

INTRAPARTUM CARDIOTOCOGRAPHY

by

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Submitted for the M.D. Examination

June 1978

ABSTRACT

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The history of fetal heart rate (FHR) monitoring is traced from the first described auscultation to the introduction of intrapartum cardiotocography approximately ten years ago. The relationship between FHR monitoring and other modern obstetric techniques is discussed and 'consumer' attitudes to these techniques are described.

The discourse proceeds to show that complications attributable to cardiotocography are uncommon but that, in 1974, misinterpretation of FHR traces and misuse of cardiotocographic techniques were significant problems indicating the need for improved teaching or automated FHR analysis.

Studies are described by which it was hoped to determine which FHR variable indicates most reliably the presence of fetal acidosis and whether, by using that variable, there remained a need for fetal blood sampling as the final diagnostic test.

The information obtained during the studies highlights the multiple shortcomings encountered in conventional FHR classifications. These are discussed together with the pathophysiology of FHR changes during labour. The objectives of intra-partum cardiotocography are re-iterated and its efficacy discussed before the aforementioned studies are appraised critically; conclusions are then drawn which imply that baseline variability is the most reliable of FHR features in the detection of fetal acidosis but that fetal blood sampling remains a necessity.

Methods of measuring baseline variability are then discussed and reference is made to the possibility of automated measurement which would reduce the need for expertise in the analysis of FHR traces.

Other possible developments are then discussed and the need for continuous intrapartum biochemical assessment of the fetus is emphasised.

In the appendices the apparent cost-effectiveness of intrapartum cardiotocography is shown and the safety of diagnostic ultrasound is reviewed; problems of automated management of labour are demonstrated and a diagnostic regimen for the learner of FHR trace analysis is presented. Finally there is an author's bibliography.

TABLE OF CONTENTS

	Page No.
ABSTRACT	i
TABLE OF CONTENTS	ii
LIST OF TABLES	iv
LIST OF ILLUSTRATIONS	vii
INTRODUCTION	1
<u>SECTION I</u> : The History of Fetal Heart Rate Monitoring including a discussion of common terminology	3
<u>SECTION II</u> : The Obstetric Perspective	15
<u>SECTION III</u> : Complications of Intrapartum Cardiotocogra- phic and Fetal Blood Sampling Techniques	26
<u>SECTION IV</u> : Misinterpretation and Misuse of Intrapartum Cardiotocography	39
<u>SECTION V</u> : Fetal Distress (Fetal Acidosis) including an examination of the fetal heart rate traces in 41 acidotic or pre-acidotic fetuses	65
<u>SECTION VI</u> : Part 1 : Introduction	77
Part 2 : The Analysis of Continuous Fetal Heart Rate Traces in the First and Second Stages of Labour	81
<u>SECTION VII</u> : The Aetiology, Characteristics and Diagnostic Relevance of Late Deceleration Patterns in Routine Obstetric Practice	101
<u>SECTION VIII</u> : Progression in Response Patterns of Fetal Heart Rate throughout Labour	112
<u>SECTION IX</u> : General Discussion	131
<u>SECTION X</u> : Baseline Variability	164
<u>SECTION XI</u> : Future Considerations	178
CONCLUSIONS	191
<u>APPENDIX A</u> : The Cost Effectiveness of Intrapartum Fetal Heart Rate Monitoring	193

	Page No.
<u>APPENDIX B</u> : The Safety of Diagnostic Ultrasound	196
<u>APPENDIX C</u> : A Controlled Trial of the Cardiff Automatic Infusion System in the Management of Induced labour	208
<u>APPENDIX D</u> : A Diagnostic Regimen for the Inexperienced Observer	218
<u>APPENDIX E</u> : Publications by Gareth Thomas, M.B.BS.,M.R.C.O.G.	220
ACKNOWLEDGEMENTS	223

LIST OF TABLES

Page No.

SECTION I

TABLE I:1	Fetal heart rate classification as proposed by Huntingford and Pendleton, 1969. (Journal of Obstetrics and Gynaecology, <u>76</u> , 586)	9
-----------	--	---

SECTION III

TABLE III:1	Known complications of intra-partum fetal monitoring techniques	27
TABLE III:2	The organisms isolated from fetal scalp abscesses attributable to intrapartum fetal monitoring techniques in twenty-two reported cases	30

SECTION IV

TABLE IV:1	Indications for intrapartum fetal heart rate monitoring	44
TABLE IV:2	General statistics - June 1973-June 1974 - U.C.H.	48
TABLE IV:3	Stillbirths - June 1973-June 1974 - U.C.H.	49
TABLE IV:4	Neonatal deaths - June 1973-June 1974 - U.C.H.	50
TABLE IV:5	Factors associated with low Apgar scores - June 1973-June 1975 - U.C.H.	52
TABLE IV:6	Showing the indications for FHR monitoring which were present in the unmonitored low Apgar cases - June 1973-June 1974 - U.C.H.	54
TABLE IV:7	Apparent birth asphyxia (Base excess greater than -10) - June 1973-June 1974 - U.C.H.	55
TABLE IV:8	Miscellaneous signs of neonatal morbidity - June 1973-June 1974 - U.C.H.	56
TABLE IV:9	The use of cardiotocographic monitoring in low Apgar fetuses - June 1973-June 1974 - U.C.H.	58
TABLE IV:10	Comparing written observations made in labour with the true cardiotocographic appearances in thirteen wrongly interpreted traces - June 1973-June 1974 - U.C.H.	59
TABLE IV:11	Distribution of Apgar scores in thirty-nine low pH fetuses - June 1973-June 1974 - U.C.H.	61

SECTION V

TABLE V:1	The percentage incidence of clinical and FHR signs in 37 of the 41 acidotic and pre-acidotic fetuses detected at U.C.H. between June 1973 and June 1974	72
-----------	---	----

SECTION VI Part 2

TABLE VI:1	List of measured parameters (relating to trace analysis)	82
TABLE VI:2	Classification of baseline variability	86

SECTION VII

TABLE VII:1	The factors associated with the appearance of late decelerations	104
TABLE VII:2	The incidence of abnormal fetal heart rate patterns associated with late decelerations	106

SECTION VIII

TABLE VIII:1	Definition and description of fetal heart rate response patterns observed in the 70 traces	114
TABLE VIII:2	The significance of five minute segments of constant response pattern in the two groups of infants (low Apgar and high Apgar groups)	118
TABLE VIII:3	Types of first response pattern following a normal trace in the two groups of infants	120
TABLE VIII:4	Progression of response patterns through transient acceleration in the two groups of infants	121
TABLE VIII:5	Distribution of patterns following pre-acceleration dips, early decelerations and saltatory responses	123
TABLE VIII:6	Distribution of episodes of basal rate and FHR variability recorded in the two groups of infants	123

SECTION IX

TABLE IX:1	Classification of FHR changes in relation to fetal scalp blood pH and Apgar score as suggested by Wood and colleagues - American Journal of Obstetrics and Gynecology (1969) 105, 942	150
TABLE IX:2	O'Gureck's proposed practical classification designed to assist international communication - O'Gureck, J.E. et al. (1972). Obstetrics and Gynecology, 40, 356	151

APPENDIX A

TABLE I: Appx.A	Quinquennial costs of an intrapartum fetal heart rate monitoring service for 2,000 deliveries per year	194
-----------------	--	-----

APPENDIX B

TABLE I: Appx.B	Showing the field intensities produced by diagnostic equipment in routine use at University College Hospital in 1973/1974	198
-----------------	---	-----

APPENDIX C

TABLE I: Appx.C	Comparison of the age, parity, management of labour, length of labour and method of delivery in the fifteen matched pairs (Cardiff pump v Ivac drip counter)	213
TABLE II: Appx.C	Relative frequencies of the alarms and situations responsible for the triggering of false alarms in the Cardiff system.	214

LIST OF ILLUSTRATIONS

Page No.

SECTION I

- | | | |
|----------|--|---|
| Fig. I:1 | The title page of Lejumeau's thesis on auscultation of the fetal heart | 5 |
|----------|--|---|

SECTION III

- | | | |
|------------|--|----|
| Fig. III:1 | Showing the modern spiral electrode and the older type of clip electrode which may be used for hemostasis following fetal blood sampling | 35 |
|------------|--|----|

SECTION IV

- | | | |
|-----------|--|----|
| Fig. IV:1 | Showing pre-acceleration | 41 |
| Fig. IV:2 | Showing rebound accelerations | 41 |
| Fig. IV:3 | Showing saltatory baseline fluctuations of fetal heart rate (FHR) | 41 |
| Fig. IV:4 | Showing an FHR trace, obtained by Doppler, in which decelerations were mistakenly attributed to loss of signal during contractions | 42 |

SECTION V

- | | | |
|----------|--|----|
| Fig. V:1 | Classic 'cord-compression' decelerations | 68 |
|----------|--|----|

SECTION VI : part 2

- | | | |
|-----------|--|----|
| Fig. VI:1 | Lag times | 84 |
| Fig. VI:2 | Rates of change in fetal heart rate | 84 |
| Fig. VI:3 | Scatterograph of baseline variability scores and corresponding pH values in fifty instances in the first stage of labour | 89 |
| Fig. VI:4 | Graph relating slope of recovery with corresponding slope of deceleration in decelerations observed in the first stage of labour | 90 |
| Fig. VI:5 | Scatterograph of baseline variability scores in the last hour of each of fifty labours and their corresponding one-minute Apgar scores | 92 |

SECTION VIII

- | | | |
|-------------|---|-----|
| Fig. VIII:1 | Showing the percentage distribution of fetal heart rate responses and comparing the low Apgar group with the total group, comprising both high and low Apgar neonates | 117 |
| Fig. VIII:2 | Postulated cycle of fetal heart rate response patterns during the transient hypoxic stress imposed by uterine contractions | 127 |

SECTION IX

Page No.

Fig. IX:1	Showing the way in which decelerations attributed to head compression return to baseline well before the tocographic recording	134
Fig. IX:2	FHR trace from an anaemic fetus during early labour (pH:7.14)	146
Fig. IX:3	Showing some of the more confusing aspects of FHR classification	148
Fig. IX:4	Showing the use of different-sized 'observation windows' in the assessment of baseline variability	154

SECTION X

Fig. X:1	Showing the two types of variation seen in baseline fetal heart rate variability	166
Fig. X:2	Showing the predictive capability, in terms of pH, of a '5-10 minute observation window' when applied to the traces analysed in Section VI	170
Fig. X:3	Showing an enlargement of FHR trace over one minute (X:3a) and comparing the degree of information obtained regarding variability using a '10-20 second observation window' and a '30-60 second observation window' respectively (X:3b)	172

SECTION XI

Fig. XI:1	Showing how the use of a '30-60 second observation window' for the assessment of baseline variability particularly facilitates the detection of a reduced <u>frequency</u> of long term baseline variability	181
Fig. XI:2	Showing a profound deceleration of fetal heart rate caused, in this instance, by supine hypotension	186

APPENDIX C

Fig. I (App.C)	In which are compared the respective rates of increase of oxytocin dosage using the Cardiff automatic infusion system and an Ivac-501 drip counter operated manually	210
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INTRODUCTION

This discourse on the utilisation of intrapartum cardiotocography is an attempt by the author first, to assess the usefulness of the technique in lowering perinatal mortality and morbidity and second, to determine the problems associated with the technique and to seek out ways of overcoming them.

