Clinical Validity of Fetal ECG Waveform Analysis

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Our ability to identify the fetuses at risk of intrapartum asphyxia depends on either
monitoring the actual level of hypoxemia using biochemical means or, by different
techniques, interpreting the reactions caused by hypoxemia. The fetal ability to adapt
to hypoxemia involves multiple defense mechanisms. These consist primarily of car-
diovascular compensation which increases blood flow to the most important or-
gans—the brain, the heart, and the adrenals, as well as the placenta—and counter-
acts the decreasing oxygen content. A second line of defense is the metabolic compensatory mechanisms. This chapter presents data of the fetal reaction to hy-
poxemia as interpreted from ECG analysis.

During labor, the ECG signal is easily obtainable and additional information may
be gathered without the need to change patient handling routines. Changes in the
ECG waveform have been the subject of much clinical interest throughout the last
30 years (1,2). Throughout the past 10 years, this interest has been focused on de-
tection of specific hypoxia-related phenomena (3).

BASIC RESEARCH—ST WAVEFORM

Since the initial experimental observations in 1972, there has been a continuous
line of basic research to clarify some of the pathophysiological mechanisms involved
in the elevation of the ST segment and T wave amplitude as a fetal response to
hypoxia. In summary (Fig.1), the increase in T wave height, quantified by the ratio
between T wave and QRS amplitudes (T/QRS ratio), occurs when the cardiovascular
adaptation to hypoxia (increased coronary blood flow) is no longer sufficient. Myo-
cardial metabolic adjustments are needed to cover for a negative energy balance.
Glycogenolysis, stimulated by adrenaline surge, becomes the most important source of energy and at the same time there is a change in membrane potential due to potassium release causing the ST elevation (4,5). Perhaps the best way of displaying these changes experimentally is during spontaneous labor in the chronically instrumented fetal sheep. Figure 2 gives an example of such a case where labor ended in fetal death at the time of delivery and a number of parameters were recorded in parallel with the ECG waveform. Eleven hours before fetal death the T/QRS ratio increased and during the last hour a further increase was noted with an increase in T/QRS ratio from 0.6 to 1.0. Figure 3 shows changes in cardiovascular parameters and somatosensory evoked EEG potentials during the last hours. Evoked potentials could serve as a measure of the ability of the fetal brain to operate during hypoxia (6) and it is obvious that from recordings like this, as well as from other data (7,8), ST waveform changes appear before loss of brain function. Figure 3 also indicates the capacity of the fetus to compensate for asphyxia with adequate brain signal processing in spite of marked acidosis. There are only a few minutes between the loss of cerebral activity and the last heart beat in this mature fetal lamb.

Further experimental work in the experimentally growth-retarded guinea pig fetus indicates the development of ST depression with a negative-positive T wave during hypoxemia. Thus, a “biphasic” ST waveform seems to be a most significant response.

**ECG WAVEFORMS RECORDED FROM THE SCALP LEAD**

All the animal experiments have used the precordial ECG lead which is optimal to identify all waveform components in the ECG. The QRS complex as well as the P wave are robust parameters, whereas the amplitude of the T wave is greatly influenced by the ECG vector. Lindecrantz *et al.* (9) have presented data to indicate
FIG. 2. Recordings from a chronically instrumented sheep fetus during spontaneous labor ending in fetal death at time of delivery. FHR, fetal heart rate; MAP, mean arterial pressure; SaO₂, oxygen saturation. Data modified from Rosén KG, et al. (7).

that the standard bipolar scalp lead (an exploring electrode inserted in the scalp with a reference electrode placed close by and in contact with the vaginal fluid) was only optimal in identifying T wave changes occurring in the horizontal (x + z) plane of the fetal body. The x-axis has consistently been unable to detect changes in the T wave amplitude, which means that the standard scalp ECG used for cardiotocography (CTG) monitoring is not optimal for ST analysis. On the other hand, the y-axis, which identifies changes in the longitudinal plane of the fetal body, is optimal for detection of changes in T wave height. This y-lead is simply recorded by using
a unipolar scalp lead. Our model for creating this unipolar scalp lead during labor is to place the reference electrode far away from the scalp, i.e., on the maternal thigh. Apart from increased sensitivity for changes in T wave configuration, there is also an increase in signal quality, due to less ECG baseline shift (10). This is not of minor importance as any further attempt to reduce this low frequency shift by signal filtering could seriously affect the low-frequency components of the ECG—the ST period (11,12). Furthermore, our experience with different scalp electrodes has consistently indicated that one helix scalp electrode gives the least baseline shift as well as the optimal signal amplitude.

