series of 2500 patients, Larks and Larks (1965b) showed that right axis deviation commonly occurs in the presence of postmaturity, cord problems, and pre-eclamptic toxaemia. The authors suggested that these changes represent chronic hypoxia. An analogous situation occurs in infants born at high altitudes, with persistent right axis deviation.

In a further comparative study of ECGs obtained from abdominal leads, scalp leads and direct from the neonate, Roche and Hon (1965) confirmed the morphological similarities between the foetal leads and lead II of the neonatal ECG.

Using their new method of foetal radioelectrocardiography, Kendall, Farell, Kane and Ostrand (1964) reported the presence of abnormal wave forms in seven patients with clinical foetal distress. The characteristic features were prolongation of conduction times and depression of the ST segment.

Foetal electrocardiography was used about this time to study a variety of situations. Kendall and Farell (1965) recorded the foetal ECG from abdominal leads in 20 patients having delivery by elective Caesarean section.

Thirteen patients showed significant foetal bradycardia at some stage during delivery, four of these patients developing bradycardia at the time the skin incision was made.

Stander, Banden, Thompson, Pugh and Werts (1964) and, later, Shenker (1965) studied the effects of maternal isoxsuprine infusion on the foetal heart rate. Both studies showed that mild elevation of the foetal heart rate is a common sequel to the use of this drug.

In an interesting case report, Hess and Davis (1964) recorded the effects of hypothermia on foetal heart rate in a patient requiring craniotomy. Marked foetal bradycardia occurred without any apparent ill effects to the foetus.

The question of recording artefacts, first reviewed by Sureau, Trocellier, Chavinie and Cannan in 1964, was investigated again in 1966. Kawtor, Bowman and Abbott reported two instances of false foetal ECGs recorded from abdominal leads with the foetal RKG500 radioelectrocardiograph, obtained in the presence of intrauterine death of the foetus. Whitfield (1966), in an excellent review of the subject of ECG artefacts, reported a further seven patients in whom an unusual series of rapid impulses was seen using maternal abdominal leads. He concluded that

the impulses were maternal in origin and probably represented the summated action potentials of a small number of contracting motor units in the rectus abdominis muscles.

Further technical refinements were also reported in 1966. Offner and Moisard described the use of a group of four amplifiers and a coincidence technique which resulted in a clear trace of the foetal ECG recorded from abdominal leads with elimination of much of the background noise. Walden and Bimbaum described a simple method of cancelling the maternal complex by using two separate preamplifiers with leads from the chest and abdomen. The sensitivity of the two amplifiers was balanced with the maternal ECG being reversed, in order to cancel each other, but preserve the foetal ECG. Larks and Larks (1966a), in a comparative study of foetal electrocardiograms obtained from abdominal leads and neonatal electrocardiograms, investigated 1606 cases of singleton pregnancies and divided these subjects into abnormal and normal groups. The purpose of this investigation was to find out whether the two groups differed with respect to correlation coefficients relating to their electrical axes and associated measurements. The findings showed that the difference between correlation coefficients of normal and abnormal subjects was significant in comparisons between the foetal R wave and newborn R wave, in the newborn R wave versus the foetal cardiac

axis, and where the foetal cardiac axis was compared with the newborn cardiac electrical axis. In other words, correlation coefficients in the parameters measured tended to be lower in the abnormal groups than those in the normal groups. This suggested greater variability and birth process changes in the abnormal cases.

In a further publication in 1966(b), Larks and Larks reinforced their previous claims on the validity of assessing foetal electrical cardiac axis by a comparative study of foetal ECGs obtained by abdominal leads and lead II of the neonatal ECG. The foetal cardiac axis in utero was assessed at 135.7^{o} and the neonatal cardiac axis was 134.9^{o}. The findings were consistent with right ventricular predominance and muscularity, and with relatively high right ventricular and pulmonary arterial pressures.

In 1967, Larks, Webster and Larks presented further quantitative data from the foetal electrocardiogram in relationship to condition at birth. The amplitude and width of the QRS complex and the QRS ratios were studied. The foetal ECG complex was the most significant variable studied.

Kendall, in the same year, reported a series of 19 patients in whom abnormal foetal arrhythmias were recorded using abdominal foetal electrocardiography. The

abnormalities included atrial extrasystoles, sinus tachycardia, sinus bradycardia, bigeminal rhythm and heart block. The author concluded that arrhythmias did not necessarily indicate foetal acidosis, although no acid-base measurements were made in this study. A variety of technical modifications were described in 1967. Schuler, Glass and Whyte described a simple self-contained battery operated octave bandwidth amplifier which can be adapted to a standard electrocardiograph. This equipment was particularly suitable for the inexperienced operator because of low noise, high input impedance and good differential rejection ratio of the amplifier section. Steerman, Garra and Catalaud (1967) stressed the advantages of multiple abdominal leads in increasing the accuracy of diagnosis of foetal life and recommended that six leads should be used in each patient.

Levine and Weiss (1967) made a comparative study between standard and radioelectrocardiographic techniques and found no difference in the accuracy of diagnosis of foetal life, although only 13 subjects were studied by the latter technique.

Copher and Huber (1967) induced maternal hypoxia in a series of 473 patients antenatally using 15% oxygen and 85% nitrogen. Using abdominal leads to record the foetal

ECG, they showed that oxygen deprivation produced little change in normal subjects, but in subjects with reduced placental function, produced profound bradycardia. These findings were related to the subsequent development of the signs of foetal distress.

In a study on the individual differences in heart rate variability, Welford, Sontag, Phillips and Phillips (1967) investigated beat-to-beat heart rate records from 10 foetuses in the last six weeks of pregnancy and showed considerable variability between individuals, but a tendency to consistency within any single individual.

From 1967 to 1969, further advances in the registration and interpretation of foetal electrocardiographic data obtained from maternal abdominal leads have been made. The use of computer techniques has made it possible to handle large collections of data. For example, in 1968, Larks, Webster and Larks reported a variety of parameters of the foetal ECG (34 variables in all) in 2028 cases. The study revealed some surprising findings. A sex difference was shown to exist in foetal ECG values. It was shown that the peak to peak amplitude of the complex, the foetal R wave and the newborn R_2 were all larger in the female than in the male, and comparable differences were observed in electrical axis studies. In 1969, the authors

extended this study to include 5000 pregnancies. They showed that the diagnosis of breech presentation from electrocardiography is restricted by the sex differences in configuration of the ECG. It was suggested that rotation of the foetus in utero takes place "under the influence of an optimal endocrine environment" - a theory which will surely be difficult to substantiate.

Further studies in the quantification of foetal ECG data by Behrer, Glaeser, Cox and Woolf (1968) was obtained by computer processing with a study of the trends occurring through pregnancy. Once again, an averaging technique was used and the duration and amplitude of the QRS complexes recorded.

Nielsen and Moestrup (1968) and, in the same year, Takemura, added to the literature on foetal cardiac arrhythmias without arriving at any very definite conclusions as to their significance.

Finally, in 1968, a series of publications concerning methods of improving recording techniques from abdominal leads appeared.

Berard, Katz and Uner performed a comparative study of different types of electrode gels for improving skin contact. The importance of high salinity in these gels

was emphasised and the merits of silver-silver chloride electrodes confirmed.

The value of multipolar registration of the foetal electrocardiogram was studied by Gielin, Schmidt and Megens, and the method was shown to be particularly reliable in detecting foetal complexes in early pregnancy. Schuler, Puddicombe and Park (1968) used a selective combination of signals obtained from several abdominal sites to attenuate random noise and the maternal ECG, and obtained a relative improvement in foetal signal amplitude. Rhyne (1968) has described a new signal enhancement device that computes the average ECG, but gives more weight to more recent complexes. This permits the observation of short-term anomalies in the foetal complex.

Van Bemmel, Peeters and Hengeveld (1968) showed that averaged complexes can be distorted by maternal ECG waveforms. They suggested that it was wise to average only those foetal ECG complexes in which the foetal and maternal R waves do not coincide. A considerable improvement in the representation of the average foetal complex from abdominal leads was obtained by observing this principle.

SECTION 2. The Use of Direct Foetal Electrodes

The advantages of using abdominal leads to record the foetal electrocardiogram are readily apparent. Electrodes can be easily applied to the maternal abdomen, and do not constitute any risk to the mother or foetus. However, the disadvantages of this method are equally apparent. The amplitude of the signal obtained is often very little more than background noise. In addition, the signal obtained is always affected by the maternal electrocardiogram. P and T waves are rarely clearly visible and only the QRS complex is consistently seen. New computer averaging techniques have resolved many of these difficulties, but these techniques are expensive and not readily available.

The simple answer to many of these problems is to apply electrodes directly to the foetus. This can be achieved easily and safely when labour has started and the cervix is dilated.

It was not until 1953 that Smyth first described the use of an electrode applied directly to the foetus. In one patient, a silver wire electrode was passed into the amniotic sac through a polythene catheter. This gave an excellent foetal electrocardiogram which clearly showed foetal complexes, including P and T waves. The signal was free of maternal complexes and was five times the amplitude of the

signal obtained from abdominal leads. The electrode was not attached to the foetus, but lay in contact with it in the amniotic sac. In 1956, Sureau described a more extensive application of the use of a vaginal electrode. A scalp electrode was used which consisted of a silver chloride plate on a wire conductor isolated from maternal tissues by a polythene sheath. The electrode was introduced through the cervical os and held manually against the foetal scalp in a series of 34 patients. Recordings similar to those presented by Smyth were obtained.

In 1960, Hunter, Lansford, Kroechel and Braunlin described an electrode made of insulated wire covered by a sliding plastic sleeve. The tips of the sprung steel wire were sharpened so that the electrode could be inserted and retained in the foetal scalp. A similar electrode was introduced into the skin of the maternal perineum and an additional indifferent grounding electrode attached to the right leg. Good quality foetal ECGs were obtained. However, in view of the discomfort to the mother of the perineal electrode, it was unlikely that such a technique would achieve wide acceptance.

In 1958 and 1959, Hon and Chung, and Hon recorded the use of abdominal electrodes and abdominal foetal ECGs for studies on heart rate in response to normal and abnormal

labour, and in response to compression of the foetal skull. The heart rate was recorded by manual reduction technique of the ECG data, or by cancellation of the maternal ECG and the use of a cardiotachometer. It was shown that the foetal heart rate did not fall significantly during contractions in normal breech labours and normal vertex deliveries. It was suggested that, where foetal bradycardia did develop during contractions, this might be the result of foetal head compression. This was subsequently investigated by applying pressure directly to the foetal skull.

In 1961, Hon, Bradfield and Hess presented a study of five subjects. In four of these, there were clinical signs of foetal distress. The foetal electrocardiogram was recorded by attaching clip electrodes to the foetal scalp. Heart rates were plotted logarithmically. In four subjects, the cord was shown to be around the neck at birth, and in one subject the cord was prolapsed. In each case, profound bradycardia developed during contractions. This bradycardia was clearly modified by administration of atropine to the mother. During bradycardia, there appeared to be a shift in pacemaker with P waves absent before some QRS complexes. The authors suggest that sinus arrest occurs and nodal rhythm sometimes supervenes. It is also suggested that foetal bradycardia may be a compensating mechanism associated with increased vagal tone. This hypothesis was developed further

by Hon in 1962 in a study of foetal distress and the importance of increased vagotonia in the causation of foetal bradycardia and the passage of meconium was emphasised. In other words, the accepted signs of clinical foetal distress do not always indicate foetal hypoxia.

Hon and Huang (1962) also reported a series of 25 patients with foetal cardiac arrhythmias during labour caused by premature and missed beats. In 22 patients, the arrhythmia disappeared almost immediately after birth, and in two patients it persisted for 48 hours. All infants in this series were delivered in good condition and it was therefore suggested that vagotonia possibly associated with mild hypoxia was the main cause of the arrhythmias.

In 1963, a great upsurge of interest in the techniques and applications of direct foetal electrocardiography occurred. A variety of technical modifications and improvements were described. Stander, Barden and Braunlin described the use of multi-channel recorders for measuring multiple parameters during labour, including maternal blood pressure, intra-amniotic pressure, foetal electrocardiography and foetal heart rate. Scalp clips constructed from Michel wound clips were used but a second electrode was still attached to the maternal perineum. This particular discomfort was eliminated by the electrode design of Hon described in 1963, which

eliminated the necessity for using a skin electrode by placing the second lead in the vagina. These electrodes were constructed from insulated wire, with one lead soldered to a surgical skin clip and the other to a coil of 22 gauge fine silver wire. With adequate insulation of the clip, this electrode minimized interference from the electromyogram of the abdominal wall, and had the additional advantage of being almost free of discomfort to the patient. Labours were monitored for as long as 23 hours. Of 249 patients monitored, only three developed mild uterine infections. Only one scalp infection occurred, and occasional scalp ecchymoses were seen.

Vasicka, Hutchinson, Rylander and Murray (1967) inserted platinum electrodes directly into the foetus through polyethylene tubing at the fundus and lower segment of the uterus. Where two intrauterine leads were used, foetal signals alone were obtained, but the quality of the ECG was variable, depending presumably on the adequacy of contact with the foetus.

Kelly (1963) used scalp and abdominal leads to record the foetal heart rate during instrumental delivery. Strain gauges fitted into the forceps blade measured the stresses obtained during forceps delivery. Slowing of the foetal heart rate was commonly seen during traction of both forceps

and vacuum extractor. Forceps deliveries in which individual traction forces exceeded 50 pounds were associated with a high rate of depressed infants.

Further advances occurred in data processing techniques. Hon and Lee (1963f) described a method of averaging foetal complexes obtained from scalp electrodes. Signals were recorded on magnetic tape with an Ampex FR1100 recorder. This was subsequently played back and further amplified by another preamplifier to raise the voltage to the required peak-to-peak level of six volts for a CAT computer. The filtered ECG was used as an input to a pulse generator which provided a pulse of 1.5 volts to act as a trigger to instruct the computer to begin averaging. A marked improvement in signal-to-noise ratio was obtained.

As a starting point of an automatic data-processing and computer analysis programme, a working classification of foetal heart rate was defined by classifying the limits of normal heart rate, and ranges for moderate and marked bradycardia and tachycardia were defined.

Hon (1963b) described a series of heart rate patterns in labour. The appearance of bradycardia confined to the time of a contraction did not indicate hypoxia. Artificially induced maternal hypoxia resulted in foetal hypoxia and more

severe hypoxia resulted in bradycardia persisting after the end of a contraction, and eventually persisting without remission. In a further elucidation of the mechanism of foetal bradycardia, Lee and Hon (1963) demonstrated the effect of cord artery and cord vein compression at Caesarean section in 11 patients. Compression of the umbilical vein or the whole cord produced prompt and profound foetal bradycardia.

To relate these changes to clinical conditions of asphyxia in the foetus had, to this stage, proved difficult. Hon and Lee (1963e) presented data on foetal heart rate patterns and ECG configuration in a series of cases preceding foetal death. As death approached, foetal bradycardia with onset at the latter part of the contraction of the uterus became progressively prolonged, until the bradycardia became persistent. In all instances, foetal tachycardia was noted 30 to 40 minutes before delivery. High peak and often biphasic or inverted P waves were noted, and the PR intervals were shortened. In addition, wandering pacemaker, false bundle branch block, widened QRS complexes and ST segment depression were noted. Newman (1963), however, showed that bradycardia was a common finding during delivery and cardiac arrhythmia also commonly occurred. In cases of placental insufficiency, tachycardia occurred before contraction, followed by the late onset of bradycardia in the contraction phase.

These findings were confirmed in 1965 by Ginsburg and Gerstley, in a study of 102 cases of foetal tachycardia in labour, compared with a control group of 251 cases. In this study, a phonocardiotachometer was used. The authors suggested that there was little difference in the immediate neonatal condition between the tachycardia group and the control group. However, there appeared to be a profound worsening in the neonatal condition when episodes of foetal bradycardia took place in labour in addition to tachycardia.

Bieniarz, Fernandez-Sepulveda and Caldeyro-Barcia (1965a and b) recorded the effects of drug-induced hypotension on the foetal heart rate in labour using direct foetal electrocardiography. Normal uterine contractions had no effect on the foetal heart rate, either during hypotension or during normotension, in the dorsal or lateral position of the mother. The only noticeable influence of hypotension was a slight tendency to accelerate the foetal heart rate. This tachycardia returned to normal with restoration of normal blood pressure. In a further similar study in two labours complicated by cord prolapse, and one labour complicated by toxaemia, transient bradycardia occurred late in the uterine contraction phase (described by Hon as type II dips). The amplitude of these dips was significantly greater during maternal hypotension and decreased when the

mother breathed oxygen. These results suggested that this pattern of heart rate change in the foetus was hypoxic in origin.

Publications at this time were mainly directed at patterns of foetal heart rate changes, but further attention was also directed at the configuration of the foetal electrocardiogram. Lee and Hon (1965) described seven cases in which abnormal QRS complexes were recorded during labour. Sporadic marked changes in the QRS configuration were associated with manipulation of the umbilical cord.. The authors suggested that these changes may reflect interference with the usual excitation pattern of the foetal heart by sudden ventricular dilation and strain secondary to profound alteration in foetal haemodynamics.

Persistent changes in the QRS complex which were unaltered by the events of labour were thought to represent organic conduction defect. However, changes in the QRS complex present during labour may not be indicative of adverse foetal environment and a note of caution in attempting to relate such changes to foetal well-being was sounded.

In a comparative study of lead systems, Roche and Hon (1965) showed that the configuration of QRS complexes obtained from vaginal and abdominal electrodes in vertex presentation was similar, and that both resembled lead II

of the neonatal electrocardiogram. In the same year, Hon and Lee described new display techniques for reducing total labour records by employing group averaging techniques for ECG data and by storing the same data on microfilm.

In 1966, Hon described a significant improvement in instrumentation with the use of a modified scalp electrode. Nickel silver wound clips were used, covered by silastic adhesive and with a positive electrode made by soldering a fine coil of silver wire to the cable. Signal voltages between 300 to 500 microvolts were obtained. In the same year, Figuero-Longo, Poseiro, Alvarez and Caldeyro-Barcia obtained foetal electrocardiograms by inserting electrodes directly into the foetus through the maternal abdominal and uterine wall. Nine patients were included in this study and 1364 foetal ECG cycles studied. The greatest amplitude of signal and the lowest maternal interference were obtained when two direct electrodes were used by insertion of one into the foetal buttocks and attaching the other to the foetal scalp. The mean and standard deviations were recorded for the duration and amplitude of the different components of the ECG waveform.

In a study of the dynamics of foetal cardiac activity, Persianinov, Ilyin, Karpman and Savelieva (1966) recorded the foetal ECG and foetal phonocardiogram in labour. Scalp

electrodes were used in 15 subjects. The authors showed that the length of mechanical systole (time interval between first and second heart sounds) was directly related in a linear fashion to the foetal heart rate. It was also demonstrated that the QT interval became prolonged during bradycardia and that the second foetal heart sound sometimes preceded the T wave under these circumstances. After delivery, heart rates during the first few hours were much the same as before delivery. Mechanical systole was longer after delivery despite the similarities in heart rate, and there was a shortening of diastole. Phase shifts in the newborn infant indicated increased stress prolongation of systole.

Bradycardia occurring during contractions was thought to be a vagal effect, but against this theory is the constancy of PQ interval in conditions of fluctuating heart rate. The authors also suggested that prolongation of the QT interval may take place as a result of hypoxia.

The somewhat vexed question of the clinical value of foetal monitoring in labour was reviewed by Millican, Urbach, Carrington and Lambert in a report of 873 patients monitored by direct electrocardiography. Using the criteria previously established by Hon in reference to heart rate patterns, the authors showed that the clinical management

of 12.5% of these patients was influenced by the monitoring techniques, and they concluded that the expense and effort were warranted.

By 1967, more emphasis was being placed on methods of assessing foetal acid-base status, and on joint studies of foetal heart rate and acid-base status, and the number of significant publications on foetal electrocardiography per se declined. Hon described in detail modifications of the scalp electrode previously mentioned. These nickel-silver electrodes produced a marked reduction in noise level, in addition to increasing the amplitude of the foetal ECG.

In 1968, Crosby described the recording of the foetal electrocardiogram with a fine teflon-coated steel wire inserted through the intra-abdominal catheter placed in the foetal abdomen during intrauterine transfusion of the erythroblastic foetus. The author suggested this as a useful method of monitoring the foetus during the course of such transfusions.

In an interesting study on the inaccuracies of clinical auscultation of the foetal heart rate, Day, Maddern and Wood compared clinical auscultation with electronic readings of the foetal heart rate. A random error, an error biased towards normality when the heart rate was fast or

slow, and an error based on the inability to count the heart rate during contractions was noted.

In spite of the limitations, a clinically observed foetal heart rate of more than 160 beats per minute was shown to be associated with significantly lower Apgar scores at birth. In contrast, a steady foetal heart rate of 100 to 120 beats per minute was not associated with low Apgar scores.

Takemura, in a study of abdominal and scalp lead recordings on 200 women, produced evidence to suggest that the configuration of the foetal ECG obtained from scalp leads most closely resembled the augmented V lead from the foot, rather than lead II, as suggested by Hon.

SECTION 3. Foetal Acid-Base Balance

Acidosis and alkalosis are terms used to describe opposite types of disturbance of the body fluids. Acidosis may be defined as the condition in the blood characterised by the reduction of the $BHCO_3/HHCO_3$ ratio, and alkalosis as an increase in the ratio. When $BHCO_3$ is primarily affected, the disturbance is referred to as metabolic in origin. When $HHCO_3$ is first to be altered, the resulting change is considered to be respiratory in origin.

Primary changes of either $BHCO_3$ or $HHCO_3$ usually stimulate compensatory changes in the other, which tend to restore the original normal ratio. When compensation for acidosis is incomplete, an increased hydrogen ion concentration of the blood results.

For absolute diagnosis of acidosis or alkalosis, two of three variables - $BHCO_3$, $HHCO_3$ and pH - have to be derived. Since pH and $BHCO_3$ usually move in the same direction, it was common practice to determine only $BHCO_3$, using the Van Slyke manometric method. Although this measured the total CO_2 content of serum or plasma, it did represent principally $BHCO_3$ as 20/21 of the CO_2 comes from this source, the remainder coming from $HHCO_3$. Carbon dioxide tension represents $HHCO_3$ in plasma. The development of CO_2 electrodes which can rapidly measure carbon

dioxide tension and the development of glass micro-electrodes for measurement of blood pH have dramatically altered the technical availability of acid-base assessments in clinical practice. Whilst it is not the purpose of this review to discuss in any depth the experimental work that has led to the description of the metabolic adaption of the foetus to chronic asphyxia, some reference to the accepted description of these changes is included as a working basis for further observations. The major portion of this section is concerned with a description of the clinical observations and clinical assessment of foetal acid-base balance.

Dawes has summarised the major aspects of foetal vascular and metabolic changes in asphyxia in his book entitled "Foetal and Neonatal Physiology" (1968).

Under asphyxial conditions, the foetus can obtain some energy for survival by anaerobic glycolysis, at the expense of converting relatively large amounts of carbohydrate into lactic acid. The oxidation of glucose is the major energy source for the metabolic needs of the foetus. In the anaerobic phase, one molecule of glucose is oxidized to two molecules of pyruvic acid. In the absence of oxygen, pyruvic acid is reduced to lactic acid. In the presence of oxygen, pyruvic acid is oxidized in the citric

acid cycle to CO_2 and H_2O instead of being reduced to lactic acid. Anaerobic glycolysis provides only 1/19 of the potential energy of a glucose molecule. In addition, the resultant accumulation of pyruvic acid and lactic acid causes an increase in hydrogen ion concentration. This is reflected in foetal blood by a falling pH, reduction in blood buffer and, in particular, a reduction in blood bicarbonate.

These changes have provided a method for studying the severity of foetal asphyxia in a simple and rational fashion.

Interest in the chemical nature of foetal blood first arose in relationship to factors determining the onset of repiration in the newborn. As far back as 1888, Ahlfeld delivered several women in warm saline baths in order to demonstrate that irritation of the skin was not the factor initiating respiration in the newborn.

In 1932, Eastman published a classic paper entitled "The Chemical Nature of Asphyxia neonatorum and its Bearing on Certain Practical Problems". This paper merits consideration in some detail, as the conclusions reached at this time have formed the basis of knowledge of foetal acid-base balance. In this study, Eastman measured

the carbon dioxide content, the carbon dioxide tension and the hydrogen-ion concentration of maternal venous blood and umbilical arterial and venous blood at the time of delivery in 12 normal labours and six asphyxiated foetuses. The pH of the serum was measured with a Michaelis U electrode on a Leeds and Northrup potentiometer. Total carbon dioxide content was determined by the method of Van Slyke and Neill and CO_2 tension was calculated from the pH and total carbon dioxide content. It was shown that the carbon dioxide tension of foetal blood is higher than that in the maternal arm vein, even in normal subjects - the gradient being 1.3 to 1.

In severe asphyxia, the oxygen content of the blood was low, and carbon dioxide content was low " as a result of the displacement of this gas from base by the large amounts of lactic acid present". The pH of the serum fell and the carbon dioxide tension rose. Although the conclusions in this paper were limited by the extent of medical knowledge at that time, the basic description of the significant changes in blood biochemistry in foetal asphyxia have not really altered in the next 37 years. From these findings, Eastman concluded that the administration of carbon dioxide to the asphyxiated infant was not justified, and that the major requirement of such an infant was the administration of oxygen.

A similar study was performed by Noguchi in 1937, with certain modifications. For example, Noguchi pointed out that the formula used by Eastman in calculating carbon dioxide tension was incorrect. The author used a micro-glass electrode for measurement of blood pH and emphasised the importance of constant temperature in making pH measurements. The findings in this paper basically confirmed the conclusions of Eastman. However, the author did point out that "a low pH can be regarded as one of the characteristic changes in the blood properties of asphyxiated newborns - a poor oxygen content of the foetal blood cannot be regarded as one of such characteristics."

Kaiser continued this study in 1953 and measured the pH of maternal venous blood, uterine venous blood and umbilical arterial and venous blood in a series of patients undergoing elective Caesarean section. The mean peripheral vein pH was 7.38, uterine vein pH 7.36, umbilical vein 7.32, and umbilical artery pH 7.26.

By 1953, the specific nature of foetal adaption to hypoxia was being realised. Villee studied the metabolism of foetal liver and placental tissue in vitro. The glycogen content of foetal liver was shown to increase as gestation proceeded, and placental glycogen decreased as pregnancy advanced. Experiments with C_{14} labelled glucose showed that

the ability of the placenta to produce glucose decreased as pregnancy proceeded and the ability of the liver to produce glucose increased with gestation.

Cross, Killick and Huggett, in a review article of foetal hypoxia in 1954, stressed the ability of the foetus to exist for long periods without oxygen, the considerable powers of anaerobiosis being dependent upon glycolysis. This process, however, leads to the accumulation of fixed acids such as lactate and pyruvate.

Goodlin and Kaiser (1957) induced maternal acidosis by administering ammonium chloride to a series of patients. This produced foetal acidosis with reduced foetal pH, increased pCO_2 and reduced oxygen saturation. In some cases, the changes were disproportionate to those observed in the mothers. The data also suggested a time lag in the initial adjustment of the foetus to maternal acidosis. This study was of considerable importance as it emphasised that asphyxia is not the only cause of acidosis in the foetus, and that the foetal acid-base status is, at least in part, dependent on the maternal acid-base balance.

In 1960, Astrup, Jorgensen, Andersen and Engel described a new approach to the assessment of acid-base balance. This method introduced certain important advantages to the

clinical assessment of acid-base values. Firstly, it allowed rapid assessment of all relevant blood values for the identification of disturbances in acid base metabolism, and secondly, it allowed such an assessment to be made on capillary samples of blood. The relevance of these advances in relationship to studies on the foetus and newborn will be reviewed further in this section.

James (1960) studied oxygen saturation in cord venous and cord arterial blood in a series of vigorous and asphyxiated infants. A wide range of oxygen saturation was demonstrated, but higher values were not seen in the depressed group. The effect of asphyxia on blood pH was demonstrated. Depressed infants had lower pH values than normal infants and higher serum potassium levels. Cardiovascular collapse was shown to be imminent if blood pH fell below 6.9.

Further publications in 1961 contributed to the knowledge of acid-base values in the newborn and the neonate. Oliver, Denis and Bates described acid-base values in cord arterial blood samples following normal deliveries, forceps deliveries and elective Caesarean sections. Serum electrolyte values were also measured. The validity of the extraordinarily high potassium values must be questioned. McCance and Hatemi (1961) demonstrated that the ability of the newborn infant to excrete hydrogen ions is demonstrably

under-developed, and that the infant is therefore prone to acidosis when it is fed on unsuitable artificial foods.

In 1962, Vermelin, Dellenstable and Muller reported a valuable study on the blood gas and pH values of umbilical venous blood in 160 cases of foetal distress. These authors came to the conclusion that these cases could be divided into four groups:

(1) Normal oxygen saturation and normal pH - no anoxia.

(2) Low oxygen saturation and normal pH - recent anoxia of short duration.

(3) Low oxygen saturation and low pH - intense anoxia of longer duration.

(4) Normal oxygen saturation and low pH - anoxia before birth, which has led to acidosis.

The authors concluded that measurement of blood gases and pH can provide evidence of the duration and severity of hypoxia.

Spackman, Fuchs and Assali (1963) studied acid-base status and partial oxygen pressures in early and full term pregnancies. The authors showed that the foetus, regardless of its maturity, presents a certain degree of acidosis compared with maternal values.

These findings were confirmed by Low (1963) in a study of acid-base assessment in a series of normal obstetric patients. The pH estimation in the umbilical vein of 7.287, and umbilical artery of 7.196, demonstrated a degree of acidosis in the normal newborn infant which the author suggested was primarily respiratory in origin.

In 1964, Stenger, Eitzman, Gessner, Andersen and Prystowsky investigated the relationship between maternal and foetal acid-base balance. Maternal arterial and uterine venous blood were compared with values for pCO_2, pH and HCO_3, in cord arterial and cord venous blood. Although the actual values of pCO_2 were higher in cord arterial blood than either maternal arterial or uterine venous blood, the foetal pCO_2 was shown to vary directly with maternal pCO_2. A similar relationship was shown to exist between maternal and foetal blood bicarbonate values. Levison, Boston, Muirhead, Wang, Weiss and Smith (1964) attempted to relate maternal acid-base status and neonatal respiratory distress. Fourteen infants in this study developed respiratory distress. Neither maternal hypoxia nor acidosis at delivery, nor intrauterine foetal asphyxia could be shown to be significant aetiological factors in the development of respiratory distress in the neonate. However, acidosis in the infant was associated with maternal acidosis and tended to

persist for one hour after delivery.

Vedra measured oxygen and lactic acid values in normal and asphyxiated infants at birth, but was unable to establish any significantly different values in the normal and asphyxiated infants. Marse and Greene measured maternal lactate and pyruvate values during labour and showed that peak levels of lactate, pyruvate and excess lactate were reached at the time of parturition. Cord blood values were not measured in this investigation.

The biochemical changes resulting from acute or chronic asphyxial processes in the foetus were well documented by 1964, although most of these findings clinically were based on observations made on cord blood samples. An important development in the assessment of foetal acid-base balance was described in 1964. Saling described a method for obtaining capillary blood from the foetus during labour. Blood samples were collected from the presenting part of the foetus. The skin was first sprayed with chlorethylene to induce hyperaemia of the skin and arterialisation of the capillary blood. A small drop of silicon grease was wiped on the skin to prevent smearing of blood and the skin was incised to a depth of 2mm and 1.5mm wide. Blood was aspirated into heparinised tubing and oxygen saturation, carbon dioxide tension, actual pH value, buffer base and standard

bicarbonate measured. The influence of caput formation on the sample did not appear to be significant. Oxygen saturation was shown to rise during uterine contractions.

Because the blood sample was not obtained in a strictly anaerobic manner, the influence of air on blood gases was studied, using an experimental model. Oxygen content was not influenced by contact with air, but carbon dioxide tension diminished by a fraction. A small increase in pH therefore occurred, and this could be corrected by subtracting 0.024 pH unit and adding 9% to pCO_2 values.

A high spread of values was found in pH measurements of scalp blood, the range extending from 7.209 to 7.415 in the 77 deliveries included in this study.

This work provided a method which was basically simple for a new approach in the management of foetal asphyxia. The technique was not ideal for measuring blood gases, but it was an improvement on any methods available up to this time in the general acid-base assessment of the foetus, and it has been the basis of a considerable amount of further work. At the same time, the technique of amnioscopy for screening the colour of amniotic fluid in "at risk" infants was introduced as an adjunct to foetal scalp blood sampling.

In 1965, two further publications introduced Saling's method into the English-speaking world and expanded the application of this technique. Morris and Beard reviewed the rationale of biochemical monitoring of the foetus, and went on to describe a series of 26 patients in labour in whom the assessment of foetal and maternal acid-base status was studied. Using maternal venous blood samples - a source of later criticism in relationship to the significance of blood gas values in these samples - and scalp bloods, a constant maternal-foetal acid-base difference was shown to exist at all stages of labour. The mean foetal pH in early labour was 7.29 $\pm$ 0.05, in late first stage 7.30 $\pm$ 0.05 and a fall in pH to 7.28 occurred at full cervical dilatation. Maternal pH and base deficit showed a similar pattern.

A series of papers on cord blood values in a variety of situations appeared in the same year. MacRae and Palavradji studied cord arterial blood acid-base values in normal pregnancies and in patients with suspected placental insufficiency and/or foetal distress. In normal pregnancy, 80% of cases had a cord blood arterial pH > 7.20, whereas in pregnancies complicated by foetal distress, half the cases were associated with significant acidosis and hypercapnoea. Patients delivered by elective and emergency Caesarean section were also included in this paper. Low (1965) measured

oxygen saturation and acid-base values in 149 patients and infants delivered by Caesarean section under general anaesthesia and epidural anaesthesia. Oxygen saturation was shown to be lowest in the umbilical artery and vein in patients having delivery under epidural anaesthesia, whereas carbon dioxide tension was higher in patients being delivered under general anaesthesia - a finding which could be related to the method of ventilation of the mother during general anaesthesia. Crawford (1965) presented a similar study on 38 subjects delivered by Caesarean section under spinal anaesthesia, and showed that the length of time from induction of labour to delivery was related in a systematic fashion to increasing foetal asphyxia.

An interesting observation on the effects of positive pressure ventilation during anaesthesia for Caesarean section was also made in 1965 by Morishima, Daniel, Adamsons and James. These authors showed that artificial ventilation of the mother could induce a respiratory alkalosis in the foetus, but if this was excessive, i.e. to a degree that the arterial pCO_2 in the mother was reduced to a value of less than 17mm Hg, a rapid and severe metabolic acidosis occurred in the foetus. The suggested mechanism for this observation was that excessive ventilation of the mother induced a respiratory alkalosis in the mother which, if it was of sufficient severity, led to a reduction in the placental bed blood flow

and hence to acidosis in the foetus.

The method described by Astrup for measuring acid-base values which has been used extensively in the studies already discussed has not escaped criticism. Brackett, Cohen and Schwartz (1965), in a paper on the carbon dioxide titration curve of normal man, showed that blood in vivo does not behave the same way as blood in vitro. The addition of hydrogen ions generates less bicarbonate in the intact human than in whole blood in a test tube. This is, perhaps, not surprising, in view of the various methods available to the body to deal with hydrogen ions. The claims of these authors, however, whilst technically correct, tends to exaggerate the significance of these differences. The authors state that titrations in vitro on which buffer base, standard bicarbonate and base excess are based, are invalid. However, on examination, the carbon dioxide and bicarbonate titration curves in vivo and in vitro are very similar, excepting at very high values of carbon dioxide. Astrup's method would, therefore, appear to be valid in the assessment of metabolic and respiratory disorders of blood over the range of values commonly seen under clinical circumstances.

In 1966, further publications of foetal scalp sampling by Saling and a single case report by Suranyi et al. appeared

in the literature. Beard, Morris and Clayton applied scalp blood sampling techniques in 220 patients and also obtained maternal venous blood samples from these patients. The authors claimed that the use of maternal venous blood, as opposed to maternal arterial blood, was justified on the grounds of greater convenience and because a comparison of a brachial artery and cubital vein blood during both stages of labour showed that there was no significant difference in the degree of metabolic acidosis. The authors showed that a scalp blood pH value of less than 7.20 was associated with asphyxial depression of the foetus, and that the difference between maternal and foetal base deficit also provided an index for the severity of aphyxial depression in the newborn child.

Crawford (1966) analysed acid-base parameters in cord venous and cord arterial blood samples and analysed these results in relationship to various parameters of the Apgar score at 1 and 5 minutes. From a series of 162 patients, the conclusions made were that points allotted for colour at 1 and 5 minutes do not provide significant information of the condition of the foetus and the muscle tone is the first factor to be affected by asphyxia. Thalme (1966) presented an excellent paper on electrolyte and acid-base balance in foetal and maternal blood, in which a fall in blood pH and

a rise in serum potassium were demonstrated in foetal blood in rats and man. During the first 15 minutes after birth, umbilical arterial plasma showed a fall in plasma potassium and bicarbonate, but with an increase in phosphate and total protein.

An extensive evaluation of Saling's technique appeared in a series of articles in 1967.

Newman, Braid and Wood used the scalp blood sampling technique to observe the relationship between maternal and foetal blood pCO_2 and bicarbonate concentration. By artificially increasing and decreasing maternal pCO_2 a positive linear relationship between pCO_2 in maternal capillary blood and foetal scalp blood pCO_2 was established. A positive correlation was also found between heart rate and foetal blood pCO_2, and in four patients with foetal hypercapnia, the foetal heart rate pattern showed features consistent with cord compression. A positive relationship was also established between maternal and foetal base excess in the first stage of labour, whereas in the second stage of labour, the maternal base excess increased significantly without a concomitant rise in foetal values. Maternal infusion of sodium bicarbonate increased both foetal and maternal bicarbonate levels. The opposite effect was achieved by maternal infusion of ammonium chloride. McDonald and Kelman (1967)

stressed the value of foetal acid-base assessment in predicting impending danger to the foetus, and Bretscher and Saling (1967) defined the basic limits of pH for general clinical employment of this method. The lowest limit of normal pH values was found to be 7.20 and values between 7.20 and 7.24 were considered to be "prepathologic". Newman, McKinnon, Phillips, Paterson and Wood (1967) used Saling's technique to study oxygen transfer from mother to foetus during labour and demonstrated that inhalation of oxygen enriched gaseous mixtures by the mother increased both maternal and foetal partial oxygen pressures in normal subjects and subjects with placental insufficiency.

Surprisingly few adverse publications appeared up to this time on foetal scalp sampling. MacDonald and Kelman (1967) discussed some of the technical problems of the technique with particular reference to prevention of sample clotting and the loss of carbon dioxide through P.V.C. tubing. Paul, Garem and Whetham (1967) described the results obtained in 146 cases followed in labour by means of foetal scalp samples. Of these patients, 56 showed clinical signs of foetal distress but no difference could be demonstrated in the acid-base values of this group as compared with the clinically normal group. Very few cases of severe biochemical acidosis were included in the study and, in view of subsequent publications, the scope of this

work was insufficient to constitute a significant criticism of the technique. Saling and Schneider, in the same year, reported the results of a study of 850 labours selected out of 4396 deliveries, and showed that foetal acidosis occurred in 28% of the total number of deliveries and in 14.4% of the abnormal group of subjects. In 99 of 122 cases, the metabolic disorder was regarded as chronic in nature.

Beard, Morris and Clayton (1967) studied 250 patients with clinical signs of foetal distress and showed that a scalp pH greater than 7.25 in a sample taken within 30 minutes of delivery was associated with an Apgar score of 7 to 10 in 92% of infants, and a foetal scalp blood pH of less than 7.15 was associated with an Apgar score of less than 6 in 80% of infants. Wood, Lumley and Renou (1967) showed that, in babies born with low Apgar scores, abnormalities of foetal heart rate tended to precede a significant fall in foetal blood pH, but the foetal blood pH was a more useful prognostic measurement than either pO_2 or pCO_2.

In an interesting study of the effects of induced maternal metabolic acidosis on the human foetus and newborn infant, Blechner , Stenger, Eitzman and Prystowsky (1967) studied pH, bicarbonate and pCO_2 in maternal arterial, uterine venous and cord arterial blood at Caesarean section

in 15 patients before and after the administration to the mother of ammonium chloride. As maternal pH fell, the usual transplacental pH gradient was virtually eliminated. The authors suggested that maternal and foetal bicarbonate levels act independently, and that a limitation in placental permeability to hydrogen and bicarbonate ions appeared to exist.

In 1968, Persianinov and Savelieva studied acid-base and lactate-pyruvate levels in 3000 subjects and showed high lactate values with normal pyruvate levels in asphyxiated infants. Levels of carbonic anhydrase were shown to be low, suggesting that anaerobic glycolysis is, to a high degree "physiological" in the foetus. Renou, Newman, Lumley and Wood analysed foetal scalp blood during and between contractions in 28 patients and showed that, when the foetal heart rate remained constant, no significant change occurred in the mean pH, pO_2 or pCO_2. In eight cases, foetal heart rate fell during contractions and in five of these patients, foetal scalp pO_2 also fell, suggesting that the heart rate changes may be due to alterations in partial oxygen pressures in the placenta and foetus.

Wood, Lumley, Hammond and Newman (1968) measured scalp blood pH, pCO_2 and pO_2 in 147 patients with hypertension in pregnancy. Of this group, 69 showed hypertension and proteinuria and, in this group, the pH was significantly lower

than in normal subjects and in hypertensive subjects without proteinuria.

Beard (1968) reviewed the clinical application of scalp sampling over a 40 month period at Queen Charlotte's Hospital and showed that the Casesarean section rate for foetal distress over this time fell. Twentysix stillbirths occurred in 10,400 deliveries over this time and 13 of these were due to placental insufficiency. Three of these patients had scalp samples taken and two of these values were abnormally low, but were ignored. This paper establishes the clinical value of foetal scalp sampling in the management of foetal distress. In 1968, Walker, Phillips, Powe and Wood described a new electrode for measurement of scalp tissue partial oxygen pressures. An increase in these levels occurred following the administration of oxygen to the mother, and a decrease in partial oxygen pressure was noted during episodes of foetal bradycardia. Whilst qualitative changes in partial oxygen pressures could be demonstrated, quantitative measurements of these values were difficult, as the electrode could not be calibrated once it was inserted into the scalp tissues.

In 1969, further confirmation of the practical value of foetal scalp sampling appeared. Coltart, Trickey and Beard investigated 295 out of 1668 patients in a six month

period who developed clinical signs of foetal distress. Foetal acidaemia, defined as any foetus in whom the scalp blood pH fell to 7.25 or less, developed in 45 patients. Foetal acidaemia in association with clinical signs of foetal distress occurred with twice the frequency in patients who had existing complications of pregnancy before the onset of labour. No cases of acidaemia were detected in any of the foetal blood samples performed routinely on "at risk" patients in the absence of clinical signs of foetal distress.

The assimilation of these methods into clinical practice has yet to gain momentum. Garard, May and Simmons (1969) described some of the practical difficulties involved in organising biochemical foetal assessment in regional hospitals. However, by 1969, it was quite apparent that Saling's method of scalp sampling had research and practical value, despite the technical difficulties and potential for error in the technique itself. Hon and Khazin (1969), in a study of 194 patients, suggested that foetal scalp samples needed to be collected at no longer than 10 minutely intervals if a true measure of foetal acidaemia was to be obtained. This study was unique in the number of scalp blood samples collected from each patient, and it is probable that the frequency of sampling provided a major source of sample variability, in view of the possible effect

that this would have on the scalp tissues. Certainly, the evidence available from all other workers so far does not suggest that significant changes in acid-base status could occur so quickly, and the transient changes in pH that occur in the contraction phase are probably of little significance.