The reports contained herein are the result of observations made on the Labour Ward at University College Hospital, London between November 1972 and December 1975 during which time the author was first, a Research Registrar in Fetal Monitoring and then, a Clinical Lecturer in Obstetrics and Gynaecology. The original investigative work which is reproduced in Section IV to VIII inclusive and in Appendix C was carried out mainly by this author but assistance with statistical testing was provided by a medical physicist, R. J. Blackwell, who therefore appeared as a co-author when Sections VI and VIII and Appendix C were published. A half-share of the trace assessment involved in the preparation of Section VIII was undertaken by D. T. Y. Liu, Research Fellow, but the majority of the published text was attributable to this author.

Although the work described was completed in December 1975, clinical pressures have prevented the final preparation of the manuscript until the present time. However, every attempt has been made to upgrade the commentary so that it pertains to the utilisation of intrapartum cardiotocography in December 1977.

The work was carried out and the manuscript prepared during a period of apparent unrest between an outspoken minority of the lay public and the profession. As the use of intrapartum cardiotocography has increased so, too, has the demand for so-called 'natural childbirth', Le Boyer delivery and home confinement. Whilst the proponents of these latter techniques remain in a minority group, they succeed in engendering a more generalised suspicion of modern obstetric management. With this sort of atmosphere prevailing there are obstetric units which strive to keep investigative procedures to a minimum

and although cardiotocography is now a routine procedure in all but a very few units in Great Britain, the use of fetal blood sampling as a complementary procedure is by no means universal. One of the aims of the investigations described in this discourse was to determine whether or not fetal blood sampling was necessary in order to make a definitive diagnosis of so-called 'fetal distress'.

A second aim was to seek out a single variable or a small number of variables of fetal heart rate patterns which might be used to screen out fetuses which are at risk of becoming acidotic. Such simplification, were it possible; would facilitate both teaching and automated fetal heart rate analysis.

The third aim was to see if there might be any prognostic value in studying the less common fetal heart rate patterns such as accelerations associated with decelerations, or in studying the progression of patterns occurring in any one particular patient with especial reference to the response to the first monitored contraction.

Fourthly, it was intended to determine whether cardiotocography was being used to the best of its advantage, with the least possible complication rate and without an unnecessary rise in Caesarean section rate.

It should be stressed at this juncture that all the studies described were carried out on purely clinical material in an attempt to assess current clinical techniques.

Each section has been written as a separate paper so that there is, inevitably, some repetition of references. Repetition within the text has been avoided as much as possible.

I: THE HISTORY OF FETAL HEART RATE MONITORING
including a discussion of common terminology

Everything has been said before by somebody who did not discover it.

ALFRED NORTH WHITEHEAD

It is proposed to describe the discovery of the fetal heart beat and the evolution of fetal heart rate monitoring as an aid to the diagnosis of fetal distress. Before so doing it is important to define what is meant by the term 'fetal heart rate monitoring' and to place it within its true perspective in relation to overall obstetric management. During the last decade the technique of continuously recording the fetal heart in conjunction with uterine activity (cardiotocography) has come to be known simply as fetal monitoring. This is incorrect; the terms 'fetal heart rate monitoring' and 'fetal monitoring' are not synonymous. Fetal heart rate monitoring should be seen as part of the technique of cardiotocography which, in turn, should be considered as part of a larger programme of fetal monitoring which also includes antenatal clinical assessment of fetal growth, placental function tests, serial ultrasound scanning for growth, fetal movement charts, fetal breathing movement studies and, in labour, fetal blood sampling.

Fetal monitoring and neonatal medicine, collectively known as perinatology, are relatively new and rapidly developing fields of medicine. This is demonstrated by the fact that the oldest method of antenatal fetal monitoring, that of regular clinical assessment of fetal growth, was not introduced until Dr. Ad Pinard's treatise on malpresentations was published in 1878 (1). There are earlier references to antenatal care but these refer purely to the health and well-being of the mother (2, 3, 4, 5). In 1895 Pinard described the establishment of an institution for the care of unmarried, and usually destitute, expectant mothers. Although this institution was not the first of its kind (6) Pinard was the first to record that babies born to the inmates were, in general, healthier than those born to mothers who had worked until labour commenced (7). Similar benefits are said to have been noted by Haig-Ferguson at the Lauriston Prematurity Home in Edinburgh and as a result of both his efforts and

those of Ballantyne (8) the introduction of preventative, antenatal supervision of both mother and fetus took place between 1901 and 1915 at the Royal Maternity Hospital, Edinburgh (9).

It is not certain when auscultation of the fetal heart became a routine part of an antenatal or intra-partum examination. Ballantyne referred to antenatal auscultation in his paper and the simple fetal stethoscope is attributed to Pinard, himself. It is highly likely, therefore, that the technique became routine as soon as the concept of antenatal care of the fetus became accepted and that the use of the fetal heart rate as an indicator of fetal well-being has developed in parallel with antenatal care.

It is intriguing that, despite several historic references to fetal life in utero (10a, 10b, 11a, 11b), the biological concept of a living fetus as an individual in utero, whose welfare might differ from that of the mother, is less than a hundred years old. Leonardo da Vinci described the fetus as a living entity but had no appreciation of its individuality in utero (12). The earliest record of a fetal heart being heard is dated 1650 and appears in a satirical French poem written by a physician named Phillipe le Goust who taunted a colleague for claiming to have heard the heart of a fetus beating 'like the clapper of a mill' (13). The first scientific reference to auscultation of the fetal heart appeared in 1822 in a thesis written by J. A. Lejumeau de Kergaradec (14). He had become convinced of the usefulness of auscultation and sought to apply its use to pathological states other than those described by Laennec (15). Lejumeau was under the impression that fetal movements gave rise to amniotic fluid thrills and while listening for such a thrill he heard what he described as double pulsations. 'Étonné, j'hésitais à porter un jugement sur la cause de ces pulsations. Je les comptai pendant quelque temps et je trouvais qu'elles se renouvelaient de cent quarante-trois à cent quarante-huit fois par minute..... Il fallut donc bien reconnaître qu'ils étaient produits par les contractions du coeur du foetus'. [Please see Fig. I:1]

In a postscript to his thesis, Lejumeau officially withdrew his claim to being the first to discover and record auscultation of

167
MÉMOIRE

SUR

L'AUSCULTATION

APPLIQUÉE A L'ÉTUDE DE LA GROSSESSE

ou

RECHERCHES

SUR DEUX NOUVEAUX SIGNES PROPRES A FAIRE RECON-
NAÎTRE PLUSIEURS CIRCONSTANCES DE L'ÉTAT DE
GESTATION ;

*Lu à l'Académie royale de médecine, dans sa séance
générale du 26 décembre 1821.*

PAR M. J.-A. LEJUMEAU DE KERGADEDEC,

Docteur en médecine de la Faculté de Paris, médecin du Bureau de
charité du septième arrondissement, membre de la Société de
médecine et de l'Athénée de médecine de Paris.



PARIS,

CHEZ MÉQUIGNON-MARVIS,

LIBRAIRE, RUE DE L'ÉCOLE DE MÉDECINE, N° 3.

1822.

Fig. I:1

*The title page of Lejumeau's thesis on
auscultation of the fetal heart*

the fetal heart. Since writing his thesis he had read that the observations of one M. Moyat (16) showed that one could recognise if an infant at term was alive or not and 'qu'il est mort si l'on n'entend rien'. This latter statement caused Lejumeau some concern, not unnaturally, but on reading the original article himself (17) he could find no such statement and, indeed, found that the author was one M. Maior, a close colleague of Laennec.

There were very few significant developments in the understanding of fetal heart rate during the remainder of the nineteenth century. In 1833 Kennedy (18) described the fetal heart sounds and the funic souffle in considerable detail and in 1859 Frankenhauser (19) described a sex difference in fetal heart rates, a difference which was later confirmed by McClintock (20). Perhaps more important was Naegele's description in 1838 of fetal bradycardia associated with head compression (21).

In 1907 Von Winckel reported a poor fetal outcome associated with fetal bradycardia and tachycardia (22) and in 1917 similar reservations with respect to fetal tachycardia were published by Baumm who considered that tachycardia occurred secondarily to intracranial haemorrhage (23). Despite these earlier investigations the full potential of listening to the fetal heart rate in labour in order to assess fetal welfare was not appreciated. Indeed, fetal welfare was not of prime importance at that time. It was not until the massive loss of young life in the 1914-1918 war and the consequent passage of the Maternity and Child Welfare Act (England and Wales) in 1918 that a greater concern for the fetus developed. Auscultation of the fetal heart became routine not only at antenatal visits but also during labour.

There were very few English language publications concerned with fetal heart rate monitoring in the first half of the twentieth century. Bartholomew in 1925 (24) and Freed in 1927 (25) reported on the incidence and prognostic importance of fetal tachycardia and both authors suggested that it was neither as frequent nor as serious as had been supposed by earlier writers. In 1936 Richardson attempted to relate fetal tachycardia and bradycardia in terms of placental

separation, suggesting that such an event caused fetal tachycardia unless the area of separation was greater than one quarter of the total surface area in which case fetal bradycardia became the predominant pattern (26). King presented some personal observations to the New Orleans Gynecological and Obstetrics Society in 1939 (27); he considered 160 beats per minute as too low a level at which to set the upper limit of normal and suggested that the limit be raised to 180 beats per minute. Similarly he questioned the rate of 100 beats per minute as the correct lower limit of normal and considered that it, too, should be raised. In 1943 Lund questioned again the reliability of fetal tachycardia as a sign of fetal distress partly because of an apparently low related neonatal morbidity and partly because the administration of oxygen to the mother had no effect on the tachycardia (28).

From the account so far it is apparent that, other than Naegele's early observations, there were virtually no references to any human fetal heart rate variables other than the basal rate and that even the limits of normal rate were little understood.