APPROPRIATE TECHNOLOGY

There has been a continuous development in technology in the last 10 years with the aim of developing a robust, user-friendly monitor. Throughout this process, data have been collected using currently available technology. In our initial study (13), standard equipment was used (oscilloscope and Mingograph ECG-recorder) and with further development, a purpose-built microprocessor for ST-ANalysis (STAN) was constructed.

STAN—A CTG Monitor with ST Waveform Analysis

The ST analyzing system is displayed schematically in Fig. 4. The fetal ECG is picked up differentially between a one helix scalp electrode and a skin electrode.
placed on the maternal thigh. Both electrodes are connected to a patient isolating box (PIB), via an ECG clip connector. In the PIB the ECG signal is amplified and this unit also contains the necessary galvanic isolation between the patient and the rest of the system. The PIB also has an input connector for a uterine activity signal from an external toco transducer or an internal uterine pressure transducer. From the PIB, which is kept close to the patient, both toco and ECG signals are led to the main unit through a cable that can be of several meters' length.

The main unit, where most of the signal processing and the computations are performed, is furnished with a one-color printer plotter for data presentation, a graphic LC display and five push buttons for software control functions. Via a serial interface (RS 232) the signals can be presented to a personal computer for further processing and storage. The ECG signal from the PIB passes through an adaptive line frequency filter that eliminates 50 or 60 Hz interference in the signal. The bandwidth used for waveform analysis is 0.05–100 Hz and the signal is A to D converted at the rate of 500 Hz with a resolution corresponding to 8 bits. For further details of the system, see Rosén et al. (14).

Once the machine is connected to the patient and turned on, it automatically adjusts gain level and the processed data are presented on both the LC display and the printer/plotter. Fetal heart rate and toco activity are presented on the printer according to accepted standards for CTG monitors. The T/QRS ratios are also presented as a function of time, and every second minute an average fetal ECG complex is plotted (Fig. 5).

The Nottingham group, headed by M. Symonds, has developed a separate system of ECG time interval analysis (15). This system has been based on a minicomputer system with an ongoing effort to develop a microprocessor-based unit. The aim of the Nottingham system is to enable on-line assessment of a multitude of ECG parameters—time constants as well as alterations in waveforms. This approach has
been fruitful as it has empirically, during labor, identified changes in the PR time constant which shortens during hypoxemia in spite of a lengthening of the RR (decreasing heart rate).

**CLINICAL STUDIES—ST WAVEFORM**

In the initial study (13) we were able to demonstrate, with simple technology, that it was possible to detect ST waveform changes during labor and these changes occurred independently of CTG changes. Furthermore, a linear relationship was found between T/QRS ratio and umbilical lactate levels. The number of patients in the study was small ($n = 46$) and no clinically asphyxiated babies were found. In a subsequent study (16) the first microprocessor version of STAN was tested with a continuous recording of T/QRS ratios using the group averaging technique. Only one fetus showed a T/QRS ratio >0.30 throughout the 2.5 h recording. Umbilical artery pH was 7.05 and lactate was 7.73 mmol/l with normal Apgar scores and neonatal outcome. There were two more fetuses with ST waveform changes lasting more than 30 min; both of these had CTG changes as well as high lactate and low pH values. There was one case with biphasic T waves showing a marked respiratory acidosis (pH 7.06). In eight cases blood was available for catecholamine analysis. A significant correlation, $r = 0.73$, $p<0.05$, was found between adrenaline concentration and T/QRS ratio, but no correlation was found between noradrenaline and T/QRS. Both studies were small by their numbers and did not incorporate any child with clinical asphyxia, but they still provided information on the technology needed and some
indications were found of a parameter, the T/QRS ratio, which might independently provide information of fetal response to hypoxia in labor.