CHAPTER II

COMBINED STUDIES IN FOETAL ELECTROCARDIOGRAPHY AND FOETAL ACID-BASE STATUS

The clinical assessment of the neonate at the time of delivery as an index of foetal asphyxia has always been fraught with difficulties. Neonatal depression may be caused by a variety of factors, and, excepting the gross example of foetal death, has had obvious limitations when used as a method of assessing the significance of foetal heart rate changes and the configuration of the foetal electrocardiogram. In 1962, a series of studies appeared relating foetal heart rate changes to acid-base status of the neonate at birth. Brady and James recorded the foetal heart rate using foetal electrocardiography from abdominal leads and correlated their findings with the clinical status and the acid-base balance of the neonate at birth. Bradycardia lasting for more than a minute after the end of a contraction was invariably associated with asphyxia. Brady and James reported acid-base findings in four cases with persistent foetal tachycardia in labour. All cases showed clinical depression at birth and low cord arterial blood pH and low oxygen saturation.

In 1964, Quilligan, Katigbak, Nowacek and Czarnecki presented a similar study. Sixty-seven patients were

monitored during labour using foetal scalp electrodes and heart rate changes compared with cord blood acid-base values. Heart rate at the height of each contraction and mid-way between contractions was recorded and the mean values and standard deviations were calculated. There was evidence of mild respiratory and metabolic acidosis in the umbilical arterial blood of those infants who showed bradycardia late in labour, as compared with those patients where the foetal rate showed no significant difference during and between contractions, and a mean heart rate less than 160 beats per minute.

In the following year, the same group reported a further study of 54 patients with bradycardia in labour. A slight increase in acidosis in umbilical arterial blood was noted in those infants with bradycardia during contractions. In those infants who developed bradycardia between contractions, marked acidosis was demonstrated in the umbilical arterial blood. In a separate publication in the same year, Quilligan and Katigbak showed that persistent foetal tachycardia was also associated with acidosis at the time of delivery.

In 1967, Mendez-Bauer, Arnt, Gulin, Escarcena and Caldeyro-Barcia presented a report of the first joint study of foetal acid-base status and foetal heart rate obtained

during labour. The foetal heart rate was monitored throughout labour using scalp electrodes and scalp blood samples were obtained using the method of Saling. Nine patients were included in this study. Their findings showed that a low scalp pH (pH 7.20) preceded depression of the infant at delivery. Foetal acidosis was associated with typical foetal heart rate patterns characterised by a high basal heart rate and transient falls of heart rate occurring towards the end and following contractions. In normal labours, where the scalp blood pH remained higher than 7.20, "type II" dips in heart rate did not occur and the foetal rate varied between 130 to 150 beats per minute.

In the same year, Komaromy, Gaal, Mihaly, Mocsary and Suranyi studied five foetuses showing extrasystoles in labour. None of the infants were shown to be acidotic using blood obtained from the foetal scalp during labour.

Wood, Ferguson, Leeton, Newman and Walker (1967) performed an extensive study of foetal heart rate patterns and foetal acid-base status during labour. The mean Apgar score at two minutes was 3.6 in those subjects where the foetal scalp blood pH was less than 7.2, and 6.4 in those subjects with scalp blood pH greater than 7.2. There appeared to be no obvious risk to the foetus where no heart rate change occurred in relationship to uterine

contractions, even if bradycardia or tachycardia occurred during resting phases. Foetal acidosis caused by foetal asphyxia was associated with marked slowing of the heart rate during contractions, with a heart rate greater than 160 beats per minute during the resting phase. Slowing of the heart rate at the end of contractions was also associated with asphyxia.

Althabe, Schwarcz, Pose, Escarcera and Caldeyro-Barcia (1967) inserted oxygen electrodes in a series of foetuses during oxytocin-induced labours. Administration of oxygen to the mother produced a rise in foetal muscular partial oxygen pressure, although it was not possible to calibrate the foetal electrodes. A rise in foetal muscular pO_2 in normal subjects did not produce any alteration in the foetal heart rate. However, in patients showing type II dips in foetal heart rate, and foetal tachycardia, oxygen administration to the mother corrected the foetal heart rate changes, thus providing good evidence for an anoxic basis for these heart rate patterns.

In 1968, Gennser, Johansson and Kullander studied a group of foetuses delivered by hysterotomy from 16 to 23 weeks gestation by recording ECGs from these foetuses and measuring lactate-pyruvate ratios. With increasing

time and a rise in lactate-pyruvate ratio, the P-R interval lengthened, but the duration of the QT interval remained reasonably constant. Half an hour after clamping the cord, increasing prolongation of the P-R and QRS intervals was noted. However, relevance of such observation in relationship to the "in vivo" situation in foetal distress is probably remote. With the removal of placental circulation, the haemodynamics of the foetus are radically changed and the possibility of a differential factor in failing placental function is eliminated.

In 1969, Hon and Khazin reported a study on 194 foetuses monitored during labour, from whom an incredible total of 1392 scalp samples was collected. The authors showed that the pH system in the foetus has a rapid response and a short memory for insults such as cord compression.

In one patient with normal pH values, marked S-T segment depression and late deceleration heart rate patterns were shown. Also, low pH readings were demonstrated in the presence of normal foetal heart rate patterns in the second stage of labour. Variation in scalp circulation is suggested as an important factor in introducing a local "error" factor in pH assessment.

The relationship between scalp pH and Apgar score

at 1 minute and 5 minutes was also studied in this series. With a foetal scalp pH greater than 7.20, 20.1% of the samples were associated with Apgar scores of 1 to 6 at 1 minute, but where the foetal pH was less than 7.20, 42.3% of the samples were associated with an Apgar score of 1-6. The authors suggest that many of the discrepancies between scalp pH and Apgar score can be explained on the basis of the timing of the foetal scalp sample.

The importance of sampling in the resting uterine phase is clearly apparent from this article.

In general, the data available on the relationship between foetal acid-base status and the foetal heart rate and foetal electrocardiography is still limited and by no means comprehensive. It was for this reason that this present thesis was undertaken, in order to obtain a more comprehensive account of the influence of the foetal acid-base status on the foetal electrocardiogram, and to explore the biochemical pathways by which these changes might be induced.

CHAPTER III

SUBJECTS AND METHODS OF STUDY

SECTION I. Selection of Subjects

108 subjects were included in this study and biochemical measurements and electrocardiographic data were obtained in 103 subjects.

In five patients, all studied in the early phase of this work, satisfactory recordings of the foetal electocardiogram could not be obtained and these subjects were therefore included only because of the available biochemical and neonatal follow-up information.

One hundred and two patients were selected where the foetus was considered to be at risk from foetal asphyxia and, as the electrocardiographic data was studied in relationship to acid-base values early in labour and at the time of delivery, these measurements were used as the proper indication of the presence or absence of significant foetal asphyxia.

It was initially decided to include a group of normal control subjects in this work and, to this end, six patients were included who had no previous abnormal pregnancy and whose current pregnancy had been uncomplicated. To the

point where monitoring was started, no clinical signs of foetal distress had been noted. However, in each patient, subsequent continuous monitoring of the foetal heart rate revealed variations in the foetal heart rate which were clinically outside the generally accepted range of normality (110 to 160 beats per minute) at some stage during labour, and there seemed no value in pursuing this method of selection.

The indications for monitoring are shown in Table I and Table II. In Table I, the factors existing antenatally are listed, and in Table II, the clinical manifestations of foetal distress are recorded. In many subjects, multiple indications were present and the total number of indications therefore exceeds the number of subjects.

A brief summary of all subjects is included in Appendix 8.

Although a history of previous miscarriages is included in Table I, in all subjects an additional complication was present apart from the history of previous miscarriages. Indeed, in the group of subjects selected for this study because of the indications outlined, there was a surprisingly high incidence of history of miscarriage in previous pregnancies.

TABLE I

ANTENATAL AND INTRAPARTUM INDICATIONS FOR SELECTION OF SUBJECTS

Indication	Number of Subjects
Mild pre-eclamptic toxaemia (Hypertension without proteinuria)	28
Moderate to severe pre-eclamptic toxaemia (Hypertension + proteinuria)	22
Essential hypertension	8
Essential hypertension + P.E.T.	7
Antepartum haemorrhage	6
Previous history of anoxic stillbirths	3
Previous history of neonatal deaths	5
Previous miscarriages	23
Rhesus isoimmunisation	3
Suspected retarded intrauterine growth	1

Table II shows the number of subjects developing the various clinical signs of foetal distress. These signs were observed before monitoring was started and the patient was therefore assessed clinically as having signs of foetal distress.

TABLE II

CLINICAL SIGNS OF FOETAL DISTRESS

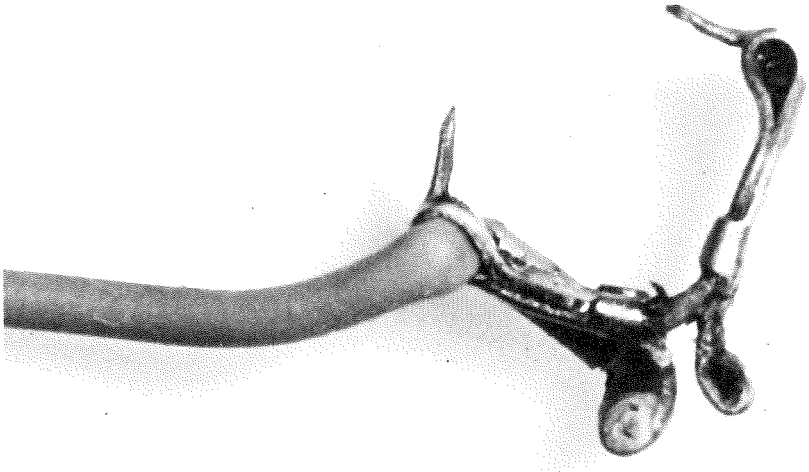
Clinical Signs	Number of Subjects
No clinical signs of foetal distress	48
Meconium stained liquor	32
Foetal tachycardia (FHR 160)	20
Foetal bradycardia (FHR 120)	17
Foetal heart irregularity	12
Foetal heart difficult to hear*	1
Excessive foetal movements	3

*In the one subject in whom the foetal heart was difficult to hear, meconium stained liquor was also present.

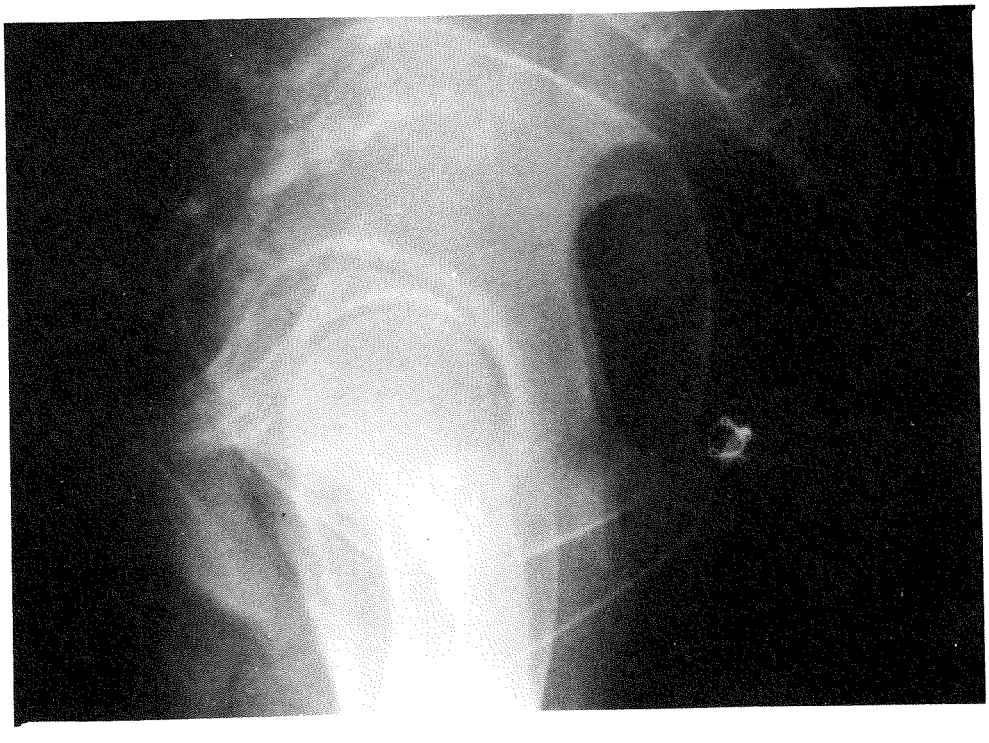
SECTION 2. Method of Foetal Electrocardiography

The vaginal electrodes were constructed on a design similar to that suggested by Hon (1963a). Some consideration was given to the use of suction electrodes such as described by Ross in 1961. These electrodes give excellent recordings of the foetal electrocardiogram but suffer from the disadvantage that their attachment tends to be unstable. It was therefore decided to use scalp clip electrodes in view of the long periods of monitoring involved. Nickel-silver wound clips were soldered to a three foot length of single core low resistance shielded and insulated coaxial cable 0.07" in diameter, with a sheath of P.V.C. tubing covering the lead. The scalp clips were covered in liquid araldite and subsequently allowed to dry over the next 24 hours. Later, silicone varnish was used, in view of its quick drying characteristics. A silver wire sleeve attached to the lead approximately 1 cm from the clip served as an indifferent electrode. The points of the scalp clips were filed clean of araldite and were then sharpened to enable easy skin penetration. All electrodes were sterilised before use by autoclaving, and electrodes were generally used in three or four subjects before becoming unserviceable. Figure 1(a) shows a view of an electrode clip. The attachment of the clip is shown radiologically in Figure 1(b).

FIGURE 1



(a): Foetal scalp electrode constructed from a nickel-silver wound clip. The outer surface of the clip is insulated with liquid araldite.



(b): Erect lateral X-ray of the pelvis of a patient in labour with scalp electrode in situ.

The electrodes were attached to the scalp under direct vision through the amnioscope at the same time as the scalp blood was collected. The scalp clip was inserted, using uterine packing forceps.

The foetal electrocardiogram was recorded using a Gilson Medical Electronics Model M5P mini-polygraph. The scalp electrode was attached to a 3-tail patient cable which was plugged into an EEG module. The frequency response of the EEG module is flat to approximately 30 cycles, above which point it drops rapidly. The restriction of high frequency response limits its use for the study of details of notching and slurring in the QRS complex. The time constant is sufficiently long that broadening of the QRS complex, S-T deviation and change in T waves are readily apparent.

Recording of the foetal ECG was made at a paper speed of 2.5 cms per second and samples were recorded, where possible, every ten minutes throughout labour. The recording was in rectilinear coordinates, eliminating curvilinear distortion to permit correlation of events in time without corrections for the arc produced by the pens. Whilst there was considerable variation in the amplitude of the ECG complexes, a voltage of 100uV produced a pen deflection of 1 cm. Care was taken to ensure that the lead polarity was kept constant.

In all, some 600 hours of foetal electrocardiographic records were obtained. Within 24 hours of delivery, the neonatal electrocardiogram was recorded with a Both electrocardiogram. The three standard leads and augmented limb leads were recorded, and three chest leads from positions 1, 3 and 6 were also recorded.

In order to obtain ECG readings which could be reliably related to acid-base values, two foetal ECG samples were studied in each subject. The first sample was obtained as soon after collection of scalp blood as possible. This time varied between five minutes and 30 minutes, depending on the amplitude of the signal. It was frequently found that the foetal ECG signal improved in amplitude during the first 30 minutes after attachment of the scalp electrode, a finding which may be explained by local tissue reaction around the scalp clip. The clip was not removed until the foetal head was delivered and the last foetal ECG sample was obtained shortly before delivery.

ECG intervals were measured manually and all samples were measured during uterine resting phase for the general data analysis. A separate analysis of ECG intervals was also performed in a limited number of subjects during uterine contractions and in the resting phase.

SECTION 3. Monitoring of Uterine Contractions

A sterile, open-ended fine Portex P.V.C. catheter was inserted into the amniotic sac at the time of attachment of the scalp electrode. Where the flow of amniotic fluid was insufficient to fill the catheter, it was filled with a dilute hibitane solution to ensure continuity of the fluid column. The catheter was then attached to a Model P23AA Statham pressure transducer. This was attached to a CH-CBPP chopper amplifier as a modular unit in the Gilson MP5 mini-polygraph, and uterine contractions were recorded in strength and frequency throughout labour.

SECTION 4. Foetal Biochemical Assessment

Foetal Scalp Blood

Foetal scalp blood samples were collected using the method described by Saling (1964). The samples were collected during the first stage of labour and were repeated subsequently where electrocardiographic monitoring indicated a change in foetal condition. For purposes of simplifying the data, only the first sample taken was used for comparison with the ECG data.

Scalp blood samples were collected in glass pipettes which were heparinised with sterile sodium heparin (strength 5000 units/ml) immediately preceding collection of the sample. Clotting of the specimen was sometimes troublesome, but there was always a sufficient column of blood free of clot to allow estimations to be performed. Where the complete sample was clotted, it was discarded and a further sample collected, or the subject was excluded from the study. Analysis of all parameters was completed within 10 minutes of sample collection. The sample was taken immediately to a laboratory adjacent to the labour ward, and acid-base assessment, including actual pH, carbon dioxide tension, standard bicarbonate and base excess was estimated in a type AMEI Astrup Micro Equipment (Radiometer), using the method of Siggard Andersen, Engel, Jorgensen and Astrup (1960). Standard bicarbonate was used throughout

this thesis as an index of metabolic acidosis. The reasons for this decision are discussed in Chapter 6. At the same time, 0.1 ml of whole blood was collected into a diluting fluid for subsequent true blood glucose estimation using the method of Marks (1959) and Watson (1962).

Cord Blood Samples

Samples of cord arterial and cord venous blood were collected into sterile 10 ml glass syringes which were heparinised with lithium heparin (5000 units/ml). All air bubbles were excluded from the syringe and the syringes were sealed with metal caps. Acid-base assessment was performed as soon after collection as possible, and individual samples were kept at $4^{o}C$ whilst awaiting measurement (usually within 15 minutes of collection). Blood samples were also collected for true blood glucose estimation, as for the scalp blood.

The blood samples remaining after acid-base assessment were centrifuged at 2500 RPM for ten minutes and the plasma stored for measurement of sodium and potassium using an Intrumentation Laboratory Model 143 Flame Photometer with the IL144 Diluter. Chloride values were determined by titrating plasma samples against mercuric nitrate using diphenylcarbazone as an indicator.

The umbilical cord was clamped as soon as the infant

was delivered, before the onset of respiration, and blood samples were drawn immediately from the placental end of the cord.

SECTION 5. Maternal Biochemical Assessment

Maternal venous blood samples were drawn from the cubital vein at the time of the initial scalp blood sampling and at the time of delivery. The use of a tourniquet was avoided and 10 mls of blood were collected anaerobically into a sterile glass syringe. The same methods and procedure were employed as with the cord blood samples with the measurement of pH, pCO_2, standard bicarbonate, base excess, sodium, potassium, chloride and true glucose.

SECTION 6. Assessment of the Neonate

The assessment of the neonate was started one minute after delivery and the infant was scored using the method described by Apgar (1953). This assessment was completed at the end of the second minute after delivery. The Apgar score was estimated again starting at the fifth minute after delivery.

For purposes of subsequent statistical analysis, the Apgar scores were classified into three groups:

Group I - Apgar score 0 - 5
Group II - Apgar score 6 - 8
Group III - Apgar score 9 - 10

The Apgar score was assessed by the labour ward sister and the medical attendant.

Twenty-four hours after delivery, an independent assessment was made by one paediatrician, who studied all the infants in this series. The infant was assessed and scored on a system described by Dr. D. Craven (1964) in her thesis on the effects of antepartum haemorrhage on neonatal development. A general assessment of the central nervous system, respiratory system and general condition of the infant was made and coded as shown in Appendix 4 and Code to the Appendices.

At six weeks after delivery, the infant was again seen

with the paediatrician and an independent assessment of the central nervous system, respiratory system, general condition and social quotient was made. These assessments were coded as described in the Code to the Appendices.

SECTION 7. Methods of Data Analysis

The data collected in this study are recorded in the appendices. A brief summary of the clinical details of each subject is presented in Appendix 8. As the study consisted of observations of 108 patients and involved documentation of 105 items of data for each patient, it was clear that analysis would present a major problem.

For this reason, the assistance of Mr. K.M. Cellier, Senior Research Scientist, C.S.I.R.O., was sought and the author of this thesis is deeply indebted to his help in this analysis.

Data were transferred onto punch cards and analysed on the CDC3200 computer, Division of Computing Research, C.S.I.R.O., Adelaide. A complete matrix of linear relationships between all variables was established, and the significant correlation coefficients abstracted. In some cases, quadratic relationships were established and curvature in the relationship between variables was examined. A total of approximately 16,000 analyses were performed in this study and from this matrix of data, only those values where the p value was less than 0.01 were included in the results concerning linear and quadratic relationships.

Where quantitative measures were not available (e.g. where infants were grouped in classes according to their

condition or appearance) then single classification analyses of variance were carried out in order to determine whether the mean values of particular variates were different in the different classes.

Correlation coefficients, means and standard errors have been presented where appropriate.

Variance of Foetal ECG Time Constants

Foetal ECG time constants were measured manually from samples recorded during the resting uterine phase at a period during which heart rate showed little variation. The mean of four or five complexes was taken for each sample.

The standard deviation and variance of these measurements in two subjects are shown in Appendix 7.

CHAPTER IV

BIOCHEMICAL RESULTS

SECTION I. Foetal Blood pH and Acid-Base Values in Labour and at Delivery

Foetal scalp blood samples were collected during the first stage of labour and, at the time of delivery, from the umbilical cord as previously described.

The mean values for foetal acid-base variates are shown in Table III.

The mean values for scalp blood pH, pCO_2 and standard bicarbonate were similar to those values measured in cord venous blood. The cord arterial blood pH was significantly lower than the cord venous blood pH, the acidosis being partly respiratory in origin with a high pCO_2, and partly metabolic with a lowered value for standard bicarbonate.

Scalp blood pH showed a significant positive linear correlation with cord arterial blood pH ($r = +0.38$ $p < .001$) but did not show a significant relationship with cord venous blood pH, despite the similarity in mean values.

Scalp blood pH also showed a significant linear relationship with cord arterial blood standard bicarbonate ($r = +0.44$ $p < .001$) and cord venous blood standard

TABLE III

FOETAL ACID-BASE VALUES

Variate	Number of Subjects	Mean	Maximum	Minimum	Standard Deviation (Mean)
Scalp blood pH	106	7.308	7.410	7.050	.005
Scalp blood standard bicarbonate (meq/l)	100	19.976	26.00	15.40	.213
Scalp blood pCO_2	100	41.735	77.00	24.00	.752
Cord Arterial blood pH	103	7.209	7.420	7.030	.008
Cord arterial blood standard bicarbonate (meq/l)	103	17.551	22.500	12.600	.226
Cord arterial blood pCO_2	103	51.28	85.00	30.50	1.084
Cord venous blood pH	70	7.304	7.410	7.070	0.007
Cord venous blood standard bicarbonate (meq/l)	70	19.289	24.500	13.300	.258
Cord venous blood pCO_2	70	39.68	61.00	26.50	0.78

bicarbonate ($r = +0.42$ $p < .001$).

The scalp blood standard bicarbonate showed a significant positive linear relationship with cord venous and cord arterial blood standard bicarbonate ($r = +0.42$ and $+0.39$ respectively), the relationship being highly significant ($p < 0.001$) in both instances (see Figure 2).

An analysis of the relationship between cord arterial and cord venous blood values is shown in Figure 3. There is a highly significant relationship between cord arterial and cord venous blood pH values ($r = +0.65$ $p < .001$), between cord arterial pH and cord venous blood standard bicarbonate ($r = +0.65$ $p < .001$) and cord venous blood pH and cord arterial blood standard bicarbonate ($r = +0.54$ $p < .001$). A high significant relationship was demonstrated between cord arterial blood standard bicarbonate and cord venous blood standard bicarbonate ($r = +0.81$ $p < .001$). No relationship was established between pCO_2 values, either in the study of scalp blood and cord blood values, or between cord arterial blood and cord venous blood values.

The group mean acid-base data for various clinical parameters of foetal distress are shown in Table IV. Within the major groups examined, it can be seen that the lowest values of pH and standard bicarbonate in the scalp blood

Figure 2

INTERRELATIONSHIPS BETWEEN SCALP BLOOD AND CORD BLOOD ACID-BASE VALUES

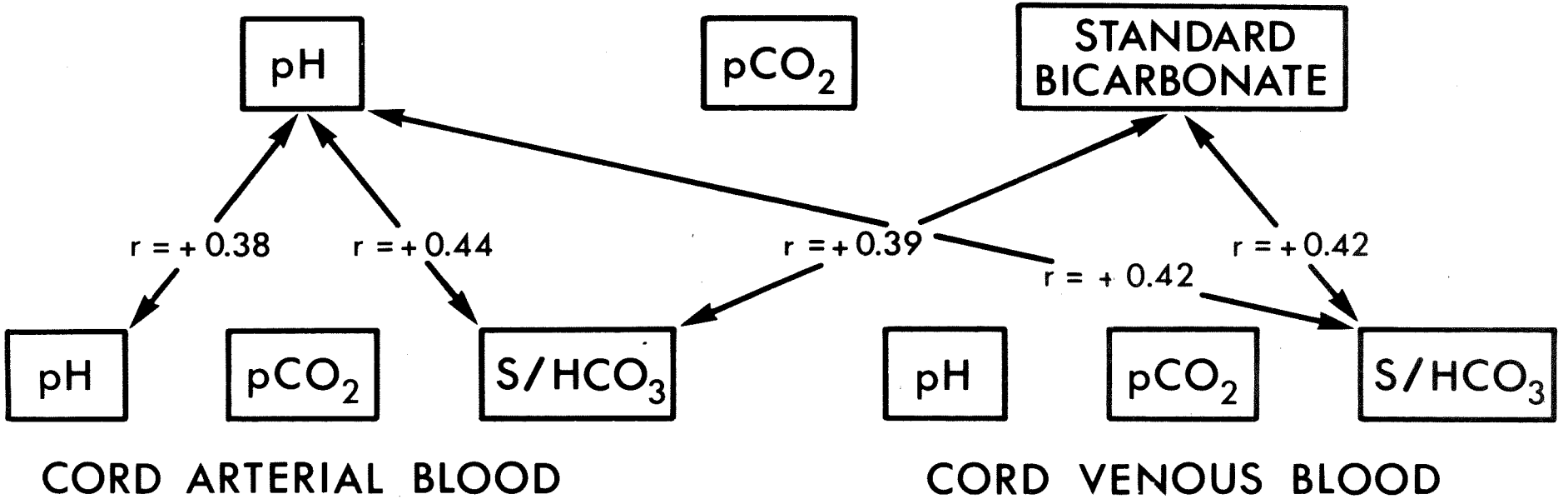


TABLE IV

CLINICAL SIGNS OF FOETAL DISTRESS AND FOETAL ACID-BASE STATUS

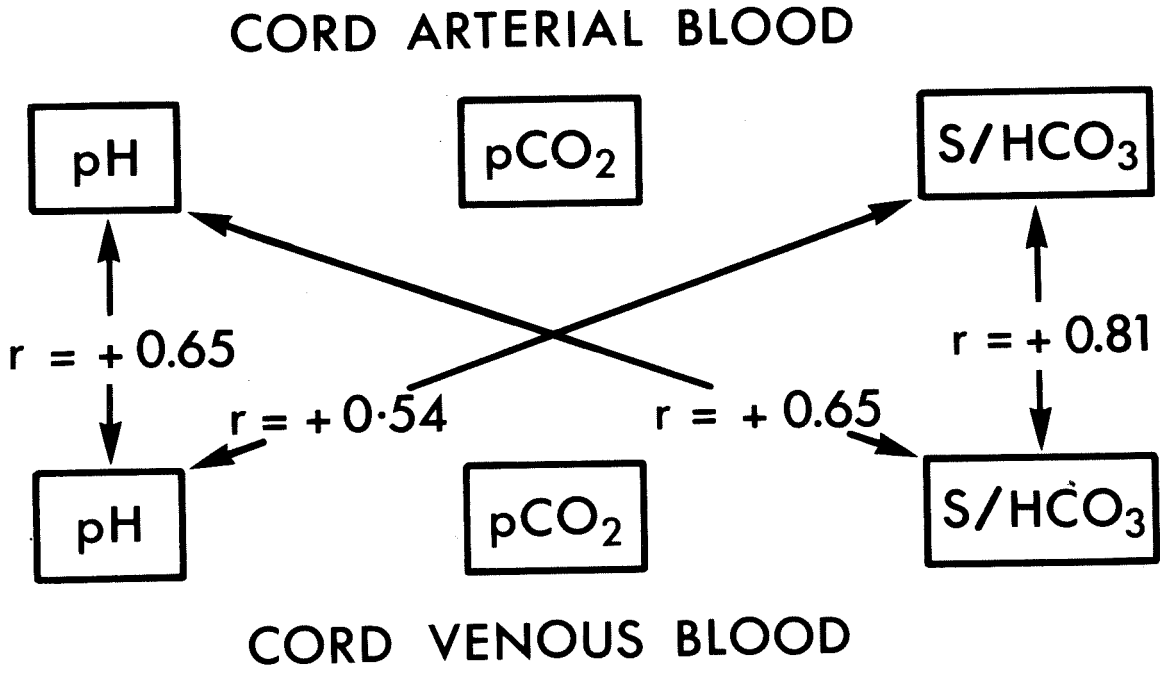
Variate	Group			
Foetal Blood	2 (19)	2 & 3 (5)	2 & 4 (3)	2,3 & 4 (3)
Scalp pH	mean 7.31 s.e. .015 Pooled estimate of s.e. = .01872	7.296 0.0263	7.267 0.0624	7.31 .0216
Scalp pCO_2	mean 39.94 s.e. 1.552 Pooled estimate of s.e. = 2.675	41.4 4.75	45.5 (2) .05	50 1.633
Scalp standard bicarbonate	mean 19.82 s.e. .424 Pooled estimate of s.e. = .511	19.54 .739	17.5 (2) .5	21.93 1.58
Cord arterial pH	mean 7.18 s.e. .018 Pooled estimate of s.e. = .0306	7.244 .055	7.163 .0998	7.12 .0735
Cord arterial pCO_2	mean 54.82 s.e. 3.085 Pooled estimate of s.e. = 3.922	47.2 7.47	51.33 7.76	65 10.61
Cord arterial standard bi-carbonate	mean 16.94 s.e. .533 Pooled estimate of s.e. = 1.607	18.1 0.92	15.87 2.45	15.93 1.552

Number of subjects shown in parenthesis.

2 = meconium stained liquor
3 = foetal tachycardia
4 = foetal bradycardia

Figure 3

INTERRELATIONSHIP BETWEEN CORD BLOOD SAMPLES – ACID – BASE VALUES



sample occurred in those patients who developed meconium stained liquor and foetal bradycardia.

A significant difference ($p < 0.05$) was shown between scalp blood pCO_2 in subjects where meconium-stained liquor alone occurred, and in those subjects with meconium stained liquor, tachycardia and bradycardia. A significant difference ($p < 0.05$) was also seen between scalp bicarbonate values in the group of subjects showing meconium stained liquor and bradycardia, and in subjects showing meconium stained liquor, foetal tachycardia and foetal bradycardia.

An analysis of scalp blood and cord blood acid-base and electrolyte values showed no significant differences between subjects with mild and severe toxaemia.

Discussion

A comparison between the mean values of pH, pCO_2 and standard bicarbonate in this present study and other studies is difficult because of the nature of selection of subjects. In a review of several studies of cord blood pH, Kaiser (1959) reviewed cord venous blood samples and cord arterial blood pH values in studies by Kaiser (1953) and Goodlin and Kaiser (1957). The pH values obtained in the cord venous blood samples in normal infants was 7.32 and the cord arterial samples 7.26 and 7.28 in the two studies.

Low (1963) studied 100 normal subjects delivered under general or epidural anaesthesia and demonstrated mean values of 7.287 (general anaesthesia) and 7.275 (epidural anaesthesia) for pH of cord venous blood. The values of pH for cord arterial blood were 7.196 and 7.179 respectively. Standard bicarbonate values in cord venous blood in subjects delivered under general and epidural anaesthesia were 18.17 meq/litre and 17.41 meq/litre respectively. In this study, values of pCO_2 in the cord artery were 61.8 mm Hg and 61.9 mm Hg in the two anaesthetic groups.

The values stated for pH are similar to those obtained in this present study, as are the values for standard bicarbonate. The values for pCO_2 are slightly higher in the study of Low. This may be explained by the anaesthesia in this series.

In a study of acid-base values of cord arterial bloods obtained in 150 cases of various complications, and including some normal subjects, MacRae and Palavradji (1965) showed a mean cord arterial blood pH of 7.209, a value which is identical with that obtained in this present series.

Beard and Morris (1965) reported scalp blood values for pH in 26 normal patients in labour of 7.29 in early

labour, 7.30 in late first stage of labour, and 7.285 in the second stage of labour.

The majority of subjects in this present study were in established labour and the mean pH of 7.308 is similar to that obtained by Beard and Morris in samples taken at 3-5 cms dilatation of the cervix.

The mean values for pH in all foetal blood samples show, as all previous studies have shown, that the foetus exists in a state of acidosis relative to maternal blood values. Whilst some authors have stated that this was predominantly a respiratory acidosis, it was apparent in this study that there was also a significant metabolic acidosis.

Consideration of the interrelationship between scalp blood and cord bloods showed that a significant positive linear relationship existed between scalp blood pH and cord arterial blood pH. However, the most consistent and significant relationship was demonstrated between scalp blood pH and standard bicarbonate and the standard bicarbonate values of cord arterial and cord venous blood. The metabolic influence on acid-base status was the most significant and consistent factor seen in this study. The variability of pCO_2 tended to weaken the relationship between scalp pH and cord blood values.

A highly significant relationship was demonstrated between cord arterial and cord venous blood standard bicarbonate. Despite the difference in mean values, the close relationship between cord arterial and cord venous blood pH and standard bicarbonate values implies that assessment of foetal acid-base status can be validly deduced from studies on cord venous blood, and that such assessment need not necessarily be performed on cord arterial blood. This is a practical point of some significance, as cord arterial blood is often difficult to obtain, particularly when the foetus is severely asphyxiated and cord arterial vasoconstriction may be present at the time of delivery.

SECTION 2. Maternal and Foetal Acid-Base Values in Labour and at Delivery

Maternal venous blood samples were collected from a cubital fossa vein at the time of scalp blood sampling and at the time of delivery. The mean values for pH, pCO_2 and standard bicarbonate in maternal venous blood are shown in Table V.

The relationship between foetal scalp blood and maternal acid-base values in labour are shown in Figure 4.

A significant relationship existed between foetal scalp blood pH and maternal venous blood pH, and between foetal scalp blood pH and maternal venous blood standard bicarbonate. A highly significant relationship existed between foetal scalp blood bicarbonate and maternal venous blood bicarbonate ($r = +0.55$, $p < 0.001$), but no relationship could be demonstrated between foetal scalp blood pCO_2 and maternal venous blood pCO_2.

A similar pattern emerged at the time of delivery between maternal venous blood and cord venous blood. Standard bicarbonate values in maternal venous blood were significantly related to standard bicarbonate values in both cord arterial and cord venous blood ($r = +0.49$ and $+0.54$ respectively, $p < 0.001$) (see Figure 5). Carbon dioxide tension in maternal venous blood was shown to be related to carbon

Figure 4

RELATIONSHIP BETWEEN FOETAL SCALP BLOOD ACID-BASE VALUES AND MATERNAL VENOUS BLOOD VALUES COLLECTED AT THE SAME TIME

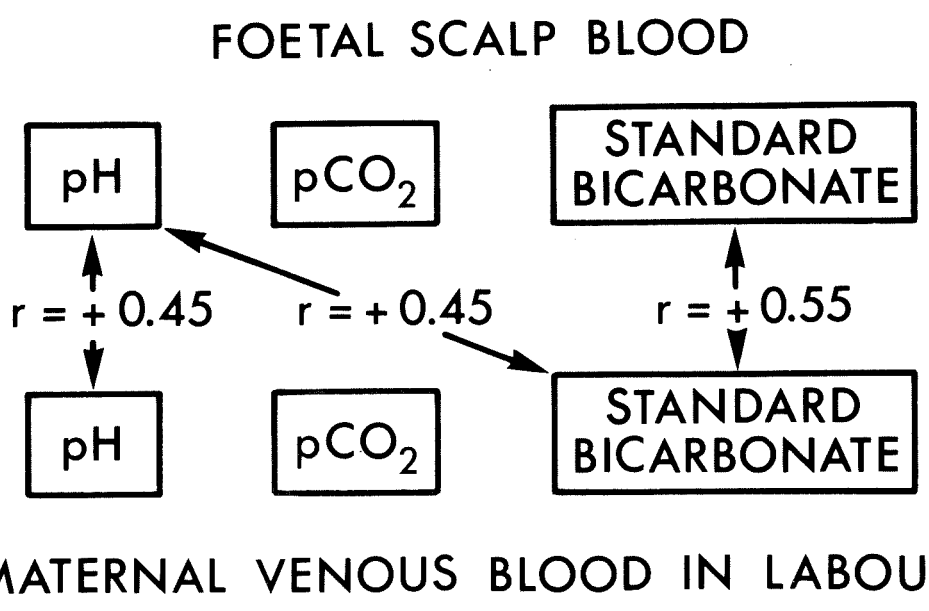


TABLE V

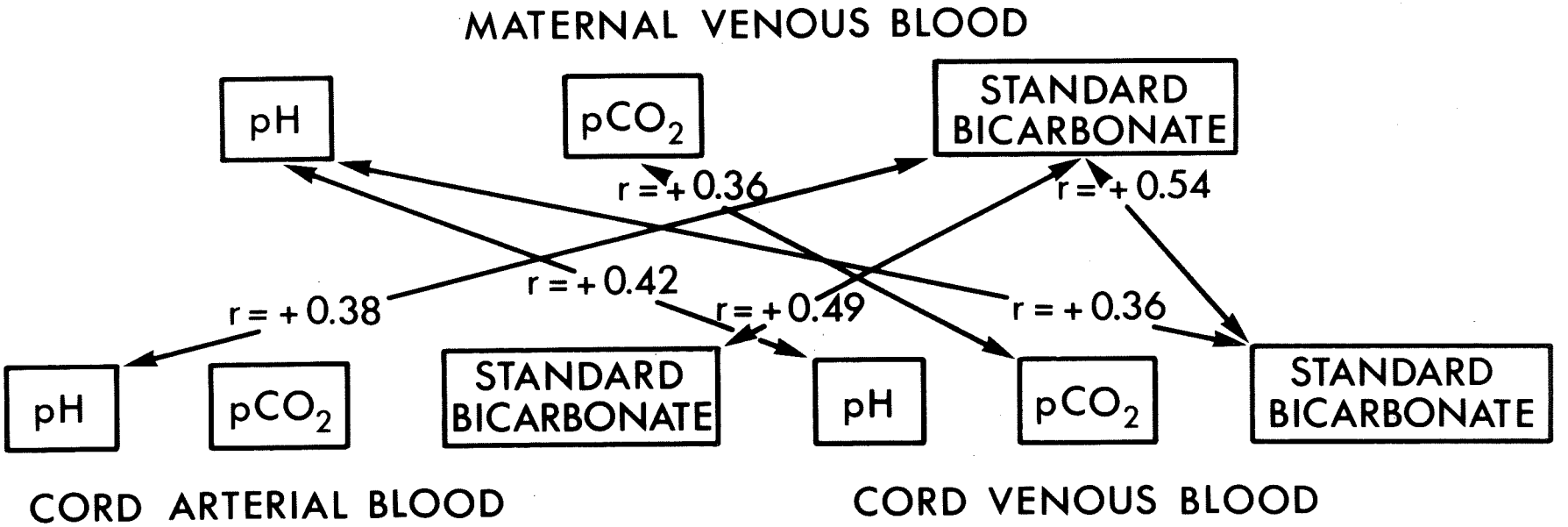
MATERNAL VENOUS BLOOD ACID-BASE VALUES

Before Delivery

Variate	Number	Mean	Maximum	Minimum	S.D. (Mean)
pH	101	7.406	7.530	7.310	.004
pCO_2 (mm Hg)	101	32.11	45.00	18.00	.439
Standard bicarbonate (meq/litre)	101	21.17	25.00	16.50	.144
		After Delivery			
pH	104	7.351	7.62	7.24	.005
pCO_2 (mm Hg)	103	31.986	54.00	14.00	.568
Standard bicarbonate (meq/litre)	103	18.81	24.00	14.00	.185

Figure 5

RELATIONSHIP BETWEEN CORD BLOOD VALUES AND MATERNAL VENOUS BLOOD VALUES COLLECTED AT THE TIME OF DELIVERY



dioxide tension in cord venous blood ($r = +0.36$, $p < 0.01$).

Discussion

Table V shows that the mean pH of maternal venous blood and the standard bicarbonate fell during labour, but values for pCO_2 remained constant. These samples were collected during a resting phase between uterine contractions during labour and immediately following delivery of the foetus. The findings are similar to those established by Beard and Morris (1965), using maternal venous blood samples. These authors showed that a significant increase in base deficit occurs during labour, and that maternal venous blood pH fell from 7.41 to 7.35. Despite the fact that the figures in this study were based on the results of measurements taken from 26 normal subjects, a similar metabolic acidosis was demonstrated to that in the present study, which included a series of patients with both normal and prolonged labours. The metabolic acidosis which develops during labour is probably dependent on the development of a hyperlactacidaemia, as shown by Marse and Greene (1964).

A consistent relationship was demonstrated between scalp blood standard bicarbonate and maternal venous blood standard bicarbonate ($r = +0.55$), and at delivery between the standard bicarbonate of maternal venous blood and both cord arterial blood ($r = +0.49$) and cord venous blood ($r = +0.54$).

Stenger et al. (1964) showed that a consistent relationship existed between maternal and foetal bicarbonate in a study on maternal arterial and uterine venous blood and cord blood samples. Newman et al. (1967) showed that a similar relationship occurred between maternal arterialised capillary blood and scalp blood, both in relationship to standard bicarbonate and base excess. The administration of bicarbonate to the mother increased both maternal and foetal bicarbonate levels. However, Blechner et al. (1967) administered ammonium chloride to a series of patients under general anaesthesia and showed that, although maternal arterial pH fell, the usual transplacental pH gradient was virtually eliminated. The authors considered that maternal and foetal bicarbonate levels were independent, and that there was a limitation in placental permeability to hydrogen and bicarbonate ions. These findings were at variance with those published in 1957 by Goodlin et al., in a similar study.

Whether the relationship between maternal and foetal bicarbonate is due to a direct transfer of bicarbonate ions across the placenta or is secondary to the transfer of acid metabolites across the placenta, this study has confirmed that a consistent and highly significant positive linear correlation occurs between maternal and foetal blood standard bicarbonate at all stages of labour.

Similar studies have been undertaken on pCO_2 values in maternal and foetal blood. Stenger et al. (1964) and Newman et al. (1967) have both demonstrated a significant relationship between maternal and foetal blood pCO_2. In this study, no significant relationship could be shown between maternal venous blood pCO_2 and scalp blood pCO_2, or between maternal venous blood pCO_2 and cord arterial blood pCO_2. However, a significant relationship was established between the pCO_2 of maternal venous blood collected at delivery and the pCO_2 of cord venous blood ($r = +0.36$). $p < 0.01$.

The discrepancy between these findings and those of other authors may be explained by the fact that Stenger et al. used maternal arterial and uterine venous blood, and Newman et al. used arterialised capillary blood. The use of maternal venous blood for measurement of pCO_2 is of limited value, but a significant relationship was demonstrated with cord venous blood.