One reason for the slow progress in the understanding of fetal heart rate was the lack of an adequate means of recording it continuously. Amplification of fetal heart sounds was a relatively simple process but the exclusion of additional noises such as bowel sounds was extremely difficult. In 1906 Cremer (29) had described a technique of obtaining a fetal electro-cardiograph (FECG) but it was almost 1940 before further reports of fetal electrocardiography became available (30, 31). Amplification of the signal was a problem which led Edward Hon to investigate the possibility of triggering a ratemeter using FECG signals (32). By 1960 Hon and Hess were in a position to compare the relative merits of FECG monitoring and fetal heart rate (FHR) monitoring (33). They reported that the electrocardiographic abnormalities, caused by anoxia and seen just prior to fetal death, were preceded by a considerable length of time by abnormalities of fetal heart rate. These findings led Hon to concentrate his own efforts and those of his team on the correlation between FHR abnormalities and clinical events. In 1967 he published a working classification 'of FHR' (34) which was followed, in 1968, by an atlas of fetal heart rate patterns (35). About the same time

Caldeyro-Barcia's team presented similar findings but the method of classification was different (36). Meanwhile, in Germany, Hammacher had designed a fetal phonocardiograph which, because of its particular filter levels and the rapid response time of the recorder pen, gave better records of the fetal heart rate than had been achieved before with phonocardiography (37). The production and distribution of this instrument was taken up on a commercial basis by Hewlett-Packard and the design was such that the ratemeter and recorder pen could be triggered either from phonocardiography or from electrocardiography. Hammacher considered that the baseline irregularity was the most important variable to be considered when interpreting FHR (38). In 1969 Huntingford and Pendleton published a classification of fetal heart rate which took into account the classifications of both Hon and Caldeyro-Barcia and also the classification proposed by Hammacher (Table I:1) (39); this has been the basis of the British understanding of fetal heart rate over the last seven to eight years. The authors of that paper made a plea for further assessment of the clinical significance of different patterns of fetal heart rate and particularly for clarification of the significance of different degrees of baseline irregularity.

In 1971 the results of the first clinical assessment carried out in Britain were reported by Beard and colleagues (40) who had analysed fetal heart rate traces obtained from defined high risk labours over the course of one year. They concluded that, in terms of clinical results, i.e. pH and Apgar score, the important features to observe in fetal heart rate traces in order to diagnose the possibility of fetal distress were

- a) the appearance of decelerations
- b) the amplitude and timing of those decelerations relative to uterine contractions
- c) changes in beat-to-beat variation which was a term then used to describe baseline irregularity, and
- d) baseline tachycardia.

The authors also concluded that continuous fetal heart rate monitoring, used alone, would result in false positive diagnoses of fetal asphyxia and advised that, in practical terms, an abnormal fetal heart rate should be regarded as a warning sign indicating the need

TABLE I:1 FETAL HEART RATE CLASSIFICATION AS PROPOSED BY
HUNTINGFORD AND PENDLETON. 1969

FHR Feature	Classification
Baseline rate	Normal : 120-160 b.p.m. <u>Tachycardia</u> : Moderate : 161-180 b.p.m. : Marked : More than 180 b.p.m. <u>Bradycardia</u> : Moderate : 100-119 b.p.m. : Marked : Less than 100 b.p.m.
Baseline irregularity	<u>Silent</u> : 5 or less b.p.m.² <u>Narrowed undulatory</u> : 6-10 b.p.m.² <u>Undulatory</u> : 11-24 b.p.m.² <u>Saltatory</u> : 25 or more b.p.m.²
Periodic (transitory) changes of rate	Changes lasting less than 10 minutes and more than 20 seconds. Either outside the normal range or more than 30 b.p.m. change within the normal range. <u>Accelerations</u> : Moderate : 161-180 b.p.m. : Marked : Greater than 180 b.p.m. <u>Decelerations</u> : Moderate : 100-119 b.p.m. : Marked : Less than 100 b.p.m.
Decelerations	<u>Early</u> : Hammacher type I : Onset coincides with contraction <u>Late</u> : Hammacher type II : Delay of more than 30 seconds between contraction and deceleration <u>Variable</u> : Hammacher type 0 : No constant time relationship between contractions and decelerations.

to take a fetal blood sample in order to determine the fetal pH. This latter technique had been introduced to Britain by Morris and Beard in 1965 (41) but because of technical difficulties and its considerable potential inaccuracy it failed to become as universally acceptable as fetal heart rate monitoring. As a consequence there developed a school of thought which, using fetal heart rate monitoring alone, claimed results similar to, if not better than, those achieved using the combined method of monitoring (42). Most of the relevant literature of the last four years has been concerned with a deeper understanding of fetal heart rate patterns aimed either at avoiding the need to carry out fetal blood sampling or at allowing a stricter definition of the abnormal so that the need for fetal blood sampling might be minimised. With respect to the complete avoidance of fetal blood sampling Shelley and Tipton introduced the concept of dip area measurement (43) and Goodlin suggested a classification of fetal heart rate which was based on the 'reactivity' of the fetus (44) and which was, therefore, totally different to the original concepts of Hon and of Caldeyro-Barcia. Other than the work of Shelley and Tipton and the suggestions of Goodlin there has been nothing 'revolutionary' during the last five years. Much of what has been published will be referred to in the subsequent text which is concerned with the effectiveness of FHR monitoring, the possibility of avoiding fetal blood sampling, the recognition of the abnormal trace and the role of baseline irregularity in the diagnosis of fetal distress.

Before studying these factors it is necessary to examine the place of fetal heart rate monitoring in the total obstetric perspective which, when viewed over the last decade, also includes the development of many other obstetric investigative procedures and a rapidly changing consumer attitude towards maternity hospitals, their staff and their techniques. It is also necessary, before any attempt is made to assess the usefulness of FHR monitoring, to examine critically any possible ill effects or complications of the techniques involved, whether they be emotional or physical.

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II: THE OBSTETRIC PERSPECTIVE

When parents order a baby, they order a good one.

ALFRED WHITE FRANKLIN

The history of the fetal heart has been traced from the time of its first recognition (1) up until 1971 when a basic classification of fetal heart rate patterns was tested in terms of neonatal outcome and fetal scalp blood pH (2). Prior to 1971 intrapartum cardiotocography had been a clinically experimental technique with limited availability in Great Britain, but during that year, as a result of an increasing number of relevant publications (2, 3, 4, 5) together with a Study Group on Fetal Monitoring arranged by the Royal College of Obstetricians and Gynaecologists (6), the technique became acceptable and available to many hospitals as a labour ward routine. At the time of writing in 1977 there are thought to be very few maternity units in Great Britain without at least one available monitor (7). The relative rapidity with which the technique has been introduced may be appreciated by referring to a leading article in the British Medical Journal in February 1966, just over 12 years ago, in which it was stated that 'the time may be coming when the medicine of the unborn foetus will occupy doctors almost as much as the medicine of post-natal life; there was no reference to fetal heart rate monitoring (8).

There have been many other developments in obstetrics in the last ten years but these have not been without their difficulties, of which two are particularly worthy of note. The first has been the method of assessing these developments, a problem which has resulted in a growing obsession of obstetricians and paediatricians with the perinatal mortality rate. The second has been the rapidly changing and somewhat strained relationship between obstetricians and their patients.

THE PERINATAL MORTALITY RATE

This rate expresses the number of deaths occurring after the 28th week of pregnancy, and before the end of the first week after

delivery, per 1,000 total births; it is a rate which reflects the standard of obstetric and neonatal care and has been used as such since it was first introduced by the Registrar General in 1928. At that time, in England and Wales, the rate was 61 per 1,000 total births (9). By 1958 the rate was only 35 per 1,000 (10); the significant reduction was attributed to improved maternity and neonatal services associated with the introduction of antibiotics between 1939 and 1945 and the setting up of the National Health Service in 1948. In 1958 a perinatal survey was carried out by the National Birthday Trust in order to ascertain which maternal factors were associated with higher perinatal mortality rates (11). Since that time the perinatal mortality rate has been used increasingly as a means of obstetric and neonatal audit but, whilst the motive should not be criticised, it should be appreciated that perinatal morbidity rates would be far more relevant, were they more easily measurable. Similarly it must be realised that any such rate is only of use in assessing the overall obstetric/neonatal standard of care and cannot be used to demonstrate the effectiveness of any single development. The perinatal mortality rate (U.K.) was 23 per 1,000 in 1970 (12) and by 1975 it had fallen to an average of 19.3 per 1,000 (13), the figure in many individual centres being almost as low as 10 per 1,000 (14, 15, 16). The dramatic fall is attributable to a multitude of factors, including the further development of placental function tests, the increasing use of ultrasonography in assessing gestational age and localising the placenta, the facility to prevent lung immaturity at birth by the measurement of lecithin/sphingomyelin ratios or palmitate levels in the liquor, the development of cardiotocography and fetal blood sampling, the prevention of Rhesus iso-immunisation using anti-D gamma-globulin, the use of genetic amniocentesis when indicated by maternal age or a history of congenital abnormality and the measurement of alpha-feto protein in maternal blood and amniotic fluid in order to detect anencephaly and spina bifida. In addition there have been many significant improvements in neonatal care. There have been so many simultaneous developments that it is impossible to determine the effect of any one of them on the perinatal mortality rate and this applies as much to cardiotocography as it does to any of the others.

THE DOCTOR-PATIENT RELATIONSHIP IN OBSTETRICS

The dramatic fall in perinatal mortality rate which has taken place since 1935 has already been described. The forty year span in which this fall occurred is the equivalent of nearly two reproductive generations, a time span which has made the majority of the current obstetric population totally unaware of the risks associated with childbirth prior to the 1940's. It is now taken for granted that for each individual who conforms with the current system of maternity care the chance of either losing a baby or delivering a 'faulty' one is extremely low. That the system has been developed or, more accurately, has been allowed to evolve in order to minimise the chance of tragedy is no longer appreciated. As a result, there is an almost constant barrage of criticism from a minority group which would prefer a return to older, less safe, methods, purely in the interests of maternal psychology (17). The majority of obstetric patients appear happy to accept the more modern and safer methods especially if the underlying rationale is explained, preferably at antenatal classes. However, a small nucleus of 'medical journalists' persist in their attempts to drive a wedge between obstetricians and their patients, using commentaries which are usually anecdotal and uninformed (18) or, alternatively, highly edited (19). Of the commentators who have been conscientious in their attempts to gain information and to present it accurately, Shearer is well-known. A prologue from one of her recent commentaries is quoted here because it highlights many of the apparently small, but nevertheless accumulative, points which lead to an unpleasant patient-doctor divergence.

"..... She panted quickly and rubbed her abdomen, her husband holding her other hand. At this point the door was propped open and a cabinet-sized machine was rolled into the room, pushed by a nurse. It stopped next to the bed, with a brief pause for the removal of the husband's chair to the corridor outside. The hand which had held the husband's was searched for a vein and an intravenous drip of glucose installed. The other arm was connected to a continuous blood pressure gauge. The abdomen was encircled with two belts and the electronic devices on the belts were squirted with jelly and planted firmly

on the mother's abdomen, the belts then tightened securely. The monitor was turned on and out rolled a continuous strip of paper bearing the fetal heart rate tracing, the uterine contraction tracing and the blood pressure tracing. The husband found a perch on a window sill across the room. For the next seven hours a nurse or resident would come in at 30 minute intervals, study the trace carefully, and tell the mother that all was well." (20).