With the need for a larger population of high-risk pregnancies, collaboration was started with the National University of Singapore and the University of Colombo, Sri Lanka. The first major data base on ST waveform analysis during labor using the STAN monitor has recently been collected (10,17), and the normal range of T/QRS ratios during labor in healthy fetuses was shown to be 0.15 ± 0.05 (mean ± 1 SD) (10). The ratio was found to be most stable during labor with an occasional increase during contraction, but this was not a regular finding as can be seen in Fig. 4.

The second study (17) is based on 201 deliveries, 25% at high risk. Cord artery pH and buffer balance together with Apgar scores were used as end points of perinatal asphyxia. During first stage of labor 45% had suspicious or abnormal heart rate traces, whereas only 27% had a baseline T/QRS ratio >0.25 (+2 SD). Twenty-seven operative deliveries were performed for fetal distress diagnosed on CTG findings, and in three of these the cord artery blood pH was <7.15. Among these 27 fetuses, 11 (41%) displayed elevated baseline T/QRS, which identified all fetuses with cord artery pH <7.15 and/or standard bicarbonate <15.0 mmol/l.

It was of special interest to correlate the reduction in buffering capacity of the fetal blood as identified by a standard bicarbonate <15.0 mmol/l with the increase in T wave amplitude. The normal ST waveform identified with a probability of 99.3% a fetus with normal buffering capacity. Five fetuses developed a metabolic acidosis (pH <7.15 and standard bicarbonate <15.0 mmol/l), all of whom showed high T/QRS ratios. Acute hypoxia emerging during second stage of labor was identified by rapid rise in T/QRS. There were three cases with a clinical diagnosis of asphyxia, all were identified by changes in T/QRS, and the lowest 5-min Apgar was 6. An evaluation test between T/QRS during first stage of labor and reduced buffer capacity in the cord artery blood showed a sensitivity of 94% and a specificity of 80%. Kappa index was 0.40.

CTG on its own did not reflect the acid-base status in cord artery. However, in the group of fetuses (five cases) with abnormal CTG and increased mean T/QRS, four had a low bicarbonate concentration and metabolic acidosis always occurred when there were changes in both ST waveform and CTG (Table 1). Thus, it seems possible to increase the specificity and positive predictive value by combining T/QRS with fetal heart rate.

The first independent trial on STAN has been performed by Murphy, Valente, and Johnson in Oxford (18). Their data are based on 86 recordings using a STAN prototype monitor which they found was robust and suitable for clinical application. Of the recordings, 97% were of a quality to allow ECG waveform analysis. Only one fetus developed clinical problems related to perinatal asphyxia, identified from metabolic acidosis in the cord artery and vein (cord artery pH 6.96, standard bicarbonate 13.6 mmol/l, standard base excess −16.0 mmol/l). Apgar scores were 2 and 7, with some respiratory distress during the neonatal period. This case revealed a high T/QRS ratio during first stage (mean T/QRS 0.31). During the end of the first
stage and in the second stage a "biphasic" ST waveform with a negative T wave component with ST segment depression occurred. The authors also analyzed the relationship between CTG, using Krebs intrapartum CTG score, and T/QRS ratio, and showed a significant relationship between high T/QRS values and low-scoring (abnormal) segments of the intrapartum CTG (p<.01).

Of the ongoing trials, the largest series of cases has been collected in Colombo. At the time of writing, 485 recordings have been analyzed, showing a success rate of 97% using the one helix scalp electrode. There has been one case of intrapartum death, which was monitored. Monitoring means heart rate from the STAN, but not ECG waveform for clinical management. This fetus was severely growth-retarded (birth weight 1.700 g at term) and displayed negative T waves. This pattern was already evident at the beginning of the 8-hour recording. Figure 6 shows the recording 1 h before fetal death demonstrating increasingly negative T waves. Figure 7 shows another recording lasting 20 min, in a full-term, appropriately grown fetus. The baseline bradycardia was noted in first stage and after a manual dilatation of the cervix a vacuum extractor was applied. The baby was delivered with Apgar scores of 2 and 5 and showed seizures at 24 hours after birth. The subsequent neonatal outcome was uneventful.