Maternal venous blood pH was significantly related to scalp blood pH during labour ($r = +0.45$) and to cord venous blood pH ($r = +0.42$) at the time of delivery. ($p < 0.001$).

In summary, this study of the relationship between acid-base values of maternal venous blood and foetal blood has shown that a significant relationship exists between actual pH of both foetal scalp blood and cord venous blood

and maternal venous blood. Although no valid conclusions can be made concerning the respiratory influence where maternal venous samples have been used, a significant relationship in metabolic factors is apparent. There is evidence that metabolic acidosis in the mother influences the acid-base status of the foetus.

SECTION 3. Foetal and Maternal Plasma Electrolyte Values and Acid-Base Status at Delivery

Cord blood samples were collected at the time of delivery into glass syringes heparinised with sodium heparin. On measurement of the sodium content of the heparin and on estimating the amount remaining in the syringe after heparinising the walls, it was calculated that the potential error in plasma Na measurement due to this method of heparinisation was 0.5 to 1 meq/Na/litre. As this was considerably less than the range of error of the method, it was disregarded as a significant factor in sodium measurements.

Sufficient blood could not be collected from scalp samples to perform plasma electrolyte measurements.

Table VI shows the mean values of cord arterial and cord venous blood electrolyte values.

It can be seen that sodium and potassium concentrations are marginally higher in cord arterial blood than in cord venous blood, a finding that could be explained on the basis of water transfer across the placenta. A study of the inter-relationship between acid-base measurements and electrolyte values in cord arterial blood and cord venous blood showed that K^+ values were the only values that were related in a linear fashion to acid-base values. Cord arterial blood

TABLE VI

CORD ARTERIAL AND VENOUS PLASMA ELECTROLYTES

	Na(meq/litre)					K(meq/litre)					Cl (meq/litre)				
	Number	Mean	Max.	Min.	S.D.	Number	Mean	Max.	Min.	S.D.	Number	Mean	Max.	Min.	S.D.
Cord arterial blood	74	130.4	167.0	110	1.4	74	5.33	10.6	3.2	0.15	58	101.6	140	70	1.6
Cord venous blood	67	128.6	152.0	113.0	0.929	67	5.1	10.7	3.4	0.147	65	100.1	134	76	1.2

standard bicarbonate showed a significant negative relationship with K values ($r = -0.38$, $p < 0.001$) and cord venous blood pH was related to cord venous blood K in a negative manner ($r = -0.35$, $p < 0.01$). In other words, acidosis in the foetal blood resulted in high potassium values but was not related to Na^+ and Cl^- values.

The interrelationships of electrolyte values both between cord arterial and cord venous blood samples are shown in Figure 6. No significant relationships were demonstrated within the electrolyte values, i.e. Na^+ to K^+, Na^+ to Cl^-, Cl^- to K^+ within cord arterial or cord venous blood samples, and no cross correlations, other than those demonstrated in Figure 6, were demonstrated in this study.

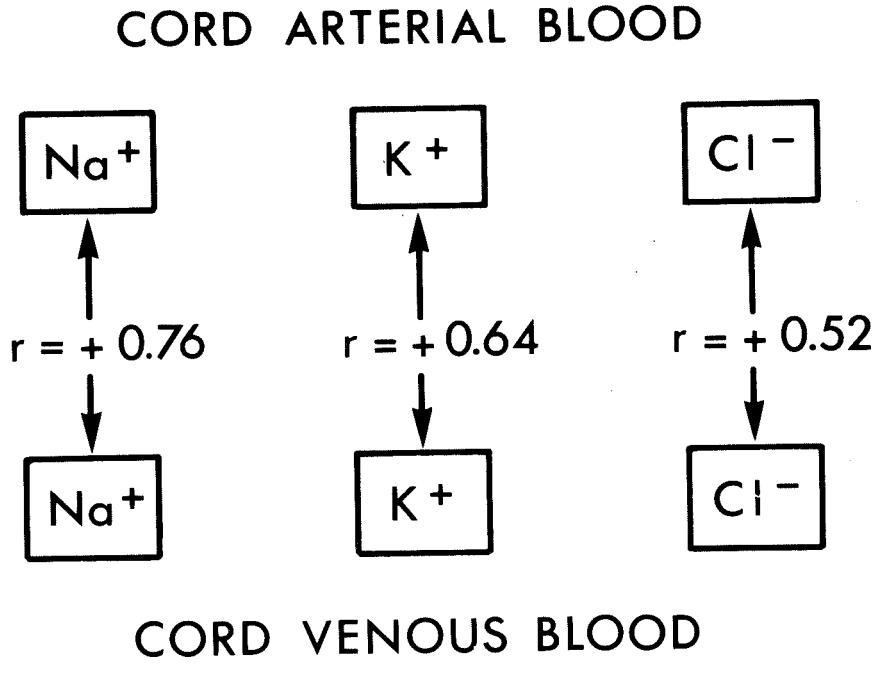
Maternal and Foetal Electrolyte Relationships

The mean values for Na^+, K^+ and Cl^- in maternal venous blood at the time of delivery are shown in Table 7.

The extreme maximum and minimum values in Na^+ and Cl^- estimations were by no means representative of the scatter of the data, and were well outside the range of the remainder of the values. The Na^+ values were, however, checked on different flame photometers, and the values remained constant. No difference in the sampling procedure could be detected, and the syringes used were cleaned and heat sterilised. These values have, therefore, been left in the data,

Figure 6

INTERRELATIONSHIP OF ELECTROLYTE VALUES IN CORD ARTERIAL AND CORD VENOUS BLOOD



although their significance remains uncertain.

Examination of the chloride values of maternal venous blood and cord arterial and cord venous blood showed that no relationship existed between these values. However, Figure 7 shows the relationship between Na^+ and K^+ values. A highly significant positive relationship was demonstrated between the potassium values of cord arterial and cord venous blood and maternal venous blood at the time of delivery ($r = +0.51$, $p < 0.001$ and $r = +0.50$, $p < 0.001$ respectively). However, in the case of sodium, a positive relationship was only shown between maternal plasma sodium and cord venous plasma sodium ($r = +0.59$, $p < 0.001$).

Discussion

A significant relationship has been shown between maternal and foetal plasma Na^+ and K values at the time of delivery. However, whilst K^+ values in the cord plasma were dependent on maternal values, potassium levels in the foetus were also clearly dependent on the acid-base status of foetal blood, a rise in plasma K occurring in response to increasing acidosis. A significant difference between the mean values of maternal plasma K^+ and cord arterial and cord venous plasma K^+ suggests that a positive gradient to potassium ions must be maintained across the placental barrier. The higher potassium values in cord blood are

Figure 7

RELATIONSHIP BETWEEN Na^+ AND K^+ VALUES IN MATERNAL AND CORD BLOOD

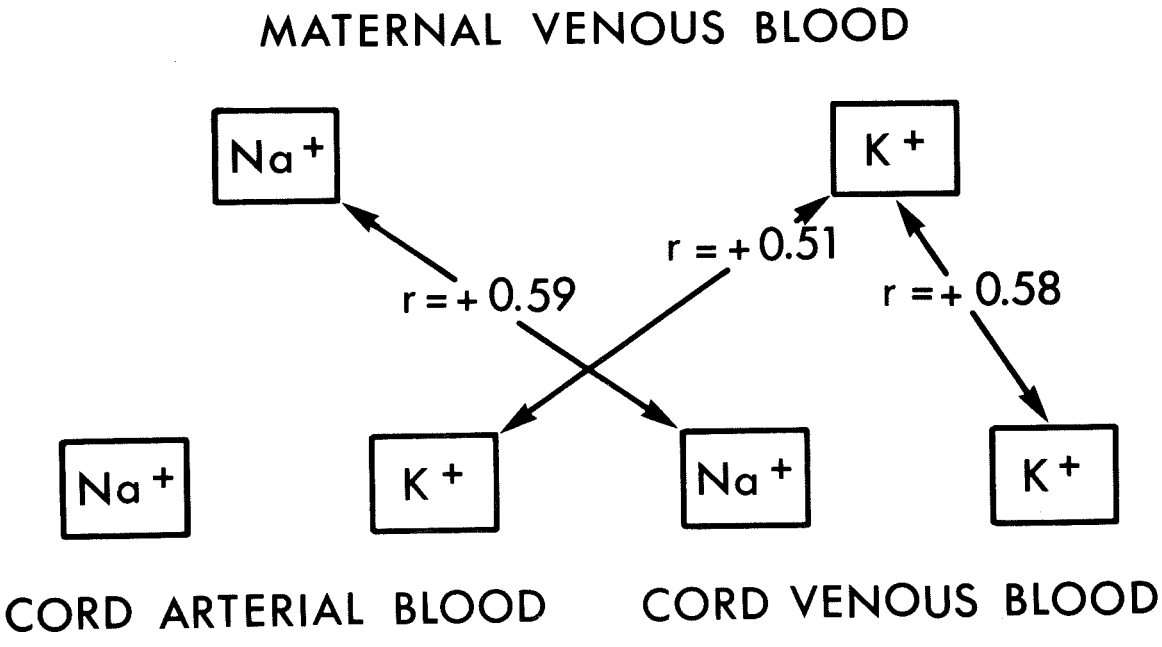


TABLE VII

MATERNAL VENOUS BLOOD ELECTROLYTE VALUES

AT DELIVERY

(Values in meq/litre)

Variate	Number of Subjects	Mean	Maximum	Minimum	S.D.
Na	81	131.3	192.0	91.0	1.42
K	81	3.77	5.70	2.50	0.065
Cl	77	100	138	85.0	1.075

associated with the relative foetal acidosis. In a situation where severe and prolonged acidosis occurs in the foetus and the maternal acid-base status remains normal, this transplacental potassium gradient is widened and could act as a significant source of potassium depletion in the foetus, even though the foetal plasma K^+ level could remain high.

Further investigation into this situation of potential K^+ loss in relationship to electrocardiographic findings in the foetus is considered in the electrocardiographic data.

SECTION 4. Maternal and Foetal Blood Glucose Levels

True blood glucose measurements were included in this study because of the possible importance of hypoglycaemia and glycogen depletion on myocardial action.

Using a micromethod as previously described, true blood glucose levels were measured on all foetal and maternal blood samples.

The mean values are shown in Table VIII.

The wide variation in the range of values in both maternal and foetal blood is, in part, explained by the use of intravenous infusions of dextrose used in many of these subjects. This is also apparent from the mean values in maternal blood, which are higher at the time of delivery than during labour. The mean values in maternal blood are significantly higher than those found in foetal blood, the difference being more obvious in labour when the maternal values are more than twice the levels found in scalp blood.

Blood being delivered to the foetus through the cord vein contained significantly more glucose than blood being returned to the placenta via the cord artery.

The relationship between maternal venous blood glucose before delivery and scalp blood glucose was significant, with a positive correlation coefficient ($r = +0.49$, $p < 0.001$).

TABLE VIII

MATERNAL AND FOETAL BLOOD GLUCOSE LEVELS

(mgm per cent)

Variate	Number	Mean	Maximum	Minimum	S.D. (mean)
Maternal venous blood in labour	71	74.9	245.0	21.00	5.04
Foetal scalp blood	42	33.9	114.0	5.00	3.106
Maternal venous blood at delivery	71	88.39	215.00	21.00	4.59
Cord arterial blood	70	54.4	155.0	7.00	3.64
Cord venous blood	67	65.7	160.0	15.0	3.96

This relationship was clearer at the time of delivery, as can be seen in Figure 8.

No significant relationships could be shown between maternal and foetal blood glucose values and acid-base and electrolyte values, with the exception of maternal venous blood glucose in labour, which showed a significant negative linear correlation with plasma potassium levels ($r = -0.4$, $p < 0.001$).

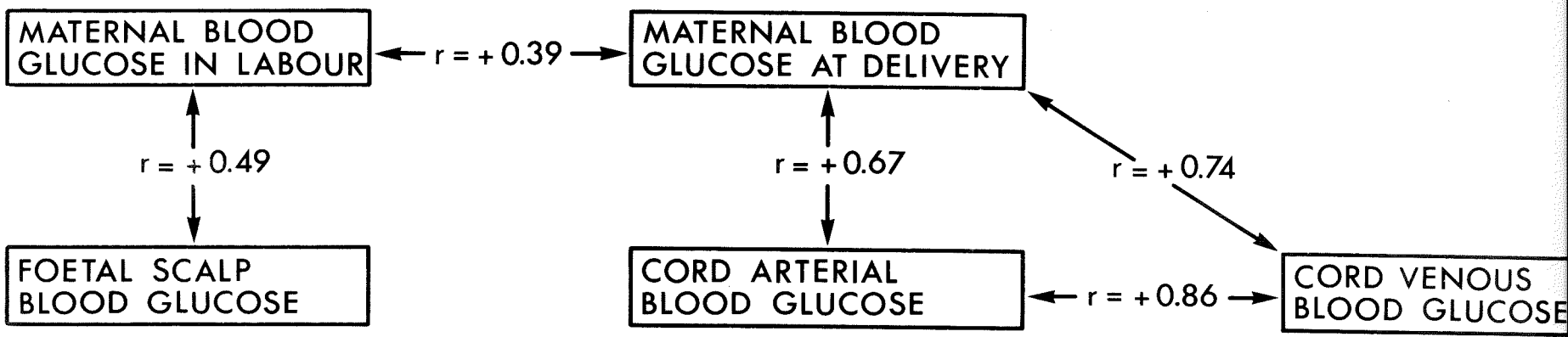
Discussion

It is difficult to relate the mean values obtained in this study to those obtained in other studies, because of the difference in selection of subjects and because of the difference in management with the administration to the mother of intravenous dextrose solutions. The values for scalp blood glucose were certainly lower than those obtained by Paterson, Phillips and Wood in 1967. However, this study did confirm the highly significant relationship between maternal and foetal blood glucose levels, both during labour and at delivery. It also confirmed the later findings of Phillips, Lumley, Paterson and Wood (1968), that foetal blood glucose was not related to foetal acid-base status.

If capillary blood glucose levels can be taken as an

Figure 8

INTERRELATIONSHIPS BEWEEN MATERNAL AND FOETAL BLOOD GLUCOSE LEVELS



indication of circulating blood glucose values, it is also apparent that severe hypoglycaemia can occur in the foetus in utero, with values recorded as low as 5 mgm per cent. At the other extreme, very high blood glucose levels can be induced in the foetus by inducing high blood glucose levels in the mother.

SECTION 5. (a) Foetal Biochemical Measurements and Subsequent Clinical Progress of the Neonate

The relationship between the various biochemical measurements made on foetal blood were studied in relation to the condition of the foetus at birth, and of the neonate 24 hours after delivery, and six weeks after delivery.

The methods of assessing the infant at birth and at the two subsequent intervals after delivery represent a relatively crude index of assessment. Nevertheless, with the Apgar scores graded as previously described (see Section 6, p83), a significant relationship was shown between Apgar score at five minutes and scalp blood pH ($r = +0.41$, $p < 0.001$), and the condition of the central nervous system at 24 hours after delivery and scalp blood pH ($r = +0.34$, $p < 0.01$).

A similar pattern emerged with cord arterial blood pH and subsequent progress of the infant, a positive correlation existing with Apgar score at one minute ($r = +0.30$, $p < 0.01$), Apgar score at five minutes ($r = +0.291$, $p < 0.05$) and central nervous system at 24 hours ($r = +0.26$, $p < 0.05$). Cord arterial blood pCO_2 showed a significant negative relationship with the Apgar score at one minute ($r = -0.29$, $p < 0.05$).

No other significant relationships could be shown between electrolyte and glucose values and Apgar score, or

any of the indices of assessment of the neonate.

(b) The Relationship of Apgar Score and Scalp and Cord Arterial Blood Acid-Base Values

Subjects were grouped according to the Apgar score from 1 minute and the Apgar score from 5 minutes as shown in the next table (Table IX).

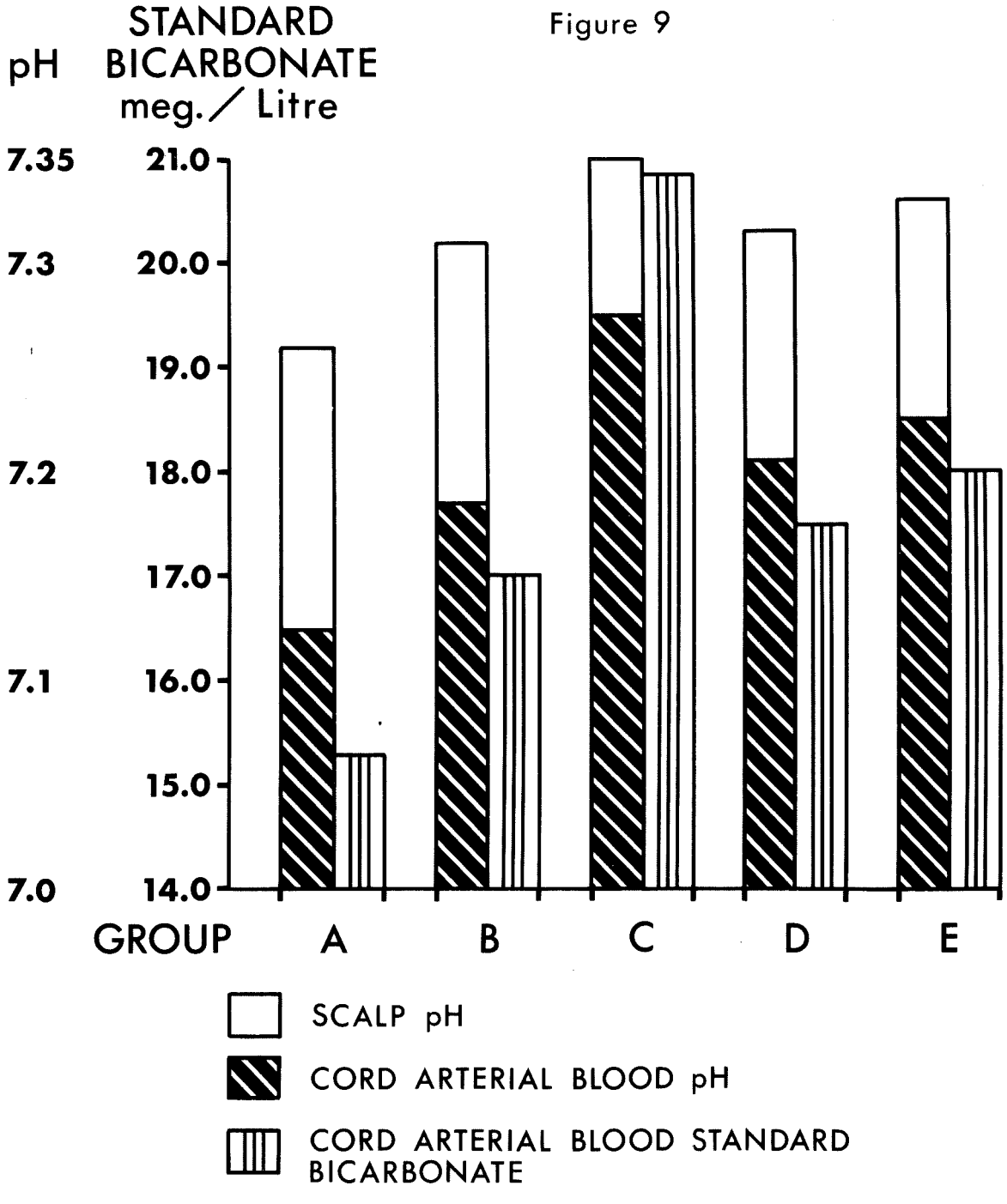
TABLE IX

Code	1 Minute Apgar Score	5 Minutes Apgar Score
A	1 - 5	6 - 8
B	1 - 5	9 -10
C	6 - 8	6 - 8
D	6 - 8	9 -10
E	9 -10	9 -10

The mean values for scalp blood pH, cord arterial blood pH and cord arterial blood standard bicarbonate can be seen in Figure 9.

Group A with a mean scalp blood pH of 7.254 showed a significantly lower value than in all other groups, but there was no difference between Groups B, C, D and E. An analysis of scalp blood pCO_2 showed no significant difference between groups. However, the mean pCO_2 for Group A

Figure 9


STANDARD
pH BICARBONATE
meg./Litre
7.35
7.3
7.2
7.1
7.0
21.0
20.0
19.0
18.0
17.0
16.0
15.0
14.0
GROUP
A
B
C
D
E
SCALP pH
CORD ARTERIAL BLOOD pH
CORD ARTERIAL BLOOD STANDARD BICARBONATE

(pCO_2 47.61 mm Hg) was higher than in the other groups (Group B, 39.10 mm Hg, Group C, 40.83 mm Hg, Group D, 42.87 mm Hg, Group E, 40.76 mm Hg) and was significantly higher than the pooled mean of all other groups.

An analysis of mean values for standard bicarbonate in scalp blood showed no significant difference between groups.

Cord arterial blood pH was significantly lower in Group A than in Groups B, D and E. As there were only three subjects in Group C, the difference between Group A and Group C was not significant.

Whilst no differences were seen between Groups in cord arterial blood pCO_2, the value for standard bicarbonate in Group A (mean 15.41 meq/litre) was significantly lower than in Groups C (20.67 meq/litre), D (17.43 meq/litre) and E (17.91 meq/litre).

Discussion

This study confirms previous findings that increasing acidosis is associated with deteriorating condition of the infant at birth.

Prospectively, scalp blood pH alone stands out as the most consistent single prognostic factor for assessing the condition of the neonate at birth.

The importance of metabolic acidosis becomes apparent at the time of birth when it is clear that foetal condition is linked with pH and standard bicarbonate values and not with pCO_2 in the cord arterial blood. The respiratory component of any acidosis therefore appears to have little significance in relation to the condition of the infant at birth. Further discussion of the significance of these findings is included in Chapter VI.

CHAPTER V.

FOETAL ELECTROCARDIOGRAPHY

SECTION 1. Time Constants and Configuration of the Foetal Electrocardiogram in Normal and Acidotic Foetuses

Numerous reports have appeared in the literature on the time constants of the foetal electrocardiogram, and these studies were reviewed by Shenker in 1966.

The purpose of the present study was to obtain values for time constants of the foetal electrocardiogram in the whole group studied with samples measured early and late in labour, and to obtain mean values for the whole sample. In addition, subjects have been grouped in relation to known biochemical data obtained at the same time, in an attempt to obtain a more accurate idea of the influence of acid-base changes on the foetal electrocardiogram. The group mean data for time constants in the foetal electrocardiogram are shown in Table X and XI.

TABLE X

FOETAL ELECTROCARDIOGRAM TIME CONSTANTS IN SECONDS

FIRST SAMPLE OF FOETAL ELECTROCARDIOGRAM

Time Constant	Number	Mean	Maximum	Minimum	Standard Deviation (Mean)
PR	101	0.11	0.14	0.08	0.002
RR	101	0.439	0.56	0.28	0.004
QRS	101	0.028	0.04	0.02	0.001
QT	97	0.233	0.300	0.200	0.002
$*QT_c$	97	0.350	0.440	0.29	0.003

$*QT_c$ in this and all subsequent tables is the QT interval corrected for heart rate.

TABLE XI

FOETAL ELECTROCARDIOGRAM TIME CONSTANTS IN SECONDS

SECOND SAMPLE OF FOETAL ELECTROCARDIOGRAM

Time Constant	Number	Mean	Maximum	Minimum	Standard Deviation (Mean)
PR	95	0.114	0.160	0.08	0.002
RR	96	0.448	0.72	0.30	0.008
QRS	96	0.028	0.04	0.02	0.001
QT	95	0.243	0.400	0.18	0.003
QT_c	95	0.363	0.47	0.29	0.004

The standard deviation of QRS values in Table X did not record on the computer programme, as the values were recorded only to the third decimal place.

With the exception of QRS values, all the other intervals were slightly prolonged at the time of the second ECG sample, collected shortly before delivery.

It should be remembered that electrocardiograms measured in this study were sampled during a resting uterine phase at a time when the heart rate was constant. An analysis of the linear correlation of variants was performed on all foetal time constants and acid-base parameters. No significant linear relationships were established within the foetal acid-base values and foetal electrocardiographic values. The scalp blood pH was shown to have a significant quadratic relationship with PR intervals measured at the same time.

Time constants of the foetal electrocardiogram were grouped according to the degree of acidosis as measured by scalp pH and standard bicarbonate values and cord arterial and venous blood pH and standard bicarbonate.

The results of these analyses are shown in Tables XII to XVII.

TABLE XII

GROUP MEAN DATA

SCALP BLOOD pH AND FOETAL ECG CONSTANTS - FIRST SAMPLE

(Seconds)

ECG Variant	Group I			Group II			Group III		
	No.	Mean	S.E.	No.	Mean	S.E.	No.	Mean	S.E.
PR	12	0.113	.005	29	0.112	.003	58	.109	.002
RR	12	0.437	.013	29	0.44	.008	58	0.44	.006
QRS	12	.026	.001	29	.029	.001	58	.029	.001
*R/S	12	1.314	0.309	29	1.276	.199	58	1.497	.141
QT	12	0.235	.007	28	0.234	.004	55	0.233	.003
QT_c	12	0.353	.009	28	0.348	.006	55	0.350	.004

Group I - scalp pH $\leq$ 7.25

Group II - " " 7.26 - 7.30

Group III - " " $\geq$ 7.31

*R/S in this and subsequent tables refers to the ratio of the R wave to the S wave.

TABLE XIII

SCALP BLOOD STANDARD BICARBONATE AND FOETAL ECG CONSTANTS - FIRST SAMPLE

Variant	Group I			Group II		
	No.	Mean	S.E.	No.	Mean	S.E.
PR	57	0.110	.002	39	0.113	.003
RR	57	0.434	.006	39	0.446	.007
QRS	57	.028	.001	39	.028	.001
R/S	57	1.399	.143	39	1.441	.173
QT	55	0.231	.003	37	0.235	.004
QT_c	55	0.349	.004	37	0.351	.005

Group I - scalp blood standard bicarbonate $\leqslant$ 20.5 meq/litre
Group II - " " " " $\geqslant$ 20.6 meq/litre

TABLE XIV

CORD ARTERIAL BLOOD pH AND FOETAL ECG CONSTANTS - SECOND SAMPLE

Variant	Group I			Group II			Group III		
	No.	Mean	S.E.	No.	Mean	S.E.	No.	Mean	S.E.
PR	47	0.116	.003	17	0.113	.004	28	0.114	.004
RR	48	0.432	.011	17	0.472	.019	28	0.460	.015
QRS	48	.028	.001	17	.026	.001	28	.027	.001
R/S	48	1.238	.089	17	1.56	.149	28	1.212	.116
QT	47	0.242	.005	17	0.248	.008	28	0.240	.006
QT_c	47	0.367	.005	17	0.362	.008	28	0.354	.007

Group I - cord arterial blood pH $\leqslant$ 7.20
Group II - " " " " 7.21 - 7.25
Group III - " " " " $\geqslant$ 7.26

TABLE XV

CORD ARTERIAL BLOOD STANDARD BICARBONATE AND FOETAL ECG CONSTANTS - SECOND SAMPLE

Variant	Group I			Group II			Group III		
	No.	Mean	S.E.	No.	Mean	S.E.	No.	Mean	S.E.
PR	9	0.107	.006	74	0.115	.002	9	0.116	.006
RR	10	0.444	.025	74	0.450	.009	9	0.438	.026
QRS	10	.028	.002	74	0.027	.001	9	.03	.003
R/S	10	1.413	.198	74	1.283	.073	9	1.206	.208
QT	9	0.247	.011	74	0.243	.004	9	0.236	.011
QT_c	9	0.372	.012	74	0.361	.004	9	0.359	.012

Group I - cord arterial blood standard bicarbonate $\leqslant$ 14.5 meq/litre

Group II - cord arterial blood standard bicarbonate 14.6 - 20.5 meq/litre

Group III - cord arterial blood standard bicarbonate $\geqslant$ 20.6 meq/litre

TABLE XVI

CORD VENOUS BLOOD pH AND FOETAL ECG CONSTANTS - SECOND SAMPLE

Variant	Group I No.	Mean	S.E.	Group II No.	Mean	S.E.	Group III No.	Mean	S.E.
PR	10	0.112	.006	21	0.119	.004	35	0.117	.003
RR	10	0.449	.025	21	0.446	.017	35	0.469	.014
QRS	10	0.029	.001	21	0.027	.001	35	0.028	.001
R/S	10	1.468	.207	21	1.440	.143	35	1.326	.111
QT	10	0.260	.011	21	0.247	.008	35	0.243	.006
QT_c	10	0.384	.010	21	0.372	.007	35	0.354	.005

Group I - cord venous blood pH $\leqslant$ 7.25
Group II - cord venous blood pH 7.26 - 7.30
Group III - cord venous blood pH $\geqslant$ 7.31

TABLE XVII

CORD VENOUS BLOOD STANDARD BICARBONATE AND FOETAL ECG CONSTANTS - SECOND SAMPLE

Variant	Group I No.	Mean	S.E.	Group II No.	Mean	S.E.	Group III No.	Mean	S.E.
PR	1	0.160	.017	47	0.114	.002	17	0.120	.004
RR	1	0.600	.079	47	0.455	.012	17	0.465	.019
QRS	1	.030	.004	47	.027	.001	17	.030	.001
R/S	1	2.00	0.656	47	1.384	.096	17	1.324	.159
QT	1	0.300	.035	47	0.247	.005	17	0.244	.008
QT_c	1	0.390	.033	47	0.366	.005	17	0.355	.008

Group I - cord venous standard bicarbonate $\leqslant$ 14.5 meq/litre
Group II - " " " " 14.5-20.5 "
Group III - " " " " $\geqslant$ 20.6 meq/litre

A complete analysis of these groups of biochemical data was also studied in relationship to electrocardiographic time constants as sampled early and late in labour, and following delivery. The significant differences of ECG variants in the various acid-base groups are summarised in Table XVIII.

The configuration of the foetal electrocardiogram based on the mean data of the intervals measured for time constants in relation to grouping of the scalp pH values is shown in Figures 10 and 11. Figure 10 shows the ECG values in normal foetuses and Figure 11, the values in acidotic subjects. Figure 12 shows 2 examples of normal foetal ECGs and one example of the foetal ECG in an acidotic foetus.

ST Segment and T Wave Configuration

ST segment changes and T wave configuration were recorded on all of the foetal electrocardiographic measurements. For further analysis, changes were classified in three groups, as follows:-

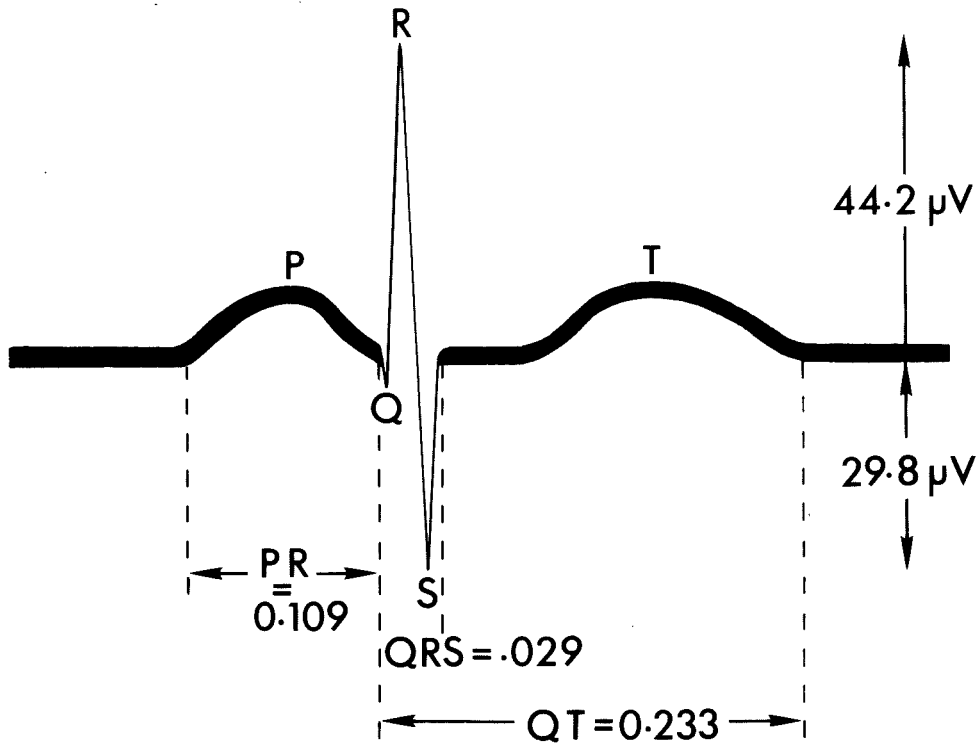
ST segment	1 = normal
	2 = depressed
	3 = elevated
T waves	1 = flat
	2 = upright
	3 = inverted

LEGEND TO FIGURE 10

Mean values for foetal E.C.G. time constants in relationship to scalp blood pH values within the normal range. The amplitudes of the P, Q and T waves are not drawn to scale.

Figure 10

SCALP BLOOD pH ⩾ 7·31



SCALP BLOOD pH
7·26 – 7·30

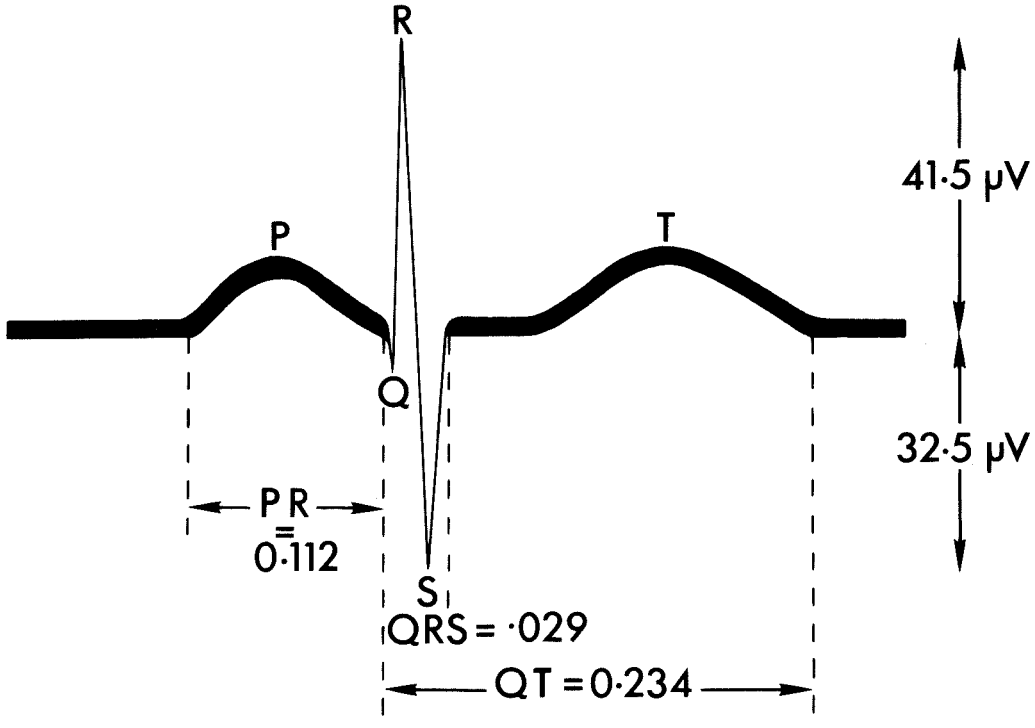


TABLE XVIII

SIGNIFICANT ECG DIFFERENCES IN GROUPED ACID BASE DATA

Biochemical Variant	ECG Variant	Grouping	Mean	S.E.	Level of Significance
Scalp blood pH	Post delivery heart axis	≤7.25	142.92	6.837	5%
		7.26-7.30	118.65	5.335	
		≥7.31	133.09	3.514	
Scalp blood pH	R/S aVR (post delivery)	≤7.25	8.214	1.133	5%
		7.26-7.30	5.007	.801	
		≥7.31	5.279	.572	
Scalp blood standard bicarbonate	R/S_2	14.6-20.5*	1.175	.084	5%
		≥20.6*	1.446	.099	
Cord arterial blood pH	R/S Lead III (post delivery)	≤7.20	8.100	.434	5%
		7.21-7.25	5.940	.679	
		≥7.26	7.889	.585	
Cord venous blood standard bicarbonate	PR_2	≤14.5*	0.160	.017	5%
		14.6-20.5*	0.114	.002	
		≥20.6*	0.120	.004	
Cord venous blood standard bicarbonate	QRS_2	≤14.5*	0.130	.004	5%
		14.6-20.5*	0.027	.001	
		≥20.6*	0.030	.001	
Cord venous blood pH	QTC_2	≤7.25	0.384	.010	5%
		7.26-7.30	0.372	.007	
		≥7.31	0.354	.005	

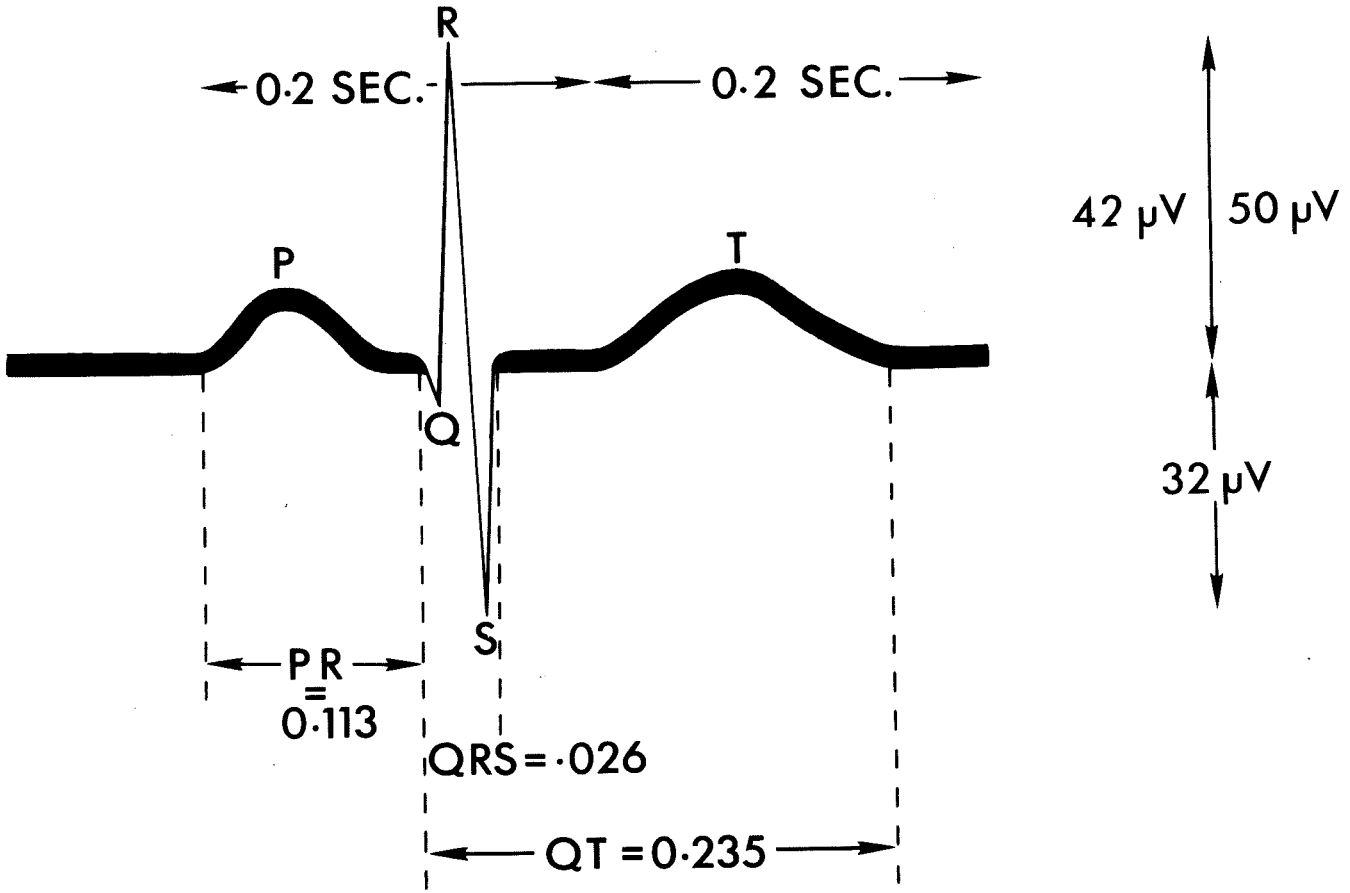
*Values marked with an asterisk are in meq/litre.

LEGEND TO FIGURE 11

Mean values for foetal E.C.G. time constants in relationship to scalp blood pH values where scalp blood pH measurements showed the presence of foetal acidosis.

Figure 11

SCALP BLOOD pH
pH ⩽ 7·25



Inverted T waves were noted in 14 of the foetal ECG samples and depressed ST segment was noted in five samples.

Analysis of ST segment and T wave changes in relation to foetal acid-base and electrolyte parameters showed no consistent linear or quadratic relationships. An increased tendency to flattened T waves was noted in the relationship to hyperkalaemia ($r = -0.38$).

Peak-to-Peak Voltage of the foetal Electrocardiogram

Peak-to-peak voltages were measured of the foetal ECG complex early and late in labour.

The mean value in early labour was 87.2 μV (S.E. of mean 3.83), and in late labour 105.7 μV (S.E. of mean 5.22).

There was a significant increase in peak-to-peak voltage towards the end of labour.

Range of Values of the Normal Foetal Electrocardiogram

To establish the range of values of foetal electrocardiographic time constants in a group of foetuses known to have normal acid-base status, the maximum and minimum values for all time constants for foetuses with a scalp blood pH greater than or equal to 7.31 are shown in Table XIX.

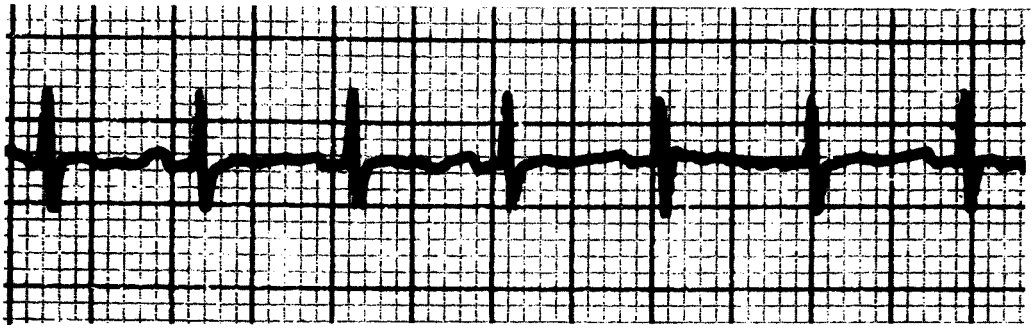
FIGURE 12

Case Numbers 16 and 20 Foetal electrocardiogram in two subjects with known normal acid-base status. Configuration shows normal P waves and normal T waves. Paper speed 2.5 cms/second.

Case Number 51 Foetal electrocardiogram recorded shortly before delivery. Cord arterial blood pH 7.12. T waves are flat and sometimes inverted. QT_c = 0.35 second. Paper speed 2.5 cms/second.