There was an obvious failure of communication in this particular instance. This situation can only be avoided by closer attention to patient psychology, not only by the patients' doctors and attendants but by architects and designers alike, and by increasing antenatal education by informed personnel who can define the concept of a normal, modern labour.

There is nothing more conducive to a straightforward labour and delivery than for the patient to feel that she is normal. Currently the use of a fetal heart rate monitor or of an intravenous infusion is considered by many patients to be abnormal. It is hoped that with the increasing use of antenatal classes this attitude may soon be eradicated. Unfortunately the classes are often addressed to a self-selected audience or to patients who have sought advice elsewhere and there is an argument for introducing the concept of a normal, monitored labour in biology classes at secondary schools. The normality of an electronically monitored labour will be further promoted by the increasing use of monitors; there may be other arguments in favour of monitoring all labours (21) but there are far reaching implications which involve finances and staffing structures and these will be discussed later in the text.

JUSTIFICATION FOR THE USE OF ELECTRONIC FETAL HEART RATE MONITORING

Through the last decade in which electronic fetal heart rate monitoring has slipped into the obstetric perspective there have been requests for its justification from several quarters, first from ob-

stetricians and then from patients. Currently the greatest demand for justification comes from financial administrators.

It is unfortunate that there is no objective, scientific evidence to support the role of electronic fetal heart rate monitoring in the reduction of perinatal mortality or morbidity. While its importance would seem obvious there are no controlled trials to support that contention. With regard to cardiotocography, there should have been, at some time, a controlled multicentre trial but the technique is now accepted as routine to such an extent that a trial of that nature would be open to severe criticism on ethical grounds. The closest approximation was a comparison of two hospital practices in Melbourne (22). There was a highly significant difference in results in favour of the monitored practice but the poor results in the unmonitored practice were attributable to traumatic and prolonged deliveries rather than to a failure to detect abnormal fetal heart traces. In 1975 Walker (23) reported a series of 3,659 patients in his own multi-racial South African practice in which he 'pays no attention to fetal monitoring'; he compared his perinatal mortality rate with that observed at the Groote Schuur Hospital in Cape Town (36 per 1,000), the overall rate in the United Kingdom (22.3 per 1,000) and a two year report from Potiuncle Hospital, Ballinasloe, Ireland (31.6 per 1,000). His figure of 18 per 1,000 (actually 17.2) compared favourably but the latter two comparisons were very unsuitable and there was no attempt made to describe the obstetric population of the Groote Schuur Hospital. Close study of Walker's tables suggests that intrapartum fetal heart rate monitoring may have reduced the perinatal mortality rate by between 1 and 3 per 1,000 and the author, himself, confesses that such monitoring may have expedited delivery in at least five out of 32 infants who showed evidence of distress at birth and required special neonatal care.

Gabert and Stenchever (24) conducted a trial which showed 'very little difference' in fetal mortality as a result of FHR monitoring but there was a statistically significant lowering of neonatal mortality after the introduction of a monitoring programme. This result either reflects a simultaneous improvement in neonatal special care facilities or it suggests that the monitored infants were deli-

vered in a generally better condition than those who were unmonitored, but this might well be due to the secondary intensification of care in labour as much as to the detection of abnormal heart rates. Paul and Hon (25) reported a large series of patients in which the intrapartum mortality rate in 28,208 unmonitored, low-risk patients was compared with the rate in 9,759 monitored, high-risk patients. The data was corrected for deaths attributable to congenital malformation and birth trauma and the respective rates were 1.1 per 1,000 and 0.6 per 1,000. The expected rate in an unmonitored high-risk group would be 2.2 per 1,000 (26), so the effect of fetal heart rate monitoring appears to be considerable. The effect appeared most marked in the 1,000-1,500 Gram group. There is, of course, a danger in correcting data for deaths attributable to birth trauma since hypoxia and susceptibility to trauma are so closely linked. In 1975 Tuter and Newman (27) reported their experience of monitoring high-risk patients over the course of four years. Their monitored group was small (608 patients) but the striking aspect of the results was the single neonatal death in that group together with the absence of intrapartum fetal loss. The poor figures in the unmonitored group suggest that their monitoring programme was insufficiently aggressive and it is interesting to note that, as a result of their experience, the authors now monitor 98 per cent of their patients.

In Britain there seems to be general agreement that the incidence of intrapartum stillbirth is reduced (21, 28) and in University College Hospital there were no intrapartum stillbirths during the three year observation period reported in this thesis, other than those attributable to non-viable congenital abnormalities.

The knowledge that electronic intrapartum fetal heart rate monitoring reduces the intrapartum stillbirth rate has been extrapolated to provide the most important justification for FHR monitoring, the reduction of perinatal morbidity secondary to intrapartum hypoxia. Quilligan has recently reiterated that 'it seems not unreasonable to assume that the stress of labour may be a factor in producing brain damage, since it is clearly capable of causing fetal death' (29). There is evidence which shows that severe intrapartum hypoxia in the sub-human primate can cause cerebral palsy (30) and a survey of all cases of cerebral palsy occurring in Sweden between 1954 and 1970

has shown that birth asphyxia played a prominent role (31). Despite this and the fact that both circumstantial evidence and logic would suggest a spectrum of end results attributable to varying degrees of hypoxia, Quilligan's assumption is not an entirely safe one. Two studies, in 1967 and 1970 respectively, have reported the follow-up of infants who were hypoxic at birth (32, 33) and in neither study was there evidence to suggest long term sequelae, other than death. There appears to be an all-or-nothing relationship between hypoxia and perinatal consequence; either the hypoxia is 'very severe' in which case the infant may be asphyxiated and death, or, perhaps, severe brain damage may result, or the hypoxia is of a lesser degree in which case it is apparently of no long term consequence. Unfortunately, it is not known at what level hypoxia becomes 'very severe' or what relation that cut-off point has with human fetal heart rate patterns. This does not detract from the apparent value of FHR monitoring in the prevention of perinatal death, spasticity and, presumably, severe mental retardation but it does lead to doubt as to the effectiveness of FHR monitoring in preventing the occurrence of low grade retardation or educational subnormality and it certainly raises a doubt as to the value of IQ tests in assessing the effectiveness of FHR monitoring.

FINANCIAL CONSIDERATIONS

Electronic fetal heart rate monitoring was introduced as a service commodity at a time when the National Health Service was undergoing tremendous expansion. However, the present economic climate presents strict financial limitations and there has arisen a need to cost many of the more modern techniques. The need to cost the technique of fetal heart rate monitoring has become more acute since the suggestion that all labours should be so monitored.

Figures quoted by Quilligan and Paul in the United States of America (34) are much in agreement with those obtained in a costing exercise carried out by the author at University College Hospital early in 1975. (Please see Appendix A). The only item which showed discrepancy was that of added personnel and this obviously varies between centres because of its dependence on pre-monitoring staffing

levels. Any unit using monitoring techniques should have the full or part-time use of an electronics technician and a supervisory midwife who is able to maintain the equipment and supplies. Even allowing for such an expensive item as additional personnel, the cost per patient of electronic fetal heart rate monitoring is negligible compared with the cost to a family and to the State of severe mental or physical handicap (Appendix A).

An attempt has been made to present both clinical and financial justification for the use of fetal heart rate monitoring in labour. It is, of course, necessary to ensure that the techniques used present no added risk to the fetus.

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III: COMPLICATIONS OF INTRAPARTUM CARDIOTOCOGRAPHIC AND FETAL BLOOD SAMPLING TECHNIQUES

Experience is the name everyone gives to his mistakes

OSCAR WILDE

The preceding text has outlined the reasoning behind the use of FHR monitoring and has demonstrated that the advantages of monitoring, although logically apparent, are difficult to prove statistically because of the lack of control data and the simultaneous influence of so many other recent developments in obstetrics. It is 'doubly' important, therefore, to ensure that cardiotocographic monitoring techniques are entirely safe for both mother and fetus and that they present no additional hazard during labour.

The forthcoming text indicates that complications are either rare or rarely reported and that the more serious complications appear to be preventable by ensuring adequate training in monitoring techniques. Since the incidence of complications is small it is vital that any which do occur are reported and published; in this way the maximum experience may be gained with the minimum number of mistakes or complications.

Known complications are listed in Table III:1. Since one of the briefs of this thesis is to discuss the role of complementary fetal blood sampling (FBS), complications attributable to that technique are included.

SCALP ELECTRODE COMPLICATIONS

In a preliminary clinical trial of monitoring techniques, in which clip electrodes were used in 249 patients, Hon (1) reported occasional minor scalp ecchymoses, one 2-mm pustule on the scalp and one scratch on a neonate's right arm, a total incidence of fetal complications of 0.82 per cent. Three instances of maternal endometritis and one superficial vaginal laceration were also attributed to the use of

TABLE III:1 KNOWN COMPLICATIONS OF INTRA-PARTUM FETAL
MONITORING TECHNIQUES

COMPLICATIONS ATTRIBUTABLE TO SCALP ELECTRODES:

- Bruising at applications site
- Infection at application site
- Bleeding from application site
- Penetrating injuries - fontanelle
- eye
- Other fetal lacerations
- Maternal lacerations
- Endometritis

COMPLICATIONS ATTRIBUTABLE TO INTRA-UTERINE CATHETERS:

- Uterine perforation
- Endometritis
- Pelvic abscess
- Retained portions of catheter

COMPLICATIONS ATTRIBUTABLE TO FETAL SCALP BLOOD SAMPLING:

Infection at sampling site
Haemorrhage from sampling site - free
- haematoma formation
Penetrating injuries - fontanelles or suture lines
Retained broken sampling blades
Alopecia

electrodes. Two more recent reports, each from Hon's department, gave incidences of 0.24 and 0.15 per cent, respectively, for fetal scalp abscesses following the use of scalp electrodes (2, 3).

Other, less frequent complications include bleeding from the site of application and mild cellulitis of the scalp (4). In 1975 Tuter and Newman (5) reported one cephalhaematoma and one scalp abscess in a series of 608 patients in whom scalp electrodes were used.

Most disturbing is the case reported by Goodlin and Harrod (6) in which cerebrospinal fluid drained for three days from the posterior fontanelle of a neonate following the use of a spiral electrode. According to Turbeville (7) a similar case was reported by Wilkinson and colleagues in the same journal but this proves to be a misquote.

At University College Hospital, London, between December 1972 and August 1974, there were 691 patients in whom FHR monitoring was carried out using scalp electrodes. During that time only two scalp abscesses were found at routine neonatal assessment within the first week of life. Both infants were born by caesarean section on the same day and both swab cultures grew bacteroides. There were no fetal bleeding disorders and there were no known maternal complications, although it is possible that mild episodes of endometritis occurred but were unnoticed or unreported. There was one complication which caused considerable concern- that was the penetration of a fetal eyelid and a consequent corneal scratch by a spiral electrode. The case was reported by the author in the American Journal of Obstetrics and Gynaecology (8).