**ONGOING TRIALS**

Through the European Community concerted action project "New Methods for Perinatal Surveillance," a multicenter prospective study is now starting, involving 10 to 15 European perinatal centers. The ECG is collected "blind" and the purpose is to build a European ECG data base. STAN is used as an ordinary CTG monitor with a PC backup system for data handling and subsequent storing, which uses optical disks to handle the large amount of data generated. The plan is to collect the ECG in a format that will allow off-line analysis of all ECG-related parameters like power spectrum analysis of heart rate variability and time interval analysis. The latter is of special interest as the Nottingham group (15) has demonstrated the relevance of
a change in correlation between PR and RR time constants with intrapartum hypoxia. Normally this correlation is positive, i.e., with increasing RR (lower heart rate) there is also an increase in PR but with hypoxia the PR shortens in spite of lengthening of RR and the correlation becomes negative. Sheep experimental data support this finding and there are now data (15,17) to indicate that by combining different aspects of the ECG one should be able to improve the diagnostic properties.

**TENTATIVE MODEL FOR ST WAVEFORM ANALYSIS**

On the basis of approximately 1,500 cases where ST waveform monitoring has been undertaken, together with our experience from the animal data base, there is a possibility of making a tentative model of the relationship between the ability of the fetus to react to hypoxia and the appearance of the ST waveform changes. Figure 8 summarizes this model, and the reaction of the fetus to hypoxia is separated into three groups:

1. Those with intact defense mechanisms
2. Those with reduced defense mechanisms
3. Those lacking defense mechanisms.
Intact Defense Mechanisms

These are the majority of fetuses, approaching 99%. Those fetuses have developed all the resources needed to handle asphyxia, including energy stores, optimal reactivity regarding the sympathetic system and catecholamine release, optimal receptor sensitivity, etc. This gives optimal hypoxia reactivity and full compensation over long periods of hypoxemia and also an ability to handle severe asphyxia. The occurrence of ST segment elevation and increase in T wave height signifies a situation in which glycogenolysis is occurring and an important defense mechanism is in operation. How could we then identify the point at which these mechanisms no longer fully support the fetus? At the moment the data indicate that such a situation can be identified by a combination of increasing and/or sustained high T/QRS ratio with signs of a reduced reactivity in the fetal heart rate pattern.

What is happening to the fetus when these patterns emerge? A case showing such marked changes is presented in Fig. 7. This baby responded to resuscitation and
NORMOXIA | HYPOXEMIA
---|---

**DEFENCE MECHANISMS**
Cardiovascular & Metabolic adjustments
- Increased sympathetic activity
- Redistribution of blood flow
- Glycogenolysis

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<tr>
<th>INTACT</th>
<th>REDUCED</th>
<th>LACKING</th>
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<tr>
<td>Optimal hypoxic reactivity</td>
<td>Decreased hypoxic reactivity</td>
<td>Minimal hypoxic reactivity</td>
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<tr>
<td>Full compensation</td>
<td>Reduced compensation</td>
<td>Noncompensation</td>
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Corresponding pattern of ST waveform changes in the fetal ECG

<table>
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<tr>
<th>T/QRS 0 - 0.25</th>
<th>T/QRS &gt; 0.25</th>
<th>T/QRS &lt; - 0.10</th>
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<tr>
<td>ST segment elevation and high T waves</td>
<td>Biphasic ST waveform</td>
<td>Negative T waves</td>
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**Phase of noncompensation**

- T/QRS increasing
- Decreased FHR reactivity

- T/QRS variable
- FHR?

- T/QRS < - 0.20
- FHR?

**FIG. 8.** A tentative model of interpretation of ST waveform and heart rate changes. (Figure used with permission from Cinventa AB.)

survived the neonatal period with only a short-lasting seizure 24 hours after birth. The Singapore data also tell us that the combination of elevated T waves and abnormal CTG will identify fetuses with a decreased buffer capacity but who are clinically normal at birth.

**Reduced Defense Mechanisms**

This is a situation that could be identified experimentally using the growth-retarded guinea pig model and subjecting these fetuses to hypoxemia. They are moderately
growth-retarded which means that they survive labor but show a decrease in their hypoxic reactivity and ability to compensate. Their ability to maintain cellular integrity is curtailed and the risk for decompensation and asphyxia will increase. In his experimental work Carl Widmark has demonstrated “biphasic” ST waveform as a characteristic response to hypoxemia.