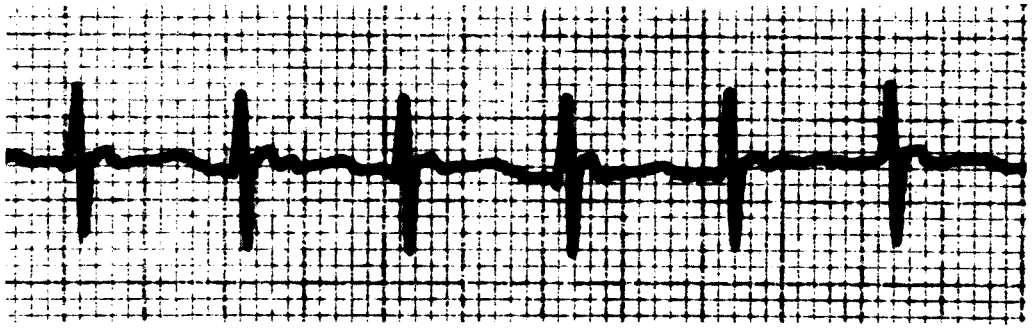
FIGURE 12

Case Number 16.



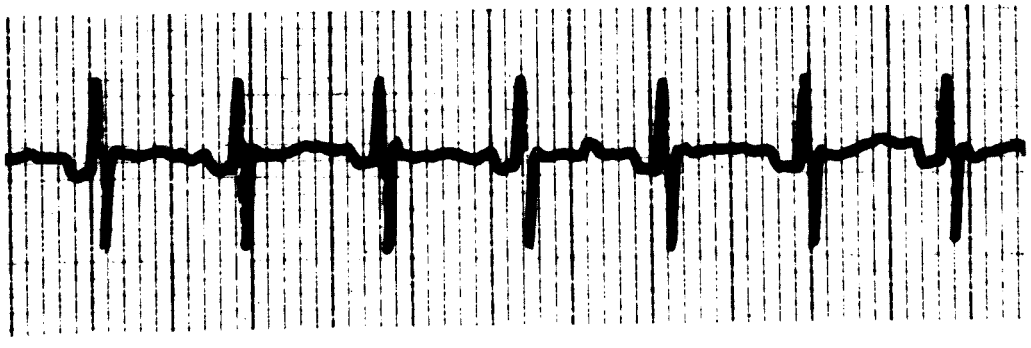
Foetal E.C.G. Scalp electrode. Normal acid-base status.

Case Number 20.



Foetal E.C.G. Scalp electrode. Normal acid-base status.

Case Number 51.



Foetal E.C.G. Scalp electrode. Acidotic foetus.

TABLE XIX

RANGE OF TIME CONSTANTS OF THE "NORMAL" FOETAL ELECTROCARDIOGRAM

	Mean	Maximum	Minimum	S.D. (Mean)
PR	0.109	0.14	0.08	.002
RR	0.44	0.56	0.28	.006
QRS	0.029	0.04	0.02	.001
R/S	1.497	10	0.5	.141
QT	0.233	0.3	0.2	.003
QT_c	0.350	0.43	0.31	.004

It can be seen from Table XII and Table XIX that there is considerable overlap of values between normal and acidotic groups. This overlap is apparent from the maximum or minimum values of the normal group or within the range of 2 standard deviations of the mean values.

Discussion

Various methods of analysis have been used to examine the relationship between acid-base changes in the foetus and the time constants and configuration of the foetal electrocardiograph. Analysis of the linear correlation of variants and regression analyses were disappointing in demonstrating very little significant data. The only factor that emerged from this extensive analysis was a significant quadratic relationship between scalp blood pH values and PR values for the first foetal ECG sample recorded.

However, an analysis of the group mean data of foetal acid-base showed some differences in the electrocardiographic values.

Significant differences occurred principally in the cord venous blood data. In the presence of metabolic acidosis, as indicated by low standard bicarbonate levels in cord venous blood, PR intervals of the foetal ECG samples taken immediately before delivery were significantly prolonged. A significant prolongation of the corrected QT time in the "pre-delivery" foetal ECG was shown in relation to low pH values in cord venous blood.

The mean values of the cardiac axis after delivery showed that there was a significant increase in right axis deviation in those foetuses who showed low scalp blood pH

values, the mean axis being 142.9 in the group with scalp blood pH values less than 7.25.

Excluding those cases with cardiac arrhythmias, no consistent changes were seen in P and R wave configuration with respect to notching or broadening.

ST segment depression has been described by Southern (1957) and Kendall et al. (1962 and 1964), as being associated with intrauterine foetal anoxia. In these studies, the assessment of anoxia was based on the clinical signs of foetal distress. No significant pattern of ST segment and T wave configuration has been shown in this study in relation to known acid-base alterations in the foetus.

Various studies reporting the range of time constants for the foetal electrocardiogram have been made, and further discussion of this subject is included later in this thesis. Takemura (1968) analysed time constants of the foetal electrocardiogram in relation to the subsequent outcome for the foetus in 105 cases, and showed a range of PQ values of 0.09 to 0.13 second, and a QRS range of 0.06 to 0.08 second.

The range of values in the group mean data in this present study showed a PR range of 0.14 to 0.08 second, and a range of 0.04 to 0.02 second for QRS values. Very little variation was shown in the mean values for QRS measurements within the normal and acidotic groups. The most

significant changes occurred in QT intervals, with prolongation of QT intervals corrected for heart rate in the presence of significant acidosis in cord venous blood samples.

SECTION 2. Changes in Foetal Electrocardiographic Time Constants and Configuration in Normal and Acidotic Foetuses in Relation to Uterine Contractions

All of the electrocardiographic data presented in the study of foetal acid-base status and the foetal electrocardiogram was recorded during the resting uterine phase. In order to study the effect of uterine contractions on the characteristics of the foetal electrocardiogram, five subjects in whom the foetal acid-base status was known to be normal throughout labour and five subjects in whom the foetus was known to be acidotic throughout labour were selected for further study.

Time constants and R/S values were measured during the resting phase and during uterine contractions in both groups. Measurements during the contracting uterine phase were made on samples taken from the electrocardiogram immediately following the measurements made during the resting phase.

The individual results of this study are shown in Tables XX and XXI.

The group mean data from this study showed that there was no significant difference in the mean values of the time constants during uterine contraction or in the resting phase, both in the acidotic and normal subjects. Furthermore, no significant difference could be shown between the mean values

TABLE XX

FOETAL ECG VALUES DURING RESTING AND CONTRACTING UTERINE PHASES

IN 5 NORMAL FOETUSES

		PR	RR	PR/RR	QRS	R/S	QT	QT_c
CASE 89	Resting	0.1	0.42	0.24	0.02	1.7	0.24	0.37
	Contracting	0.1	0.46	0.22	0.02	1.7	0.26	0.37
CASE 95	Resting	0.1	0.42	0.24	0.02	1.0	0.2	0.31
	Contracting	0.1	0.44	0.23	0.02	1.0	0.2	0.3
CASE 72	Resting	0.08	0.42	0.19	0.04	1.2	0.2	0.31
	Contracting	0.08	0.46	0.18	0.04	1.0	0.22	0.32
CASE 43	Resting	0.12	0.44	0.27	0.03	2.0	0.24	0.36
	Contracting	0.12	0.46	0.26	0.03	2.8	0.26	0.38
CASE 14	Resting	0.12	0.48	0.25	0.02	1.1	0.26	0.37
	Contracting	0.12	0,52	0.23	0.02	1.1	0.26	0.36

TABLE XXI

FOETAL ECG VALUES DURING RESTING AND CONTRACTING UTERINE PHASES

IN 5 ACIDOTIC FOETUSES

		PR	RR	PR/RR	QRS	R/S	QT	QT_c
CASE 44	Resting	0.08	0.42	0.2	0.03	2.0	0.24	0.37
	Contracting	0.08	0.41	0.2	0.03	1.5	0.24	0.37
CASE 65	Resting	0.12	0.46	0.26	0.02	1.7	0.22	0.32
	Contracting	0.12	0.54	0.22	0.02	1.0	0.24	0.38
CASE 70	Resting	0.12	0.46	0.26	0.03	2.7	0.26	0.38
	Contracting	0.1	0.44	0.23	0.03	3.0	0.24	0.37
CASE 88	Resting	0.12	0.46	0.26	0.02	0.75	0.24	0.35
	Contracting	0.12	0.44	0.27	0.02	0.63	0.24	0.36
CASE 105	Resting	0.1	0.44	0.23	0.02	1.2	0.24	0.36
	Contracting	0.1	0.4	0.25	0.02	1.3	0.24	0.38

within the normal and acidotic groups.

Discussion

A significant difference in time constants was not apparent in relation to the stress placed on the foetal cardiovascular system by the increase of intrauterine pressures. An analysis of these measurements therefore suggests that the changes that occur in these factors are dependent on chronic or long-term factors such as metabolic acidosis and associated electrolyte changes, rather than possible short-term effects of alteration in blood gas parameters.

Various authors have shown a relationship between heart rate patterns and foetal acid-base status (Wood et al. 1967), Mendez-Bauer et al. 1967), and it has not been the intention of this study to investigate foetal heart rate patterns. It was of interest to note, however, that the foetal heart rate slowed during contraction in all of the normal cases, and in the acidotic group the heart rate slowed in four cases and increased in one case during contractions.

SECTION 3. Changes in Foetal Electrocardiographic Time Constants and Configuration in Relation to Foetal Blood Plasma Electrolytes

Plasma sodium, potassium and chloride values were measured in cord arterial and venous blood samples. These parameters were not measured in scalp blood as there was usually insufficient sample. In the analysis of the biochemical data, a significant negative correlation was established between potassium levels in relation to pH values in both cord arterial and cord venous blood. It was considered possible that the influence of chronic asphyxial changes on the foetal electrocardiogram might be effected through changes secondarily induced in plasma electrolyte values. A linear and regression analysis was performed on all foetal electrocardiographic and electrolyte measurements. The significant linear relationships in this study are shown in Table XXII.

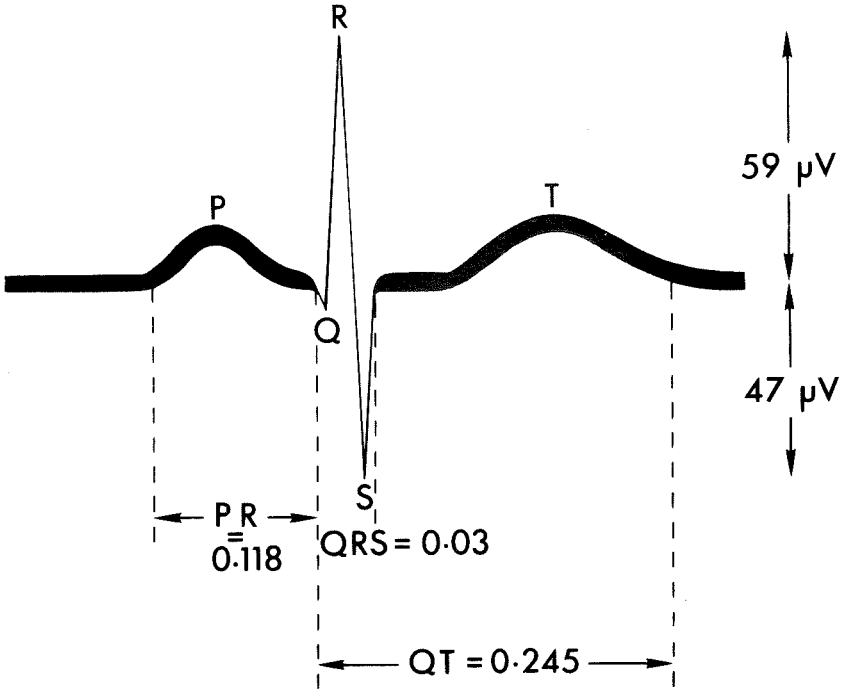
The relationship between cord venous blood plasma K^+ and T wave configuration showed that "flat" T waves tended to occur in the early ECG samples as plasma potassium levels increased ($r = -0.38$, $p < 0.01$). The opposite relationship was noted between cord venous plasma sodium and T wave configuration. The relationship between cord venous plasma potassium values and foetal ECG configuration is represented in Figures 13 and 14.

LEGEND TO FIGURE 13

Mean values for foetal E.C.G. time constants within normal ranges of cord venous plasma electrolyte values. The amplitudes of the P, Q and T waves are not drawn to scale.

Figure 13

CORD VENOUS PLASMA
K ≤ 4·0 meq./ L.



CORD VENOUS PLASMA
K 4·1 – 6·0 meq./ L.

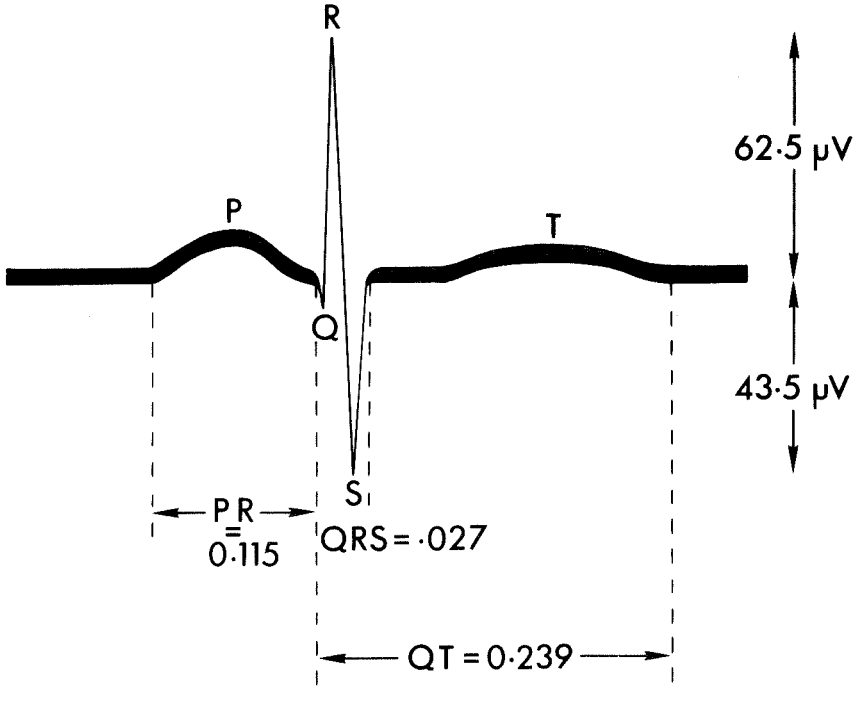


TABLE XXII

FOETAL PLASMA ELECTROLYTE VALUES AND ELECTROCARDIOGRAPHIC DATA

SIGNIFICANT LINEAR RELATIONSHIPS

Plasma Electrolyte	Foetal Electrocardiographic variant	Correlation Coefficient
Cord arterial plasma chloride	PR interval (sample 1)	-0.3212
Cord arterial plasma chloride	PR interval (sample 2)	-0.2868
Cord arterial plasma chloride	Post-delivery ECG. ST segment	+0.3505
Cord arterial plasma chloride	R/S ratio. Lead III (post-delivery ECG)	+0.3166
Cord arterial plasma potassium	R/S ratio. Lead III (post-delivery ECG)	-0.2587
Cord venous plasma sodium	QT_c (sample 1)	+0.2731
Cord venous plasma sodium	T wave configuration (sample 2)	+0.3388
Cord venous plasma sodium	Post-delivery ECG. ST segment	-0.2549
Cord venous plasma potassium	T wave configuration (sample 1)	-0.3830

Regression analysis showed a significant quadratic relationship between cord venous plasma sodium concentration and the R/S ratio in lead aVL of the delivered infant.

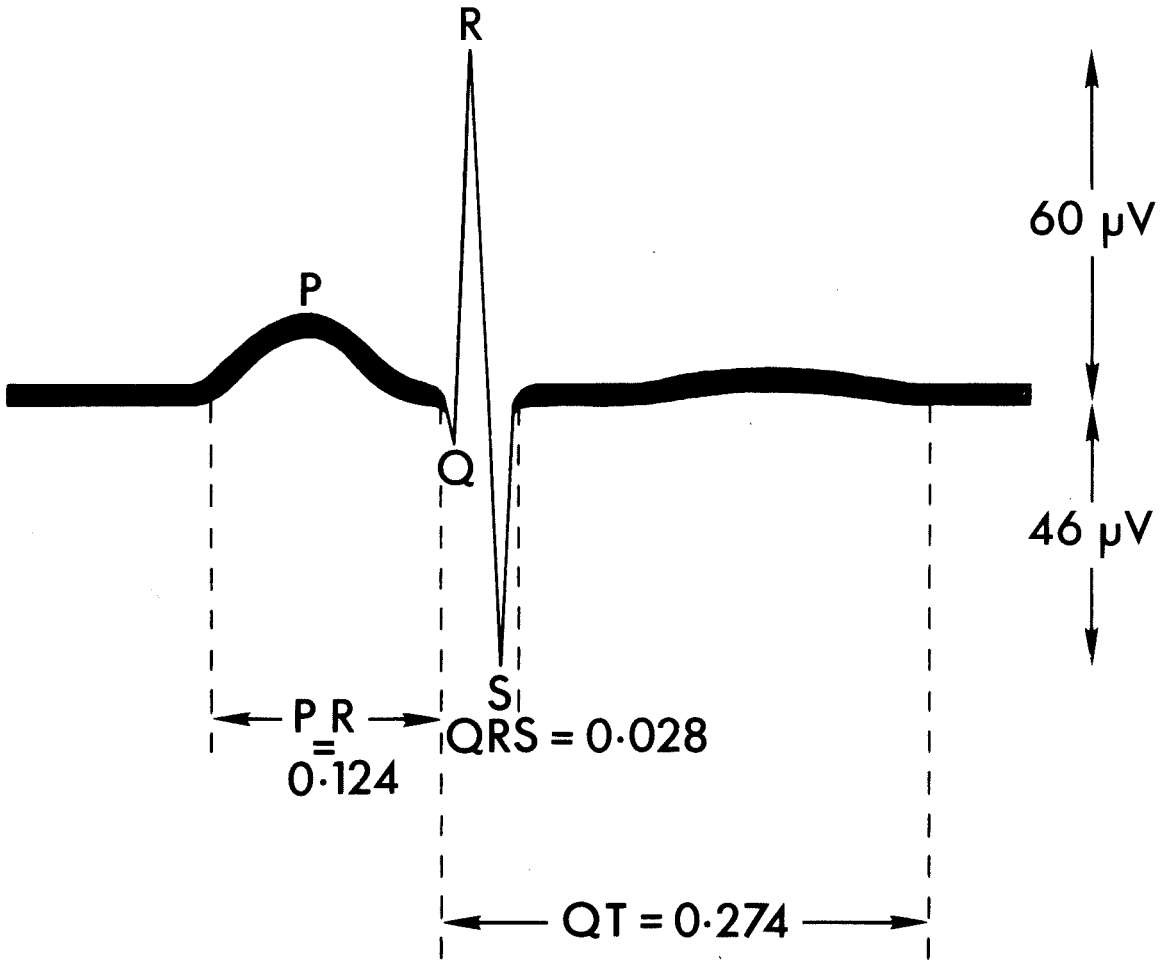
All relationships shown in Table XXII were significant at a 5% level, and for all relationships where $r \pm 0.3$, the p value was < 0.01.

LEGEND TO FIGURE 14

Mean values for foetal E.C.G. time constants in the presence of hyperkalaemia in cord venous plasma potassium. K^+ 6.1 meq/l. Note significant prolongation of QT interval.

Figure 14

CORD VENOUS PLASMA
K ⩾ 6·1 meq. / L.



Calcium balance was not measured in this study, but the importance of this factor has not been overlooked. Hypocalcaemia is generally associated with hypokalaemia and, for this reason and the importance of potassium balance in its own right, further studies have been performed in relation to foetal electrocardiographic time constants.

The mean values for the time constants and configuration of the foetal electrocardiogram immediately before delivery in data grouped according to plasma potassium levels in cord arterial and cord venous blood are shown in Tables XXIII and XXIV respectively.

The significant differences between these groups are summarised in Table XXV.

Discussion

Certain significant factors have emerged in the analysis of the electrolyte data. Some of the relationships are difficult to explain. It can be seen, for example, that plasma chloride values in cord arterial blood showed a significant relationship with a variety of electrocardiographic measurements before and after delivery.

In fact, the most significant relationship in the matrix of linear relationships was between cord venous plasma potassium and T wave depression ($r = -0.3830$).

TABLE XXIII

CORD ARTERIAL BLOOD PLASMA POTASSIUM AND FOETAL ECG CONSTANTS - SECOND SAMPLE

Variant	Group I			Group II			Group III		
	No.	Mean	S.E.	No.	Mean	S.E.	No.	Mean	S.E.
PR	8	0.108	.007	50	0.114	.003	15	0.121	.005
RR	8	0.475	.028	50	0.447	.011	15	0.440	.020
QRS	8	.026	.002	50	.026	.001	15	.028	.001
R/S	8	1.444	0.233	50	1.345	.093	15	1.272	0.170
QT	8	0.243	.011	50	0.245	.005	15	0.235	.008
QT_c	8	0.349	.011	50	0.366	.004	15	0.355	.008

Group I - cord arterial plasma potassium $\leq$4.0 meq/litre
Group II - cord arterial plasma potassium 4.1-6.0 meq/litre
Group III - cord arterial plasma potassium $\geq$6.1 meq/litre

TABLE XXIV

CORD VENOUS BLOOD PLASMA POTASSIUM AND FOETAL ECG CONSTANTS - SECOND SAMPLE

Variant	Group I			Group II			Group III		
	No.	Mean	S.E.	No.	Mean	S.E.	No.	Mean	S.E.
PR	11	0.118	.005	42	0.115	.003	10	0.124	.006
RR	11	0.465	.024	42	0.447	.012	10	0.500	.025
QRS	11	.030	.001	42	.027	.001	10	.027	.001
R/S	11	1.245	0.201	42	1.437	0.103	10	1.296	0.211
QT	11	0.245	.010	42	0.239	.005	10	0.274	.010
QT_c	11	0.360	.009	42	0.356	.005	10	0.393	.009

Group I - cord venous plasma potassium $\leq$4.0 meq/litre
Group II - cord venous plasma potassium 4.1-6.0 meq/litre
Group III - cord venous plasma potassium $\geq$6.1 meq/litre

TABLE XXV

SIGNIFICANT FOETAL ELECTROCARDIOGRAPHIC CHANGES

IN RELATION TO CORD PLASMA POTASSIUM VALUES

Biochemical Variant	ECG Variant	Grouping (meq/l)	Mean	S.E.	Level of significance
Cord arterial plasma potassium	QRS (sample 1)	≤ 4.0	0.110	.005	1%
		4.1-6.0	0.109	.002	
		≥ 6.1	0.124	.004	
Cord venous plasma potassium	QT (sample 2)	≤ 4.0	0.245	.01	5%
		4.1-6.0	0.239	.005	
		≥ 6.1	0.274	.010	
Cord venous plasma potassium	QT_c (sample 2)	≤ 4.0	0.360	.009	1%
		4.1-6.0	0.356	.005	
		≥ 6.1	0.393	.009	

The subsequent analysis of group mean data has shown that hyperkalaemia in cord venous blood is associated with a significant prolongation of QT intervals. The significance of this relationship is even more apparent where corrections for heart rate have been made. It was of interest that this pattern emerged most clearly in cord venous blood, whereas, although the same trend was seen in cord arterial blood, once a correction factor for heart rate had been introduced, the difference did not achieve significance. This difference may be explained by the fact that, by the time the foetal blood has passed through the placenta, some correction of potassium and acid-base values should have occurred. If the hyperkalaemia still persists at this stage, it indicates that more severe and persistent changes had occurred which were no longer being adequately corrected by the placenta.

Hyperkalaemia in the foetus has been shown in this study to be related to foetal acidosis, both in the cord arterial blood and cord venous blood, and a fall in cord venous blood pH has also been shown to be related to a prolongation of the corrected QT interval.

It is apparent that an anomalous situation has been described with regard to the electrocardiographic features in the foetus and the acid-base and potassium values in foetal blood.

Hyperkalaemia in the adult is characterised by exceptionally tall and slender T waves, widening and slurring of the QRS complex, and cardiac arrhythmias associated with depression of the normal pace-makers. The characteristic features of the electrocardiogram in the hyperkalaemic and acidotic foetus, as shown in this study, consist of slight prolongation of the QRS interval, although there is little significant change in the QRS configuration, with prolongation of the QT interval and depressed T waves.

These findings are those found in hypokalaemia in the adult heart. Similar findings are also seen in hypocalcaemia and myocardial ischaemia.

U waves were occasionally seen in the foetal electrocardiogram, but interpretation and identification of U waves was too uncertain to justify inclusion in the general analysis of data.

It is therefore possible that these changes may result from chronic myocardial ischaemia. However, the foetus is in a unique position in relation to electrolyte balance, as it must be dependent, to some degree on placental exchange of electrolytes to maintain electrolyte homeostasis. Potassium levels have been shown to be related to acid-base values, and metabolic acidosis is associated with hyperkalaemia. In the situation where the foetus has become

acidotic because of chronic asphyxial changes, a potassium gradient develops across the placental barrier, and the possibility for potassium loss from the foetus is established. Under these circumstances, a unique paradox is created whereby intracellular potassium depletion occurs in the presence of hyperkalaemia in the foetus, and the characteristic features of the electrocardiogram of the acidotic foetus are therefore those of hypokalaemia.

SECTION 4. Interrelationships Between Foetal and Neonatal Electrocardiographic Data and Neonatal Assessment

The mean values for the electrocardiographic data of the foetus have been described in Tables X and XI. The mean values for neonatal electrocardiographic time constants are shown in Table XXVI. These values were measured on Lead II of the neonatal electrocardiogram. Recordings were made within eight hours of delivery and, in most cases, within one hour of delivery. R/S ratios and electrical axis values are discussed in a later section.

TABLE XXVI

POST-DELIVERY ELECTROCARDIOGRAPHIC VALUES (LEAD II)

Variate	Number	Mean	Maximum	Minimum	S.D. (Mean)
PR	98	0.115	0.160	0.080	.002
RR	98	0.476	0.660	0.300	.007
QRS	98	0.031	0.050	0.020	.001
QT	98	0.280	0.400	0.160	.004
QT_c	98	0.404	0.530	0.310	.005

A significant difference was apparent between the actual QT intervals and QT intervals corrected for heart rate in the values before delivery and following delivery. PR values appear to remain constant, and there is a slight increase in QRS measurement after delivery.

The significant linear relationships ($p < 0.01$) between time constants of the foetal electrocardiogram and the neonatal electrocardiogram are shown in Table XXVII. Significant linear relationships were shown between the first sample RR interval and post-delivery PR intervals, first sample QT intervals and post-delivery RR intervals, and first sample RR intervals and first sample QT intervals.

A significant relationship was shown between all time constants measured in the first and second samples of the foetal electrocardiogram. In other words, the time constants apparent early in labour bear a significant positive relationship to time constants late in labour. However, no consistent relationship was shown in the time constants of the foetal electrocardiogram and the neonatal electrocardiogram. QT intervals were shown to be related to RR intervals in both foetal electrocardiographic samples, but not in the post-delivery sample.

QT intervals showed a highly significant relationship with RR intervals in both early and late samples of the foetal electrocardiogram ($r = +0.5$ and $+0.63$, $p < 0.001$ respectively). Prolongation of QT intervals in sample 1 was shown to be related to T wave inversion ($r = +0.36$, $p < 0.001$).

Atrioventricular conduction times before delivery, as

TABLE XXVII

ELECTROCARDIOGRAPHIC VARIANTS

Variant 1	Variant 2	Correlation Coefficient
PR_1	PR_2	+0.49
PR_1	PD.RR	+0.46
PR_2	PD.RR	+0.51
PR_2	QRS_2	+0.36
PR_2	RR_1	+0.32
QRS_1	QRS_2	+0.45
QRS_1	PD.QRS	+0.34
QRS_2	RR_1	+0.30
RR	QT_1	+0.50
RR_1	RR_2	+0.35
RR_1	QT_2	+0.37
RR_2	QT_2	+0.63
QT_1	QT_{c1}	+0.86
QT_1	T_1	+0.36
QT_1	QT_2	+0.57
QT_2	QT_{c2}	+0.76
QT_{c1}	T_1	+0.32
QT_{c1}	QT_{c2}	+0.45
T_1	T_2	+0.47
PD.QRS	PD.QT	+0.37
PD.QRS	$PD.QT_c$	+0.33
PD.QT	$PD.QT_c$	+0.90

1 = Sample one of the foetal ECG

2 = Sample two of the foetal ECG

P.D. = post-delivery ECG

measured by both foetal samples of PR values, showed highly significant relationships with post-delivery heart rate.

A detailed linear and regression analysis of all electrocardiographic time constants in relation to Apgar scores and all neonatal assessment factors did not reveal any significant correlations.

Discussion

The mean values for QT intervals were shown to be significantly prolonged after delivery. This difference was clearly apparent when a correction was made for heart rate using the method described by Kissin et al. in 1948. A significant relationship was also demonstrated between QT intervals (electrical systole) and heart rate in both foetal and neonatal electrocardiograms. These findings confirmed those described by Persianinov et al. in 1966. Thus, the length of systole becomes prolonged after delivery, and diastole becomes shortened. A significant relationship was demonstrated between T wave inversion and QT prolongation. It was not possible to define whether this change in QT intervals was real, or whether it was associated with the presence of U waves. However, no definite U waves were identified.

The mean values of PR intervals representing the atrioventricular conduction time showed similar and consistent values in all ECG samples. This pattern was also confirmed by the study of the relationship between PR intervals before and after delivery, a significant linear relationship being shown between PR_1 and PR_2, and PR_1, PR_2 and post-delivery PR intervals.

Although the previous analysis of group mean data showed a significant prolongation of QT intervals in relation to low pH values in cord venous blood, no significant relationships were established in the regression and linear analyses of neonatal assessments and electrocardiographic data.

SECTION 5. Vectorcardiography in the Foetal and Neonatal Electrocardiogram

In view of the studies by Larks on the electrical axis of the foetal heart, as assessed from maternal abdominal recordings of the foetal electrocardiogram, a comparative study was undertaken between foetal and neonatal electrocardiographic configuration. This investigation was designed to determine which lead in the neonatal electrocardiogram most closely resembles the configuration of the foetal electrocardiogram.

Initially, all assessments of the configuration of the QRS complex were based on the ratio of the positive R wave deflection to the negative S wave deflection. Where there was no recordable negative deflection, a value of 10 was allotted, and where there was no recorded positive deflection, a value of 0.1 was allotted. These values therefore represented the maximum and minimum readings in this study.

Table XXVIII shows the mean values for the R/S ratio in both foetal electrocardiographic samples and neonatal R/S ratios in all leads.

From the mean values in Table XXVIII, it can be seen that the mean R/S ratios for the foetal ECG samples most closely resemble the R/S ratio in Leads I and II and V_1,

TABLE XXVIII

MEAN VALUES - FOETAL AND NEONATAL R/S RATIOS

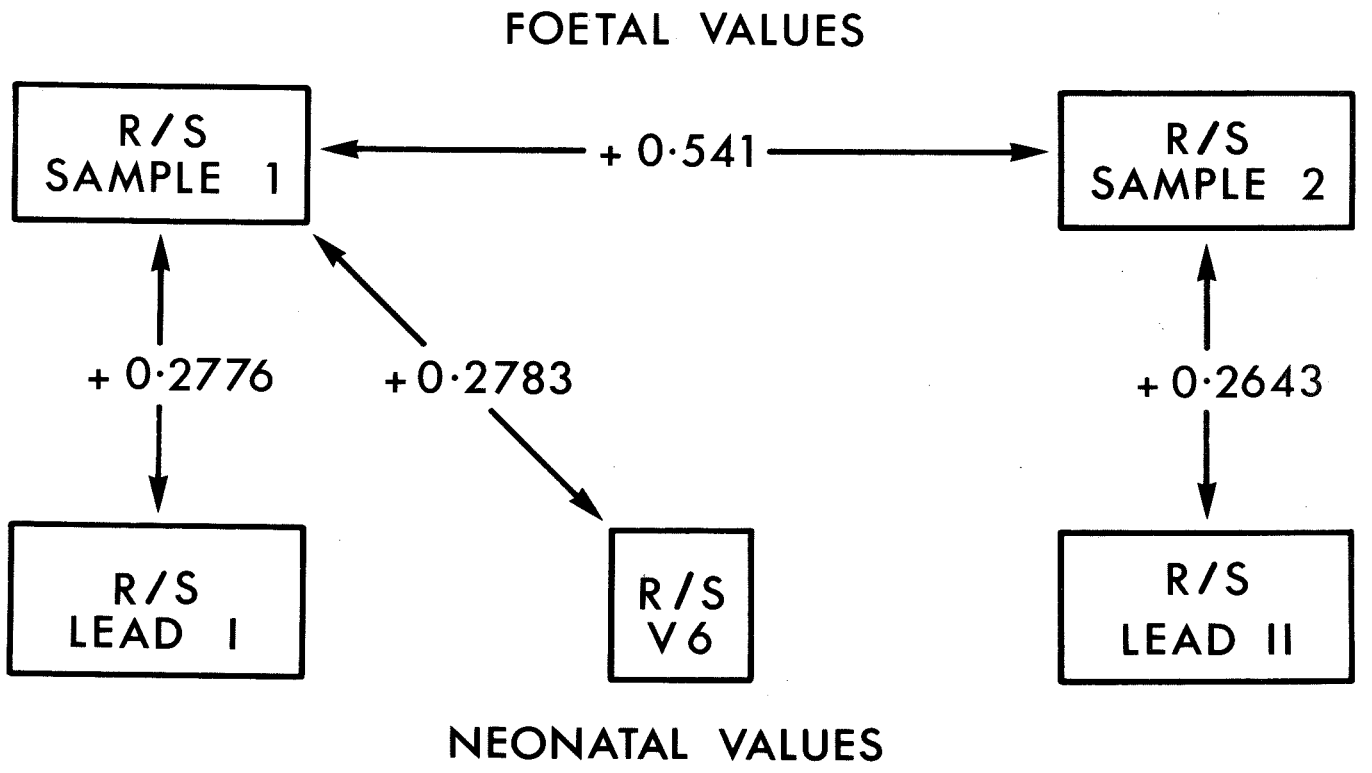
Variate	Number	Mean	Maximum	Minimum	S.D. (Mean)
R/S (sample 1)	101	1.407	10.00	0.50	0.105
R/S (sample 2)	96	1.285	5.00	0.200	0.062
R/S Lead I	99	0.922	10.00	0.100	0.254
R/S Lead II	98	3.835	10.00	0.100	0.394
R/S Lead III	99	7.532	10.00	0.100	0.318
R/S aVR	99	5.713	10.00	0.100	0.435
R/S aVL	99	0.690	10.00	0.100	0.218
R/S aVF	99	6.281	10.00	0.100	0.402
R/S V_1	98	1.399	10.00	0.220	0.142
R/S V_3	96	1.217	10.00	0.100	0.154
R/S V_6	96	2.056	10.00	0.100	0.308

V_2 and V_3 in the neonatal electrocardiogram, sample 1 being closest to V_1, and sample 2 being closest to V_3.

The R/S data was also subjected to linear and quadratic regression analyses, in order to determine the most consistent relationship shown within the data. It was apparent that, although the mean values of foetal R/S values resembled mean R/S values in certain of the neonatal leads, this did not necessarily imply that these values were related to each other in any consistent fashion. Figure 15 shows the significant correlations between foetal and neonatal R/S values.

Figure 15

THE RELATIONSHIP OF R/S VALUES IN THE FOETAL AND NEONATAL ELECTROCARDIOGRAM



A significant quadratic relationship was also shown between R/S values in sample 1 of the foetal electrocardiogram and lead V_3 on the neonatal electrocardiogram.

Whilst Larks et al. (1966) have produced convincing evidence that the foetal electrocardiograms obtained from abdominal electrodes closely resemble lead II of the neonatal electrocardiogram, these authors have not studied the relationship with scalp electrode ECGs. Roche and Hon, in 1965, stated that the QRS complex of the vaginal and abdominal foetal electrocardiogram appeared to be similar, and resembled lead II of the neonatal electrocardiogram.

Although the picture is not clear when relating the foetal electrocardiogram early in labour to subsequent post-delivery electrocardiograms, it does appear, in this study, that the only significant linear relationship to emerge near the time of delivery was between the scalp ECG configuration and lead II of the neonatal electrocardiogram.

Studies in Electrical Axis of the Foetal and Neonatal Heart

Using the method described by Larks in 1965 for estimating the foetal axis, based on the algebraic sum of R wave and Q or S waves corrected to an R wave value of 10, the electrical axis of the foetal heart was measured in early and late labour.

Because all R and S wave values were expressed as a ratio, it was necessary to convert them to an algebraic sum. This was done using the formula as follows:

$$\text{Algebraic sum} = 10 - \left(\frac{S \times 10}{R}\right),$$

where S and R are the negative and positive deflections of the foetal complex measured in millimetres.

A conversion nomogram derived from this formula is shown in Figure 16.

These values were then converted to electrical axis using the graph described by Larks (1965) for this purpose. This graph was derived from the fact that the algebraic sum of positive and negative deflections of the foetal electrocardiogram is directly proportional to the sine of the angle of the foetal cardiac axis.

The mean values for cardiac axis of the foetus and neonate are shown in Table XXIX.

No relationship could be established between electrical axis before delivery and cardiac electrical axis following delivery.

These findings show that there is right axis deviation in the foetus and in the neonate.

Figure 16

CONVERSION OF R/S RATIO TO ALGEBRAIC SUM OF R & S VALUES IN THE FOETAL ELECTROCARDIOGRAM

(versus R–S converted to a factor of 10)

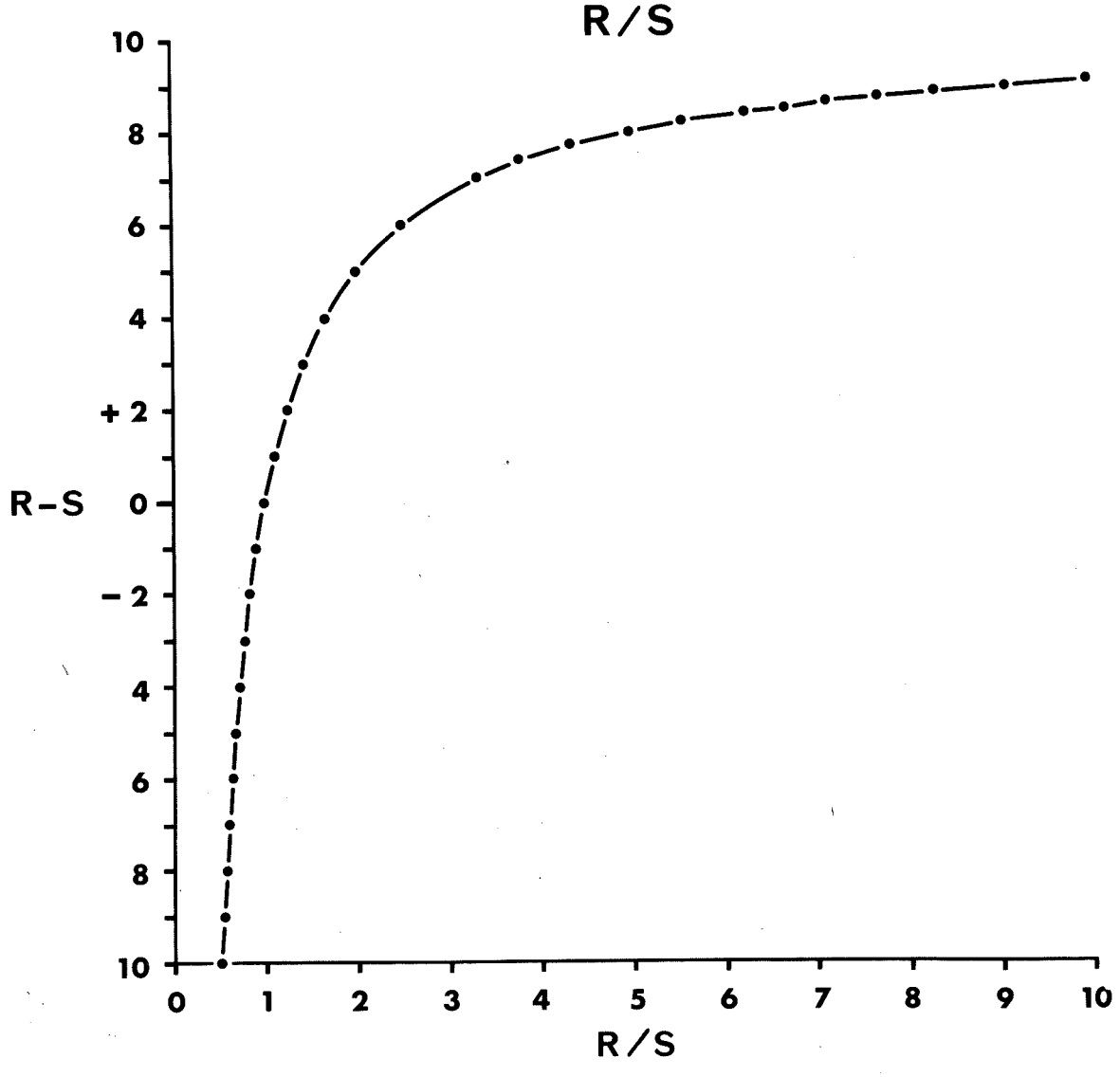


TABLE XXIX

GROUP MEAN VALUES OF THE FOETAL AND NEONATAL CARDIAC AXES

Variate	Number	Mean	Maximum	Minimum	S.D. (Mean)
Sample I Foetal ECG	99	144.1°	178°	94°	-
Sample II Foetal ECG	94	143.7°	178°	116°	-
Neonatal cardiac axis	92	131.62°	180.00°	30.00°	2.781

The Relationship Between R/S and Electrical Axis Values and Apgar Score

R/S values in sample 2 of the foetal electrocardiogram showed a significant negative linear relationship with the Apgar score at one minute ($r = -0.2655$, p 0.01) and five minutes ($r = -0.3094$, p 0.01). The fact that the correlation coefficient is higher with the five minute Apgar score than the one minute score suggests that this is a valid observation, and that a tendency towards left axis deviation is associated with neonatal depression immediately after delivery. R/S and calculated axis values were now considered in relation to Grouped Apgar data, and these results are shown in Table XXX.

TABLE XXX

APGAR SCORE VERSUS ELECTRICAL AXIS OF THE FOETUS AND NEONATE

Group	Axis 1			Axis 2			Axis 3		
	Number	Mean	S.E. (Mean)	Number	Mean	S.E. (Mean)	Number	Mean	S.E. (Mean)
A	9	131.1	8.407	8	130.9	8.024	9	123.11	23.71
B	5	154.4	11.28	5	135.8	10.15	6	137.5	29.04
C	3	132.0	14.56	3	134.0	13.10	3	118.0	41.07
D	28	140.75	54.76	24	143.3	4.63	25	108.8	14.23
E	54	147.8	3.43	54	147.08	3.09	55	117.07	9.59

	1 Minute	5 Minutes
Group A = Apgar	1-5	6-8
Group B = Apgar	1-5	9-10
Group C = Apgar	6-8	6-8
Group D = Apgar	6-8	9-10
Group E = Apgar	9-10	9-10

An analysis of these figures showed no significant difference in electrical axis between groups of Apgar scores.

Axis 1 = theoretical axis estimated on the first sample of the foetal electrocardiogram.

Axis 2 = theoretical axis estimated on the second sample of the foetal electrocardiogram shortly before delivery.

Axis 3 = neonatal cardiac axis.

Discussion

Measurements of the R and S waves of the foetal electrocardiogram provide a simple method of assessment of the electrocardiographic configuration which, when expressed as a ratio, is related to the electrical axis of the foetal heart.

On the basis of the resemblance of the foetal electrocardiogram (as obtained by scalp electrodes), to lead II of the neonatal electrocardiogram, electrical axis can be estimated. The mean values for foetal cardiac axis obtained in this study using recordings obtained with scalp electrodes are very similar to those obtained by Larks in 1965 using abdominal electrodes, the mean value in early labour in this study being $+144^{\circ}$ and late in labour $+143^{\circ}$, as compared with a mean value of 134° described by Larks. The foetus shows pronounced right axis deviation throughout labour. However, in this study, right axis deviation was less pronounced in those subjects with low Apgar scores at birth.