Case report : The patient, a 25 year old primigravida, was admitted to the labour ward for surgical induction of labour at 42 weeks gestation. The pregnancy had been normal and the dates were certain. The lie was longitudinal and the presentation was cephalic, left occipito-lateral, not engaged but fixed in the pelvic brim. The cervical os was reported as 2 centimetres dilated and soft, but posterior and thick. The membranes were ruptured without difficulty but the clinician was unable to determine the position of the fontanelles or the suture lines prior to the application of a spiral electrode (15130A, Hewlett-Packard). An open-ended intrauterine catheter

was inserted after the application of the electrode and an intravenous infusion of oxytocin was commenced. The fetal heart rate was regular. Labour became established 90 minutes after induction and the head engaged after 150 minutes. The labour was normal in all respects and a spontaneous vaginal delivery of a live male infant took place after 9 hours. The Apgar score was 9 at one minute.

At delivery the scalp electrode was found attached to the medial end of the left lower eyelid. One of the two spirals had penetrated the substance of the eyelid and had caused considerable oedema. A minute scleral haematoma was present. The electrode was removed immediately and the infant was referred to the paediatricians for special surveillance. The oedema subsided rapidly and the haematoma resolved during the first 24 hours of life.

It is difficult to imagine the electrode becoming applied to the eyelid by any means other than by direct application at the time of induction. The most likely explanation is that the head was deflexed, that the clinician palpated the frontal bone and that the fetus moved before the electrode was applied. This stresses the importance of the clinician having the tip of his guiding finger actually against the presenting part at the time of application. In difficult cases where the cervix is long and uneffaced electrode application should be carried out only by experienced staff.

This last described case, together with the two aforementioned scalp abscesses, constituted an electrode complication rate of 0.43 per cent at University College Hospital during the twenty-month observation period.

The reviewed literature contains one fetal fatality which was apparently related to an infected application site (9). Briefly, the problem arose in an 1880 gram male infant born at 34 weeks gestation to a 23-year-old primigravid, pre-eclamptic patient. Labour lasted six hours, there was no fetal blood sampling and the infant was delivered with the aid of forceps. The one and five-minute Apgar scores were 8 and 9 respectively. Physical examination was compatible with a 34 week infant. There were no abnormalities other than a 1-2 mm puncture mark on the right occipital area at the site of electrode

TABLE III:2 THE ORGANISMS ISOLATED FROM FETAL SCALP ABSCESSSES
ATTRIBUTABLE TO INTRAPARTUM FETAL MONITORING
TECHNIQUES IN 22 REPORTED CASES

Literature source	No. of abscesses	Reported organisms	No. supposedly sterile
Paul, R.H. and Hon, E.H. Obstetrics and Gynecology <u>37</u> , 779. (4224 patients)	6	All 'sterile' (6)	6
Cordero, L. Jr. and Hon, E.H. Journal of Pediatrics <u>78</u> , 533. 1971 (4750 patients)	7	Beta-hemolytic streptococcus (1) Klebsiella (1) 'Sterile' (5)	5
Balfour, H.H. et al. American Journal of Obstetrics and Gynecology <u>107</u> , 288. 1970 (678 patients)	3	Escherichia coli (1) Aerobacter aerogenes (1) Staphylococcus albus (1)	
Messer, J. and Willard, D. European Congress of Perinatal Medicine. 1-1/7 Prague. 1974	4	Escherichia coli (1) 'Sterile' (3)	3
	2	Bacteroides (2)	
	22		14

placement. At 14 hours of age the infant became temporarily apnoeic; examination revealed crepitus in the occipital region and a skull X-ray showed air density in the same region. Cultures of blood, cerebrospinal fluid, urine and gastric acid were taken and the infant was treated with ampicillin and kanamycin. The crepitus increased only slightly but at 44½ hours of age the baby died after losing bright red blood from the mouth. Autopsy gave the cause of death as pulmonary haemorrhage. *Escherichia coli* were grown from the scalp and the blood; the authors suggest that the site of electrode application was the portal of entry for a fatal *E.coli* septicaemia. They advised that external monitoring should be used when feasible and that infants who are monitored using an invasive electrode should be observed closely for possible sequelae.

It is interesting to note that the high vaginal swab from the infant's mother grew *E.coli* also. Table III.2 shows the organisms which were isolated from the scalp abscesses reported in the foregoing papers. Of the 22 reported abscesses, 14 were supposedly sterile. Details of culture techniques are not given in the reports but it is a recognised fact that bacteroides species are easily missed (10) and that this species, although normally non-pathogenic and only commensal in nature, may give rise to serious septicaemia. It is therefore of extreme importance that any neonate demonstrating a scalp or soft tissue abscess is observed thoroughly during the first week of life regardless of the culture achieved and that an attempt is being made in the laboratory to isolate bacteroides and to determine sensitivities.

COMPLICATIONS FROM THE USE OF INTRA-UTERINE CATHETERS

The practice of intra-partum cardiotocography in Great Britain has tended away from the use of intra-uterine catheters except in some research environs. Some of the arguments for and against this method of tocography will be presented later when discussing the interpretation of fetal heart rate patterns. Complications are reported at rates which are similar in magnitude to the reported rates of electrode complications. Chan and colleagues (11) reported three symptomless uterine perforations in a series of 1,150 patients, a rate of 0.26 per cent, whilst Tutera and Newman (12) reported one perforation occurring in 608 patients, a rate of 0.16 per cent.

There were no fetal complications in these two series. Of the four reported perforations, two were found at caesarean section, one was suspected because of pressure changes caused by the Valsalva manoeuvre and one was detected by palpation of the catheter per abdomen. In 1973 Okada and Hobel (13) described an apparently avoidable hazard which had occurred twice in a series of 2,500 patients. In each case the disposable catheter had been resterilised and the catheter tip had broken off at the site of a side perforation. One tip was found at caesarean section, the other was passed at spontaneous vertex delivery.

Most uterine perforations appear to be asymptomatic and uncomplicated but Haverkamp and Bowes (14) reported an exception. A perforation had been suspected because of pressure changes observed during a Valsalva manoeuvre. The patient experienced right iliac fossa pain immediately post-partum but she was afebrile and was allowed home on the third day. On the ninth day she was readmitted with a continuous abdominal pain and a pyrexia; laparotomy revealed a 10 cm x 9 cm x 4 cm intraperitoneal abscess from which bacteroides and anaerobic cocci were isolated. The authors suggested that softer catheter guides than those described by Paul and Hon (15) would reduce the chance of perforating the uterus.

Maternal infections secondary to the use of intra-uterine catheters appear to be uncommon, being confined to the last mentioned case and rare instances of endometritis (16, 17).

PROBLEMS ASSOCIATED WITH THE USE OF EXTERNAL TOCOGRAPHY

There are no recorded complications associated with this technique although certain makes of tocodynamometer can be most uncomfortable, requiring to be worn beneath a tight abdominal strap in order to produce reasonable records.

THE POSSIBLE COMPLICATIONS OF DOPPLER ULTRASOUND

The biophysical effects of high intensity ultrasound may be thermal,

cavitation or direct by inactivation of enzymes or degradation of tissues. The aim of relevant research in latter years has been to ensure that low intensity ultrasound, such as that used in diagnostic methods in obstetrics, does not have these biophysical effects. Attempts have been made to define a threshold at which insonation may be considered safe. A full report concerning the safety of diagnostic ultrasound is presented in Appendix B. It may be seen that, despite considerable controversy, the safety of diagnostic ultrasound is assured provided that the equipment is cared for and frequently serviced and tested. There remains the burning question of the safety of a damaged ultrasound transducer (18, 19). This is a particularly important question if only because of the increasing realisation of the importance of FHR monitoring in premature labour. Direct access to the fetal scalp is rarely available and long term Doppler ultrasound monitoring is often necessary. The transducers are in constant use and are subjected to repeated trauma by labour ward staff. The quoted reports suggest that damaged transducers may be a potential danger to the fetus.

COMPLICATIONS OF FETAL BLOOD SAMPLING

As with scalp electrodes the main complications of fetal blood sampling are infection and haemorrhage. The sampling blades currently used are designed in such a way as to reduce the penetration to 2 millimetres so that deep penetrating injuries are unlikely to occur. In 1970 Balfour (20) described three scalp abscesses associated with sampling techniques used in 678 patients, an incidence of 0.44 per cent. It was suggested that such complications could be avoided by strict adherence to aseptic technique and careful neonatal surveillance. Similar localised abscesses had been described by Kubli (21). In view of the apparently sterile nature of most of the abscesses it is probable that the most common offending organism was bacteroides species. At University College Hospital, during the twenty-month observation period referred to earlier, there were 323 fetuses monitored with the aid of fetal blood sampling- there were no complications other than one instance of prolonged localised alopecia (22).

Although Saling referred to a fetal fatality associated with fetal

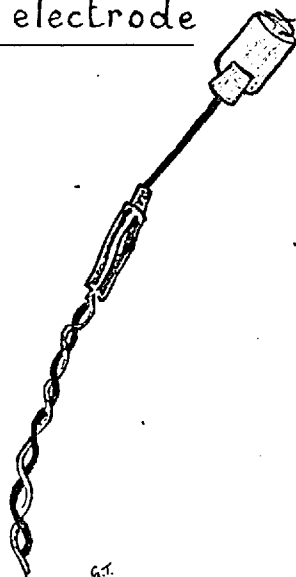
blood sampling (23), the problem was first highlighted by Beard and colleagues (24) who carried out 1025 fetal blood samples in 640 patients without any complications. Then two fetal deaths occurred. In the first case sampling was followed by free bleeding from the site and a fetal tachycardia. Eventually the fetal heart slowed and stopped and the fetus was stillborn eighteen hours later, having bled from a 4 millimetre incision which lay over the interparietal suture and included the wall of a vein draining into the longitudinal sinus. In the second case vaginal bleeding following scalp sampling was attributed to an ante-partum haemorrhage since there was no free bleeding from the site. There followed a marked fetal tachycardia which eventually gave way to a bradycardia and death. The neonate showed no physical abnormalities but there was evidence of a clotting defect.

Soon after Beard's report the 2-millimetre guarded blade was developed and marketed and the use of Singer's test was advocated in order to differentiate between maternal and fetal blood when vaginal bleeding follows fetal blood sampling.