**Biphasic T Wave**

Clinically as well as experimentally the ST waveform shows “biphasic” T wave with a negative T wave component occurring together with a positive component. This pattern has only been recorded in a few cases but all of those have developed asphyxia, identified by metabolic acidosis base deficit >12 mmol/l), decrease in Apgar scores and neonatal problems (seizures or respiratory distress). It seems as though biphasic patterns may either be sustained throughout labor or develop into high T waves. The T/QRS ratio will vary but usually goes beyond 0.25 as the positive T wave will dominate.

**CTG**

The relationship with CTG has, so far, been one of parallel changes. It could be anticipated, however, that heart rate would not show distinct changes because there is still some reactivity and heart rate is poor when it comes to quantifying the reactive patterns. The incidence of these changes is probably on the order of 0.5–1%.

**Lacking Defense Mechanisms**

Here we have severely growth-retarded fetuses who may not be able to handle labor at all, probably due to the long-term stress. Reactivity to hypoxia is minimal and the risk of decompensation is imminent, leading to extreme risk of asphyxia. As there is no capacity to compensate by using metabolic mechanisms, the degree of metabolic acidosis might be less. It also seems likely that these fetuses could suffer permanent damage at a far lower degree of hypoxemia than would be the case with a fetus who was able to compensate. Due to the work in Colombo, we have been able to identify the pattern of negative T waves that seems to reflect this grossly adverse situation. This may be a unique piece of information obtainable only from ECG waveform analysis. It seems logical that an indicator of organ function should be able to signify a situation of extreme risk. Identifying such situations is of vital importance in the reduction of perinatal mortality and morbidity.

This model of ECG data interpretation during labor is presently tested in a randomized controlled trial that is taking place in Plymouth. The data obtained from the study of 600 cases show that ST and CTG monitoring in combination could reduce the rate of operative deliveries by more than 50% with no increased risk of perinatal
asphyxia as compared with the use of CTG alone (K. R. Greene and J. Westgate, personal communication).

ACKNOWLEDGMENTS

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REFERENCES

Dr. Dawes: In your presentation you mentioned a randomized controlled trial. What is the basis for clinical management in the group of fetuses where the STAN monitor is used?

Dr. Rosen: In this study we are comparing standard management, including CTG and fetal blood sampling in one leg of the study with CTG, and fetal blood sampling and ST waveform analysis in the other. Using the ST waveform we have one variable that is independent of heart rate. The aim of the trial is to test whether ST waveform could add to clinical management, reducing the number of operative interventions without increasing the risk of intrapartum asphyxia. In the protocol it is stated that with a normal ST waveform, blood sampling should be postponed until the trace becomes persistently abnormal. If there is a preterminal fetal heart rate pattern, operative delivery is performed. A normal ST waveform means that interference can be delayed. When there is a normal fetal heart rate with a T/QRS ratio between 0.25 and 0.5 we just observe closely. When both variables identify changes, further information is needed from fetal blood sampling. If there are marked changes, immediate delivery is indicated.

Normally the T/QRS ratio is stable during labor, which means that a long-term increase in the ratio signifies that the fetus needs additional energy from stored myocardial glycogen.

On the extremely rare occasions when there are biphasic T waves, fetal blood sampling is performed even if there is a normal fetal heart rate pattern. I believe that this situation signifies a fetus lacking the ability to compensate for asphyxia and rapidly approaching the stage of decompensation.

Dr. Saling: When do you think this method of monitoring should be considered? After all, it is a traumatic procedure requiring the introduction of the electrode and sometimes the rupture of the membranes.

Dr. Rosen: The advantage of the ECG waveform analysis is that we do not need to change our patient-handling procedures, since a scalp electrode is already required for cardiotocographic analysis during labor. Naturally it would be a great advantage if we could do waveform analysis before labor, using the transabdominally recorded ECG. Unfortunately I do not think this is possible because of the vectorcardiographic aspects. It is possible to obtain the ECG waveform analysis antenatally via a needle used for transfusing the fetus, though unfortunately the cord does not seem to give an ECG signal.

Dr. Marini: Changes in the T wave indicate that the metabolism of the heart is impaired; this can be either because of hypoxia or because of impairment of the coronary circulation. Do you know which heart chamber you are actually reading?

Dr. Rosen: The unipolar system provides a stable signal that is independent of the position of the head. It should identify the Y vector which reflects events in both ventricles.