These findings may be explained by the fact that, when the myocardium is impaired by chronic asphyxial changes and glycogen depletion, cardiac dilatation occurs, with some counteracting left axis deviation. The mean value for cardiac electrical axis after delivery was $+131.6^{\circ}$, showing the typical right heart preponderance of the newborn infant.

Larks and Larks (1965) have suggested that the normal range of values extends from 100° to 160°, and that values outside of this range are associated with postmaturity, cord entanglement and pre-eclamptic toxaemia. In this study, 26 subjects showed foetal axis values outside of this range, but the incidence of toxaemia and post-maturity was no higher than in the whole group.

In conclusion, R/S values in the foetal electrocardiogram recorded shortly before delivery showed a high value in relationship to low Apgar scores at 1 minute and 5 minutes. Electrical axis values based on the similarity of the foetal electrocardiogram recorded using scalp leads to lead II of the neonatal electrocardiogram showed cardiac axis values that would be expected in the foetus. However, the measurement of foetal cardiac axis does not appear to provide a useful method of predicting foetal condition at birth.

SECTION 6. Foetal Cardiac Arrhythmias

Three subjects in this study showed cardiac arrhythmias of various types. The clinical details of these subjects are described in the Appendix of case reports (see case numbers 8, 55 and 79). Foetal acid-base values for these three subjects are shown in Table XXXI.

Two of these three subjects show significant acidosis in the scalp sample, and all three showed significant evidence of acidosis at the time of delivery.

The Apgar scores in these cases showed that two infants were seriously depressed at the time of birth and were still significantly depressed at the end of five minutes. The abnormal foetal electrocardiograms are shown in Figures 17, 18 and 19.

In the three cases, the arrhythmia consisted of nodal extra-systoles.

Transient changes occurred in the foetal electrocardiogram in Case 79 shortly before delivery. The changes consist of a series of rapid impulses and resemble the changes described by Whitfield in 1966. Whitfield concluded that these appearances were produced by summated action potential of a small number of contracting motor units in the rectus abdominis muscles. However, Whitfield obtained these recordings using abdominal electrodes, and the recording in this

TABLE XXXI

FOETAL CARDIAC ARRHYTHMIAS AND FOETAL ACID-BASE STATUS

Case No.	Scalp Blood			Cord arterial Blood			Cord Venous Blood		
	pH	pCO_2 mm Hg	S/HCO_3 meq/litre	pH	pCO_2 mm Hg	S/HCO_3 meq/litre	pH	pCO_2 mm Hg	S/HCO_3 meq/litre
8	7.24	48	17.5	7.11	60	14	-	-	-
55	7.20	25.5	17.5	7.16	55	16.5	7.28	34	17.0
79	7.30	39.5	18.8	7.11	68.5	14.4	7.27	40	18.2

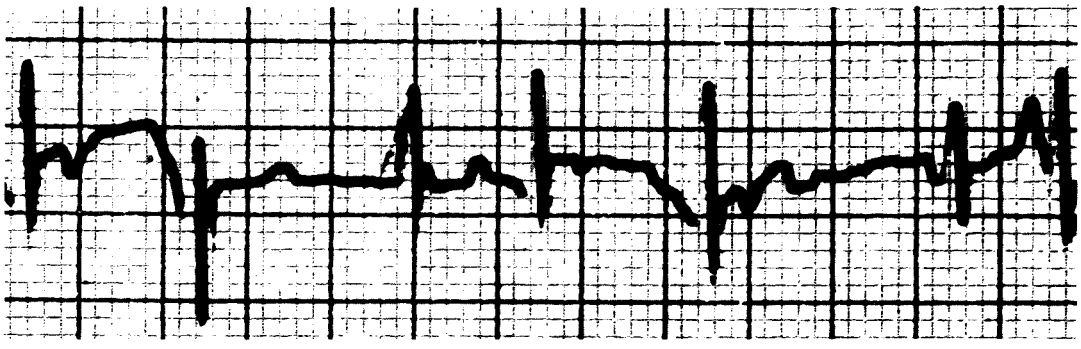
FIGURE 17

Case Number 8

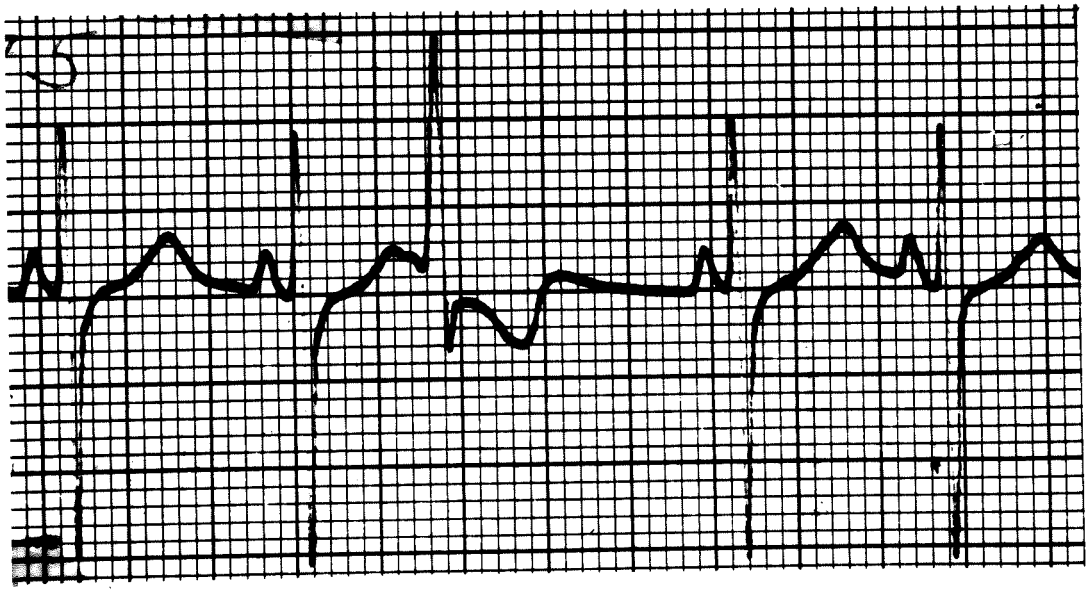
Variations in PR intervals and the size, shape and direction of P waves in the foetal electrocardiogram denote a wandering pacemaker. Missed beats occur, depending on the refractory state of the atria and ventricles. The same changes are seen in the neonatal electrocardiogram. Paper speed 2.5 cms/second.

FIGURE 17

Case Number 8.



Foetal E.C.G. Scalp lead.



Neonatal E.C.G. Lead V1. Recorded 5 hours after delivery.

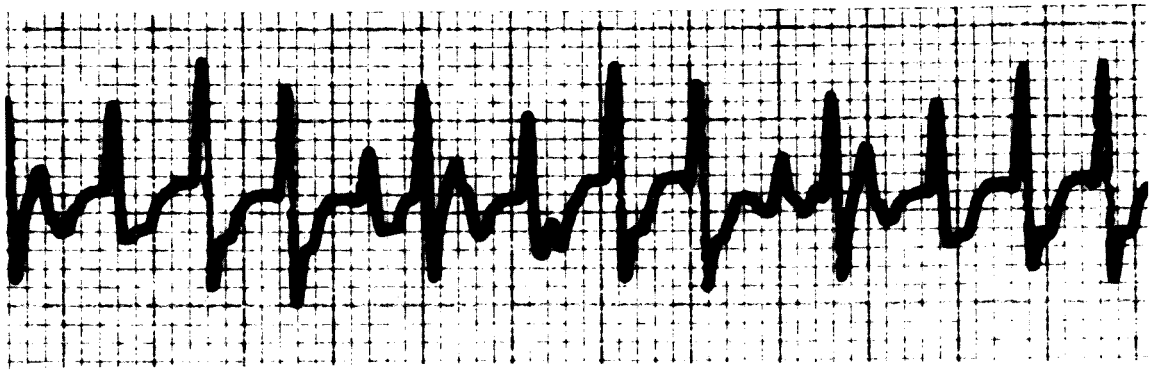
FIGURE 18

Case Number 55

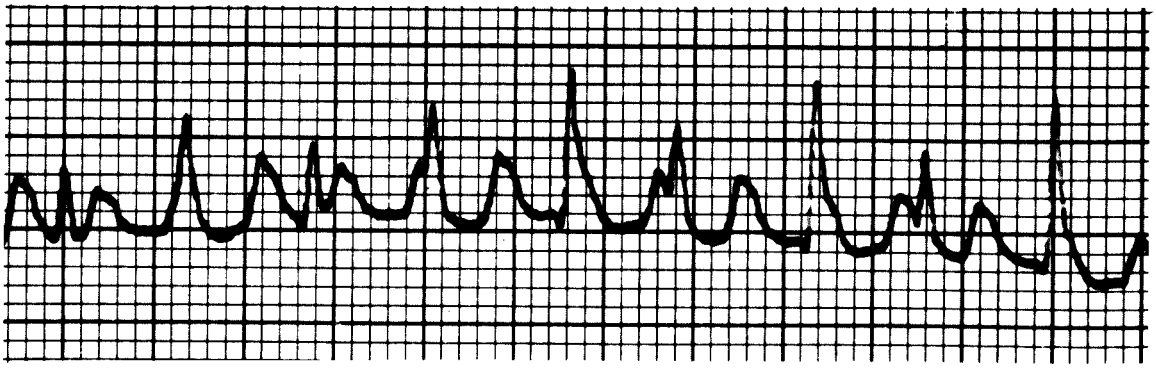
Nodal tachycardia with occasional impulses arising in the sino-auricular node. P waves can be seen to follow QRS complexes in some instances. Variations in P wave occurrence can also be seen in the neonatal electrocardiogram, but some of these complexes appear to be artefacts. Paper speed 2.5 cms/second.

FIGURE 18

Case Number 55.



Foetal E.C.G. Scalp lead.



Neonatal E.C.G. Lead II. Recorded 2 hours after delivery.

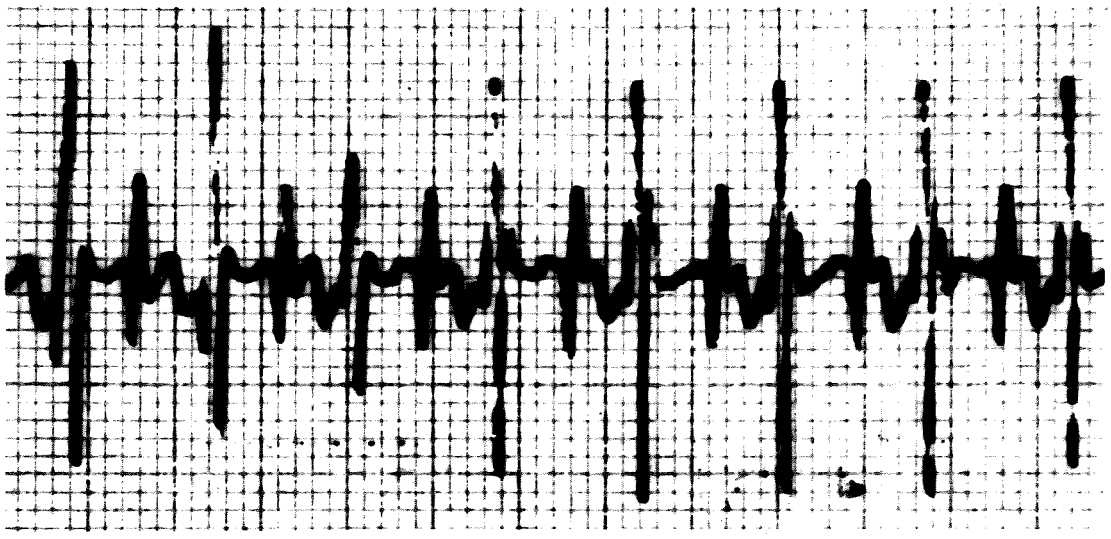
FIGURE 19

Case Number 79

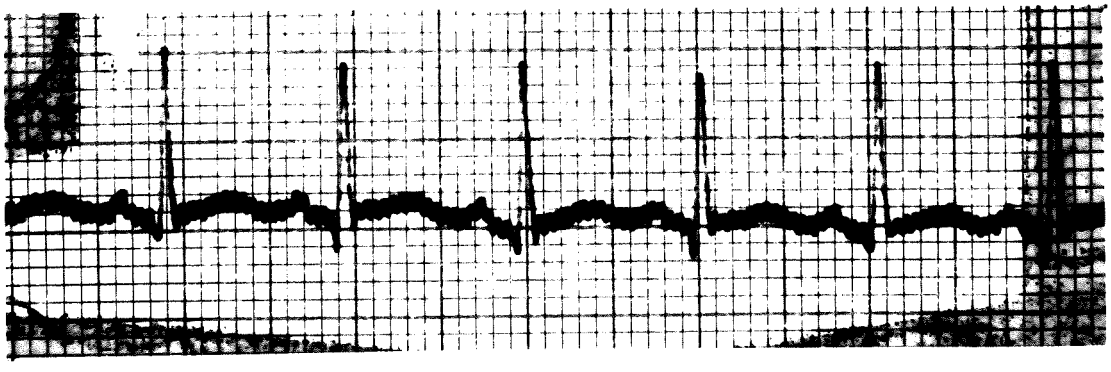
Foetal electrocardiogram shortly before delivery. The appearances are those of auricular flutter with a variable 2:1 3:1 block. The neonatal electrocardiogram (Lead III) four hours after delivery was normal. Paper speed 2.5 cms/second.

FIGURE 19

Case Number 79.



Foetal E.C.G. Scalp lead recorded shortly before delivery



Neonatal E.C.G. recorded 4 hours after delivery. Lead III.

instance was obtained from a vaginal electrode.

Discussion

The number of cases with cardiac arrhythmias in this study was too limited to draw any conclusions concerning the significance of foetal cardiac arrhythmia in general. However, in all instances, there was evidence of acidosis at some time during the labour.

Previous studies by Hon and Huang in 1962, and Kendall in 1967, reported much larger series of foetal cardiac arrhythmias, but these findings were not related to acid-base changes and it is therefore difficult to draw any final conclusions from this data.

Case 79 was of particular interest because of previous work on this subject. It seems likely that the abnormal foetal electrocardiogram in this subject was not due to the phenomenon described by Whitfield. There was certainly no evidence of the maternal electrocardiogram in this recording, and the voltage of the maternal electrocardiogram is considerably greater than the spike potentials produced by the abdominal musculature. Whitfield reported seven similar cases and, in three subjects, recordings were obtained simultaneously from vaginal and abdominal electrodes. In each instance, the abnormal tracing was not reproduced from the vaginal electrode. In this present case, it is

therefore suggested that this recording is not artefactual and shows evidence of auricular flutter, with a variable 2:1, 3:1 block.

Case number 8 showed evidence of a wandering pacemaker which was present throughout labour, and persisted after delivery. This infant clearly showed evidence of asphyxial changes in the biochemical findings.

Case number 55 showed gross changes in the foetal electrocardiogram consistent with nodal tachycardia. The appearances of the neonatal electrocardiogram following delivery were difficult to interpret, but variations in P wave occurrence were still seen. Some of the bizarre complexes seen appear to be artefacts. These changes are occasionally seen in the electrocardiogram of apparently normal children (personal communication, Dr. E. Goldblatt, Paediatric Cardiologist, Adelaide Children's Hospital), and their aetiology remains obscure. This infant was also grossly acidotic and showed obvious signs of cardiac failure following delivery, which responded to digitalis therapy.

CHAPTER VI

CONCLUSIONS

The configuration and time constant of the foetal electrocardiogram have been the subject of several studies. However, most of the studies have suffered from the disadvantage that the changes in the foetal electrocardiogram have not been related to specific biochemical changes. Much of the data presented in these studies has therefore been of limited value and, in many instances, the conclusions have been frankly speculative. At the same time, intensive investigation into the relationship of foetal heart rate changes to acid base values has been undertaken since scalp blood sampling techniques have become available, and it is in this area that the most clearly documented factual information is now available.

It was for these reasons that this present investigation was undertaken on the configuration and time constants of the foetal electrocardiogram, and not on the patterns of heart rate change.

This study was undertaken to answer the following questions:

1. What are the characteristics of configuration and time constants in the normal foetal electrocardio-

gram as defined by a normal range of acid base values?

2. Does a consistent pattern of change emerge in the foetal electrocardiogram through a known range of acid-base values with the spectrum covering normal and acidotic infants?
3. If specific changes do occur, are they the direct result of acid-base changes or are they a reflection of electrolyte alterations produced by prolonged acidosis?
4. Can the changes that occur in the foetal electrocardiogram be used as a useful and practical guide to the subsequent outcome for the foetus?

The evidence that chronic asphyxia results in metabolic acidosis in the foetus has been extensively reviewed by Saling (1964), and at present this evidence appears to be unassailable. Anaerobic metabolism leads to the accumulation of lactic acid as a result of anaerobic glycolysis, and hence to metabolic acidosis.

In reviewing the significance of foetal acidosis in 1959, Kaiser concluded that "in all likelihood, it is a finding much like an elevated white blood cell count. It may be entirely incidental and without pathological significance, although this is distinctly unusual. It may be

the response to an exogenous stimulus such as ammonium chloride, and well within the adaptive capacities of the newborn. Finally, it may reflect a serious morbid state. Only an estimation of the clinical state in which the lowered foetal pH is found, and a determination of the other variables which are inextricably related to it in respiratory chemistry, can make possible a proper evaluation of its significance."

Since 1959, considerable evidence has been accumulated that the clinical condition of the infant at birth is related both in a retrospective sense (as assessed by cord blood estimations), and in a prospective sense (as assessed by scalp capillary blood) to the acid base status of the foetus. However, the final assessment of these factors can only be determined by consideration of the subsequent intellectual development of the child many years later.

Examination of the infant six weeks after delivery in this study did not add to information obtained within the first twenty-four hours of delivery. This may be explained by the relative crudity of the methods of clinical assessment at this age, but it may also be due to the great difficulty in detecting subtle neurological changes in a six-week old infant. For this reason, long-term follow-up studies are being undertaken on these children in relation to their subsequent intellectual development and social

maturity. This material is not the subject of this thesis.

The relation of Apgar score to foetal scalp blood pH has been the subject of several studies. Beard, Morris and Clayton (1967) studied 250 patients with clinical signs of foetal distress where scalp blood samples were obtained within 30 minutes of delivery, and related to Apgar scores recorded two minutes after delivery. A foetal pH of less than 7.15 was associated with a score of 6 or less in 80 per cent of cases, whereas a pH more than 7.25 was associated with an Apgar score of 7 - 10. Values between 7.15 and 7.25 were not reliable in predicting the condition of the infant. Wood, Lumley and Renou (1967) studied 31 cases in whom the Apgar score was 0-3 at birth, and showed that the foetal blood pH and heart rate were usually abnormal in the second stage of labour, but changes in the first stage of labour were less common. Abnormalities of the foetal heart rate nearly always preceded a significant fall of foetal blood pH.

Hon, Khazin and Paul (1967) studied 194 patients from whom a total of 1392 foetal scalp blood samples were obtained at various times during labour and related these to Apgar scores at 1 and 5 minutes. Samples were obtained, both between and during uterine contractions. These authors concluded that "while foetal pH correlates in a general way with Apgar scores, there is considerable overlap between pH values

for high and low score babies." The timing of the sampling, both in relation to uterine contractions and subsequent delivery, was also important in relating the scalp pH to subsequent Apgar scores.

In this present study, the time of sampling varied from many hours to half an hour before delivery, and all samples were collected between uterine contractions. In view of the variability in sample delivery time, it was therefore of considerable interest to note that, although no significant relationship was established between scalp blood pH and Apgar score as assessed between the first and second minute after birth, a highly significant linear relationship was demonstrated between the Apgar score at 5 minutes and scalp blood pH ($r = +0.4113$ $p < 0.001$). The possibility of observer bias is obviated by the fact that assessment at 24 hours by an independent observer also showed a significant relationship between scalp blood pH and assessment of the central nervous system ($r = +0.3355$ $p < 0.01$). *Furthermore, scalp blood pH was shown to have a significant positive linear relationship with cord arterial blood pH* ($r = +0.38$ $p < 0.01$) *and cord arterial blood pH showed a significant relationship with Apgar score at 1 minute* ($r = +0.3045$ $p < 0.01$), *Apgar score at 5 minutes* ($r = +0.2909$ $p < 0.01$) *and the central nervous system at 24 hours* ($r = +0.2596$ $p < 0.05$).

The use of standard bicarbonate estimations in this study was chosen for a variety of reasons. Astrup, Jorgensen, Siggaard, Andersen and Engel (1960), in describing their method for estimation of plasma standard bicarbonate, emphasised that standard bicarbonate values have, like any other bicarbonate values, the drawback that it does not show directly the amount of fixed acid or base causing a change in the base content of a blood sample. This is because the carbon dioxide/bicarbonate system is responsible for only about 75 per cent of the buffer action of blood against fixed acids and bases when the pCO_2 is kept constant. Buffer base values could not be reliably estimated, as the haemoglobin values could not always be correctly estimated on scalp blood samples.

A serious criticism of Astrup's work has been advanced by Brackett, Cohen and Schwartz (1965), when these authors showed that CO_2 titration curves in vivo differ from values obtained in vitro. As these findings appear to affect standard bicarbonate estimations principally at very high levels of pCO_2, it was felt that, in this study, standard bicarbonate was still the best and simplest value to use for assessment of the non-respiratory or metabolic component of foetal acidosis within the range of partial carbon dioxide pressure found in the majority of foetal samples. Saling and Schneider (1967) assessed the metabolic component of

foetal acidosis by measuring the pH of foetal blood with carbon dioxide at a tension of 40 mm Hg and claimed that the values for "pH qu 40" had the same significance as standard bicarbonate. In a study of 850 labours complicated by clinical signs of foetal distress, metabolic alterations of foetal acid base balance occurred in 122 cases - an incidence of 14.4 per cent. These cases were selected from a total of 4396 deliveries and the overall incidence of foetal metabolic acidosis in this series was 2.8 per cent. Perinatal death occurred in 13 of 21 babies showing severe metabolic acidosis.

These findings have some relevance to the present study, as it was important to select a group of subjects likely to provide a wide spectrum of acid-base values in order to observe potential alteration in the time constants and configuration of the foetal electrocardiogram.

The reliance for many years on clinical signs of variation in foetal heart rate inevitably led to dissatisfaction in these methods for assessing foetal prognosis in relation to foetal hypoxia. This was hardly surprising, in view of the number of possible factors that influence foetal heart rate. With the introduction of continuous electronic monitoring of the foetal heart beat, it became apparent that certain patterns of heart rate change in relation to uterine contractions were associated with depression of the newborn

infant at birth.

Hon and Lee (1963) described patterns of prolonged and profound bradycardia following uterine contractions preceded by tachycardia in five cases of foetal death. Typical patterns of foetal bradycardia due to skull compression and cord compression were described by Hon and his co-authors (1961, 1962, 1959), and these patterns were also described by Caldeyro-Barcia et al. (1966).

In an attempt to obtain a better "end-point" for assessing foetal hypoxia, Brady and James (1962) and later Quilligan and his co-workers (1964 and 1965a and b) measured acid base and lactate and pyruvate values in the cord blood and related this to heart rate changes. Heart rate patterns of bradycardia towards the end of uterine contractions, and sometimes during contractions, were shown in relation to foetal acidosis, these findings thus tending to confirm the earlier observations of Hon.

With the availability of scalp blood sampling techniques, the relationship between foetal acid-base status and foetal heart rate changes was further clarified by Mendez-Bauer et al. and Wood et al., in 1967. These workers both showed that the most significant changes occurring in the asphyxiated infant were persistent foetal tachycardia followed by bradycardia occurring at the end of uterine

contractions (i.e. "type II" dips in heart rate). It has been suggested by Mendez-Bauer et al. (1967) that the patterns of foetal heart rate change are adaptive mechanisms resulting from increased sympathetic tone with increased cardiac rate and output associated with the redistribution of foetal circulation. The reduction in foetal heart rate after uterine contractions was described as an "energy-saving" mechanism aimed at reducing cardiac output at a time when a low yield in foetomaternal exchange would be likely to occur.

This hypothesis, even accepting its frankly teleological nature, is based on the assumption that the myocardium itself is functioning normally in the foetus by the time a state of chronic metabolic acidosis has occurred. Evidence that the cells of the conduction system and the myocardium are affected can only be obtained from a study of the configuration and time constants of the foetal electrocardiogram.

Experimental studies on the foetal electrocardiogram after removal of the foetus from the uterus, as performed by Enhorning and Westin on previable foetuses (1964), can have little relevance to the alterations in the foetal electrocardiogram under conditions of chronic asphyxia in the uterus.

Once the foeto-placental circulation is interrupted the situation becomes simplified, in that the potential for electrolyte changes induced by disparity of the acid base status of mother and foetus to exert an influence on the foetal myocardium is lost.

The first comprehensive study of the foetal electrocardiogram in relation to clinical signs of foetal distress and arterial oxygen saturation at birth was reported by Southern in 1957. In this study, it was shown that low oxygen saturations at birth were associated with prolongation of PR intervals, the QRS duration and S-T segment duration. T waves were also noted to be isoelectric or inverted in 35% of the anoxic cases. The S-T segment was lengthened also at comparable heart rates.

Southern stated that "the electrocardiographic abnormalities reflect the myocardial ischaemia and disturbance in the action potential induced by the anoxic state but, without further evidence, this can be held only as a theoretical postulate and therefore cannot be interpreted in terms of pathology." Gruenwald (1949) demonstrated coronary lesions in the anoxic stillborn infant resulting in damage to the media and amounting, in severe cases to complete destruction. Necrotic changes in the coronary vessels were shown to occur seven times as frequently in infants with asphyxial

haemorrhages in the epicardium as in those without such haemorrhages.

Hon and Lee (1963) described the foetal electrocardiogram from nine foetuses that died during or shortly after parturition. Four of these foetuses were anencephalic and two were premature. The changes described consisted of high peaked, biphasic or inverted P waves and shortened P-R intervals. Wandering pacemaker, false bundle-branch block, widened QRS complexes, and S-T segment depression were also noted. In a subsequent publication in 1965, Lee and Hon showed that changes in the foetal QRS complex were not necessarily indicative of asphyxia in the foetus.

Kendall et al. (1964) reviewed 400 cases of clinical foetal distress monitored from abdominal leads using the foetal radioelectrocardiogram. The characteristics of the abnormal foetal electrocardiogram as related to foetal depression at birth were prolongation of the S-T segment associated with notching and depression, and prolongation of the QRS complex with notching. Larks and Longo (1962) also suggested that morphologic changes in the QRS complex and S-T segment depression were "unfavourable omens" for the foetus.

Studies on cardiac arrhythmias in the foetus by Kendall (1967) showed that arrhythmias do not necessarily indicate

foetal asphyxia. Komaromy et al. (1967) studied five foetuses with cardiac arrythmias and noted that none of these cases showed evidence of acidosis in foetal scalp blood samples.

With the exception of the excellent study by Southern, most of the studies on foetal electrocardiographic configuration are difficult to evaluate, as they are not related to biochemical changes in the foetus. Conclusions drawn concerning those changes in the electrocardiogram in relation to the condition of the foetus at birth are of limited value because of the various causes of depression of the neonate. The changes described by Hon and Lee preceding foetal death represent the terminal events of anoxic damage to the foetus, but provide no evidence as to the nature of changes which occur with less advanced asphyxial changes.

Despite the difficulties with previous studies, the general pattern of changes that has emerged is particularly related to changes in ST or QT segments and T wave configuration. *This present study has shown clearly that foetal acidosis is related to prolongation of the QT interval, a change which cannot be accounted for by alterations in foetal heart rate. These changes can be seen during labour, as assessed by scalp blood samples, although the differences do not achieve significance at this stage. However, the same pattern emerges clearly at the time of*

delivery in studies on cord venous blood acid-base measurements.

It was of considerable importance to note that the same pattern of changes emerged in relation to hyperkalaemia. In fact, these changes were more significant in relation to potassium studies, and it was only in these studies that T wave changes - flattening of the T wave and T wave inversion - emerged as a significant factor. Unfortunately, it was not possible to perform the same electrolyte studies on scalp blood samples because of sample quantity, but it is reasonable to assume that the same pattern would emerge.

The evidence of Gruenwald, that damage occurs to the coronary vessels in asphyxial deaths, represents gross terminal changes in the foetal heart and does not necessarily explain the mechanism by which electrocardiographic changes occur. It is suggested, on the evidence presented in this study, that these changes may be explained by electrolyte alterations rather than direct asphyxial damage to the cells. This concept is substantiated by studies such as those of Niswander et al. (1966), who showed that anoxia resulting from cord prolapse caused depression at birth, but there was no greater risk of neurologic damage at one year than in control infants. Whilst there may be fallacies in extrapolating from this type of data, it is a

generally valid observation that acutely asphyxiated infants subject to acute acid-base changes of considerable severity recover and thrive better than those infants subjected to more chronic changes induced by conditions of chronic placental failure. These differences in behaviour may be explained by a variety of biochemical changes, and the present study suggests that alterations in potassium balance may be a significant factor.

The rise in plasma potassium values in relation to asphyxia has been established in experimental studies such as those described by Dawes (1968). A significant linear relationship was demonstrated in this study between standard bicarbonate values and plasma potassium levels in both cord arterial and cord venous blood. The suggestion previously discussed, that persistent hyperkalaemia in the foetus may lead to intracellular potassium depletion can only be confirmed by measurements of total body potassium and red cell potassium in infants known to have been subjected to prolonged asphyxia. However, the evidence available in this study has shown that prolongation of the QT interval is related to both increasing acidosis and hyperkalaemia, and that these changes emerge more significantly in potassium studies. Furthermore, the only significant changes to occur in relation to T wave configuration were shown in relation to plasma potassium

values. However, it must be emaphsised that calcium and magnesium levels have not been measured in this study, and these ions may also show significant variations in relation to ECG configuration.

Hypokalaemia has been shown to develop in neonates subjected to prolonged asphyxia "in utero" when the metabolic acidosis is corrected following delivery (personal communication, Dr. W.H. Tooley, Associate Professor of Pediatrics, University of San Francisco), and this suggests that cellular potassium depletion has occurred in these infants.

No significant changes were shown in QRS configuration or in PR intervals in acidotic infants, with the one exception that a weakly significant ($p < 0.05$) prolongation of PR intervals was seen in the second sample of the foetal ECG in relation to foetal acidosis as measured by low standard bicarbonate values in cord venous blood.

Definition of the time constants in the foetal electrocardiogram has been the aim of several studies, and these have been summarised by Figueroa-Longo et al. (1966). With the exception of the work of Southern, these studies have not been related to specific biochemical changes. Figueroa-Longo et al. used a direct multiple electrode system. Electrodes were placed directly into foetal buttocks and

scalp and a large number of complexes were analysed. The mean time for QT intervals was 248.4 ± 8.75 milliseconds. The values for the different ECG components were shown to be 60 to 80 per cent of adult values. With the exception of QT intervals, which were shown to be shorter in the foetus than in the neonate, the values were shown to be almost identical to those of the newborn.

A comparison of the values obtained in this study in the presence of known normal acid-base values is shown in Table XXXII.

TABLE XXXII

DURATION OF THE ECG COMPONENTS OF THE FOETAL HEART
(Milliseconds)

	Present series	Figueroa-Longo et al	Kaplan & Toyama and Sureau	Southern
PR interval	109	104	80 - 130	60
QRS complex	29	65	34 - 52	20
QT interval	233	248	200 - 276	-
Corrected QT interval	350	-	-	-

The values for P-R intervals and QT intervals in this study are very similar to those defined by Figueroa-Longo, but it is apparent that very wide ranges of QRS values occurred in various series, with the present series most

closely resembling those described by Southern. The immediate reason for this is not apparent, although the intervals being measured are short, and precise measurement is difficult unless high paper speeds are used.

However, the evidence in this study shows that QT prolongation in excess of 0.3 second is abnormal and indicates acidosis or hyperkalaemia. QT values greater than 0.26 second, or 0.4 second when corrected for rate, should be considered as abnormal.

Studies on vectorcardiography based on the concept suggested by Larks have shown a weak but significant correlation between the configuration of the foetal electrocardiogram and lead II of the neonatal electrocardiogram. A similar relationship was shown with lead II and lead V6 of the neonatal ECG. The R/S ratios related poorly to foetal condition at birth, and when these values were converted to electrical axis, on the assumption that the scalp lead relates to lead II of the neonatal ECG, no further significant information was obtained. However, the mean electrical axis values obtained by using these methods were very similar to those obtained by Larks, using abdominal electrodes. Larks' method for calculating

foetal cardiac axis appears to bevalid with tracings from scalp electrodes.

Analysis of the foetal electrocardiogram has been shown in this study to provide useful information concerning the acid-base and electrolyte values in the foetus. The potential importance of potassium balance in producing these electrocardiographic changes has been emphasised.

The changes in the electrocardiogram induced by metabolic acidosis have been shown to be present in early labour, but generally appear to be more obvious at the time of delivery.

The limitations for clinical application of these findings are the overlap of values in normal and abnormal cases, and the practical difficulties of obtaining good tracings suitable for adequate interpretation.

In conclusion, the findings of this study are briefly summarised by answering the four questions asked at the beginning of this chapter.

1. *The characteristics of the foetal electrocardiogram have been defined within known normal ranges of acid-base status during labour.*
2. *A subtle pattern of change does emerge in the time constants of the foetal electrocardiogram with some evidence of prolongation of PR intervals in relation to metabolic*

acidosis as assessed by cord venous plasma standard bicarbonate, and prolongation of QT intervals occurred in relation to low pH values in cord venous blood.

3. The specific changes that occur in the foetal ECG in the presence of foetal acidosis are related at a higher level of significance to hyperkalaemia. T wave depression also occurs in the presence of hyperkalaemia. Whilst the changes in the foetal electrocardiogram are those usually described in the presence of hypokalaemia, it is suggested that prolonged hyperkalaemia in the acidotic foetus may lead to intracellular potassium depletion by the loss of potassium across the placenta, and hence, the changes in the foetal ECG are those of hypokalaemia. In other words, there is evidence that the changes in the foetal electrocardiogram in the presence of metabolic acidosis may be a result of electrolyte changes secondary to chronic foetal asphyxia.

4. The changes that occur in the foetal electrocardiogram show evidence of foetal acidosis but these changes do not constitute a practical guide to foetal condition as there is too great an overlap between normal and abnormal values. Left axis deviation relative to the normal right axis deviation in the foetus has been shown to be related to foetal depression. However, biochemical measurements of foetal acid-base status show significant prognostic value in relation to foetal

condition at birth and for 24 hours after delivery in a pattern which emerges more clearly than seen in the extensive analysis of electrocardiographic data.

CODE TO THE APPENDICES

Complications (1-10):

1. Mild toxaemia - blood pressure raised without proteinuria.
2. Moderate to severe toxaemia - blood pressure raised with proteinuria.
3. Essential hypertension.
4. Essential hypertension + toxaemia.
5. Antepartum haemorrhage.
6. Previous history of stillbirths.
7. Previous history of neonatal deaths.
8. Previous miscarriage.
9. Rhesus isoimmunisation.
10. Suspected placental insufficiency.

Clinical Signs of Foetal Distress:

1. None.
2. Meconium stained liquor.
3. Foetal tachycardia.
4. Foetal bradycardia.
5. Foetal heart irregularity.
6. Foetal heart difficult to hear.
7. Excessive foetal movements.

Method of Delivery:

1. Normal.
2. Forceps.
3. Caesarean section.

Apgar Score (at 1 minute and at 5 minutes):

1. 0-5
2. 6-8
3. 9-10

Neonatal Assessment:

CNS (Central nervous system):	3 = normal
	2 = mild cerebral disorder, e.g. hypertonic, increased irritability
	1 = signs of severe cerebral impairment
RS (Repiratory system):	3 = normal
	2 = mild respiratory distress
	1 = severe respiratory distress
GC (General condition):	3 = normal
	2 = fair
	1 = poor
SQ (Social quotient):	3 = responds well to people and voices
	2 = limited response
	1 = mask-like - no response

E.C.G.:

The intervals of PR, RR, QRS, QT and QT_c are all measured in seconds. R/S values are numerical expressions of the ratio of positive to negative deflections on the ECG complex.

ST segment:	1 = normal
	2 = depressed
	3 = elevated
T waves	1 = flat
	2 = upright
	3 = inverted

Electrical axis - this is expressed in degrees and is positive unless specifically preceded by a negative sign.

R/S ratios. Appendix 6 includes the ratio of positive to negative deflections in the 9 neonatal E.C.G. leads.
10 = all positive. 0.1 = all negative.

Abbreviations in Appendices

pCO_2 = partial pressure of carbon dioxide (mms of mercury).
S.B. = standard bicarbonate - milliequivalents/litre.
Na^+, K^+ and Cl values are expressed in milliequivalents/litre.
G = blood glucose (mgms per 100 ml).
L.M.P. = First day of last menstrual period.
E.D.C. = estimated date of confinement.

APPENDIX 1

MATERNAL ACID-BASE STATUS AND PLASMA ELECTROLYTE VALUES

Case No.	Maternal Venous Blood Before Delivery							Maternal Venous Blood After Delivery						
	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
1	7.35	45	23	-	-	-	-	7.39	42	24	-	-	-	-
2								7.45	28	20	-	-	-	-
3								7.35	27	16.8	-	-	-	-
4	7.35	35.5	20.2	-	-	-	-	7.36	42	22.5	-	-	-	-
5	7.52	27	23.2	-	-	-	-	7.40	32	21	-	-	-	-
6	7.40	32	21	-	-	-	-	7.39	32	20.5	-	-	-	-
7	7.40	37	22.3	-	-	-	-	7.39	32	21	-	-	-	-
8	7.34	30	18	-	-	-	-	7.35	32.5	18.7	-	-	-	-
9	7.39	35.5	21.7	-	-	-	-	7.47	14	19.5	-	-	-	-
10								7.33	32	18	-	-	-	-
11														
12	7.38	35.5	21.5	-	-	-	-	7.25	36	16	-	-	-	-
13	7.42	30	21	-	-	-	-	7.36	27	17.5	-	-	-	-
	7.44	22	20	-	-	-	-							
14	7.37	35.5	20.6	-	-	-	-	7.33	32	17.7	-	-	-	-
15	7.42	28.5	20	-	-	-	-							
16	7.40	35.5	22.7	-	-	-	-	7.38	31	20	-	-	-	-
	7.42	38	24	-	-	-	-							
17								7.34	16	16.5	-	-	-	-
18								7.25	43	17.5	-	-	-	-

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
19	7.41	26.5	20	136	3.4	-	-	7.37	31.5	19	130	4.5	-	-
20	7.42	28.5	20.5	158	3.9	-	-	7.41	29	20.2	136	4.7	-	-
21	7.42	29.5	21	-	-	-	-	7.34	24	19	-	-	-	-
22	7.32	37	19	-	-	-	-	7.32	33	17.6	-	-	-	-
	7.37	33.5	20	-	-	-	-							
23	7.39	30	20	-	-	-	-	7.28	33.5	16.5	-	-	-	-
24	7.35	35	19.5	-	-	-	-	7.32	25.5	16.2	-	-	-	-
25	7.4	36	22.4	138	4.0	114	-	7.37	28	21	-	-	-	-
26	7.39	30	19.7	140	4.5	110	-	7.32	26	16.3	152	3.6	96	-
27	7.36	34	19.5	-	-	-	-	7.31	36	18	140	3.2	-	-
28	7.45	25	20.5	131	3.4	98	-	7.38	25	18	91	3.3	92	-
29	7.40	33	22	134	3.85	96	-	7.36	31	18.5	137	4.4	92	-
30	7.34	24	16.5	-	-	-	-	7.31	28	16.5	-	-	-	-
31	7.38	30	19.8	130	4.05	87	-	7.34	28	17.5	148	4.45	94	-
32	7.39	28.5	20	153	4.3	113	-	7.37	31	20	148	4.1	112	-
33	7.47	32	25	128	3.75	110	-	7.36	32	19	122	3.65	123	-
34	7.40	32	21	156	4.65	106	-	7.32	31.5	17.5	192	3.85	112	93
35	7.35	30	18.7	125	5.0	109	32	7.24	27	14.7	120	5.4	112	101
36	7.41	29	19.5	-	-	100	120	7.26	25.5	14.0	144	4.2	98	95
37	7.31	38.5	19.0	130	3.7	109	207	7.30	28.5	16.3	142	3.5	104	215
38	7.53	18	20.0	126	4.5	112	79	7.37	24.5	17.0	176	4.6	98	55
39	7.45	32.5	23.5	162	3.8	115	90	7.24	54	21.5	111	2.5	108	81
40	7.39	35	21.2	120	4.2	115	63	7.32	37	19.0	124	3.4	98	94
41	7.39	35	21.5	-	-	-	-	-	-	-	-	-	-	-

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
42	7.42	35	22.8	175	6.1	114	36	7.27	35	16.4	154	3.8	101	40
43	7.42	34	22.2	138	4.4	103	102	7.37	29	19.0	137	3.8	102	57
44	7.37	39.0	22.0	148	3.9	100	64	7.38	37	21.5	132	3.6	96	80
	7.39	41.0	24	133	3.8	98	80							
	7.39	37.5	22.9	-	-	-	-							
	7.44	33.0	18.0	-	-	-	-							
46	7.42	28.5	20.5	138	3.6	106	-	7.37	28	17.8	135	3.4	94	-
47	7.41	31	21.0	136	4.2	100	65	7.38	32	19.5	149	4.1	96	74
48	7.47	28	23.2	131	3.1	96	222	7.44	34	23.5	129	2.7	95	120
	7.545	17	24.5	131	3.2	97	93							
	7.47	28	24.0	132	2.8	80	73							
49	7.39	32	20.5	139	3.9	87	100	7.41	46	20.2	135	3.2	85	88
	7.33	25.5	16.7	-	-	0	-							
	7.48	34	25.5	-	-	-	-							
45	7.36	34	20	132	3.6	103	85	7.35	32	18.7	133	3.4	104	57
50	7.35	41	21.6	143	3.9	100	113	7.36	42	22.5	158	3.4	104	75
51	7.47	27	22.0	133	4.2	98	96	7.37	31	18.8	126	4.0	94	92
	7.40	28	20.0	-	-	-	-							
52	7.44	30	21.5	133	3.5	101	97	7.30	31	16.6	135	3.1	104	120
53	7.48	24.5	21.0	154	4.5	102	45	7.385	34	21.0	147	3.3	-	38
54	7.38	41	23.0	133	3.4	94	70	7.40	29.5	19.8	126	3.35	88	157
55	7.43	25.5	20.0	137	3.35	99	70	7.36	31	18.0	132	3.6	110	87
56	7.48	25.0	22.0	133	3.6	113	75	7.43	35	18.0	133	3.2	98	95
	7.43	24.0	25.0	112	2.7	115	87							
57	7.42	25.0	20.3	136	4.30	100	57	7.32	33	18.0	132	3.4	98	67
58	7.41	39.0	25.0	135	3.6	100	87	7.34	30.5	17.8	121	3.6	93	147