In 1972 Hull reported a small study (25) in which at least 125 stabs were made in the scalps of three fresh stillbirths; aponeurotic punctures appear to have been made by almost 45 per cent of the stabs which suggests that such punctures are of little significance clinically, assuming, of course, that a similar percentage occur in vivo ! Despite Beard's clear description of a main venous laceration in his first case, Hull questioned the explanation and postulated that blood clotting defects, possibly associated with intrauterine hypoxia, are responsible for the majority of haemorrhagic complications. Balfour reported three haemorrhagic complications in his 678 patients; of these, one presented as a subcapsular haematoma and the infant eventually died. The other two bled postnatally and required sutures. Only one of the three infants showed evidence of a bleeding disorder (26). Hull and Wilson (27) described an instance of subaponeurotic bleeding in utero associated with a coagulation defect. Fortunately delivery occurred in time to prevent exsanguination by treatment with fresh blood and vitamin K1.

Two further cases of fetal haemorrhage associated with sampling in the presence of coagulation abnormalities were reported by Modanlou (28)

Spiral electrode



Clip electrode

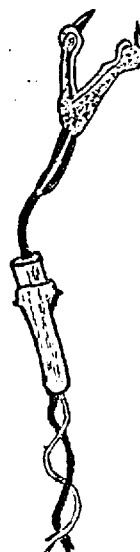


Fig. III:1

Showing the modern spiral electrode and the older type of clip electrode which may be used for hemostasis following fetal blood sampling.

who also reported an instance of a retained, broken FBS blade. This latter complication was also reported by Nelson (29) but in his case the problem was due entirely to technique. McDonald (30) described a simple laceration occurring without fetal blood loss and in the same paper he reported two cases of scalp hematomata attributable to fetal blood sampling.

It is apparent from the foregoing text that, provided there is adequate training of labour ward staff, regular maintenance and testing of equipment and appropriate microbiological investigation of all abscesses, direct complications of intrapartum cardiotocographic techniques are uncommon and of little significance. Fetal blood sampling seems potentially more dangerous but an increased awareness of the role of coagulation disorders should prevent fetal death from exsanguination. The application of a clip electrode (please see Fig. 3.1) to a sampling site often provides increased or complete haemostasis; it is important that a small number of these 'older' electrodes are available in units where the current practice is to use spiral electrodes for fetal heart rate monitoring.

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IV: MISINTERPRETATION AND MISUSE OF INTRAPARTUM

CARDIOTOCOGRAPHY

It behoves us, when reviewing the balance sheet of medical progress, not only to look at the credit side but to cast our eyes critically and honestly down the debit column.

RICHARD ASHER

The complications which were described in the last section are caused mainly by sepsis or by faulty technique. It is not always appreciated that further complications may result if the information obtained by cardiotocography is misinterpreted or misused, either by automated equipment or by labour ward personnel.

Several attempts have been made to automate the interpretation of fetal heart rate patterns (1, 2) but, for reasons which will be discussed in the section on future considerations, such attempts must be viewed with suspicion. Automated interpretation of intra-uterine pressure measurements is less fraught with hazard and it has been used to govern the administration of Syntocinon, in the Cardiff automatic infusion system, for instance (3). Complete safety within such a system depends on a hypersensitive alarm mechanism but this can lead to a high percentage of false alarms and, as the Syntocinon infusion is automatically discontinued each time the alarm is triggered, an apparent lengthening of labour can result (4) - (please see Appendix C).

Non-automated methods of fetal heart rate (FHR) interpretation also present problems because the predictive and diagnostic success of FHR monitoring in labour depends on continuity of both write-out and interpretation. Ironically, the initial interpretation of any FHR pattern is made, often, by a person with limited experience, for instance a pupil midwife, a medical student or an obstetric nurse. Suspicious traces are reported to a houseman whose experience may be equally limited and so, time passes by before someone with experience of FHR monitoring may be summoned to comment upon the observed FHR patterns. This anomaly should gradually disappear as qualified midwives learn

more about the subject but the difficulty lies with knowing what to teach them and being able to define clearly the features which render an FHR trace significantly abnormal.

The classifications of Caldeyro-Barcia, Hammacher and Hon (5,6,7) serve well as theoretical guidelines but a considerable number of modifications are required for practical purposes in a busy labour ward where medical and midwifery staff are in short supply. For instance, is it safe to assume that early decelerations are entirely innocuous and, if they are, is it safe to assume that attendants with varying levels of experience are capable of distinguishing accurately between early and 'so-called' variable decelerations? Which lag time should be measured when a deceleration is preceded by an initial acceleration (hereafter referred to as a pre-acceleration) such as that shown in Fig. IV:1? What is the significance of such pre-accelerations and, similarly, what is the significance of rebound accelerations - Fig. IV:2 and of saltatory baseline variation - Fig. IV:3? These are but a few of the questions which need to be answered in order to improve the understanding of FHR traces.

Many such questions were first raised at teaching sessions which were instituted on a weekly basis on the labour ward at University College Hospital in 1973. The author and a medical physicist were responsible for presenting the basic theoretical classifications at frequent intervals for the benefit of newcomers to the staff. Technical instructions on handling the monitoring machinery were also included and difficult FHR traces were discussed.

The value of these teaching sessions was self-apparent as the number of requests for repairs fell dramatically and the number of false alarms attributable to misinterpretation appeared to lessen. However, communication was not always adequate and not all errors were prevented. Fig. IV:4 shows the outline of a trace achieved using external tocography and Doppler on a Sonicaid FM₂ monitor. The attendant staff midwife had been made aware of the possibility of losing a Doppler signal during contractions and the FHR pattern seen in the figure was attributed to that; in fact, these were genuine decelerations of a sinister nature which were present for two hours before discovery by a clinician. Obviously the importance