Dr. Marini: When you did your study in animals, you probably followed the animals until death. Did you find any case of ventricular fibrillation before death or did the animals all die with cardiac arrest? I ask this because it is very uncommon for neonates to die with ventricular fibrillation, but in our work with the isolated neonatal heart (1) it was very common to induce ventricular fibrillation. It sounds as though the innervation of the heart will protect the neonatal heart from VF.

Dr. Rosen: We have not found ventricular fibrillation in our experiments, though sometimes there has been atrioventricular block.

Dr. Dawes: In the trial you are contemplating in primates, I think you said that you were
going to combine ST segment analysis with a visual analysis of fetal heart rate. However, the visual analysis of fetal heart rate patterns is suspect and the Krebs score worse than suspect — it is positively misleading. This score requires selection of an identifiable piece of the trace in which 50% is “characteristic.” This is not a logical prescription. The Krebs score has five variables, each scored from 0 to 2, so the maximum is 10. One of the variables concerns fetal heart rate, the second concerns the presence or absence of decelerations. The three other variables relate to fetal heart rate variability, including decelerations, zero crossing frequency, and amplitude. This score therefore gives an excessive weighting to heart rate variability, without justification. In the present state of knowledge such scores are best avoided. My advice in planning this proposed study in primates is to avoid scoring the cardiotocogram by visual analysis.

Dr. Rosén: We are not going to use the Krebs score. At the same time we must accept that visual analysis of the CTG is the present standard and we want to take the development step by step toward more objective analysis.

Dr. Dawes: Would Professor Saling agree with the statement that CTG visual analysis is the present standard?

Dr. Saling: When evaluating the CTG we use the Hammacher score, but this is also a complicated procedure. Up to now nothing better has been possible. But perhaps now with computer evaluation we shall be more objective.

Dr. Dawes: May I ask about the PR:RR ratio you mentioned? This was from a study by Henry Murray in Nottingham. I wonder whether you have any explanation of the curious relationship he described.

Dr. Rosén: The alteration in the relationship between the PR and RR time intervals was first observed by Henry Murray in his evaluation of approximately 300 labor recordings. The Nottingham group has used the correlation between the two time intervals as a way to identify the abnormal response of a shortening in the PR with a lengthening in the RR. A normal PR:RR correlation seemed to indicate that the fetus was healthy and had a normal acid-base status. By combining ST segment analysis with PR analysis they were able to identify fetuses with cord acidemia.

We have started experimental work to try to identify the pathophysiological mechanisms for the changes in the PR:RR correlation. A change in the relationship can be induced experimentally by a period of hypoxia lasting between one and 20 min. Under these circumstances, ST waveform and PR:RR changes occur in parallel. However, β-adrenoceptor stimulation, which causes increased T wave height, does not alter the PR-RR correlation. We are currently using the STAN monitor linked to a common PC for data collection in a multicenter study; it is hoped this will create a data base of ECG complexes that will allow us to investigate different ECG-related variables during labor, such as ST waveform, time constants, and power spectrum analysis of fetal heart rate variability.

Dr. Marini: Pardi and Brambati have analyzed the lengths of the various segments of the fetal ECG. They found in a severely damaged fetus with erythroblastosis that duration was much more indicative of lesions than the height of the waves. I believe that if you do not know precisely where the signal is coming from it is more reliable to work on duration.

In one of your cases you showed a long wave in the QT segment, like an afterpotential. This could be important in discriminating between altered repolarization because of electrolyte imbalance and altered perfusion under the papillary muscle, which is one of the major causes of neonatal cardiac problems.

Have you found any cases of wandering pacemaker? This is very common in the neonatal age group and could explain the varying PR length.
Dr. Rosén: The change in the PR interval that occurs with acute asphyxia and bradycardia is a shortening without a marked change in P wave configuration. In the preterminal trace you may see an independent lengthening of the PR interval without change in P wave configuration, in which case a wandering pacemaker could explain the finding, as you suggest. The possibility of discriminating between electrolyte imbalance and ischemia from changes in QT sounds interesting. However, the QT interval is hard to define as the end of the T wave may not be distinct. Intracellular electrolyte disturbance is the most likely explanation of the ST changes, since fetal myocardial performance and coronary blood flow are well maintained during hypoxemia in a term fetus.

REFERENCE