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
59	7.34	30	17.8	131	4.3	104	57	7.41	31	20.5	119	4.5	104	91
60								7.34	36.5	19.9	132	3.9	114	50
61	7.40	32.5	21.1	138	5.0	114	40	7.35	31	18.4	134	3.6	112	72
	7.40	35.2	21.9	132	3.9	114	-							
62	7.41	35	22.5	136	4.2	100	49	7.285	35	16.8	129	3.9	100	84
63	7.40	29	20.5	133	4.1	108	48	7.28	42.5	18.7	129	3.9	104	75
64	7.42	37.5	24	132	3.4	109	57	7.38	36	21.5	131	3.6	94	106
65	7.42	31.5	21.2	135	4.3	102	68	7.37	30.5	18.7	129	4.2	116	128
66	7.43	32	22.1	129	3.8	120	93	7.37	28	19	126	3.5	104	167
67	7.43	30	21.3	132	3.9	97	59	7.31	40	19.4	129	3.9	98	73
68	7.49	28.5	23.7	122	2.6	106	245	7.36	34	20	128	2.8	102	93
69	7.40	28.5	19.5	121	2.8	130	57	7.29	28.5	15.5	123	3.1	124	93
70	7.42	33.5	21.9	127	3.5	110	50	7.41	29	20.5	125	3.7	115	58
71	7.35	36.5	20	123	4.0	100	200	7.34	40	21	117	3.9	100	75
72	7.40	35	21.7	128	3.4	116	75	7.38	29.5	19.4	127	3.4	102	83
73	7.45	26.5	21	126	3.6	104	72	7.25	36	16.0	119	3.6	100	155
74	7.42	30.5	22	127	3.65	90	87	7.35	33	19.2	125	3.5	92	95
75	7.40	37	22.7	126	3.6	104	64	7.34	38.5	20.5	124	3.5	98	100
76	7.5	22.5	20.5	121	3.3	100	65	7.40	32	20.8	119	3.2	100	118
77	7.37	32	19.5	129	3.8	100	87	7.28	32.5	16.2	128	4.3	106	65
78	7.46	27.5	21.7	118	3.6	104	73	7.35	27.5	17.5	126	3	95	185
79	7.42	31.5	21.8	133	4	92	63	7.33	36.5	19.5	131	4.2	92	97
80	7.39	37	22.2	130	3.6	136	90	7.34	34.5	19.5	129	3.9	94	97
81	7.4	33.5	21.5	133	3.4	115	72	7.31	24	17	132	3.3	120	100

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
82	7.38	34.9	21.5	134	3.7	96	75	7.29	35	17.6	131	4.4	94	75
83	7.39	36	21.5	133	4.3	91	50	7.37	31	19	131	4.3	98	72
84	7.4	32.5	20.9	133	4.2	110	52	7.33	32	18	130	4.2	90	88
85	7.38	30.5	19.1	130	2.8	96	125	7.35	32.5	18.5	120	2.8	91	95
86	7.42	33	22	133	4.4	97	35	7.44	25.5	19.5	125	4.1	92	38
87	7.41	28	20.3	131	4.9	94	71	7.38	29.5	19.4	132	3.8	92	93
88	7.39	28.5	19.2	131	4.2	102	115	7.33	30.5	17.3	126	3.0	100	197
89	7.43	36.0	23.2	129	3.4	120	62	7.41	-	-	128	3.4	106	62
90	7.3	26.5	22.8	128	3.6	105	102							
91	7.39	33.5	20.9	133	3.7	96	80	7.39	27.5	19.0	131	3.3	92	93
92	7.43	31.5	22	128	3.6	92	50	7.38	34	20.8	122	4.4	100	75
93	7.39	32	20.3	130	3.9	91	40	7.34	37	20	131	3.7	100	45
94	7.44	35	21	132	3.7	106	83	7.3	37	18	122	3.8	95	37
95	7.40	35	22	136	3.7	92	43	7.3	38	18	131	3.4	96	55
96	7.40	37	23	138	3.2	106	21	7.36	37.5	21	126	3.6	100	57
97	7.39	31	20	130	4.0	100	43	7.31	38	17	129	5.3	96	35
98	7.4	34.5	21.7	131	3.9	98	40	7.33	34.5	18.7	130	4.6	86	67
99	7.46	26.5	21.0	130	3.8	102	50	7.37	39	18.5	130	3.4	96	47
100	7.42	31	21.6	130	3.7	90	63	7.33	36.5	19.5	126	4.1	102	175
101	7.38	32	19.8	130	4.2	105	32	7.33	31.5	17.9	129	3.7	86	50
102	7.35	37.5	20.5	130	4.6	96	46	7.62	17.5	22.9	133	4.6	95	21
103	7.43	31.5	21.7	128	3.7	-	53	7.30	26	15.2				
104	7.40	34.5	21.7	131	4.5	100	22	7.44	23	20.5	130	3.8	106	80
105	7.41	32	21.2	125	3.9	98	35	7.35	33	19.5	123	3.55	100	63

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
106	7.37	34.5	20.5	126	4.3	120	38	7.35	34.5	19.5	120	5.7	108	105
107	7.40	37	22.5	132	4.0	112	65	7.38	32.5	20.0	131	3.4	96	78
108	7.38	38.5	22.5	133	4.05	96	78	7.33	36	19.2	129	4.1	116	85

APPENDIX 2

FOETAL ACID-BASE STATUS AND PLASMA ELECTROLYTE VALUES

Case No.	Scalp Blood							Cord Arterial Blood							Cord Venous Blood						
	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
1	7.38	-	-	-	-	-	-	7.14	85	19.5	-	-	-	-	7.34	56	24	-	-	-	-
2	7.35	-	-	-	-	-	-	7.27	42	18.5	-	-	-	-							
3	7.38	25	15.4	} ½ hr time interval																	
	7.35	33.5	19																		
4	7.35	40	21.5	-	-	-	-	7.29	52	22	-	-	-	-							
5	7.38	38	22	-	-	-	-	7.42	30.5	21.5	-	-	-	-							
6	7.32	43	20.5	-	-	-	-	7.21	51	17	-	-	-	-							
7	7.32	41	20.3	-	-	-	-	7.24	44	17.8	-	-	-	-							
8	7.24	48	17.5	-	-	-	-	7.11	60	14	-	-	-	-							
9	7.35	34.5	20	-	-	-	-	7.18	57	16.5	-	-	-	-							
10	7.3	44	20	-	-	-	-	7.16	60	15	-	-	-	-							
11	7.17	-	-	-	-	-	-	7.12	55	14	-	-	-	-							
12	7.38	37	22	-	-	-	-	7.14	82	18	-	-	-	-							
13	7.28	44	19.2	-	-	-	-	7.22	40	16	-	-	-	-							
	7.27	52	21	-	-	-	-														
14	7.28	44	19	-	-	-	-	7.22	48	18	-	-	-	-							
15	7.31	35.5	18.5	-	-	-	-														
16	7.31	44	20.5	-	-	-	-	7.31	47	21	-	-	-	-							
	7.20	49	16.5	-	-	-	-														
17	7.29	36	18.6	-	-	-	-	7.11	56	14.1	-	-	-	-							
18	7.30	39	18.8	-	-	-	-	7.20	54	18	-	-	-	-							

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
19	7.31	41.5	20	-	-	-	-	7.29	44	19.7	167	5.5	-	-							
20	7.33	40	20.3	-	-	-	-	7.31	38	19	136	4.7	-	-							
21	7.33	44	21.5	-	-	-	-	7.14	60	16.5	-	-	-	-							
22	7.22	40	15.7	-	-	-	-	7.19	51	16.5	-	-	-	-							
	7.28	35	17	-	-	-	-														
23	7.27	45	19	-	-	-	-	7.19	35	13.8	-	-	-	-							
24	7.28	42	18.7	-	-	-	-	7.06	74	14.6	-	-	-	-							
25	7.33	52	23	-	-	-	-	7.12	65	16	-	-	-	-							
26	7.29	42	19	-	-	-	-	7.20	53	17.5	141	4.2	110	-							
27	7.29	-	-	-	-	-	-	7.20	48	16.5	1 50	5.3	-	-							
28	7.32	25	16.8	-	-	-	-	7.15	56	15.8	136	4.3	95	-							
29	7.24	77	23	-	-	-	-	7.14	53	15	126	4.5	-	-							
30	7.24	44	17.5	-	-	-	-	7.19	52	17	-	-	-	-							
31	7.29	35	17.5	-	-	-	-	7.17	53	16	148	6.3	84	-							
32	7.34	47	25	-	-	-	-	7.28	47	20	150	5.3	102	-							
33	7.34	43	22	-	-	-	-	7.28	43	19	112	3.2	112	-							
34	7.35	38	20.7	-	-	-	-	7.23	52	18.6	137	5.5	120	15							
35	7.28	48	19.7	-	-	-	8	7.25	50	19.0	-	-	98	90	7.35	32	19.4	-	-	102	95
36	7.18	34.5	18.2	-	-	-	-	7.10	55	14.2	154	5.1	125	155							
37	7.34	35.5	19.4	-	-	-	-	7.27	30.5	16.0	136	6.9	116	25	7.33	33.5	18	136	5.2	-	40
38	7.36	46	26.0	-	-	-	-	7.39	35.0	21.0	166	3.8	132	10							
39	7.32	40	20.0	-	-	-	-	7.10	51.5	14	120	4.2	140	22	7.23	42.5	17.0	122	3.5	120	53
40	7.32	40	20.0	-	-	-	32	7.10	51.5	14.0	120	4.2	140	22	7.23	42.5	17.0	122	3.5	120	53
41	7.28	50	20.5	-	-	-	-														

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
42			Clotted					7.14	61	18.5	152	5.0	103	7	7.32	40.5	20.1	141	5	100	15
43	7.33	44	21.8	-	-	-	-	7.33	42	21.2	148	4.8	95	76	7.33	36	21.2	148	5.7	100	64
44	7.25	56	21.0	-	-	-	-	7.33	47	22.5	131	3.9	96	60	7.33	47	22.5	131	4	99	45
	7.26	43	18.0	-	-	-	-														
45	7.28	45	19.3	-	-	-	-	7.25	44.5	18.1	141	4.4	104	53	7.29	36.5	17.6	138	4.5	102	56
46	7.31	46	21.5	-	-	-	-	7.13	66	16.9	141	4.9	98	-	7.34	38	20.2	135	4.3	98	-
47	7.20	56	18.0	-	-	-	42	7.03	61	12.6	142	5.4	92	57	7.07	61	13.3	138	7.4	92	21
48	7.33	43	21.5	-	-	-	114	7.31	42	20.5	137	4.2	98	58	7.37	38	21.5	134	3.4	98	72
	7.36	34.5	20.0	-	-	-	-														
	7.27	33	16.5	-	-	-	65														
49	7.35	43	22.5	-	-	-	-	7.17	82	19.4	139	4.2	100	37	7.35	29	21.2	142	3.8	108	67
50	7.34	48	23.4	-	-	-	55	7.26	40.0	21.7	140	4.7	96	23	7.36	44	24.5	139	4.1	106	27
51	7.32	43	20.6	-	-	-	30	7.12	64	15.5	133	5.2	99	75	7.17	55	16.5	130	5.2	76	85
	7.35	38	21.0	-	-	-	26														
52	7.38	41	23.0	-	-	-	40	7.17	52	16.2	138	4.4	94	80	7.28	35	17	135	5.5	98	87
53	7.38	34.5	20.0	-	-	-	-	7.26	46	18.6	144	4.3	98	25	7.33	41	20.7	152	4.8	-	28
54	7.32	50	23.1	-	-	-	43	7.21	52	17.8	-	-	-	49	7.24	51.5	19.0	128	6.9	91	60
55	7.20	50	17.5	-	-	-	22	7.16	55	16.5	125	4.5	92	32	7.28	34	17.0	137	5.0	104	55
56	7.36	37.5	21.8	-	-	-	23	7.30	44	20.0	135	3.5	97	34	7.40	35	22.0	134	4.0	100	97
	7.35	32.0	18.5	-	-	-	29														
57	7.36	37.5	20.5	-	-	-	47	7.25	38.5	16.3	-	-	-	17	7.35	35	19.6	128	3.8	112	27
58	7.35	40.5	21.5	-	-	-	32	7.30	42.0	19.7	123	6.5	90	135	7.31	39	19.1	126	5.1	92	160
59	7.28	42	18.7	-	-	-	23	7.22	49	17.2	117	4.8	100	50	7.29	41	19.0	114	4.6	105	57
60	7.05	-	-	-	-	-	-	7.13	52	14.3	138	5.9	-	38	7.19	48.5	16.2	137	4.4	106	50
61	7.27	47	19.7	-	-	-	40	7.15	61	17.3	118	5.1	134	53	7.71	38	19	113	4.4	111	50

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
62	7.31	47	21.1	-	-	-	-	7.23	53	18.6	116	3.8	108	83	7.33	40	20.3	113	3.9	115	90
63								7.15	58	16.5	123	7.44	-	83	7.20	52	17.5	120	5.3	106	103
64	7.33	47	23	-	-	-	-	7.30	50	21.7	120	5.1	104	110	7.35	45	23	123	3.9	106	115
65	7.24	60	21	-	-	-	-	7.21	50	17.7	129	4.1	106	50	7.29	35.5	17.3	127	4.8	102	72
66	7.32	43	20.7	-	-	-	-	7.07	56	13.2	116	5.7	-	55	7.29	35.4	17.5	126	4.6	96	66
67	7.37	40	22.2	-	-	-	54	7.19	45.5	16	135	5.5	97	30	7.34	40	20.9	126	4.4	92	41
68	7.39	35.5	21.2	-	-	-	43	7.27	59.5	22	127	3.5	96	33	7.34	44	22.2	131	4.0	100	63
69	7.28	44	19.2	-	-	-	-	7.21	34	14.2	123	6	122	55	7.27	32	15.8	122	3.9	134	66
70	7.23	53	19.4	-	-	-	-	7.25	50	18.2	121	6	-	23	7.34	39	20.5	121	6.2	-	43
71	7.31	43	20.5	-	-	-	43	7.24	51	19	-	-	-	60	7.30	38	21.2	116	4.0	92	80
72	7.31	46.5	21.7	-	-	-	20	7.31	40	19.3	129	6.7	-	55	7.36	37.5	21	121	5.6	106	60
73	7.29	31.5	16.0	-	-	-	35	7.15	31	17.0	120	4.4	-	113	7.25	38	18.5	125	4.5	105	152
74	7.33	46	22.0	-	-	-	42	7.20	57.5	18.8	132	4.8	102	72	7.34	40	20.9	131	4.2	114	87
75	7.32	46	21.8	-	-	-	47	7.31	37	18.5	117	5.5	-	84	7.33	38.5	20	129	5.2	96	98
76	7.41	34	21.5	-	-	-	53	7.26	50	19.6	123	4.0	-	70	7.35	37.5	20.5	122	5.3	104	80
77	7.31	-	-	-	-	-	-	7.12	35.5	14.6	130	5.8	100	68	7.2	42	15.5	128	6.2	96	65
78	7.34	43.5	22	-	-	-	-	7.23	46	17.5	113	6.9	-	90	7.35	34.5	19.5	127	4.9	130	118
79	7.30	39.5	18.8	-	-	-	22	7.11	68.5	14.4	131	5.6	86	90	7.27	40	18.2	124	6.5	96	132
80	7.32	51	23	-	-	-	-	7.16	74	19.2	132	5.3	100	38	7.3	43	19.8	132	4.6	101	83
81	7.30	47	21.7	-	-	-	-	7.22	57.5	20.2	139	4	103	30	7.37	36	21.1	138	4.3	92	52
82	7.32	45	21.5	-	-	-	-	7.13	59.5	16.2	110	10.6	-	40	7.27	38	17.2	130	7.2	105	47
83	7.29	41.5	19	-	-	-	-	7.11	58.5	14.8	131	5.3	98	34	7.27	35.5	16.5	132	5	95	43
84	7.29	40.5	18.7	-	-	-	12	7.15	55	15.8	115	6.5	92	52	7.3	33.5	17.2	126	5.8	96	55
85	7.34	32.5	18.1	130	2.8	-	50	7.09	64	15	113	6.0	-	40	7.23	47	18.0	123	5.4	86	72

Case No.	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G	pH	pCO_2	SB	Na^+	K^+	Cl	G
86	7.28	47	20.3	-	-	-	32	7.27	46.5	19.2	123	4.9	100	60	7.35	36.5	20.2	120	5.8	93	33
87	7.32	38.5	19.2	-	-	-	-	7.2	51	16.6	133	5.2	106	50	7.3	40	19	133	4.4	96	77
88	7.24	36.5	15.7	-	-	-	-	7.2	52	17.7	132	4.9	102	93	7.33	30.5	17.3	129	4.1	94	130
89	7.35	41	22	-	-	-	-	7.32	56	22	123	4.3	94	46	7.39	40	23	129	4.5	122	52
90	7.37	34	20.5	138	5.7	-	68														
91	7.27	42	18.3	-	-	-	40	7.31	35	17.8	127	6.3	104	70	7.37	26.5	18.0	132	5.3	101	52
92	7.36	38.5	21.0	-	-	-	20	7.16	67.5	18.5	120	6.9	85	46	7.34	39	20.5	122	6.4	82	68
93	7.31	40	19.5	-	-	-	-	7.22	53	18.7	129	5	70	38	7.30	42.5	19.7	126	7.1	96	42
94	7.30	34	17	-	-	-	-	7.23	54	19	117	5	100	38	7.29	45	20	121	4.0	98	55
95	7.31	52	23	-	-	-	-	7.22	47	17.5	127	5.2	92	45	7.23	42	19.5	131	5.5	105	52
96	7.33	42	22	-	-	-	30	7.19	53	17.4	125	4.2	93	50	7.31	33.5	17.6	122	4.7	98	52
97	7.23	32.5	16	-	-	-	-	7.17	45.5	14.8	126	9.5	91	22	7.26	40	17.2	118	10.7	91	22
98	7.40	29	19.2	-	-	-	21	7.29	38.5	18.1	113	8.5	96	60	7.29	38.5	18.4	122	6.7	85	53
99	7.32	39.5	19.7	-	-	-	15	7.26	43.5	18.3	126	6.2	-	15	7.36	33.5	19.5	129	4.5	85	33
100	7.37	32	18.9	-	-	-	-	7.24	33	20.5	132	4.3	94	140	7.26	52	20.5	129	4.7	94	157
101	7.33	35.5	18.6	-	-	-	15	7.29	36	17.3	117	6.0	118	25	7.3	34.5	17.5	127	4.4	100	30
102	7.25	51.5	19.8	-	-	-	-								7.41	30	20.7	130	5.8	96	17
103	7.29	42	19	-	-	-	30	7.20	40	15.2	-	-	-	-	7.22	38	15.4	-	-	-	-
104	7.38	37	21.5	-	-	-	-	7.28	33	16.3	-	-	-	15	7.35	45	22.8	133	5.4	104	35
105	7.24	35	15.5	-	-	-	5	7.18	44	15.0	-	-	-	30	7.29	42	14.0	131	4.75	94	53
106	7.38	24	16.0	-	-	-	-	7.21	52	18.5	127	6.8	106	70	7.26	45	18.7	123	6.3	92	60
107	7.3	42	19.7	-	-	-	8	7.28	47	20	132	5.0	100	45	7.4	30	20	132	4.6	98	62
108	7.33	30.5	16	-	-	-	-	7.30	43	20	119	5.3	-	58	7.28	45	20	128	7.4	100	73

APPENDIX 3

CLINICAL FACTORS IN NEONATAL AND INTRAPARTUM ASSESSMENT

Case No.	Gestation (weeks)	Sex of Infant	Birth Weight (Kgms)	Complication	Clinical signs of foetal distress	Time from scalp blood sampling to delivery (Hrs)	Method of Delivery
1	40	M	4.05	1	2,6	17½	1
2	40	M	2.44	2	2,4,5	14	3
3	40	M	3.27	-	3,4,5	2¼	1
4	33	F	2.18	3,7,8	1	6½	1
5	40	F	3.12	-	3	20	1
6	38	F	2.02	2,4	5	4½	2
7	40	F	2.84	-	3,5	8½	2
8	41	M	3.29	1	5	2	2
9	43	F	4.0	2,4	2,3	17¾	3
10	41	M	3.94	6	6	5	3
11	43	F	3.97	2	2	2¾	3
12	39	M	4.59	1,7,8	2	8	1
13	43	F	3.63	-	2,3	17½	2
14	41	M	3.57	-	1	1	1
15	38	M	2.83	1	1	8	1
16	41	F	3.02	8	2,3	8½	3
17	40	M	2.63	-	2	2½	1
18	40	F	3.44	-	2,3,5	12	1
19	35	M	3.21	3	2	5½	1

20	41	F	3.41	4,8	2,3	7	2
21	39	F	3.66	-	2	5½	2
22	41	F	3.52	8	2,4	4½	1
23	38	F	3.3	3,8	1	4¼	1
24	41	F	3.73	1	2	1½	2
25	42	M	3.53	2,4	2,3,4	15¾	1
26	38	F	3.41	-	2	3	1
27	41	M	4.34	-	4	7½	2
28	40	M	2.92	5	1	2½	1
29	43	F	3.41	1,8	1	5	1
30	40	F	3.54	1,8	1	12¼	1
31	42	F	3.48	-	1	2	1
32	36	M	2.17	5,7,8	1	1	1
33	42	M	3.9	-	2	3	1
34	41	F	3.8	1	1	1½	1
35	39	M	4.46	4,7,8	2,3,4	9	3
36	39	M	3.12	1	1	2	2
37	40	F	3.68	-	2	1	2
38	42	M	3.73	5	1	2	1
39	39	M	3.78	1,6	3	7½	1
40	44	F	2.8	-	2	6½	1
41	43	F	2.99	-	3,4		2
42	38	F	3.75	-	1	1¾	1
43	40	M	3.34	-	1	2	1
44	42	M	4.37	-	1	13	3
45	42	F	3.81	1	1	2¾	1

46	40	F	3.0	5,7,8	1	9½	1
47	40	M	2.71	6	2,4	½	2
48	40	M	3.38	8	4	4	1
49	40	M	3.31	2,4	4	4½	2
50	42	M	3.3	-	5	9	1
51	37	F	3.75	1,8	2	19	2
52	40	F	3.28	8	5	1½	1
53	40	M	3.82	2	1	3¼	1
54	38	F	2.76	-	2,3,4	5	2
55	43	F	3.8	1	5,2	5¼	2
56	43	M	4.11	2	2	12¾	1
57	40	F	3.00	1	1	2½	1
58	39	M	3.24	2	1	18	1
59	39	M	3.10	2	4	8	2
60	35	M	1.95	5	3	½	2
61	41	M	3.74	8	3,5	7	1
62	41	M	3.61	1,4	1	24	2
63	43	F	3.83	3	1		2
64	38	F	2.82	2	1	10	2
65	40	M	3.19	2	2	2½	1
66	41	M	3.15	3,8	3	5	1
67	43	F	3.45	1,8	4	3½	1
68	36	F	2.9	3	3	4½	1
69	42	F	3.36	-	1	3	1
70	42	F	3.84	1	1	5	2

71	35	M	2.5	2	1		2
72	40	M	3.68	-	1	5	1
73	40	F	3.21	1	1	14	1
74	36	F	3.04	1	1	29	2
75	42	F	3.13	1	1	27½	2
76	40	M	3.32	1	2	23	2
77	38	F	2.38	2	1	1	1
78	41	M	3.74	3	4,7	2½	1
79	40	F	2.95	-	1	7½	2
80	40	M	3.55	2,8	1	6½	2
81	39	M	2.9	5	1	50 Min	1
82	36	M	2.8	8,9	1	9	1
83	44	M	3.08	-	1	3¼	1
84	41	F	3.41	3	1	21	1
85	38	F	3.36	-	3	10	1
86	40	F	2.90	2	1	25	3
87	40	M	3.3	1	1	1	1
88	40	F	2.81	-	5,4	3	1
89	39	F	3.11	-	1	2	1
90	37	M	2.89	-	3	2	1
91	42	F	2.30	1,8	1	3½	1
92	41	F	3.01	2	1	14	1
93	42	F	3.24	2	1	1¼	1
94	40	M	3.24	2,8	1	4½	3
95	38	F	3.55	-	1	7	3

96	43	M	3.47	1	4	37	1
97	38	F	2.22	1	1	7	1
98	39	F	3.41	-	4	16	1
99	38	F	3.32	-	3,7	1¼	1
100	40	F	3.5	-	2	7½	2
101	41	F	5.33	1,8	2	15	1
102	39	F	3.50	2	2,5	6½	3
103	42	F	4.2	1,8	2	7	2
104	37	M	3.08	9	1	9	1
105	40	F	2.02	2,9,10	2,7	8	1
106	43	M	3.42	-	1	16	1
107	38	F	3.46	2	1	4½	1
108	42	M	2.62	-	1	7½	1

APPENDIX 4

NEONATAL ASSESSMENT

Case No.	Apgar Score Min. Min.	24 Hours CNS	RS	GC	6 Weeks CNS	RS	GC	SQ
1	3 - 3	-	-	-	-	-	-	-
2	1 - 3	3	3	3	2	3	3	3
3	3 - 3	3	3	3	3	3	2	3
4	3 - 3	2	2	2	2	3	3	2
5	3 - 3	3	3	3	-	-	-	-
6	3 - 3	3	3	3	3	3	3	3
7	2 - 3	3	3	3	2	3	3	3
8	3 - 3	3	3	3	3	3	3	3
9	1 - 3	2	3	2	1	3	2	2
10	1 - 3	3	2	3	3	3	3	3
11	1 - 1	2	3	3	2	3	3	3
12	2 - 3	3	3	3	3	3	3	3
13	3 - 3	3	3	3	3	3	3	3
14	3 - 3	3	3	3	3	3	3	3
15	2 - 3	-	-	-	-	-	-	-
16	2 - 2	2	3	3	3	3	3	3
17	3 - 3	3	3	3	2	3	3	3
18	3 - 3	3	3	3	3	3	3	3
19	3 - 3	3	3	3	3	3	3	3
20	3 - 3	3	3	3	3	3	3	3
21	3 - 3	3	3	3	3	3	3	3
22	3 - 3	2	3	3	3	3	3	3
23	3 - 3	3	3	3	3	3	3	3
24	3 - 3	3	3	3	3	3	3	3
25	1 - 2	2	3	2	2	3	2	3
26	3 - 3	3	3	3	3	3	3	3
27	2 - 3	3	3	3	3	3	3	3
28	3 - 3	3	3	3	-	-	-	-

Case No.	min. min.	CNS	RS	GC	CNS	RS	GC	SQ
29	2 - 3	3	3	3	3	3	3	3
30	3 - 3	3	3	3	-	-	-	-
31	3 - 3	3	3	3	3	3	3	3
32	3 - 3	3	3	3	3	3	3	3
33	2 - 3	3	3	3	3	3	3	3
34	3 - 3	3	3	3	3	3	3	3
35	1 - 2	1	3	2	-	-	-	-
36	3 - 3	3	3	3	3	3	3	3
37	1 - 1	1	3	2	-	-	-	-
38	3 - 3	3	3	3	-	-	-	-
39	2 - 3	3	3	3	-	-	-	-
40	2 - 3	3	3	3	3	3	3	3
41	2 - 3	3	3	3	-	-	-	-
42	2 - 3	3	3	3	3	3	3	3
43	1 - 3	3	3	3	3	3	3	3
44	2 - 3	3	3	3	3	3	2	3
45	1 - 2	3	3	3	-	-	-	-
46	2 - 3	3	3	3	3	3	3	3
47	1 - 2	2	2	2	-	-	-	-
48	3 - 3	3	3	3	3	3	3	3
49	1 - 2	3	3	3	3	3	3	3
50	3 - 3	3	3	3	-	-	-	-
51	2 - 3	3	3	3	3	3	3	3
52	2 - 3	3	3	3	3	3	3	3
53	3 - 3	3	3	3	3	3	3	3
54	3 - 3	3	3	3	-	-	-	-
55	1 - 2	3	2	1	3	3	3	3
56	2 - 3	3	3	3	3	3	3	3
57	3 - 3	3	3	3	-	-	-	-
58	2 - 3	3	3	3	2	3	3	3
59	2 - 3	3	2	2	3	3	3	3
60	1 - 2	2	3	2	2	3	3	2

Case No.	Min. Min.	CNS	RS	GC	CNS	RS	GC	SQ
60	1 - 2	2	3	2	2	3	3	2
61	3 - 3	3	3	3	-	-	-	-
62	3 - 3	3	3	3	3	3	3	3
63	3 - 3	3	3	3	3	3	3	3
64	3 - 3	3	3	2	-	-	-	-
65	3 - 3	3	3	3	3	3	3	3
66	1 - 2	3	3	3	-	-	-	-
67	1 - 2	3	3	2	2	3	3	2
68	2 - 2	2	1	1	3	3	3	3
69	3 - 3	3	3	3	3	3	3	3
70	2 - 3	3	3	3	-	-	-	-
71	2 - 2	2	2	2	-	-	-	-
72	3 - 3	3	3	3	-	-	-	-
73	3 - 3	3	3	3	3	3	3	3
74	3 - 3	3	3	3	-	-	-	-
75	3 - 3	3	3	3	-	-	-	-
76	3 - 3	3	3	3	-	-	-	-
77	1 - 3	3	3	3	3	3	3	3
78	2 - 3	3	3	3	3	3	3	3
79	2 - 3	3	3	3	3	3	3	3
80	2 - 3	3	3	3	3	3	3	3
81	3 - 3	3	3	3	3	3	3	3
82	3 - 3	3	3	3	3	3	3	3
83	3 - 3	3	3	3	2	3	3	2
84	3 - 3	3	3	3	-	-	-	-
85	2 - 3	3	3	3	-	-	-	-
86	3 - 3	3	3	3	2	3	3	3
87	3 - 3	3	3	3	-	-	-	-
88	2 - 3	3	3	3	3	3	3	3
89	3 - 3	3	3	3	-	-	-	-
90	3 - 3	3	3	3	-	-	-	-
91	3 - 3	3	3	3	3	3	3	3

Case No.	Min. Min.	CNS	RS	GC	CNS	RS	GC	SQ
92	1 - 3	3	3	3	3	3	3	3
93	3 - 3	3	3	3	-	-	-	-
94	2 - 3	3	2	2	-	-	-	-
95	3 - 3	3	3	3	3	3	3	3
96	3 - 3	3	3	3	3	3	3	3
97	3 - 3	3	3	3	3	3	3	3
98	2 - 3	3	3	3	3	3	3	3
99	3 - 3	3	3	3	3	3	3	3
100	3 - 3	3	3	3	3	3	3	3
101	3 - 3	3	3	2	3	3	2	3
102	1 - 2	1	2	1	2	3	3	3
103	2 - 3	3	3	3	3	3	3	3
104	3 - 3	3	3	3	-	-	-	-
105	3 - 3	3	3	3	3	3	3	3
106	3 - 3	3	3	3	3	3	3	3
107	3 - 3	3	3	3	3	3	3	3
108	3 - 3	3	3	3	3	3	3	3

APPENDIX 5

FOETAL ELECTROCARDIOGRAM - TIME CONSTANTS AND CONFIGURATION

	1st Sample Foetal E.C.G. Time Constants and Configuration									2nd Sample Foetal E.C.G. Time Constants and Configuration								
Case No.	PR	RR	PR/RR	QRS	R/S	QT	QT_c	ST	T	PR	RR	PR/RR	QRS	R/S	QT	QTc	ST	T
1																		
2																		
3	0.08	0.4	0.2	0.03	1.25	-	-	1	1	0.08	0.40	0.2	0.03	1.25	0.3	0.47	1	2
4	0.08	0.5	0.16	0.03	0.5	0.3	0.42	1	2	0.14	0.42	0.33	0.04	1.2	0.3	0.46	1	1
5																		
6	0.14	0.38	0.39	0.03	1.25	0.24	0.38	1	2	0.12	0.46	0.26	0.03	1.25	0.2	0.29	1	2
7																		
8	0.1	0.44	0.23	0.02	0.8	0.22	0.33	1	2	0.1	0.38	0.26	0.03	0.8	0.22	0.36	3	2
9	0.1	0.44	0.23	0.04	1.2	0.22	0.33	1	2	0.14	0.32	0.44	0.04	1.25	0.2	0.35	1	1
10	0.12	0.42	0.29	0.04	0.6	0.20	0.31	1	1	0.12	0.38	0.32	0.03	0.71	0.18	0.30	1	1
11																		
12	0.12	0.44	0.28	0.04	0.75	No T waves seen				0.12	0.48	0.25	0.03	1.75	0.22	0.31	1	1
13	0.08	0.37	0.22	0.03	0.83	0.20	0.33	1	2	No 2nd Sample								
14	0.12	0.48	0.25	0.03	1.0	0.24	0.35	1	2	0.12	0.68	0.18	0.03	0,71	0.26	0.31	1	1
15	0.12	0.4	0.3	0.03	1.0	0.22	0.34	1	2									
16	0.1	0.36	0.28	0.03	1.7	0.2	0.33	2	2	0.1	0.34	0.29	0.03	1.4	0.2	0.35	1	2
17	0.08	0.42	0.19	0.03	3	0.24	0.37	1	2	Unsatisfactory								

Case No.	PR	RR	PR/RR	QRS	R/S	QT	QTc	ST	T	PR	RR	PR/RR	QRS	R/S	QT	QTc	ST	T
18	0.1	0.41	0.24	0.03	2	0.24	0.37	1	2	0.1	0.46	0.22	0.03	2.4	0.22	0.33	1	2
19	0.08	0.4	0.2	0.03	1.25	0.24	0.38	1	2	0.1	0.38	0.26	0.02	1.33	0.22	0.35	1	2
20	0.1	0.42	0.24	0.03	1.2	0.22	0.34	1	1	0.1	0.42	0.24	0.02	1.25	0.22	0.34	1	1
21	0.08	0.42	0.19	0.03	1.0	0.24	0.37	1	2	0.08	0.4	0.20	0.03	1.0	0.24	0.38	1	2
22	0.14	0.46	0.3	0.03	0.5	0.3	0.44	1	3	0.14	0.4	0.35	0.03	0.88	0.28	0.44	1	3
23	0.1	0.43	0.23	0.03	2	0.22	0.33	1	2	-	0.46	-	0.03	1.4	-	-	-	-
24	0.1	0.46	0.22	0.03	0.5	0.24	0.35	1	3	0.11	0.44	0.25	0.03	0.4	0.24	0.36	1	3
25	0.12	0.45	0.27	0.03	1.0	0.24	0.36	1	2	0.12	0.4	0.3	0.03	1.0	0.24	0.38	1	2
26	0.12	0.48	0.25	0.03	0.6	0.24	0.34	1	2	0.14	0.5	0.28	0.03	0.5	0.26	0.37	1	2
27	0.1	0.44	0.23	0.03	0.6	0.24	0.31	1	2	0.12	0.4	0.3	0.02	0.5	0.24	0.38	1	2
28	0.1	0.4	0.25	0.02	1.0	0.22	0.35	1	2	0.1	0.44	0.23	0.03	1.25	0.24	0.36	1	2
29	0.14	0.48	0.29	0.02	2.0	0.24	0.34	1	2	0.12	0.38	0.31	0.02	2.0	0.22	0.35	1	2
30	0.12	0.42	0.29	0.03	0.66	0.22	0.34	1	2	0.1	0.44	0.23	0.02	0.54	0.22	0.33	1	2
31	0.14	0.52	0.27	0.03	0.82	0.22	0.31	1	2	0.13	0.5	0.20	0.03	1.63	0.24	0.33	1	2
32	0.08	0.4	0.2	0.02	1.25	0.2	0.31	1	2	0.08	0.42	0.19	0.02	1.4	0.22	0.34	1	1
33	0.1	0.44	0.23	0.03	0.83	0.22	0.33	1	2	0.08	0.40	0.20	0.02	1.0	0.22	0.34	1	2
34	0.1	0.46	0.22	0.02	1.8	0.24	0.35	1	2	0.1	0.4	0.25	0.02	1.25	0.22	0.34	1	2
35	0.12	0.42	0.28	0.03	1.0	0.22	0.34	1	2	0.08	0.34	0.24	0.02	1.0	0.22	0.37	1	2
36	0.1	0.54	0.19	0.03	1.33	0.3	0.41	1	2	0.1	0.54	0.19	0.03	1.33	0.32	0.43	1	2
37										0.08	0.36	0.22	0.02	1.8	0.26	0.42	2	2
38	0.08	0.46	0.17	0.03	1.0	0.24	0.35	1	1	0.12	0.52	0.23	0.03	1.0	0.24	0.33	1	1
39	0.1	0.36	0.28	0.03	1.0	0.24	0.39	1	2	0.1	0.5	0.2	0.02	1.0	0.26	0.37	1	3
40	0.08	0.48	0.17	0.03	1.25	0.22	0.31	1	3	0.1	0.5	0.2	0.04	1.4	0.28	0.4	1	2

Case No.	PR	RR	PR/RR	QRS	R/S	QT	QTc	ST	T	PR	RR	PR/RR	QRS	R/S	QT	QTc	ST	T
41	0.08	0.48	0.17	0.03	1.25	-	-	-	-									
42	0.1	0.38	0.26	0.02	1.5	0.24	0.39	1	2	0.1	0.38	0.26	0.02	1.4	0.22	0.36	1	1
43	0.1	0.46	0.22	0.03	1.7	0.24	0.35	1	2	0.14	0.44	0.32	0.03	2.0	0.24	0.36	1	2
44	0.1	0.46	0.22	0.04	1.7	0.22	0.32	1	2	0.12	0.42	0.29	0.03	1.5	0.24	0.37	1	2
45	0.14	0.46	0.3	0.03	1.75	0.26	0.38	1	2	0.14	0.48	0.3	0.03	1.6	0.28	0.4	1	2
46	0.12	0.42	0.29	0.03	3.0	0.24	0.37	1	2	0.1	0.38	0.26	0.03	2.2	0.22	0.35	1	2
47										0.16	0.6	0.27	0.03	2.0	0.3	0.39	1	2
48	0.13	0.54	0.24	0.03	0.5	0.26	0.36	1	3	0.14	0.54	0.26	0.03	0.5	0.28	0.38	1	3
49	0.12	0.5	0.24	0.03	2.0	0.24	0.34	1	2	0.12	0.44	0.27	0.03	1.25	0.24	0.36	1	2
50	0.1	0.36	0.28	0.03	1.0	0.2	0.33	1	2	0.1	0.44	0.23	0.03	1.0	0.2	0.31	1	2
51	0.12	0.36	0.33	0.03	1.5	0.24	0.4	1	2	0.08	0.34	0.24	0.03	1.25	0.2	0.35	1	3
52	0.1	0.56	0.18	0.04	1.8	0.28	0.37	1	2	0.12	0.4	0.3	0.03	1.8	0.26	0.41	1	2
53	0.14	0.5	0.28	0.03	1.0	0.26	0.37	1	2	0.14	0.4	0.35	0.03	1.25	0.26	0.41	1	3
54	0.12	0.48	0.25	0.03	1.0	0.26	0.38	1	3	0.14	0.48	0.25	0.03	1.0	0.26	0.38	1	2
55	0.14	0.42	0.33	0.03	1.2	-	-	2	2	Grossly abnormal ECG								
56	0.1	0.4	0.25	0.03	3.0	0.22	0.34	1	2	0.12	0.42	0.28	0.03	0.66	0.24	0.37	1	2
57	0.14	0.4	0.35	0.03	2.0	0.24	0.38	1	2	0.14	0.46	0.3	0.03	2.0	0.24	0.35	1	2
58	0.14	0.44	0.32	0.02	3.0	0.24	0.36	1	2									
59	0.14	0.46	0.3	0.03	2.0	0.22	0.32	1	2	0.14	0.5	0.28	0.03	2.0	0.24	0.34	1	2
60	0.12	0.36	0.33	0.03	2	0.22	0.36	1	2									
61	0.12	0.4	0.3	0.03	1.0	0.22	0.34	1	1	0.12	0.48	0.25	0.03	1.5	0.26	0.38	1	1
62	0.12	0.42	0.3	0.03	0.5	0.24	0.37	1	2	0.12	0.5	0.24	0.02	1.0	0.26	0.37	1	2
63	0.12	0.48	0.25	0.03	1.5	0.25	0.35	1	2	0.12	0.48	0.25	0.03	1.5	0.24	0.35	2	2

Case No.	PR	RR	PR/RR	QRS	R/S	QT	QTc	ST	T	PR	RR	PR/RR	QRS	R/S	QT	QTc	ST	T
64	0.12	0.46	0.26	0.03	1.0	0.22	0.32	1	2	0.1	0.32	0.31	0.03	1.0	0.20	0.37	1	1
65	0.08	0.46	0.17	0.03	1.0	0.22	0.32	1	2	0.1	0.52	0.19	0.03	1.0	0.26	0.36	1	3
66	0.12	0.4	0.3	0.02	1.7	0.24	0.38	1	2	0.1	0.72	0.14	0.02	5.0	0.3	0.35	1	2
67	0.1	0.44	0.23	0.03	2.0	0.22	0.33	1	2	0.1	0.54	0.2	0.03	1.8	0.24	0.33	1	2
68	0.1	0.42	0.24	0.03	2.7	0.24	0.37	1	2	0.14	0.44	0.32	0.03	2.7	0.22	0.33	1	2
69	0.12	0.5	0.24	0.02	1.0	0.22	0.32	1	2	0.12	0.5	0.24	0.03	0.75	0.22	0.32	1	1
70	0.1	0.46	0.22	0.02	1.5	0.2	0.29	1	2	0.1	0.4	0.25	0.02	1.8	0.2	0.31	1	2
71	0.1	0.48	0.21	0.02	0.83	0.24	0.34	1	2	0.12	0.54	0.22	0.02	0.86	0.32	0.44	1	1
72	0.1	0.38	0.26	0.03	1.0	0.2	0.32	1	1	0.1	0.44	0.23	0.03	1.0	0.26	0.39	1	2
73	0.12	0.46	0.26	0.03	3.5	0.25	0.35	1	2	0.12	0.46	0.26	0.03	2.0	0.22	0.32	1	2
74	0.12	0.28	0.42	0.03	1.7	0.24	0.37	1	2	0.12	0.3	0.4	0.02	2.0	0.26	0.4	2	2
75	0.12	0.42	0.29	0.03	1.4	0.2	0.31	1	2	0.12	0.42	0.29	0.03	1.0	0.24	0.37	1	1
76	0.12	0.42	0.29	0.02	1.0	0.22	0.34	1	1	0.12	0.38	0.32	0.02	1.0	0.22	0.34	1	1
77	0.1	0.42	0.24	0.02	1.2	0.22	0.34	1	1	0.08	0.6	0.13	0.02	1.8	0.20	0.33	1	1
78	0.1	0.52	0.2	0.03	2.0	0.3	0.42	1	2	0.14	0.7	0.2	0.03	1.75	0.4	0.47	1	2
79	0.12	0.48	0.25	0.03	1.6	0.22	0.32	1	1	0.12	0.42	0.28	0.03	1.5	0.22	0.34	1	1
80	0.12	0.48	0.25	0.03	0.67	0.3	0.43	1	3	0.12	0.38	0.32	0.03	0.63	0.24	0.39	1	1
81	0.1	0.5	0.2	0.03	1.2	0.24	0.34	1	2	0.14	0.54	0.26	0.03	1.4	0.26	0.35	1	2
82	0.1	0.44	0.23	0.03	2.0	0.24	0.36	1	2	0.1	0.44	0.23	0.03	2.0	0.26	0.36	1	2
83	0.12	0.42	0.29	0.02	2.0	0.22	0.34	1	2	0.12	0.46	0.26	0.03	1.5	0.26	0.38	1	1
84	0.12	0.42	0.29	0.03	1.25	0.2	0.31	1	2	0.14	0.36	0.4	0.02	1.0	0.2	0.33	1	2
85	0.14	0.42	0.33	0.03	1.5	0.22	0.34	1	2	0.14	0.38	0.37	0.03	1.5	0.26	0.42	1	2
86	0.1	0.44	0.23	0.03	1.5	0.24	0.37	1	2	0.08	0.35	0.23	0.03	1.33	0.2	0.34	1	2