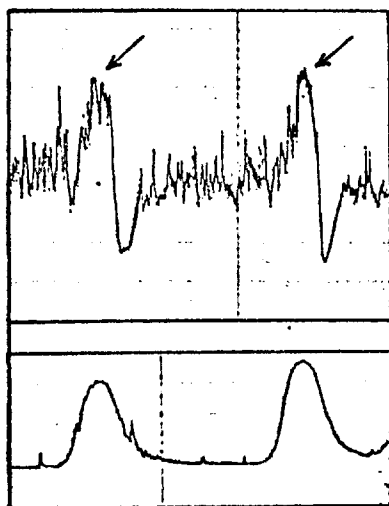


Fig. IV:1
Showing pre-accelerations

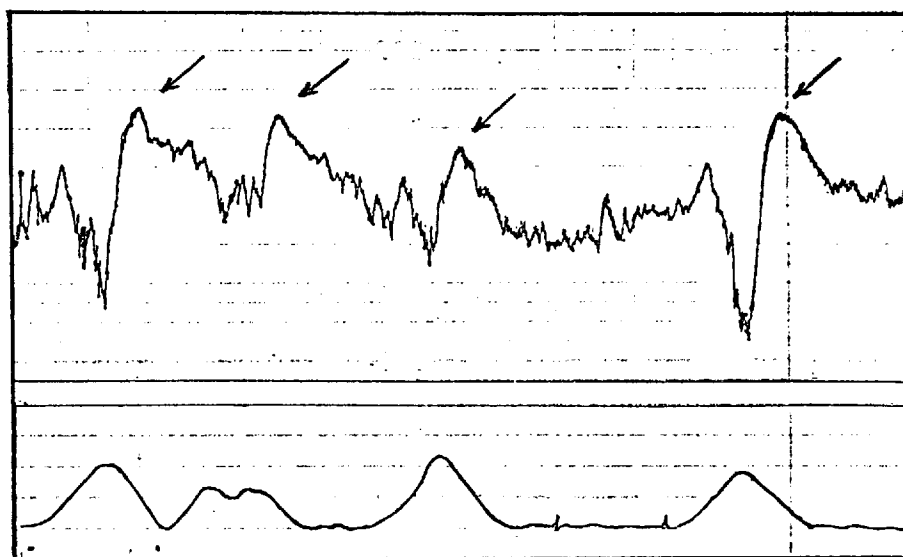


Fig. IV:2
*Showing rebound
accelerations*

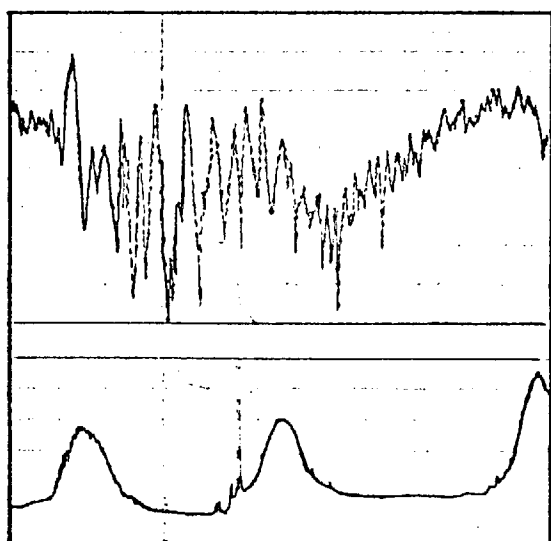


Fig. IV:3
*Showing saltatory
baseline fluctuation*

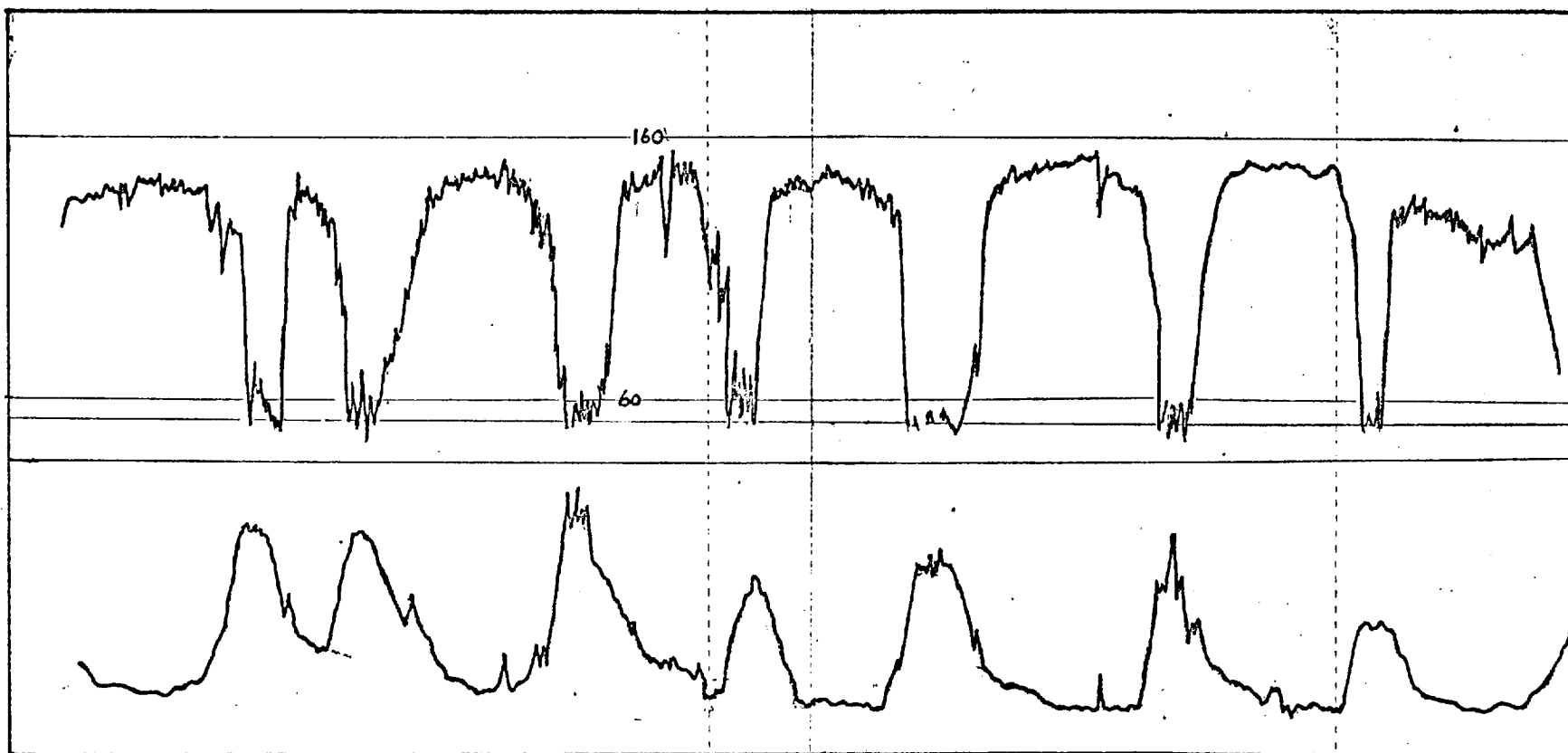


Fig. IV:4

Showing an FHR trace, obtained by Doppler, in which decelerations were mistakenly attributed to loss of signal during contractions.

of checking clinically both the contraction strength and the auscultated fetal heart rate had not been stressed sufficiently.

This was the most serious example of a misinterpreted cardiographic trace seen at University College Hospital, London during the three years of intensive study of intrapartum FHR monitoring carried out by the author. Fortunately the neonatal outcome was perfectly satisfactory following a Caesarean section and prompt intubation but the error could have given rise to serious consequences. Its occurrence led the author to carry out an audit of FHR monitoring usage in order to highlight possible errors and to ensure that the necessary information was being provided by the weekly teaching sessions. The audit was carried out retrospectively over a 12-month period between June 1973 and June 1974, without the knowledge of the labour ward staff. The unit policy at that time was to monitor all 'high risk' pregnancies based on Beard's classification and listed in Table IV:1 (8); these included fetuses defined as being 'at risk' either by antenatal assessment or from intrapartum signs. Induction and epidural rates were 40 and 22 per cent respectively. The labour ward was managed on a 1:5 rota either by a resident Senior Registrar or by a resident Registrar with at least one year of obstetric experience at that grade. Five consultants were responsible for the labour ward on a 1:5 rota. Paediatric Senior House Officers were present at all abnormal or 'at risk' deliveries and all neonates with Apgar scores of less than 4 at one minute were intubated. The majority of neonates with Apgar scores of 4 and 5 were also intubated early.

The critical assessment of an FHR monitoring programme is made difficult by the divergence of opinion amongst experts in the field and by the lack of measurable variables in the end-product - the neonate. At first glance it would seem that the perinatal results in all monitored cases should be compared with the results in all unmonitored cases but such a comparison is rendered almost meaningless by the fact that the monitored cases, by definition, were classed as 'high risk' whilst the majority of unmonitored fetuses were classed as normal. Instead it was decided to review the histories of all intrapartum stillbirths and first week neonatal deaths , together with

TABLE IV:1 - INDICATIONS FOR INTRAPARTUM FETAL HEART RATE MONITORING

<u>ANTEPARTUM</u> :	Primigravida aged 30 or more
	Multigravida aged 40 or more
	Pre-eclampsia
	Hypertension
	Diabetes
	Rhesus iso-immunization
	History of ante-partum haemorrhage during current pregnancy
	Intra-uterine growth retardation
	Poor obstetric history
	History of supine hypotension
	Cardiac disease
	Renal disease
	Anaemia
	Breech presentation
	Twins
	Oxytocics
	Epidural anaesthesia
	Trial of labour
	Trial of scar
	Hydramnios
	Uncertain dates and postmaturity (>42 weeks)
<u>INTRAPARTUM</u> :	Meconium
	Fetal bradycardia
	Fetal tachycardia
	Fetal heart rate irregularity
	Maternal ketosis
	Prematurity
	Prolonged labour (more than 18 hours in 1973-1974)
	Unbooked cases

the histories of all neonates demonstrating signs of perinatal morbidity, in an attempt to determine what role, if any, intrapartum FHR monitoring had played in each case and to assess whether better use of the monitoring facilities might have lessened the chances of mortality or morbidity.

Unfortunately perinatal morbidity cannot be adequately defined. Several criteria were used to determine whether or not morbidity had occurred, namely

- a) Neonates with Apgar scores of 6 or less at one minute and/or 7 or less at five minutes.
- b) Neonates with recorded base excesses of more than -10 on arrival in the special care baby unit.
- c) Neonates with miscellaneous signs of morbidity as listed in Table IV:8.
- d) Fetuses with scalp sample pHs of less than 7.25.

This last group will be discussed further in the next section.

The majority of neonates classed as demonstrating morbidity were said to do so because of a low Apgar score. The scoring of a neonate at one minute as described by Apgar (9) is notoriously inaccurate but there is no doubt that it gives a rough guide to the state of the newborn infant and, in the absence of any known better method of assessment, it was in routine use at University College Hospital during the audited year. Scores of 7 to 10 may be considered to represent healthy neonates whilst scores of 0 to 3 imply a pathological delay in neonatal oxygenation. Scores of 4-6 constitute a grey area which usually represents a degree of morbidity even if it is entirely due to maternal sedation. A modified system of scoring has recently been advanced (10) from which the colour of the neonate has been excluded in order to dispense with the problem of weighting. However, it is the opinion of this author that the accuracy of the score would remain unaltered as the present inaccuracy is due as much to the timing of assessment and observer variation as it is to the lack of weighting of the various clinical signs.

Indeed there is an argument for continuing to include assessment of trunk colour but to design the score to differentiate between pink and blue and white since the latter two colours differentiate between primary and secondary or terminal apnoea.

The maternal notes of the aforementioned neonates were analysed and the required information transferred to a punch card. The accumulated data were then used to determine the following information :

- a) The apparent causes of perinatal mortality
- b) The apparent causes of perinatal morbidity
- c) The relationship between perinatal morbidity and mortality
- d) The relationship between FHR monitoring usage and perinatal morbidity and mortality, i.e.:
 - i) how many of the affected neonates should have been monitored but were not
 - ii) how many affected neonates *might* have been prevented by improved monitoring usage and/or techniques.
- e) Direct complications of FHR/FBS* monitoring - see Section III

The management of each labour resulting in perinatal mortality or morbidity was assessed first by the author and then, in cases of doubt, by a Senior Registrar who was not actively involved in the labour ward routine during the period under review. The author was involved in the labour ward purely on a research basis during this time. Monitoring usage was assessed using the following guide-lines:

- A) The list of 'at risk' patients to determine who should be monitored.

*fetal blood sampling

- B) A combined FHR classification described by Huntingford and Pendleton (11) as a guide to interpretation.
- C) The use of fetal blood sampling based on Saling's description of the technique namely :
- | | | |
|-----------------------|------------|--------------------------------|
| less than 7.20 | : checked | : immediate delivery |
| less than 7.25, >7.20 | : falling | : immediate delivery |
| less than 7.25, >7.20 | : constant | : |
| | : <5 cms | : immediate delivery |
| | : >5 cms | : action dependent on progress |
| more than 7.25, | : falling | : action dependent on progress |
| more than 7.25, | : constant | : no action. |
- D) All Caesarean sections performed for supposed fetal distress without an FBS were scrutinised.

THE FINDINGS

The decision to monitor

The general statistics pertaining to the audited year are presented in Table IV:2. There were 2,072 babies born to 2,041 mothers, the perinatal mortality rate was 16.9 per thousand and the estimated perinatal morbidity rate was 185 per thousand. Intrapartum monitoring took place in almost 25 per cent of patients.

There were 18 stillbirths as shown in Table IV:3 but, of these, fourteen were ante-partum deaths. Of the 4 intrapartum deaths, three suffered congenital abnormalities which were incompatible with life and one was brought in dead having been born elsewhere with no signs of life.

The neonatal deaths, of which there were 17, are listed in Table IV:4. Five of these were born with normal Apgar scores, one dying of

TABLE IV:2 - GENERAL STATISTICS

June 1973 - June 1974 - U.C.H.

Number of neonates delivered	2072	
Number of mothers	2041	Twins: 29 Triplets: 1
<u>Mortality</u>		
Number of stillbirths	18	
Number of first week neonatal deaths	17	
PERINATAL MORTALITY RATE	16.9	per thousand
<u>MORBIDITY</u>		
Number of low Apgar scores	332	Monitored: 129 Unmonitored: 203
Number of low fetal pHs	41	
Number of neonates with base Xs of >-10	11	10 low Apgar
Number of neonates showing miscellaneous signs of morbidity	14	4 low Apgar
Total no. of fetuses showing signs of perinatal morbidity not included in perinatal mortality	384	
PERINATAL MORBIDITY RATE	185	per thousand
<u>INTRAPARTUM MONITORING</u>		
Fetal heart rate alone	278	
Fetal blood sampling alone	14	
Combined FHR and FBS	213	
Total number of fetuses monitored	505	
MONITORING RATE	24.5	per cent

TABLE IV:3 - STILLBIRTHS

June 1973 - June 1974 U.C.H.

	No.	Cause	No.	Detail
'Intrapartum' deaths	4	Congenital abnormality	3	Gross hydrocephaly Anencephaly. Down's syndrome with severe C.H.D.
		Brought in dead	1	Born elsewhere - no spontaneous respiration
Intrauterine deaths	14	Placental insufficiency	4	1 set of twins 1 long cord with 17 twists
		Antepartum haemorrhage	2	
		Severe Rhesus disease	2	
		Congenital malformation	1	Potters syndrome
		False labour	1	Nuchal cord
		Unknown	4	1 nuchal cord

TABLE IV:4 - NEONATAL DEATHS

June 1973 - June 1974 U.C.H.