Case No.	PR	RR	PR/RR	QRS	R/S	QT	QTc	ST	T	PR	RR	PR/RR	QRS	R/S=	QT	QTc	ST	T
87	0.12	0.44	0.27	0.02	1.2	0.26	0.39	1	3	0.1	0.5	0.2	0.02	1.2	0.26	0.37	1	3
88	0.1	0.44	0.23	0.03	0.75	0.24	0.36	1	2	0.12	0.42	0.29	0.03	0.83	0.24	0.37	1	2
89	0.1	0.42	0.24	0.02	1.25	0.2	0.31	1	2	0.1	0.46	0.22	0.02	1.3	0.22	0.32	1	2
90	0.14	0.4	0.35	0.03	1.0	0.24	0.38	1	2	0.12	0.36	0.33	0.03	1.0	0.2	0.33	1	2
91	0.12	0.44	0.28	0.03	1.0	0.24	0.36	1	2	0.12	0.42	0.29	0.03	1.0	0.24	0.37	1	2
92	0.12	0.48	0.25	0.03	0.7	0.24	0.35	1	2	0.12	0.48	0.25	0.03	0.7	0.24	0.35	1	1
93	0.12	0.46	0.26	0.02	1.0	0.24	0.35	1	1	0.12	0.56	0.21	0.03	1.0	0.24	0.33	1	1
94	0.1	0.44	0.23	0.03	0.6	0.22	0.33	1	2	0.1	0.4	0.25	0.02	0.67	0.22	0.4	1	1
95	0.12	0.4	0.3	0.02	1.0	0.2	0.32	1	2	0.14	0.42	0.3	0.03	1.0	0.24	0.37	1	2
96	0.12	0.46	0.26	0.03	1.26	0.26	0.38	1	2	0.12	0.44	0.27	0.03	1.25	0.26	0.39	1	2
97	0.1	0.46	0.22	0.03	0.83	0.22	0.32	1	1	0.1	0.46	0.22	0.03	0.83	0.22	0.32	1	1
98	0.14	0.46	0.3	0.03	1.33	0.24	0.35	1	2	0.14	0.44	0.32	0.03	1.75	0.28	0.42	1	2
99	0.12	0.4	0.3	0.03	1.5	0.2	0.32	1	2	0.12	0.42	0.29	0.03	1.5	0.22	0.34	1	2
100	0.12	0.42	0.29	0.03	1.0	0.24	0.37	1	2	0.1	0.35	0.29	0.02	2.0	0.2	0.34	1	1
101	0.1	0.52	0.19	0.03	0.71	0.24	0.33	1	2	0.1	0.46	0.22	0.03	0.8	0.26	0.38	1	1
102	0.12	0.48	0.25	0.03	1.33	0.26	0.37	1	2	0.1	0.52	0.19	0.03	1.2	0.26	0.36	1	1
103	0.1	0.46	0.22	0.03	0.75	0.24	0.35	1	2	0.1	0.46	0.22	0.02	1.2	0.24	0.35	1	1
104	0.12	0.46	0.26	0.03	0.8	0.24	0.35	1	2	0.12	0.58	0.21	0.03	0.6	0.3	0.39	1	2
105	0.1	0.4	0.25	0.03	1.0	0.24	0.38	1	2	0.12	0.44	0.27	0.03	1.25	0.26	0.39	1	2
106	0.12	0.44	0.27	0.03	1.5	0.22	0.33	1	2	0.12	0.42	0.28	0.02	1.8	0.22	0.34	1	1
107	0.1	0.4	0.25	0.02	1.6	0.2	0.31	1	2	0.12	0.44	0.27	0.02	1.0	0.22	0.33	1	2
108	0.1	0.4	0.25	0.02	1.33	0.2	0.31	1	2	0.1	0.5	0.2	0.03	1.0	0.26	0.87	1	2

APPENDIX 6

TIME CONSTANTS AND CONFIGURATION OF THE NEONATAL ELECTROCARDIOGRAM

Case No.	Neonatal E.C.G. Time Constants and Configuration									R/S Ratios - All Leads Neonatal E.C.G.								
	PR	RR	PR/RR	QRS	QT	QTc	ST	T	AXIS	I	II	III	AVR	AVL	AVF	V1	V3	V6
1																		
2																		
3	0.1	0.52	0.19	0.04	0.35	0.5	1	2	147	0.11	2.5	1.0	1.0	1.8	1.0	5.3	0.55	0.44
4																		
5																		
6	0.12	0.42	0.29	0.04	0.24	0.37	1	1	105	0.25	10	10	0.33	0.33	10	2.25	3	10
7																		
8	0.08	0.56	0.14	0.04	0.36	0.46	1	3	180	0.1	0.25	10	10	0.1	0.25	0.52	-	-
9	0.12	0.46	0.26	0.04	0.32	0.47	1	2	118	0.2	2.0	4.5	1.0	0.29	3.3	2	1	1.2
10		Lead II unsatisfactory							136	0.25	-	4.5	5	0.20	1.6	1.7	1.1	0.33
11								No other data										
12	0.12	0.4	0.3	0.03	0.24	0.38	1	2	146	0.17	1.2	4.3	3.0	0.25	10	1.6	0.73	0.25
13	0.12	0.6	0.2	0.04	0.32	0.41	1	2	-160	0.1	0.1	1.0	10	0.1	0.25	0.56	0.62	0.19
14	0.12	0.46	0.26	0.03	0.28	0.41	1	2	30	10	10	1.0	0.25	10	10	1.4	1.2	0.5
15								No other data										
16	0.1	0.46	0.22	0.03	0.26	0.38	1	2	113	0.29	10	10	1.0	1.29	10	0.85	0.7	1.3
17	0.1	0.55	0.19	0.04	0.24	0.33	1	2	120	0.33	0.1	10	10	0.6	0.1	1.9	0.7	0.4

Case No.	PR	RR	PR/RR	QRS	QT	QTc	ST	T	AXIS	I	II	III	AVR	AVL	AVF	V1	V3	V6
18	0.12	0.44	0.27	0.02	0.26	0.39	1	2	130	0.1	10	10	10	0.1	10	2	3.5	10
19	0.12	0.42	0.29	0.03	0.26	0.39	1	2	168	0.14	2	10	10	0.2	10	1.9	1.2	0.66
20	0.1	0.38	0.26	0.03	0.24	0.37	1	2	120	0.47	3	10	10	0.22	10	1.5	0.78	0.92
21	0.08	0.44	0.18	0.03	0.22	0.33	1	2	150	0.5	10	10	10	0.25	10	1.75	3	3
22	0.14	0.42	0.33	0.03	0.28	0.42	1	1	167	0.14	0.5	10	10	10	10	1.7	1.4	1.25
23	0.1	0.4	0.25	0.03	0.22	0.35	1	3	82	10	10	10	10	0.33	10	0.5	2.6	10
24	0.1	0.47	0.21	0.03	0.24	0.35	1	2	-175	0.1	0.1	3	10	0.1	10	0.5	0.5	0.43
25	0.12	0.48	0.24	0.04	0.24	0.34	1	3	115	0.2	0.4	10	10	0.1	10	1.3	1.6	2.00
26	0.12	0.52	0.23	0.03	0.34	0.47	1	2	105	0.5	1.5	10	1	0.4	0.25	5.5	1.3	10
27	0.12	0.42	0.30	0.03	0.24	0.37	1	2	145	0.23	2	10	10	0.1	10	0.66	0.33	0.33
28	0.1	0.54	0.19	0.03	0.24	0.32	1	2	163	0.2	1.3	10	10	0.25	10	1.3	1.2	1.0
29	0.12	0.56	0.21	0.03	0.34	0.44	1	2	130	1.0	10	10	10	0.1	10	0.86	-	10
30	0.1	0.52	0.19	0.03	0.26	0.36	1	2	180	0.1	0.1	10	10	0.1	1	1.2	0.14	0.1
31	0.14	0.42	0.33	0.03	0.2	0.37	1	2	-127	0.1	0.75	0.1	2	0.1	2	0.5	0.2	1.0
32	0.1	0.5	0.2	0.02	0.22	0.31	1	2	90	0.12	10	10	0.5	0.25	10	0.6	2.0	-
33	0.1	0.5	0.2	0.03	0.32	0.45	1	2	119	0.17	2.0	10	10	0.2	10	2.6	1.8	1.0
34	0.1	0.46	0.22	0.02	0.24	0.35	1	2	133	0.1	10	10	10	0.1	10	0.25	0.1	0.1
35	0.1	0.5	0.2	0.03	0.3	0.43	1	1	107	0.3	0.8	10	0.1	0.1	10	1.6	3	10
36	0.1	0.54	0.19	0.04	0.28	0.38	1	2	150	0.1	0.1	1.0	10	0.1	0.1	0.4	0.17	0.1
37	0.12	0.48	0.25	0.05	0.28	0.4	1	2	126	0.4	10	10	10	0.2	10	0.65	0.6	0.9
38	0.1	0.56	0.18	0.03	0.3	0.4	1	2	137	0.1	10	10	10	0.1	10	1.9	1.0	2.0
39	0.12	0.54	0.22	0.03	0.26	0.35	1	2	127	0.1	10	10	10	0.1	10	1.0	1.0	10
40	0.1	0.4	0.25	0.03	0.28	0.44	1	2	120	0.1	0.67	10	4	0.1	1.4	2.5	0.86	0.5

Case No.	PR	RR	PR/RR	QRS	QT	QTc	ST	T	AXIS	I	II	III	AVR	AVL	AVF	V1	V3	V6
41	No neonatal ECG recorded																	
42	0.1	0.46	0.22	0.02	0.28	0.4	1	1	170	0.1	0.5	10	10	0.1	10	1.6	1.3	1.0
43	0.12	0.4	0.3	0.03	0.26	0.41	1	2	143	0.25	2.0	10	10	0.13	10	1.0	1.0	0.2
44	0.1	0.5	0.2	0.03	0.34	0.48	1	2	151	0.1	10	10	10	0.1	3	4	1.0	1.0
45	0.14	0.46	0.3	0.03	0.28	0.41	1	2	137	0.1	3	10	10	0.17	10	1.1	0.8	1.25
46	0.1	0.46	0.22	0.03	0.26	0.35	1	2	112	0.1	3.5	7.0	1.0	0.25	5.0	2.2	0.8	2.0
47	0.1	0.52	0.19	0.03	0.28	0.39	1	2	114	0.1	2.0	4.5	1.0	0.25	4.0	1.0	1.0	1.5
48	0.1	0.42	0.24	0.03	0.24	0.37	1	2	168	0.1	0.33	10	3.0	0.1	2.0	0.5	0.11	0.17
49	0.1	0.44	0.23	0.03	0.26	0.39	1	2	118	10	10	10	0.1	10	10	0.85	1.5	3.0
50	0.1	0.6	0.17	0.03	0.34	0.44	1	2	109	0.5	10	4.0	1.0	0.67	3.0	2.2	2.4	3.0
51	0.1	0.58	0.17	0.03	0.3	0.4	1	3	113	0.1	2	10	1.0	0.1	3	0.67	1.0	1.0
52	0.1	0.5	0.2	0.04	0.3	0.42	1	2	-	10	10	5	0.33	10	0.1	1.0	2.0	10
53	0.14	0.66	0.21	0.04	0.38	0.46	1	2	154	0.1	0.67	3.5	10	0.1	1.0	1.6	0.6	0.2
54	0.1	0.46	0.22	0.02	0.28	0.41	1	2	94	0.1	2.0	5.0	0.33	0.33	0.25	2.0	1.6	1.0
55	Neonatal ECG grossly abnormal																	
56	0.12	0.6	0.2	0.04	0.36	0.47	1	2	163	0.25	1.0	10	10	0.1	10	0.6	0.4	0.33
57	0.12	0.5	0.24	0.03	0.32	0.45	1	2	125	0.25	10	10	0.1	0.17	10	0.86	0.6	0.33
58	0.12	-	0.31	0.02	0.22	0.36	1	3	136	0.4	2.5	4	1.0	0.33	10	0.3	0.5	0.6
59	0.14	-	0.3	0.04	0.3	0.44	1	3	150	0.1	2.0	5.0	10	0.1	3.5	0.73	0.5	0.33
60	0.1	0.42	0.24	0.04	0.28	0.43	1	1	90	10	5	3	0.1	0.1	4.0	0.75	1.0	1.0
61	0.12	0.44	0.27	0.04	0.3	0.46	2	1	117	0.17	3.0	10	1.0	0.1	4.0	0.75	0.6	0.37
62	0.12	0.54	0.22	0.02	0.32	0.43	1	2	-174	0.1	0.1	10	10	0.1	0.5	0.5	0.3	0.4
63	0.12	0.56	0.21	0.04	0.4	0.50	1	2	115	0.2	10	10	10	0.14	10	0.8	0.8	1.5

Case No.	PR	RR	PR/RR	QRS	QT	QTc	ST	T	AXIS	I	II	III	AVR	AVL	AVF	V1	V3	V6
64	0.12	0.43	0.3	0.03	0.24	0.37	1	2	155	0.1	0.1	10	10	0.1	10	10	4.0	2.5
65	0.12	0.54	0.22	0.03	0.32	0.44	1	2	159	0.25	1.0	4.0	3.0	0.33	10	5.0	1.6	1.0
66	0.14	0.46	0.3	0.02	0.3	0.44	1	2	142	0.25	10	3.5	10	0.4	4.0	6.0	3.0	1.5
67	0.1	0.4	0.25	0.03	0.26	0.41	1	2	145	0.4	2.5	3.0	3.0	0.33	8.0	1.3	1.0	1.3
68	0,12	0.42	0.3	0.03	0.24	0.37	1	1	74	10	2.0	10	10	0.1	10	0.6	0.6	10
69	0.14	0.44	0.32	0.03	0.26	0.39	1	2	136	0.25	1.5	4.0	1.5	0.25	4.0	1.4	1.0	0.8
70	0.1	0.52	0.19	0.03	0.28	0.39	1	2	100	0.1	2.0	6.0	0.5	0.1	2.5	0.5	0.6	2.0
71	0.14	0.52	0.27	0.04	0.36	0.5	1	1	167	0.1	0.5	10	10	0.1	1.0	1.0	0.5	0.6
72	0.1	0.32	0.31	0.03	0.2	0.34	1	1	150	0.2	2.0	6.0	1.0	0.17	10	1.0	0.6	0.7
73	0.12	0.52	0.23	0.04	0.26	0.36	1	2	129	0.1	3.0	9.0	10	0.1	2.0	0.7	0.8	10
74	0.12	0.4	0.3	0.03	0.26	0.4	1	2	90	0.5	10	10	0.3	0.1	10	1.0	1.0	1.5
75	0.14	0.42	0.33	0.03	0.26	0.4	1	1	161	0.2	0.1	4.5	10	0.2	2.0	0.75	0.5	0.5
76	0.12	0.48	0.25	0.03	0.24	0.35	1	2	155	0.14	0.7	3.0	6.0	0.25	5.0	1.0	1.0	1.0
77	0.12	0.5	0.24	0.03	0.28	0.4	1	2	130	0.1	3.0	10	10	0.1	10	-	-	-
78	0.14	0.42	0.33	0.03	0.26	0.39	1	2	-172	10	0.25	2.0	2.0	10	10	0.5	0.4	0.25
79	0.14	0.4	0.35	0.03	0.26	0.41	1	2	132	0.1	1.3	4.0	4.0	0.1	10	0.75	0.4	0.8
80	0.12	0.46	0.26	0.04	0.28	0.41	1	2	150	0.1	1.0	4.0	10	0.1	1.0	0.7	0.6	0.7
81	0.14	0.54	0.26	0.04	0.36	0.49	1	2	112	0.1	11	13	0.1	0.1	10	0.6	2.2	10
82	0.11	0.4	0.25	0.03	0.26	0.41	1	2	156	0.2	1.0	6.0	10	0.2	3.0	1.2	0.87	0.5
83	0.12	0.36	0.33	0.03	0.24	0.39	1	2	130	0.1	2.0	5.0	0.7	0.1	10	0.8	0.8	0.7
84	0.14	0.6	0.23	0.03	0.32	0.41	1	2	157	0.1	0.67	10	10	0.1	1.0	0.5	0.38	0.33
85	0.12	0.3	0.24	0.02	0.16	0.32	1	3	133	0.16	10	10	1.0	0.3	10	0.7	0.7	0.8
86	0.1	0.48	0.24	0.03	0.28	0.40	1	2	93	0.5	10	10	0.1	0.1	10	0.67	10	10

Case No.	PR	RR	PR/RR	QRS	QT	QTc	ST	T	AXIS	I	II	III	AVR	AVL	AVF	V1	V3	V6
87	0.12	0.5	0.24	0.03	0.3	0.42	1	2	112	0.25	1.2	10	2.0	0.16	3.5	1.3	0.5	0.5
88	0.12	0.44	0.27	0.03	0.3	0.45	1	1	150	0.2	0.4	10	10	0.1	1.0	2.8	1.0	1.0
89	0.1	0.46	0.22	0.02	0.24	0.35	1	2	117	0.2	4.0	10	10	0.1	5.0	0.9	1.0	0.6
90	0.12	0.5	0.24	0.04	0.3	0.43	1	2	120	0.38	10	10	1.0	0.1	10	1.3	1.3	1.7
91	0.16	0.56	0.28	0.03	0.3	0.4	1	2	165	0.17	1.0	10	4.0	0.14	2.5	1.0	0.61	0.67
92	0.12	0.5	0.24	0.03	0.36	0.51	1	2	172	0.2	0.6	2.0	2.5	0.1	1.25	0.5	0.48	0.65
93	0.14	0.6	0.23	0.03	0.34	0.44	1	2	110	0.25	5	9	1	0.17	7.0	0.48	0.83	1.5
94	0.1	0.44	0.23	0.04	0.26	0.39	1	2	128	0.3	2.0	10	2.0	0.14	10	0.75	0.54	0.5
95	0.14	0.4	0.25	0.03	0.24	0.38	1	2	107	0.25	7.0	10	1.0	0.14	10	0.22	0.28	0.37
96	0.12	0.48	0.25	0.03	0.3	0.43	1	2	150	0.2	1.0	4.3	10	0.1	1.0	0.7	0.5	0.63
97	0.1	0.42	0.24	0.03	0.28	0.43	1	2	142	0.1	3.0	5.0	10	0.1	4.0	0.75	0.6	0.7
98	0.12	0.56	0.21	0.04	0.3	0.4	1	2	95	1.0	16	8	0.1	0.25	16	1.1	10	7.0
99	0.12	0.4	0.3	0.03	0.24	0.38	1	2	119	0.4	3.5	10	1.0	0.2	10	0.7	0.83	2.5
100	0.14	0.42	0.29	0.03	0.28	0.43	1	2	124	0.25	2.3	4.0	2.0	0.25	5.5	3.0	3.3	3.3
101	0.1	0.54	0.18	0.03	0.3	0.41	1	2	165	0.3	0.8	4.0	3.0	0.3	1.7	0.8	0.3	0.4
102	0.1	0.42	0.24	0.03	0.26	0.4	1	2	140	0.2	10	6.0	3.0	0.2	10	1.5	2.0	2.5
103	0.1	0.6	0.17	0.03	0.3	0.39	1	2	125	0.43	2.3	10	1.25	0.33	4.0	0.67	0.9	0.8
104	0.1	0.5	0.2	0.03	0.32	0.45	1	2	180	0.25	0.4	3.0	10	0.1	0.8	1.0	1.0	0.89
105	0.12	0.46	0.26	0.03	0.28	0.41	1	2	107	0.25	3.0	10	10	0.25	10	0.75	0.67	0.7
106	0.12	0.4	0.3	0.03	0.2	0.32	1	2	132	0.1	1.5	5.0	10	0.1	10	0.5	0.5	0.7
107	0.12	0.5	0.24	0.02	0.26	0.37	1	2	132	0.33	1.0	10	3.0	0.14	5.0	2.7	1.0	0.8
108	0.12	0.5	0.24	0.03	0.3	0.44	1	2	173	0.1	0.25	10	10	0.1	1.0	1.25	0.4	0.36

APPENDIX 7

MEASUREMENTS OF TIME CONSTANTS WITHIN INDIVIDUAL SAMPLES OF THE FOETAL E.C.G. FROM TWENTY CONSECUTIVE COMPLEXES IN TWO SUBJECTS

Case 74, Mrs. Sheehan

PR	RR	QRS	R/S	QT
0.12	0.42	0.04	1.5	0.22
0.12	0.42	0.04	1.7	0.23
0.12	0.43	0.03	1.6	0.24
0.12	0.42	0.04	1.6	0.2
0.12	0.42	0.04	2.0	0.22
0.12	0.42	0.03	1.6	0.22
0.1	0.42	0.03	1.33	-
0.12	0.43	0.03	1.6	0.24
0.12	0.44	0.03	1.6	0.22
0.12	0.43	0.04	1.6	0.22
0.12	0.42	0.03	1.8	0.22
-	0.42	0.03	1.6	0.22
-	0.42	0.03	1.6	0.22
0.12	0.42	0.03	1.5	0.24
0.13	0.42	0.04	2.0	0.22
0.12	0.42	0.03	2.0	-
0.12	0.42	0.03	1.6	0.22
0.12	0.42	0.04	1.6	0.24
-	0.42	0.03	1.6	0.22
0.12	0.42	0.04	1.7	0.22

- = P or T wave missing.

Case 100, Mrs. Gill

PR	RR	QRS	R/S	QT
0.12	0.4	0.03	1.6	0.22
0.12	0.4	0.03	1.33	0.22
0.13	0.4	0.03	1.6	0.23
0.12	0.41	0.03	1.8	0.22
0.12	0.4	0.03	1.33	0.24
0.12	0.4	0.02	1.33	0.22
0.13	0.41	0.03	1.6	0.24
0.12	0.4	0.04	1.6	0.22
-	0.4	0.04	1.8	0.22
-	0.41	0.03	1.6	0.22
0.12	0.40	0.03	1.6	-
0.12	0.40	0.03	2.5	0.24
-	0.40	0.03	1.6	0.22
0.12	0.41	0.03	1.8	0.22
0.12	0.42	0.03	2.0	0.22
-	0.42	0.03	1.5	0.24
0.12	0.43	0.04	1.75	0.22
0.12	0.44	0.03	1.6	0.24
0.12	0.41	0.03	2.0	0.22
0.14	0.43	0.03	1.6	0.22

P waves not always visible.

The only significant differences between values for time constants in these two subjects occurred in the R/S values.

Case 74	R/S	Case 100	R/S
Mean	1.657	Mean	1.677
Standard deviation	0.169	Standard deviation	.0263
Variance	0.028	Variance	0.069

APPENDIX 8

CASE SUMMARIES

CASE No. 1

Mrs. P. V. L.M.P: 27.6.66

aet 37 years E.D.C: 3.4.67

Four previous pregnancies - all full term normal deliveries. The first pregnancy was complicated by toxaemia, but subsequent pregnancies were normal (1951, 1953, 1955, 1957).

Pregnancy was uneventful until 39 weeks gestation, when the blood pressure rose to 130/90. She was advised to rest at home and was admitted one week later with blood pressure of 150/90 and moderate oedema.

Forewater rupture was performed on 5.4.67 and heavily meconium stained liquor taken. Foetal scalp blood was collected and labour proceeded normally. She was delivered of a healthy male infant, birth weight 4.050 Kgms, the following morning, after a labour lasting 21 hours. Her subsequent recovery was uneventful. Placental weight was not recorded.

CASE No. 2

Mrs. V. P.	L.M.P: 10.7.66
aet 28 years	E.D.C: 17.4.67
Primigravida	

This patient was admitted at 44 weeks draining meconium stained liquor, with mild pre-eclamptic toxaemia. A scalp blood sample was taken.

There was some evidence of cephalo-pelvic disproportion and, after 21 hours in labour, she was delivered of a male infant with an Apgar of 1, by lower segment Caesarean section. Birth weight was 2.450 Kgms. This baby responded rapidly to resuscitation.

She had a mild pyrexia (100.6°) on the second day after delivery, but her recovery was otherwise uneventful.

CASE No. 3

Mrs. A. S.	L.M.P: 10.8.66
aet 21 years	E.D.C: 17.5.67

Two previous pregnancies (1964 and 1965), were both complicated by pre-eclamptic toxaemia.

This pregnancy was uneventful, apart from one episode of urinary tract infection. The patient was admitted on 22.5.57 in early labour, draining clear liquor. During labour, the foetal heart was noted to be irregular, and monitoring was therefore employed. After a labour lasting

five hours she had a normal delivery of a healthy male infant weighing 3.270 Kgms. Post-natal recovery was uneventful.

CASE No. 4

Mrs. I. O. L.M.P: 10.1066

aet 38 years E.D.C: 17.7.67

This patient had a normal pregnancy (premature labour at 36 weeks) in 1955, a premature labour with antepartum haemorrhage at 36 weeks in 1956, and in 1960, antepartum haemorrhage at 32 weeks with premature labour resulting in a child which lived only eight hours.

She was treated with medroxy-progesterone because of suspected placental insuffidency in this pregnancy and poor foetal growth.

She was admitted in threatened premature labour on 25.5.67 and was induced on 1.6.67 because of very low urinary oestriol values. The foetus was monitored during labour.

After a labour lasting six hours, she had a normal delivery of a male infant weighing 2.170 Kgms (placental weight 450 Gms). Her puerperium was complicated by persistent loss per vaginam, and lethargy and irritability, although she remained afebrile.

CASE No. 5

Mrs. O. M. L.M.P: Uncertain of dates

aet 32 years E.D.C: Probably at term

This patient was admitted on 7.6.67, having mild contractions and pushing. The cervix was only 2 cms dilated. Except for some mild pre-eclampsia in the first pregnancy in 1962, all previous pregnancies (1963, 1964 and 1966) were normal. On the day following admission, the foetal heart rate was recorded at 190 beats per minute. After a labour lasting 23 hours she had a normal delivery of a healthy female infant weighing 3.12 Kgms. Her puerperium was uneventful.

CASE No. 6

Mrs. A. H. L.M.P: 20.9.66

aet 19 years E.D.C: 27.6.67

Gravida 2, para 1

This patient had one previous pregnancy in 1964, complicated by severe toxaemia at 32 weeks gestation, and delivery by Caesarean section.

She was first seen at 26 weeks gestation in this pregnancy, with blood pressure 170/120. She was admitted for investigation at this time and considered to be suffering from essential hypertension, and treated with methyl dopa. She was readmitted on two subsequent occasions for

hypertension and proteinuria. On 9.6.67, amnioscopy because of an irregular foetal heart rate showed clear liquor. Artificial rupture of membranes on 10.6.67 produced clear liquor and the labour was monitored. A low forceps delivery was performed after a nine hour labour because of foetal bradycardia. She was delivered of a healthy female infant weighing 2.02 Kgms. Apart from persistent hypertension, her recovery in the puerperium was uneventful.

CASE No. 7

Mrs. P. K. L.M.P: 14.9.66

aet 25 years E.D.C: 21.6.67

Primigravida

This patient had an uncomplicated antenatal course and was admitted to hospital in early labour on 19.6.67. During the following day, contractions were painful but progress was slow.

Approximately 24 hours after admission, the membranes ruptured spontaneously and the liquor was noted to be thickly meconium stained.

A foetal scalp blood sample was collected at 11.00 p.m. and labour allowed to proceed normally. The following morning, low forceps delivery of a healthy female infant was performed.

In the puerperium, secondary postpartum haemorrhage occurred on the fifth day after delivery and therapeutic curettage was performed. The uterus contained only small fragments of membranes. Subsequent recovery was uneventful.

CASE No. 8

Mrs. D. L. L.M.P: 5.9.66
aet 24 years E.D.C: 12.6.67

This patient had an uneventful pregnancy until she was eight days overdue, when her blood pressure rose to 140/95. It was noted that there were "missed beats" on auscultation of the foetal heart.

She was admitted to hospital on 20.6.67 for induction of labour. Forewater rupture was performed and clear liquor obtained. After a labour lasting 19 hours and 25 minutes Kielland's forceps rotation and delivery of a healthy male infant was performed.

Her recovery in the peurperium was uneventful.

CASE No. 9

Mrs. G. D. L.M.P: 26.8.66
aet 21 years E.D.C: 2.6.67
Primigravida

This patient was noted to be hypertensive throughout her pregnancy. She was admitted to hospital for induction

of labour for post-maturity, proteinuria and hypertension, the blood pressure having risen to 160/105. Surgical induction was performed by forewater rupture, and meconium stained liquor was obtained. Foetal tachycardia (180 beats per minute) was also noted. She failed to come into labour after 24 hours and, after a further 12 hours in labour, Caesarean section was performed. She was delivered of a male infant weighing 3.997 Kgms. Her recovery in the puerperium was satisfactory.

CASE No. 10

Mrs. A. T. L.M.P: 19.9.66

aet 19 years E.D.C: 26.6.67

Multigravida - one previous stillbirth associated with antepartum haemorrhage, 1966.

This patient had an uneventful pregnancy and was admitted to hospital on 4.7.67 in early labour. After approximately nine hours in labour, the cervix was only 2-3 cms dilated. The membranes were ruptured artificially and clear liquor obtained. Transient foetal tachycardia was noted and the foetal heart was monitored.

However, progress continued to be slow and, after 20 hours, lower segment Caesarean section was performed and a male infant weighing 3.94 Kgms was delivered. The indication for Caesarean section was failure to progress in labour.

The patient developed a pyrexia on the day following delivery. This responded to antibiotic therapy, and her subsequent recovery was uneventful.

CASE No. 11

Mrs. R. G. L.M.P: 4.9.66
aet 29 years E.D.C: 16.6.67
Primigravida

This patient was admitted as an emergency admission to the Queen Victoria Hospital on 7.7.67, three weeks overdue, with mild pre-eclamptic toxaemia, and in early labour. She was noted to be draining meconium stained liquor, although the foetal heart rate at all times was within normal limits. Six hours after admission, vaginal examination showed that the cervix was effaced and 3-4 cms dilated. A scalp blood sample was taken at this time and the pH found to be 7.17.

Immediate Caesarean section was performed and a limp, flaccid, apnoeic infant was delivered. Resuscitation and intubation was necessary. The mother's progress in the puerperium was uneventful.

CASE No. 12

Mrs. G. B. L.M.P: 10.1066
aet 40 years E.D.C: 17.7.67
Gravida 8, para 7

This patient was a grand multipara known to be suffering from Thalassaemia minor. She was anaemic throughout pregnancy. She had seven previous pregnancies, three of these children having died from unspecified causes in Italy. She had also had two miscarriages.

She was admitted to hospital on 7.7.67 at 38 weeks gestation with mild pre-eclampsia, the blood pressure being 140/85, and there was considerable oedema. Surgical induction was performed on 10th July and lightly meconium stained liquor obtained. She did not establish in labour until the following day, when scalp blood was collected and monitoring commenced. After a labour lasting 20 hours, she had a normal delivery of a healthy male infant. Apart from some delay in uterine involution, her recovery in the puerperium was uneventful.

CASE No. 13

Mrs. A. P. L.M.P: 11.9.66

aet 20 years E.D.C: 18.6.67

Primigravida

This patient was first seen at the Antenatal Clinic when 15 weeks pregnant. Her pregnancy was uneventful until she was admitted to hospital on 11.7.67, when three weeks post-mature, with mild pre-eclampsia.

Surgical induction of labour was performed by fore-water rupture and thickly meconium stained liquor obtained. Some foetal tachycardia was also noted. The foetus was monitored throughout labour and, after 14 hours in labour, the patient had a low forceps delivery of a healthy female infant. In the puerperium, the patient developed a uterine infection 10 days after delivery, which responded rapidly to antibiotic therapy.

CASE No.14

Mrs. C. H.	L.M.P: 4.10.66
aet 18 years	E.D.C: 11.7.67

This patient was a primigravida who had an uneventful pregnancy. She was admitted in early labour draining clear liquor. After a labour lasting eight hours, she had a normal delivery of a healthy male infant.

Her recovery in the puerperium was uneventful.

This patient was monitored as a control normal subject.

CASE No. 15

Mrs. H. W.	L.M.P: ? 27.12.66
aet 18 years	E.D.C: End of August, 1967

This patient was first seen at antenatal clinic on 17.4.67 when she was found to be 22 weeks pregnant.

Her progress was uneventful until she was 37 weeks pregnant, when her blood pressure rose to 150/110 and some oedema developed. She was admitted to hospital and surgical induction of labour was performed.

After a labour lasting 12 hours, the patient had a normal delivery of a healthy male infant. Her recovery in the puerperium was uneventful.

CASE No. 16

Mrs. V. D. L.M.P: 25.10.66

aet 30 E.D.C: 1.8.67

Gravida 4, para 2

This patient had a normal delivery in 1960 and in 1963. She had a miscarriage at five weeks gestation in 1962. This present pregnancy was uneventful apart from some difficulties with bronchiectasis.

Labour started spontaneously on 7th August, 1967 and on the following day she was noted to be draining meconium stained liquor and the foetal heart rate was 220 beats per minute. Initial scalp sample was normal but subsequently fell to pH 7.20.

A female infant was delivered by Caesarean section and her subsequent recovery was uneventful.

CASE No. 17

Mrs. P. O'N — L.M.P: ? end October, 1966

aet 21 years — E.D.C: August 1967

Gravida 1

This 21 year old primigravida had an uneventful pregnancy and was admitted to hospital in early labour on 15th August, 1967. When examined vaginally five hours after admission, the cervix was found to be 4 cms dilated and the liquor was meconium stained. A paracervical block was inserted and a scalp blood sample collected.

Approximately two hours later she had a normal delivery of a healthy male infant. Her subsequent recovery in the puerperium was uneventful.

CASE No. 18

Miss K. S. — L.M.P: Uncertain of dates

aet 16 years — E.D.C: ? August 1967

Primigravida

This patient had an uneventful pregnancy. She was admitted to hospital on 9th August, 1967 in false labour and was discharged home again two days later. She was admitted to hospital again on 16th August in established labour. The foetal heart rate was noted to be rapid and the liquor was lightly meconium stained. A foetal scalp sample was collected. After a labour lasting nine hours,

the patient had a normal delivery of a healthy female infant. The placenta was retained and subsequently removed manually.

On the day following delivery, the patient developed a pyrexia of 38.6°C but this settled spontaneously without any specific therapy and her subsequent recovery was uneventful.

CASE No. 19

Miss M. S. L.M.P: 14.12.66
aet 24 years E.D.C: 21.9.67
Primigravida

This patient was admitted to hospital on 21st August, 1967 in early labour. She had no antenatal care throughout her pregnancy.

On admission to hospital, she was noted to be draining heavily meconium stained liquor. Labour progressed slowly, and 12 hours after admission the cervix was noted to be only 4 cms dilated. A scalp blood sample was collected and the foetal heart monitored until delivery. After a labour lasting 23 hours she had a normal delivery of a healthy male infant weighing 3.21 Kgms, which appeared to be a full term infant. Her recovery in the puerperium was uneventful.

CASE No. 20

Mrs. V. N. L.M.P: 6.11.66
aet 29 years E.D.C: 13.8.67
Gravida 3, para 0

This patient had two previous miscarriages, one in 1965 at 24 weeks gestation and one in 1966 at 12 weeks gestation.

Her third pregnancy was uneventful until she was nine days past term, when she was noted to have a mild elevation of blood pressure (130/90) and she was admitted to hospital.

On the following day, surgical induction of labour was performed and lightly meconium stained liquor obtained. After a labour lasting 25½ hours in the first stage and one hour in the second stage, she had a normal delivery of a healthy female infant weighing 3.41 Kgms.

Her recovery in the puerperium was uneventful.

CASE No. 21

Mrs. D. D. L.M.P: 29.11.66

aet 27 years E.D.C: 5.9.67

Gravida 3, para 2

The first pregnancy in 1960 was complicated by pre-eclamptic toxaemia. In 1963, this patient's second pregnancy was uneventful.

She presented to clinic for care of this, her third pregnancy, when 26 weeks pregnant. The pregnancy was uneventful until she was admitted to hospital on 31st August with ruptured membranes draining lightly meconium stained liquor. After a short labour lasting only 3½ hours, the

patient had a low forceps delivery of a healthy female infant weighing 3.65 Kgms.

Her subsequent recovery was uneventful.

CASE No. 22

Mrs. L. D. L.M.P: 24.11.66

aet 19 years E.D.C: 31.8.67

Gravida 2, para 0

This patient had one previous pregnancy in 1966, which ended in a miscarriage at eight weeks gestation.

This present pregnancy was uncomplicated and the patient was admitted to hospital on 4th September 1967, four days past term, in early labour. On the following day, labour became established and the foetal heart was noted to be 118 beats per minute. Forewater rupture was performed and the liquor found to be clear. It later became meconium stained. A scalp blood sample was taken and the pH found to be only 7.22. However, as the mother also showed signs of acidosis (pH 7.32 in venous sample), intravenous therapy was started and a further scalp sample collected four hours later. Spontaneous delivery of a healthy female child occurred. Recovery in the puerperium was uneventful.

CASE No. 23

Mrs. D. M. L.M.P: 19.12.66

aet 30 years E.D.C: 26.9.67

Gravida 3, para 1

This patient's first pregnancy in 1966 ended in a miscarriage at 2 months' gestation. In her second pregnancy in 1966 she developed toxaemia of pregnancy and delivered a normal male infant at 36 weeks gestation.

During this pregnancy, she was found to be a mild diabetic. She was admitted to hospital two weeks before term and surgical induction of labour was performed. After a labour lasting only 2½ hours, she had a normal delivery of a healthy female infant. Her recovery in the puerperium was uneventful.

CASE No. 24

Mrs. F. M. L.M.P: 2.12.66

aet 23 years E.D.C: 9.9.67

Gravida 2, para 1

This patient had one previous normal pregnancy in 1965. This present pregnancy was uneventful until five days over term, when signs of mild toxaemia were detected. The following day, the patient was admitted to hospital in early labour draining thickly meconium stained liquor. After a labour lasting 12 hours, a low forceps delivery was performed

and a healthy female infant delivered.

Recovery in the puerperium was uneventful.

CASE No. 25

Miss J. H. L.M.P: 6.12.66

aet 20 years E.D.C: 13.9.67

Primigravida

This patient was noted to have some degree of hypertension throughout pregnancy. She was an obese woman and weighed 102 Kgms when first seen at clinic.

She was admitted to hospital on 19th September, 1967 when she was six days past term, with moderately severe toxaemia (blood pressure 150/100 - cloud of proteinuria) and, on the following day, was assessed with a view to surgical induction. The cervix was unsuitable and, after another five days in hospital, a further attempt at surgical induction was made and forewater rupture performed. Meconium stained liquor was obtained and the foetal heart rate was noted to be within normal limits. On the following day, a scalp blood sample was taken and found to be normal. Despite marked variation in foetal heart rate, labour was allowed to proceed and, after a labour lasting 34 hours, she had a normal delivery of a male infant.

The patient's recovery in the puerperium was uneventful

and at six weeks the blood pressure was normal.

CASE No. 26

Mrs. O. R. L.M.P: 10.1.67

aet 27 years E.D.C: 17.10.67

Gravida 3, para 2

This patient had two previous pregnancies (1963 and 1964) complicated by antenatal anaemia. This pregnancy was also complicated by anaemia and threatened abortion at 10 weeks' gestation. She was admitted to hospital on 3rd October, 1967 in labour, draining meconium stained liquor. On vaginal examination she was found to have a face presentation which was digitally corrected to a vertex presentation and a scalp blood sample taken. After a labour lasting ten hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 27

Mrs. C. S. L.M.P: January 1967

aet 19 years E.D.C: October 1967

Primigravida

This patient was admitted to hospital as an emergency admission, having had no antenatal care. She was in early labour and was noted to have some foetal bradycardia.

Forewater rupture of the membranes produced clear liquor and, after a labour lasting 19 hours, she had a low forceps delivery of a healthy male infant. Her recovery in the puerperium was uneventful.

Case No. 28

Miss M. P. L.M.P: March 1967
aet 24 years E.D.C: December 1967
Gravida 2, para 1

This patient had one previous normal pregnancy in 1964. The estimated date of confinement was thought to be December 1967, but the actual date of confinement was 23rd October, 1967. The patient was in hospital when 30 weeks pregnant, with a small antepartum haemorrhage. She was readmitted one month later with a further small haemorrhage and one month after this she was admitted in early labour.

In view of her history of repeated small haemorrhages, the foetus was monitored during labour. After a labour lasting 20 hours, she had a normal delivery of a healthy male infant. Recovery in the puerperium was uneventful.

CASE No. 29

Mrs. H. L. L.M.P: 6.1.67
aet 32 years E.D.C: 13.10.67
Gravida 5, para 3

This patient had a normal pregnancy in 1957 and in 1959. In 1964, her third pregnancy was complicated by hypertension and foetal distress and postpartum haemorrhage. In 1965, she had a miscarriage at three months' gestation.

This pregnancy was uneventful until she reached term, when she developed mild pre-eclamptic toxaemia. This settled with bed rest but subsequently recurred. On 2.11.67 surgical induction of labour was performed and clear liquor obtained. After a labour lasting eight hours she had a normal delivery of a healthy female infant. Recovery in the puerperium was uneventful.

CASE No. 30

Mrs. A. K. L.M.P: 9.2.67

aet 25 years E.D.C: 16.11.67

Gravida 3, para 1

This patient had her first pregnancy in 1964, which ended in a miscarriage at three months' gestation. Her second pregnancy (1965) was uneventful.

This present pregnancy was uneventful, apart from iron deficiency anaemia. She was admitted to hospital in early labour with mild pre-eclamptic toxaemia two days before term and, after a labour lasting 11 hours, had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 31

Mrs. S. S. L.M.P: 4.2.67

aet 19 years E.D.C: 11.11.67

Primigravida

This patient had an uneventful pregnancy and was admitted to hospital when ten days past term, in early labour. Artificial rupture of the membranes was performed when the cervix was at half dilatation, and clear liquor was obtained. After a labour lasting 11 hours, the patient had a normal delivery of a healthy female infant.

Her recovery in the puerperium was uneventful.

CASE No. 32

Mrs. A. M. L.M.P: 10.3.67

aet 27 years E.D.C: 17.12.67

Gravida 4, para 0

This patient had a history of a miscarriage at 24 weeks in 1962. In 1963 she had a miscarriage at 20 weeks gestation and gave birth to twins which died. In 1965, premature labour occurred at 28 weeks and the infant lived only 48 hours. All pregnancies were complicated by urinary tract infections.

During this pregnancy, she was found to have a calculus

in her right ureter. At 20 weeks gestation, threatened abortion occurred and therapy with medroxyprogesterone was instituted. On 19.11.67, at 36 weeks gestation, premature rupture of the membranes occurred and, two days later, labour started. After a labour lasting 3 hours 40 minutes, normal vaginal delivery occurred of a healthy premature male infant.

Recovery in the puerperium was uneventful.

CASE No. 33

Mrs. C. G. L.M.P: 27.1.67

aet 22 years E.D.C: 3.11.67

Gravida 2, para 1

This patient had one previous pregnancy in 1963, with a forceps delivery at full term.

This pregnancy was uncomplicated until three weeks past the expected time of confinement, when she was admitted to hospital in early labour, draining meconium stained liquor. After a labour lasting 18 hours, she had a normal delivery of a living male infant. The placenta was retained and subsequently removed manually.

Recovery in the puerperium was uneventful.

CASE No. 34

Mrs. M. D. L.M.P: 17.2.67

aet 31 years E.D.C: 24.11.67

Gravida 4, para 3

This patient had three previous normal pregnancies and confinements. This pregnancy was uneventful until six days past the estimated date of confinement, when the patient developed hypertension (blood pressure 150/100). She was admitted to hospital on 29.11.67 and, on the following day, surgical induction of labour was performed and clear liquor obtained. After a labour lasting eight hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 35

Mrs. M. H. L.M.P: 2.3.67

aet 32 years E.D.C: 9.12.67

Gravida 3, para 1

The first pregnancy in 1964 ended in a miscarriage at five months' gestation. In 1967, the patient's second pregnancy resulted in a premature labour, and the child lived only one day.

The patient was admitted as an emergency when 37 weeks pregnant, having been diagnosed as a diabetic, and being treated with insulin and tolbutamide. She was also noticed

to be hypertensive (blood pressure 150/100), on admission. There was some doubt about the diagnosis of diabetes, and all insulin and tolbutamide therapy was discontinued. Glucose tolerance test showed a "long curve". The hypertension persisted and, on 4.12.67, at 39 weeks gestation, surgical induction of labour was performed. Meconium stained liquor was obtained and, after 15 hours in labour, persistent tachycardia developed. Caesarean section was performed and a large male infant delivered.

The puerperium was complicated by uterine and wound infection, and the infant progressed satisfactorily.

CASE No. 36

Mrs. C. G. L.M.P: 15.3.67

aet 28 years E.D.C: 22.12.67

Primigravida

This patient had an uneventful pregnancy until 11 days before term, when she was found to have an elevated blood pressure (160/110) and some generalised oedema. She was admitted to hospital the same day and sedated and three days later labour was induced by forewater rupture. Her urine was clear.

Clear liquor was obtained and, after a labour lasting 9½ hours, she had a low forceps delivery of a healthy male infant. Recovery in the puerperium was uneventful.

CASE No. 37

Miss M. B. L.M.P: 8.3.67

aet 15 years E.D.C: 15.12.67

Primigravida

This young single primigravida had an uneventful pregnancy. She was admitted to hospital in early labour on 18th December 1967 - three days past term. The following morning, the patient was examined vaginally to assess her progress and the membranes were ruptured. Heavily meconium stained liquor was released and a foetal scalp blood sample was taken. After a labour lasting 30 hours, low forceps delivery of a female infant was performed. Active resuscitation was necessary for the infant.

Recovery in the puerperium was uneventful.

CASE No. 38

Mrs. J. S. L.M.P: 4.3.67

aet 21 years E.D.C: 11.12.67

Primigravida

This patient had an uneventful pregnancy until five days before term, when she was admitted to hospital with suspected ruptured membranes. However, no evidence of ruptured membranes was obtained after admission to hospital, and she was discharged home on 7th December. The following day she was readmitted to hospital with a history of painless

vaginal blood loss. No further blood loss occurred after admission, and she was discharged home on 10th December. She was readmitted a third time on 21st December in early labour.

In view of her antenatal history of blood loss, forewater rupture was performed. The liquor was clear and the foetus was monitored. After a labour lasting ten hours, she had a normal delivery of a healthy male infant. Recovery in the puerperium was uneventful.

CASE No. 39

Mrs. M. C. L.M.P: March 1967

aet 32 years E.D.C: ? 2.1.68

Para 3, Gravida 4

This patient had her first pregnancy in 1960. The foetus died in labour and was stillborn. In 1962, she had a forceps delivery of a normal infant and in 1964, normal delivery of a child that showed signs of foetal distress during labour.

This pregnancy was uneventful until the patient developed mild pre-eclamptic toxaemia one week before term. She was admitted to hospital on 27th December and labour induced by forewater rupture. Clear liquor was obtained. Later the same day she was given some buccal oxytocin and, after this, some foetal tachycardia was noted. The foetus was

monitored throughout the remainder of the labour. After a labour lasting seven hours, she had a normal delivery of a healthy male infant. Her recovery in the puerperium was uneventful.

CASE No. 40

Mrs. J. S. L.M.P: Early February 1967
aet 21 years E.D.C: November 1967
Primigravida

This patient was first seen at clinic when she was eight weeks pregnant. Her pregnancy was uneventful until she was admitted to hospital when she was thought to be at term, in early labour. When she was well established in labour, vaginal examination was performed and heavily meconium stained liquor seen. The foetus was monitored and, after a labour lasting 19 hours, she had a normal delivery of a healthy female infant. Her recovery in the puerperium was uneventful.

CASE No. 41

Mrs. D. F. L.M.P: 3.3.67
aet 21 years E.D.C: 10.12.67
Primigravida

This patient was first seen at antenatal clinic when she was thought to be 30 weeks pregnant. Her pregnancy was uneventful, but there was some doubt about her expected

date of confinement. She was admitted to hospital in early labour on 4th January 1968, when it was thought that she was 25 days past term. Marked variation in foetal heart rate was noted and forewater rupture was performed. Clear liquor was obtained. After a labour lasting six hours, she had a low forceps delivery of a healthy female infant. Her recovery in the puerperium was uneventful.

CASE No. 42

Mrs. S. D. L.M.P: 6.5.67

aet 28 years E.D.C: 13.2.68

Para 1, gravida 2

This patient was delivered by Caesarean section in her first pregnancy in 1960 because of persistent and severe pre-eclamptic toxaemia.

This present pregnancy was uneventful until she was noted at clinic, when 36 weeks pregnant, to have a bl od pressure of 140/90 and some ankle oedema. This settled with rest at home. She was admitted to hospital in early labour on 1st February 1968 and, in view of her previous history, the foetus was monitored during labour. After a labour lasting ten hours, she had a normal delivery of a healthy female infant. Her recovery in the puerperium was uneventful.

CASE No. 43

Mrs. K. L. L.M.P: 2.5.67

aet 24 years E.D.C: 9.2.68

Para 3, gravida 4

This patient had three previous normal pregnancies in 1963, 1964 and 1966.

This pregnancy was uneventful and she was admitted to hospital in labour four days after term.

Monitoring of the foetus was undertaken, using this patient as a "control" normal subject. After a labour lasting ten hours, she had a normal delivery of a male infant with an Apgar score of four at one minute and 10 at five minutes. On the third day after delivery she developed a pyrexia of 101^{o}F. This was treated symptomatically and settled the following day. The cause of the pyrexia was unknown. Her recovery was otherwise uneventful.

CASE No. 44

Miss K. P. L.M.P: 20.4.67

aet 18 years E.D.C: 27.1.68

Primigravida

This young primigravida had an uneventful pregnancy. However, it was noted that she had a contracted pelvis, and this was confirmed radiologically.

She was admitted to hospital in early labour 20 days past term. The liquor was clear but scanty. After a trial of labour lasting 21 hours, it was clear that the labour had become obstructed and Caesarean section was performed. She was delivered of a healthy male infant.

She developed a breast infection in the puerperium, which responded to antibiotic therapy. Her recovery was otherwise uneventful.

CASE No. 45

Mrs. F. C. L.M.P: Uncertain of dates

aet 18 years E.D.C: February 1968

Primigravida

This patient first attended antenatal clinic when she was 34 weeks pregnant. At 38 weeks she was admitted to hospital with suspected premature rupture of the membranes and mild pre-eclampsia. No liquor was seen after admission and the pre-eclamptic toxaemia settled. The patient was allowed to go home four days later.

She was readmitted to hospital on 19th February 1968 in early labour. Forewater rupture was performed and clear liquor obtained. After a prolonged labour lasting 26 hours, she had a normal delivery of a healthy female infant.

Her recovery in the puerperium was uneventful.

CASE No. 46

Mrs. V. G. L.M.P: 18.5.67

aet 30 years E.D.C: 25.2.68

Para 1, gravida 3

This patient's first pregnancy in 1965 ended in miscarriage at 14 weeks gestation.

In 1967 she had a forceps delivery of a full term infant for foetal distress. The child died two days later from the effects of cerebral anoxia.

During this pregnancy she had a threatened miscarriage at 10 weeks gestation. Her progress was otherwise uneventful. In view of her previous history, she was admitted to hospital at 39 weeks gestation for induction of labour. Clear liquor was obtained at the time of surgical induction and, after a labour lasting seven hours, she had a normal delivery of a healthy female infant. Her recovery in the puerperium was uneventful.

CASE No. 47

Mrs. M. S. L.M.P: 19.5.67

aet 18 years E.D.C: 26.2.68

Para 1, gravida 2

This patient had her first pregnancy in 1966. At 39 weeks gestation the foetus died in utero and she was

delivered of a stillborn foetus at term. The cause of the stillbirth was unknown.

Her progress during this pregnancy was uneventful. She was admitted to hospital at term, in labour. Forewater rupture was performed and meconium stained liquor obtained. The foetus was monitored and, after a labour lasting ten hours, manual rotation and forceps delivery of a depressed male infant was performed. The infant was resuscitated by endotracheal intubation and intermittent positive pressure respiration.

Recovery in the puerperium was uneventful.

CASE No. 48

Mrs. K. W. L.M.P: ? July 1967

aet 36 years E.D.C: ? 30.4.68

Gravida 8, para 6

This patient has had six children and one miscarriage. Two previous pregnancies had been complicated by deep vein thrombosis and one pregnancy had ended in a miscarriage at 18 weeks gestation.

The present pregnancy was uneventful until she was admitted to hospital on 24th February, having some uterine contractions. She insisted on "pushing" on a closed cervix. Two days later vaginal examination revealed a cervix that was only 3 cms dilated. Foetal bradycardia was noted and

forewater rupture was performed. Clear liquor was obtained and the foetus was monitored. After a labour lasting five hours, she had a normal delivery of a healthy male infant. Her recovery in the puerperium was uneventful.

CASE No. 49

Mrs. V. P. L.M.P: 18.5.67

aet 20 years E.D.C: 25.2.68

Primigravida

This patient was admitted to hospital in the first trimester of her pregnancy for treatment of hyperemesis gravidarum. Her subsequent progress was uneventful, although she was thought to have some evidence of cephalo-pelvic disproportion at term. She was admitted to hospital on 19th February in early labour, but did not become established in labour until 29th February, when the membranes ruptured spontaneously and clear liquor was seen. Her blood pressure was noted to have risen to 145/100 at this time and the foetal heart rate was recorded at 114 beats per minute. The foetus was monitored and, after a labour lasting 30 hours, she had a forceps rotation and delivery of a living male infant. In the puerperium, she had some difficulty voiding urine and was also noted to have some infection of her perineum. When seen for her six week post-natal visit she had completely recovered.

CASE No. 50

Mrs. D. D. L.M.P: 10.5.67

aet 36 years E.D.C: 17.2.68

Gravida 9, para 8

This patient has six living children, two of her children having died in infancy. Her last pregnancy, in 1963, was complicated by pre-eclamptic toxaemia.

She did not present to antenatal clinic with this pregnancy until 36 weeks gestation. On 4th March, 15 days past term, surgical induction of labour was performed and clear liquor obtained. Contractions were infrequent and on the following day the foetal heart beat was noted to be irregular. After a labour lasting five hours, she had a normal delivery of a healthy male infant. Recovery in the puerperium was uneventful.

CASE No. 51

Mrs. A. F. L.M.P: 29.6.67

aet 42 years E.D.C: 5.4.68

Gravida 3, para 1

This patient had one previous normal pregnancy in 1950, and a miscarriage at 12 weeks gestation in 1956. This pregnancy was complicated by pre-eclamptic toxaemia at 34 weeks gestation. She was admitted to hospital on 7th March for treatment of pre-eclampsia and, on 11th March, surgical

induction of labour was performed by forewater rupture, and heavily meconium stained liquor obtained. Delivery by Caesarean section was suggested, but the patient declined. The foetus was monitored throughout labour and, after a labour lasting 9½ hours, a forceps delivery of a healthy female infant was performed. Recovery in the puerperium was uneventful.

CASE No. 52

Mrs. I. T. L.M.P: ? April 1967

aet 46 years E.D.C: ? January 1968

Gravida 4, para 2

This patient had two previous normal full term pregnancies (1945 and 1953) and a miscarriage at 10 weeks gestation in 1954. She was uncertain of the date of her last menstrual period and presented to clinic in February 1968, when she was thought to be 30 weeks pregnant.

Her pregnancy was uneventful, and she was admitted to hospital in early labour on 19th March 1968. The foetal heart beat was noted to be irregular, with wide variation in rate. Artificial rupture of the membranes was performed and clear liquor obtained. After a labour lasting two hours and 15 minutes, she had a normal delivery of a healthy female infant. Her recovery in the puerperium was uneventful.

CASE No. 53

Mrs. M. H. L.M.P: 12.6.67

aet 21 years E.D.C: 19.3.68

Primigravida

This patient had an uncomplicated pregnancy apart from excessive ankle oedema at 36 weeks gestation, which was treated by bed rest and a diuretic.

She was admitted to hospital on 21st March 1968, two days overdue, with blood pressure 130/90, gross ankle oedema and proteinuria. Forewater rupture was performed and clear liquor obtained. After a labour lasting ten hours, she had a normal delivery of a healthy male infant. On the fifth day of the puerperium, she developed a pyrexia of 100°F. This settled to normal the following day, without any specific therapy.

Her recovery was otherwise uneventful.

CASE No. 54

Mrs. M. S. L.M.P: Uncertain

aet 19 years E.D.C: End of March 1968

Primigravida

This patient was admitted to hospital as an emergency admission with ruptured membranes, draining meconium stained liquor, and a foetal heart rate of 190 beats/minute. She

was uncertain of her dates, but was thought to be 36 weeks pregnant.

The foetus was monitored but did not become established in labour until the following day, when a dilute syntocinon drip was started. On 26th March, regular contractions started and, after five hours, she had a mid-cavity forceps delivery of a healthy female infant. Recovery in the puerperium was uneventful.

CASE No. 55

Mrs. P. A. L.M.P: June 1967

aet 22 years E.D.C: March 1968

Primigravida

This patient had an uneventful antenatal history, apart from one episode of superficial thrombophlebitis at 36 weeks gestation.

She was admitted to hospital in early labour on 30th March 1968 and, at this time, the foetal heart beat was noted to be irregular. Forewater rupture was performed but no liquor was obtained. After a labour lasting 14 hours, Kielland's forceps rotation and delivery was performed under pudendal nerve block and a female infant with an Apgar score of 2 after one minute was delivered. Recovery in the puerperium was uneventful.

The infant showed a persistent tachycardia after delivery and was thought to be in cardiac failure, with an enlarged liver and spleen. The infant was digitalised but the cause of the supraventricular tachycardia remained uncertain. A cardiomyopathy or myocarditis was suspected, but the infant slowly improved.

CASE No. 56

Mrs. S. W. L.M.P: 5.6.67

aet 22 years E.D.C: 12.3.68

Gravida 3, para 2

This patient has had two previous normal confinements (1962 and 1966).

This pregnancy was uneventful until three weeks past term, when the patient developed mild pre-eclampsia with hypertension and excessive weight gain.

She was admitted to hospital on 1st April 1968. Fore-water rupture was performed the following day and meconium stained liquor obtained. After a labour lasting 11 hours, she had a normal delivery of a healthy male infant. Recovery in the puerperium was uneventful.

CASE No. 57

Mrs. O. W. L.M.P: Mid June 1967

aet 44 years E.D.C: March 1968

Gravida 10, para 9

This patient had nine previous full term pregnancies, the last two being complicated by hypertension. At the first visit to antenatal clinic, the patient was thought to be 30 weeks pregnant. The blood pressure was 160/90 and treatment was started with methyl dopa. The patient was admitted to hospital at term for induction of labour because of hypertension. On 4th April, surgical induction of labour was performed by forewater rupture of the membranes, and clear liquor was obtained. After a labour lasting two hours, she had a normal delivery of a healthy male infant.

Recovery in the puerperium was uneventful.

CASE No. 58

Mrs. A. P. L.M.P: 9.7.67

aet 32 years E.D.C: 16.4.68

Primigravida

This patient had an uneventful pregnancy until 38 weeks gestation, when she developed a urinary tract infection. She was admitted to hospital on 2nd April 1968 with persistent proteinuria and signs of mild pre-eclamptic toxaemia.

Surgical induction of labour by forewater rupture was performed one week later and clear liquor was obtained. She was later given a dilute oxytocin drip and, after a

labour lasting 17 hours, had a normal delivery of a healthy male infant.

Apart from some bruising and oedema of the perineum, recovery in the puerperium was uneventful. She was readmitted to hospital two weeks later with a secondary post-partum haemorrhage which settled with conservative management.

CASE No. 59

Mrs. M. H. L.M.P: 19.7.67
aet 22 years E.D.C: 26.4.68
Primigravida

This patient had a normal pregnancy until 34 weeks gestation, when she was noted to have developed generalised oedema and excessive weight gain.

Subsequently, the patient developed a severe hypertension of 210/130 and proteinuria. Surgical induction of labour was performed by forewater rupture on 16th April, 1968 and, after a prolonged labour lasting 30 hours, forceps delivery of a male infant was performed. The placenta was noted to be small and infarcted.

Epidural anaesthesia was used during labour and the forceps delivery was performed under general anaesthesia. Recovery in the puerperium was uneventful.

CASE No. 60

Mrs. M. P. L.M.P: 20.8.67

aet 20 years E.D.C: 27.5.68

Gravida 2, para 1

This patient had her first pregnancy in 1967 and this child was delivered by Caesarean section because of incoordinate uterine action.

Her progress during this pregnancy was uneventful, although she did not attend the antenatal clinic for the last five weeks before admission to hospital at 35 weeks gestation. At this time, she was admitted with a history of vaginal bleeding and the onset of painful contractions. Two days later, pelvic examination was performed and the cervix found to be 3 cms dilated. Forewater rupture was performed and clear liquor obtained. A foetal scalp sample was taken and 1½ hours later, low forceps delivery of an asphyxiated male infant was performed. The placenta was markedly adherent and was removed manually under general anaesthesia.

Her recovery in the puerperium was uneventful.

CASE No. 61

Mrs. J. McA L.M.P: 17.7.67

aet 31 years E.D.C: 24.4.68

Gravida 4, para 2

This patient has previously had normal pregnancies and full term deliveries in 1957 and 1958. In 1967, she had a miscarriage at 12 weeks gestation.

Her present pregnancy was uneventful. She was admitted to hospital in early labour five days past term. The following day, she was noted to have persistent foetal tachycardia and the foetal heart rate was monitored. After becoming properly established, labour lasted six hours, and delivery of a healthy male infant occurred on 30th April. Recovery in the puerperium was uneventful.

CASE No. 62

Mrs. S. B. L.M.P: 20.7.67

aet 18 years E.D.C: 27.4.68

Primigravida

This patient had an uneventful pregnancy apart from glycosuria on several occasions. Three days past term, she developed mild toxaemia of pregnancy. On 2nd May, surgical induction of labour was performed by forewater rupture and clear liquor was obtained. After a labour lasting 25 hours, she had a normal delivery of a healthy male infant. The placenta was retained, and manual removal was performed under general anaesthesia. Recovery in the puerperium was uneventful.

CASE No. 63

Miss C. C. L.M.P: 3.7.67

aet 15 years E.D.C: 10.4.68

Primigravida

This young primigravida was first seen at antenatal clinic when 36 weeks pregnant by dates. The height of the uterine fundus was thought to correspond with the dates. An abdominal X-ray was taken on 6th May, and showed a full-term foetus. Because of mild pre-eclampsia, surgical induction of labour was performed by forewater rupture on 7th May, and clear liquor obtained.

After a labour lasting 11 hours, she had a low forceps delivery under pudendal nerve block on a healthy female infant. Apart from persistent hypertension, recovery in the puerperium was uneventful.

CASE No. 64

Mrs. C. T. L.M.P: 21.9.67

aet 20 years E.D.C: 28.6.68

Primigravida

This patient had an uneventful pregnancy until 36 weeks gestation, when she was noted to have developed proteinuria and had excessive weight gain. The proteinuria persisted and the blood pressure rose to 160/90. Surgical induction of labour was performed by forewater rupture on 14th May

and clear liquor obtained. After a labour lasting 10 hours 45 minutes, the patient had a forceps delivery of a small healthy female infant. The placenta was noted to be infarcted.

The patient's recovery in the puerperium was uneventful.

CASE No. 65

Mrs. L. A. L.M.P: 5.8.67

aet 26 years E.D.C: 12.5.68

Gravida 3, para 2

This patient's first pregnancy in 1963 was complicated by pre-eclamptic toxaemia and she was delivered by Caesarean section. In 1965, she had an uncomplicated pregnancy and a normal vaginal delivery of a full term pregnancy.

This present pregnancy was uneventful until the patient was admitted to hospital on 16th May 1968, in early labour, and was noted to have mild pre-eclampsia. Forewater rupture was performed and meconium stained liquor obtained. After a labour lasting 1 hour and 15 minutes, the patient had a normal confinement of a healthy male infant. Recovery in the puerperium was uneventful.

CASE No. 66

Mrs. M. H. L.M.P: 4.8.67

aet 41 years E.D.C: 11.5.68

Gravida 13, para 9

This patient had a previous history of six normal pregnancies followed by three pregnancies complicated by hypertension. Three pregnancies ended in miscarriage.

She did not present in this pregnancy until the 38th week of gestation. At this time, the blood pressure was noted to be 190/115 but she refused admission to hospital. The patient was subsequently admitted on 17th May and surgical induction of labour was attempted the same day. No liquor was obtained.

The following day, speculum examination revealed a small quantity of meconium stained liquor. A dilute oxytocic infusion was started and, after a labour lasting seven hours, she had a normal delivery of a male infant with an Apgar score of 2 at one minute. Apart from persistent hypertension, recovery in the puerperium was uneventful.

CASE No. 67

Mrs. A. N. L.M.P: 27.7.67

aet 19 years E.D.C: 3.5.68

Gravida 3, para 1

This patient had one previous full term delivery in 1966, in which the labour was complicated by foetal distress. Her second pregnancy, in 1967, ended in miscarriage at six weeks gestation.

This present pregnancy was uneventful until three weeks past term, when the patient developed mild pre-eclampsia. She was admitted to hospital in early labour on 23rd May and foetal bradycardia was noted. Forewater rupture was performed and clear amniotic fluid was obtained. After a labour lasting seven hours, she had a normal delivery of a female infant. The infant was found to have an ABO blood group incompatibility and two exchange transfusions were necessary.

The patient made an uneventful recovery in the puerperium.

CASE No. 68

Mrs. S. M. L.M.P: 20.9.67

aet 22 years E.D.C: 27.6.68

Gravida 2, para 1

This patient had one previous pregnancy complicated by pre-eclamptic toxaemia in 1966.

This pregnancy was uneventful until 36 weeks gestation, when the patient was admitted to hospital with premature

rupture of the membranes, and draining clear liquor. The following day, the foetal heart rate was noted to be 190-200 beats/minute. Labour was stimulated with an oxytocin infusion. After a labour lasting five hours, she had a normal delivery of a healthy female infant. On the day following delivery, the infant developed respiratory distress, but subsequently made a satisfactory recovery.

Recovery in the puerperium was uneventful.

CASE No. 69

Mrs. M. D. L.M.P: 14.8.67
aet 23 years E.D.C: 21.5.68
Primigravida

This patient had an uneventful pregnancy. She was admitted to hospital on 5th June, 1968 in early labour with mild pre-eclamptic toxaemia. After a labour lasting four hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 70

Mrs. J. M. L.M.P: 15.8.67
aet 22 years E.D.C: 22.5.68
Primigravida

This patient was admitted to hospital as an emergency admission with pre-eclamptic toxaemia. Labour started

spontaneously on 6th June and the liquor was found to be clear. After a labour lasting 18 hours, the patient had a Kielland's forceps rotation and delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 71

Mrs. I. C. L.M.P: 15.10.67

aet 19 years E.D.C: 22.7.68

Primigravida

This young primigravida had an uneventful pregnancy until she developed pre-eclamptic toxaemia at 34 weeks gestation. She developed persistent proteinuria and hypertension which did not settle with conservative management. Labour was induced with a syntocinon infusion and later forewater rupture was performed and clear liquor obtained. Lumbar epidural anaesthesia was used for pain relief during labour.

After a labour lasting 22 hours, she had a manual rotation and forceps delivery of a living male infant. The infant showed signs of respiratory depression after birth and endotracheal intubation was necessary. The depression was thought to be due to the effects of heavy sedation during labour.

Recovery in the puerperium was uneventful.

CASE No. 72

Mrs. A. G. L.M.P: 8.9.67

aet 39 years E.D.C: 15.6.68

Gravida 11, para 7

The patient had six previous normal pregnancies and three miscarriages. This pregnancy was normal and the labour was normal. She was selected as a control subject.

Forewater rupture was performed early in labour and clear liquor obtained. After a labour lasting 13 hours, she had a normal delivery of a healthy male infant.

Recovery in the puerperium was uneventful.

CASE No. 73

Mrs. E. A. L.M.P: 8.9.67

aet 21 years E.D.C: 15.6.68

Primigravida

This patient had an uneventful pregnancy until she was noted at term to have mild pre-eclamptic toxaemia. She was admitted to hospital in early labour on 19th June and forewater rupture was performed. Slightly meconium stained liquor was obtained. After a labour lasting 13 hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 74

Miss G. S. L.M.P: 17.9.67

aet 17 years E.D.C: 24.6.68

Primigravida

This patient had a normal pregnancy until she was 37 weeks pregnant, when she developed mild pre-eclamptic toxaemia. The toxaemia responded to conservative management and, one week later, she was discharged from hospital. She was readmitted to hospital eight days later with a recurrence of toxaemia and, on 25th June, surgical induction of labour was performed by forewater rupture. Clear liquor was obtained. After a prolonged labour lasting 28 hours, Kielland's forceps rotation and delivery of a healthy female infant was performed.

Recovery in the puerperium was uneventful.

CASE No. 75

Mrs. B. H. L.M.P: 8.9.67

aet 31 years E.D.C: 15.6.68

Gravida 2, para 1

This patient had one previous normal pregnancy in 1959. This present pregnancy was uneventful until the patient was two weeks past the estimated date of confine-

ment, when she developed mild pre-eclamptic toxaemia.

She was admitted to hospital on 26th June, in early labour. The following day, forewater rupture was performed and clear liquor obtained. After a labour lasting 29 hours, she had a manual rotation and forceps delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 76

Mrs. J. B. L.M.P: 24.9.67

aet 21 years E.D.C: 1.7.68

Primigravida

This patient had an uneventful pregnancy until 39 weeks gestation, when she was admitted to hospital with mild pre-eclamptic toxaemia. Two days later, an attempt was made to induce labour with an oxytocin infusion. The following day, forewater rupture was performed and meconium stained liquor obtained. After a labour lasting 25 hours in the first stage and ten minutes in the second stage, manual rotation and forceps delivery of a healthy male infant was performed. Recovery in the puerperium was uneventful.

CASE No. 77

Mrs. H. S. L.M.P: 8.10.67

aet 21 years E.D.C: 15.7.68

Primigravida

This patient was noted to have mild hypertension at 34 weeks gestation. She was advised to rest at home, but the hypertension persisted. At 37 weeks gestation, she was noted to have hypertension and proteinuria and was admitted to hospital for bed rest and sedation. One week after admission, labour started spontaneously. Forewater rupture was performed and clear liquor obtained. After a labour lasting 14 hours, she had a normal delivery of a healthy female infant. Recovery in the puerperium was uneventful.

CASE No. 78

Mrs. B. M. L.M.P: 22.9.67

aet 27 years E.D.C: 29.6.68

Gravida 2, para 1

This patient had a normal pregnancy in 1964.

The present pregnancy was uncomplicated until she was admitted to hospital at term with mild pre-eclamptic toxaemia. On the day following admission, forewater rupture was performed and clear liquor obtained. After a labour lasting five hours, she had a normal delivery of a healthy male infant. Recovery in the puerperium was uneventful.

CASE No. 79

Mrs. P. C. L.M.P: 28.9.67
aet 20 years E.D.C: 5.7.68
Primigravida

This patient was admitted to hospital in labour on 9th July. She was a healthy young woman whose pregnancy had been uneventful, and she was selected as a control clinically normal subject.

Forewater rupture produced clear liquor and after a labour lasting 21 hours, she had a low forceps delivery of a healthy female infant. It was noted that persistent foetal tachycardia developed in the second stage of labour, with persistent bradycardia after contractions. These changes developed after the administration of lumbar epidural anaesthesia.

Recovery in the puerperium was uneventful.

CASE No. 80

Mrs. A. N. L.M.P: 9.10.67
aet 22 years E.D.C: 16.7.68
Primigravida

This patient was admitted to hospital on 9th July with moderate pre-eclamptic toxaemia, having previous had an uneventful pregnancy. She was noted at this time to have

hypertension and persistent proteinuria. On the following day, labour was induced by forewater rupture and clear liquor was obtained.

After a labour lasting 26 hours, she had a low forceps delivery under epidural anaesthesia of a healthy male infant. Three days after delivery, the patient developed a pyrexia of 37.9°C. This occurred on only one occasion and did not require any specific therapy.

CASE No. 81

Mrs. M. K. L.M.P: 17.10.67

aet 35 years E.D.C: 25.7.68

Gravida 5, para 4

This patient had four previous pregnancies, all of which had ended prematurely, although all of the infants had survived. She was first admitted to hospital as an emergency admission at 35 weeks gestation, with premature rupture of the membranes. Four days later she was discharged from hospital. She was readmitted to hospital at 38 weeks gestation with an unstable lie, having mild contractions, with some dark vaginal blood loss. On 16th July, forewater rupture was performed and clear liquor obtained. After a labour lasting only 2½ hours, she had a normal delivery of a healthy male infant. Her recovery in the puerperium was uneventful.

CASE No. 82

Mrs. J. P. L.M.P: 5.11.67

aet 33 years E.D.C: 12.8.68

Gravida 8, para 6

This patient had six previous full term pregnancies, the last pregnancy being complicated by rhesus iso-immunisation. This pregnancy was uneventful. However, in view of anti-Rh agglutinins present to a titre of 1/2, surgical induction of labour was performed by forewater rupture at 37 weeks gestation. A sample of scalp blood was collected and the foetal blood group was shown to be O Rhesus positive, with a positive direct Coombs test.

After a labour lasting seven hours, the patient had a normal delivery of a healthy male infant. The cord blood showed a positive direct Coombs test, but as the infant was only mildly affected, exchange transfusion was not required. Recovery in the puerperium was uneventful.

CASE No. 83

Mrs. E. M. L.M.P: 10.9.67

aet 39 years E.D.C: 17.6.68

Gravida 2, para 1

This patient had one previous normal pregnancy in 1966. This pregnancy was uneventful, apart from the fact that the patient was admitted to hospital on 18th July, one month

past term, for induction of labour, the indication being post-maturity.

Surgical induction of labour was performed on the same day by forewater rupture and clear liquor was obtained. After a labour lasting only two hours, the patient had a normal delivery of a healthy male infant.

Recovery in the puerperium was uneventful.

CASE No. 84

Mrs. M. De G. L.M.P: 7.10.67

aet 28 years E.D.C: 14.7.68

Gravida 3, para 2

This patient had two previous pregnancies, both complicated with pre-eclamptic toxaemia. The first child, in 1961, was delivered by Caesarean section, and the second child was delivered by forceps.

This pregnancy was complicated by persistent anaemia. In view of her previous obstetric history the patient was admitted to hospital for surgical induction of labour eight days past term. On 25th July, forewater rupture was performed and meconium stained liquor was obtained.

After a labour lasting 19 hours, she had a normal delivery of a healthy female infant. In the puerperium, the patient developed a uterine infection which responded quickly

to antibiotic therapy.

CASE No. 85

Mrs. C. E. L.M.P: End October 1967

aet 25 years E.D.C: July 1968

Gravida 7, para 6

This patient had six previous normal pregnancies. This pregnancy was uneventful. The patient was admitted to hospital on 26th July, 1968 in early labour. The foetal heart rate was noted to be 180 beats/minute. Forewater rupture was performed and clear liquor obtained.

After a labour lasting 13 hours, the patient had a normal delivery of a healthy female infant. Recovery in the puerperium was uneventful.

CASE No. 86

Mrs. Y. G. L.M.P: 20.10.67

aet 22 years E.D.C: 27.7.68

Primigravida

This patient had an uneventful pregnancy until term, when she developed mild pre-eclamptic toxaemia. She was admitted to hospital on 26th July. After resting for three days, surgical induction of labour was performed by forewater rupture and clear liquor obtained. At this time, the pelvis was noted to be contracted. After a labour lasting 25 hours,

Caesarean section was performed for cephalopelvic disproportion. A healthy female infant was delivered. In the puerperium, the patient developed a pyrexia and was found to have a uterine infection and peritonitis. She responded to antibiotic therapy, although she also developed some wound infection.

CASE No. 87

Miss A. B. L.M.P: 26.10.67
aet 16 years E.D.C: 3.8.68
Primigravida

This young primigravida had an uneventful pregnancy until term, when she developed pre-eclamptic toxaemia. She was admitted to hospital on 1st August, when surgical induction of labour was performed by forewater rupture, and clear liquor was obtained. After a labour lasting 11 hours, she had a normal delivery of a healthy male infant. Recovery in the puerperium was uneventful.

CASE No. 88

Mrs. E. V. L.M.P: 28.11.67
aet 35 years E.D.C: 5.9.68
Gravida 2, para 1

This patient had one previous pregnancy in 1962 which was normal.

Her antenatal care was managed by her local medical

officer and she was admitted to hospital on 15th July, with threatened premature labour. She was discharged from hospital the same day, and was readmitted on 5th August in early labour. The membranes had ruptured spontaneously, and she was draining clear liquor. She was noted to have foetal bradycardia with considerable irregularity in the foetal heart rate. After a labour lasting 6½ hours she had a normal delivery of a female infant. Recovery in the puerperium was uneventful.

CASE No. 89

Mrs. B. G. L.M.P: 8.11.67

aet 26 years E.D.C: 15.8.68

Gravida 5, para 4

This patient had four previous pregnancies, of which two had been complicated by toxaemia.

This pregnancy was uncomplicated, and the patient was admitted to hospital on 6th August in early labour. She was selected for monitoring as a control normal subject. Forewater rupture was performed in early labour and clear liquor obtained. After a labour lasting six hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 90

Mrs. E. H. L.M.P: November 1967

aet 30 years E.D.C: August 1968

Gravida 5, para 4

This patient had four previous normal pregnancies. Her progress was uneventful during this pregnancy, apart from glycosuria on several occasions. A glucose tolerance test at 34 weeks gestation was normal. On 2nd August, the patient was admitted to hospital with ruptured membranes draining clear liquor. Labour did not start until three days later. At this time, the foetal heart rate was noted to be 160 beats/minute. Pelvic examination revealed that the membranes were still intact and forewater rupture was performed. After a labour lasting 13 hours, the patient had a normal delivery of a healthy male infant.

Recovery in the puerperium was uneventful.

CASE No. 91

Mrs. R. C. L.M.P: 20.10.67

aet 26 years E.D.C: 27.7.68

Gravida 7, para 3

This patient had three previous miscarriages and three full term normal pregnancies. Her present pregnancy was uneventful until one week past term, when she developed

pre-eclamptic toxaemia. After admission to hospital on 6th August, forewater rupture was performed and clear liquor obtained. After a labour lasting only three hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 92

Mrs. P. S. L.M.P: 26.10.67

aet 28 years E.D.C: 2.8.68

Gravida 6, para 5

This patient had five previous pregnancies, the first three pregnancies being complicated by toxaemia of pregnancy. This pregnancy was uneventful until three days before term when the patient developed pre-eclamptic toxaemia. She was admitted to hospital on 29th July and the blood pressure settled to normal with bed rest and sedation. One week later, surgical induction of labour was performed by fore-water rupture. Labour did not become established until three days later, when contractions were stimulated with a dilute oxytocin infusion.

After a labour lasting only three hours, she had a normal delivery of a female infant.

Recovery in the puerperium was uneventful.

CASE No. 93

Mrs. M. V. L.M.P: 21.10.67

aet 22 years E.D.C: 28.7.68

Primigravida

This patient had an uneventful pregnancy until she was admitted to hospital one week after term, when she was noted to have excessive weight gain and proteinuria.

She was treated with bed rest and sedation and one week later labour started spontaneously. Forewater rupture was performed and clear liquor obtained. After a labour lasting eight hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 94

Mrs. F. R. L.M.P: 9.11.67

aet 37 years E.D.C: 16.8.68

Gravida 3, para 0

This patient had a history of two previous miscarriages. At first visit to the antenatal clinic, she was noted to have a generally contracted pelvis. Her pregnancy was uneventful until 39 weeks gestation, when she developed mild pre-eclamptic toxaemia. On 14th August, she was admitted to hospital in early labour.

Progress in labour was slow and the following day, forewater rupture was performed and clear liquor obtained. There was some evidence of cephalopelvic disproportion and, after a labour lasting 40 hours, delivery was effected by Caesarean section. The male infant was in a satisfactory condition at birth, but was noted to have a cleft palate and evidence of the Pierre-Robin syndrome. The patient made an uneventful recovery in the puerperium.

CASE No. 95

Mrs. G. G. L.M.P: 27.11.67
aet 25 years E.D.C: 4.9.68
Primigravida

This patient had an uneventful pregnancy and was admitted to hospital in early labour on 22nd August. She was monitored during labour as a control subject. After a labour lasting 18 hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 96

Mrs. E. K. L.M.P: 6.11.67
aet 24 years E.D.C: 13.8.68
Primigravida

This patient had an uneventful pregnancy until she

was 37 weeks pregnant, when she developed hypertension and excessive weight gain. She refused admission to hospital and the following week her blood pressure was normal. However, one week past term, she was again noted to have developed hypertension, with blood pressure 140/100. She was admitted to hospital the same day and treated with bed rest and sedation. On 3rd September, forewater rupture was performed, but no liquor was obtained. Some transient foetal bradycardia was noted. After a labour lasting 19 hours, she had a normal delivery of a healthy male infant.

Recovery in the puerperium was uneventful.

CASE No. 97

Mrs. I. S. L.M.P: 12.12.67

aet 22 years E.D.C: 19.9.68

Primigravida

This patient had an uneventful pregnancy until she was 30 weeks pregnant. She then developed mild hypertension which responded to bed rest. At 38 weeks gestation, she developed moderate toxaemia of pregnancy and surgical induction of labour was performed by forewater rupture. Clear liquor was obtained. After a labour lasting seven hours, she had a normal delivery of a premature female infant.

Recovery in the puerperium was uneventful.

CASE No. 98

Mrs. E. B. L.M.P: End November 1967

aet 19 years E.D.C: September 1968

Gravida 2, para 1

This patient had her first child in 1967. This child was delivered by Caesarean section following prolonged labour and foetal distress. The antenatal course in this pregnancy was uneventful until she was admitted to hospital in false labour on 25th August. She was discharged home the following day and readmitted on 9th September, in early labour. The foetal heart rate was noted to be slow and forewater rupture was performed. Clear liquor was obtained. After a labour lasting 14½ hours, she had a normal delivery of a healthy female infant. Recovery in the puerperium was uneventful.

CASE No. 99

Mrs. J. K. L.M.P: December 1967

aet 23 years E.D.C: September 1968

Gravida 2, para 1

This patient had one previous normal pregnancy in 1965. This present pregnancy was uneventful until the patient was admitted to hospital in early labour on 11th September 1968. The following day she was noted to have some foetal tachycardia. Forewater rupture was performed and clear liquor

obtained. After a labour lasting 19½ hours she had a normal delivery of a healthy female infant. Recovery in the puerperium was uneventful.

CASE No. 100

Mrs. S. G. L.M.P: 10.12.67
aet 35 years E.D.C: 17.9.68
Primigravida

This patient had an uneventful pregnancy until she was admitted to hospital in early labour on 17th September, and was noted to be draining meconium stained liquor. Towards the end of the first stage of labour, she developed persistent severe foetal tachycardia with a foetal heart rate of 180 beats/minute.

After 16 hours in labour she was noted to have a thick lip of anterior cervix palapable on vaginal examination. In view of the foetal tachycardia, Kielland's forceps rotation and delivery was performed under general anaesthesis and a healthy female infant delivered. Recovery in the puerperium was uneventful.

CASE No. 101

Mrs. A. Z. L.M.P: 12.12.67
aet 39 years E.D.C: 19.9.68
Gravida 9, para 4

This patient had four previous full term pregnancies, the last one in 1966 being complicated by pre-eclamptic toxaemia. She had also had four miscarriages, all at three months gestation, the last one occurring in 1962.

This pregnancy was uneventful until four days after term, when she developed pre-eclamptic toxaemia. She was admitted to hospital immediately and the following day forewater rupture was performed, and meconium stained liquor obtained. After a labour lasting 11 hours, she had a normal delivery of a healthy female infant. Recovery in the puerperium was uneventful.

CASE No. 102

Mrs. G. M. L.M.P: 10.1.68

aet 22 years E.D.C: 17.10.68

Primigravida

This patient had an uneventful pregnancy until 32 weeks gestation, when she was admitted to hospital with proteinuria for investigation. The urine became clear with bed rest and she was discharged home three days later.

She was readmitted to hospital on 18th September with pre-eclamptic toxaemia. Despite bed rest and sedation, her condition did not improve. On 25th September, labour started spontaneously. Forewater rupture was performed and

meconium stained liquor was obtained. After 12 hours in labour, little progress had been made and delivery was effected by Caesarean section. The female infant showed signs of asphyxia at delivery and required resuscitation.

Apart from an initial pyrexia on the first day after delivery, recovery in the puerperium was uneventful.

CASE No. 103

Mrs. P. T. L.M.P: 2.12.67

aet 20 years E.D.C: 9.9.68

Gravida 2, para 0

This patient had one previous pregnancy which ended in miscarriage at two months gestation.

This pregnancy was uneventful until two weeks past term. The patient developed mild pre-eclamptic toxaemia and was admitted to hospital for bed rest. Two days later, labour started spontaneously. On the following day, fore-water rupture was performed and clear liquor obtained. The liquor subsequently became meconium stained. After a labour lasting 32 hours, she had a Kielland's forceps rotation and delivery of a healthy female infant.

Recovery was uneventful until two weeks after delivery, when she developed signs of pelvic infection. This rapidly responded to antibiotic therapy.

CASE No. 104

Miss B. S. L.M.P: Mid-January 1968

aet 24 years E.D.C: October 1968

Gravida 4, para 3

This patient was first seen at clinic on 23rd September at 37 weeks gestation. She had two previous normal pregnancies and in her third pregnancy she developed rhesus antibodies. An indirect Coombs test in this pregnancy showed a titre of 1/256. Labour was induced by forewater rupture on 30th September, because of rhesus isoimmunisation. Clear liquor was obtained. After a labour lasting ten hours, she had a normal delivery of a living male infant. Exchange transfusion was performed on the child two days after delivery. Recovery in the puerperium was uneventful.

CASE No. 105

Miss R. M. L.M.P: 1.1.68

aet 21 years E.D.C: 8.10.68

Primigravida

This patient was first seen at antenatal clinic when she was 34 weeks pregnant. At 38 weeks gestation she was found to have a blood pressure of 140/100 with half protein in the urine. She was treated with bed rest and sedation. Both urinary pregnanediol and oestriol levels were low and, on 10th October, surgical induction of labour was performed

by forewater rupture on 15th October and clear liquor obtained. After a labour lasting 13 hours, she had a normal delivery of a healthy male infant. The indication for monitoring this labour was post-maturity.

Recovery in the puerperium was uneventful.

CASE No. 107

Miss J. P. L.M.P: 29.1.68
aet 18 years E.D.C: 5.11.68
Primigravida

This patient had an uneventful pregnancy until 37 weeks gestation, when she developed pre-eclamptic toxaemia, with a blood pressure of 140/95 and proteinuria. She was admitted to hospital on 17th October and treated with bed rest and sedation.

On 22nd October, 16 days before term, surgical induction of labour was performed and clear liquor was obtained. After a labour lasting only 3½ hours, she had a normal delivery of a healthy female infant.

Recovery in the puerperium was uneventful.

CASE No. 108

Mrs. A. G. L.M.P: 27.2.68
aet 19 years E.D.C: 5.11.68
Gravida 2, para 1

This patient had one previous normal pregnancy in 1967. This present pregnancy was uneventful until one week past term, when she developed mild pre-eclampsia with generalised oedema and a trace of protein in the urine. Surgical induction of labour was performed by forewater rupture on 14th November and clear liquor was obtained. After a labour lasting seven hours, the patient had a normal delivery of a healthy male infant.

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