Cause	No.	Detail	Low Apgar	Monitored
Congenital	7	C.H.D.	✓	-
		Potter's Syndrome 30 weeks	✓	-
		C.H.D. Small for dates	✓	-
		Downs' syndrome. Hypoplastic lungs	✓	-
		Multiple abnormalities. 36 weeks	✓	-
		C.H.D. Duodenal atresia	✓	-
		Operative death: pulmonary atresia	no	no indication
Prematurity	4	Breech 26 - 28 weeks	✓	-
		Breech ? weeks	✓	-
		Breech 33 weeks	no	no 2nd twin
		Amnionitis in labour 27 - 28 weeks	✓	-
Hydrops fetalis	2	Rhesus - also small for dates	✓	-
		Rhesus	✓	-
Miscellaneous	3	Necrotising enterocolitis	no	no indication
		Small for dates	no	SFD not detected no other indication
		Undiagnosed second twin	✓	no
Unknown	1	Elective caesarean section	no	no

an unknown cause after an elective Caesarean section, another dying following surgery for pulmonary atresia and a third dying from necrotising enterocolitis despite being perfectly healthy at birth. The other two were a premature second twin and an undiagnosed small for dates infant respectively, neither of whom were monitored in labour, the first because it was not the unit policy at the time to monitor the second twin and the second because the condition was not diagnosed until delivery. Twelve of the neonatal deaths occurred in neonates who were compromised at birth and the managements of their respective labours have been assessed along with all other low Apgar labours. Six of the neonates had congenital abnormalities, three were premature, two were affected by rhesus iso-immunisation and one was an undiagnosed second twin.

There were 332 neonates with low Apgar scores, approximately 16 per cent of the total number of deliveries. Table IV:5 shows the more common obstetric situations associated with low Apgar scores and compares the number monitored with the number in whom such monitoring did not take place. The figures of note are boxed. Approximately one half of neonates born at elective Caesarean section were compromised sufficiently to give them low Apgar scores. It is probable that this high incidence was related to the lack of wedging of the mothers during the immediate pre-operative period and during the general anaesthetic. At that time the importance of relative supine hypotension was appreciated far less than it is now. The table shows quite clearly that of the fifteen low-Apgar assisted breeches, twelve were unmonitored- according to the criteria for monitoring used at the time six only should have been monitored as two were undiagnosed and four others were second twins. FHR monitoring was not carried out in two cases because of extreme prematurity. This, of course, was illogical as a decision to preserve an extremely premature infant of 26-28 weeks should be accompanied by every effort to deliver it in an optimal state. The figures suggest, also, that an attempt should be made to monitor second twins.

There were 49 cases in which labour was induced but in whom there were no other factors such as malpresentation, multiple pregnancy, assisted delivery or general anaesthesia which might have been of relevance.

TABLE IV:5 - FACTORS ASSOCIATED WITH LOW APGAR SCORES

June 1973 - June 1974 U.C.H.

FACTOR	No.	No. Moni- tored	No. Unmoni- tored	Detail
ELECTIVE CAESAREAN SECTION	45			
NON-ELECTIVE CAESAREAN SECTION	78	56	22	) no wedging
ROTATIONS WITH G.A.	11	7	4	
TOTAL NON-ELECTIVE G.A.	91	63	28	
VACUUM EXTRACTION	8	6	2	
ASSISTED BREECH DELIVERIES	15	3	12	4 second twins : 1 elderly primip. 2 undiagnosed : 3 no reason given 2 extreme prematurity
TWIN DELIVERIES	6	2	4	all second twins
SECOND STAGE > 1 Hr.				
a) Total	29	8	21	
b) Only factor	19	4	15	
INDUCED LABOURS	49	22	27	including: 11 - raised B.P. 1 - I.U.G.R. 2 - Spurious labour 4 - no reason
BIRTH WEIGHT < 2500G				
a) Total	40	15	25	12 breeches or twins
b) Only factor	10	5	5	
INTRAPARTUM INDICATIONS (F.H./meconium) IGNORED	) 23	-	18	
FAILURE TO ACT QUICKLY ON FETAL ACIDOSIS		5	-	

Twenty-seven of these patients laboured without FHR monitoring despite the fact that any indication for induction is an indication for monitoring and that the use of an oxytocic, which was routine following artificial rupture of the membranes, is also an indication for FHR monitoring. Sixty-two per cent of low birth weight, low-Apgar neonates were unmonitored but half of these were breeches or twins which have already been discussed.

There were 23 cases of low Apgar in which either intrapartum indications for FHR monitoring were ignored or there was a failure to act on a diagnosis of fetal acidosis. This figure constitutes just over one per cent of the total number of deliveries.

Table IV:6 shows the primary indications for monitoring which were present in the mothers of the unmonitored low Apgar neonates. There appear to be no significant differences in the incidences of of the indications in the very low and middle grade Apgar groups although there were a disproportionately high number of breeches and premature fetuses in the very low Apgar group.

Apparent birth asphyxia, made manifest by a base excess of more than minus 10, occurred in eleven neonates as shown in Table IV:7. Ten of these had low Apgar scores and have, therefore, been included in the low Apgar statistics. The only neonate with a good Apgar score was diagnosed as having suffered intra-uterine growth retardation and died within the first week of life. The two other neonatal deaths in this group occurred in unmonitored premature breeches. There were no other cases in this group where FHR monitoring might have improved the outcome.

Miscellaneous signs of neonatal morbidity occurred in 14 babies. Only four of them had low Apgar scores and there were no neonatal deaths in the group. Table IV:8 shows that in three of them there were indications for FHR monitoring which were unheeded, one a case of maternal diabetes, one a case of intrapartum hypoxia and one a premature infant.

TABLE IV:6 - SHOWING THE INDICATIONS FOR FHR MONITORING WHICH WERE PRESENT IN THE UNMONITORED LOW APGAR CASES

June 1973 - June 1974 U.C.H.

	Very low Apgar (0-3 incl.)	Middle Grade Apgar (4,5,6)
<u>TOTAL NO. OF FETUSES</u>	51	152
-ELECTIVE CAESAREAN SECTION	10	35
-LABOURING	41	117
-NO INDICATION FOR MONI- TORING	5	39
-INDICATIONS FOR MONITORING	36	78
<u>-PRIMARY INDICATION</u>		
Raised B.P.	7	17
Maternal age	2	7
Antepartum Haemorrhage	2	5
Intrauterine growth retar- dation	1	1
Poor obstetric history	1	0
Supine Hypotension	0	2
Anaemia	1	0
Breech presentation	5	6
Twins (7 pairs)	3	11
Epidural anaesthesia	0	2
Trial of labour/scar	1	6
Hydramnios	1	0
Uncertain dates	1	2
Postmaturity	0	4
Meconium	2	6
Abnormal fetal heart	4	5
Prematurity	4	2
Prolonged labour	1	0
Unbooked cases	0	1
Debatable	0	1

TABLE IV:7 - APPARENT BIRTH ASPHYXIA

(Base excess greater than -10)

June 1973 - June 1974 U.C.H.

CAUSE	No.	DETAIL	Low Apgar	NND	Moni-tored
Difficult delivery - malpresentation	3	1. Cord prolapse L.S.C.S	1.5	-	no
		1. face presentation	3.7	-	✓
		1. S.V.D.	6.9	-	no
Intra-uterine growth retardation	1	- 2296 Grams at term	9.10	✓	no
Second twin	1		Flat	-	no
Prematurity	2	1 33-week breech	4.6	✓	no
		1 26-28 week breech	3.6	✓	no
Meconium aspiration and intra-uterine hypoxia	2	1 Failed ventouse L.S.C.S.	5.8	-	✓
		1 L.S.C.S.	2.5	-	✓
Immediate respiratory problems	1	Pneumothorax	Low	-	no
Unknown	1	Unspecified decelerations - probably intrauterine hypoxia	2.7	-	✓

TABLE IV:8 - MISCELLANEOUS SIGNS OF NEONATAL MORBIDITY

June 1973 - June 1974 U.C.H.

Sign	No.	Further Details Suspected Causes	Moni- toring Indica- ted	Moni- tored	Apgar	NND
CONVULSIONS	6	3 - unknown	no	no	all normal	-
		1 - ? mode of delivery Kjellands	yes	FBS	10.10	-
		1 - ? birth trauma S.V.D.	no	no	6.9	-
		1 - intrapartum hypoxia	yes	no	5.10	-
CEREBRAL IRRITABILITY	2	1 - unknown. ELSCS -placenta praevia	no	no	3.8	-
		1 - intrapartum hypoxia + vacuum extrac- tion	yes	yes	2.7	-
HYPERREFLEXIA	1	1 - Calcium disorder	no	no	normal	-
HYPOTONIA	1	-	no	no	normal	-
SLOW FEEDING	2	1 - Unknown	yes	yes	8.10	-
		1 - Maternal diabetes	yes	no	normal	-
BIRTH TRAUMA	1	1 - fractured parietal bone - Kjellands	no	no	10.10	-
TUMOUR	1	Prem. S.V.D. Abdominal tumour	yes	no	normal	in- fant death

The question of interpretation

By studying the cardiotocographic records in conjunction with maternal notes and partograms, together with the punch card data, it was possible to assess the accuracy of FHR interpretation in the 129 monitored, low-Apgar neonates. From Table IV:9 it can be seen that in 38 cases (29.5 per cent) FHR monitoring was carried out and interpreted absolutely correctly according to the criteria already laid down. In 35 cases (27 per cent) there were some imperfections such as faulty technique; no tocographic record, for instance, or some features of the FHR being missed. Nineteen FHR traces (15 per cent) were interpreted quite wrongly in places, one third of them by doctors, two thirds by midwives, whilst interpretation was totally incorrect in 13 cases (10 per cent); again, doctors were responsible in one third of cases and qualified midwives in two thirds. The FHR trace or the notes were missing or incomplete in 23 instances (18 per cent).

It would be impossible to prove that these misinterpretations were in any way responsible for the low Apgar scores and neither is it necessary to do so. The point is that there were occurring, in as many as 50-70 per cent of monitored cases, misuses of equipment and misinterpretations of traces which could well endanger the fetus. The types of misinterpretation which took place are shown in Table IV:10. The fact that they continued to occur despite regular teaching sessions implied that our teaching methods needed to be improved or simplified or both.

The use of fetal blood sampling.

During the year fetal blood sampling was carried out successfully in 227 patients; in 14 of these it was the only method of intrapartum fetal monitoring. In 41 samples the pH was less than 7.25 thus indicating fetal acidosis and suggesting the presence of prolonged intra-uterine hypoxia. As Table IV:5 shows there were five instances where insufficient attention was paid to the presence of acidosis and the neonates were born with low Apgar scores.

TABLE IV:9 - THE USE OF CARDIOTOCOGRAPHIC MONITORING IN
LOW APGAR FETUSES

June 1973 - June 1974 U.C.H.

Assessment of Usage	No.	Approx. Percentage of total number	Responsibility
Perfectly correct	38	29	Doctors : 6 Midwives : 13
Imperfect (some features not noted) (Faulty technique, etc.)	35	27	
Incorrect (Some features wrongly- interpreted)	19	15	
Totally Incorrect	13	10	
No FHR Trace/Incomplete Notes	23	18	
Total No.	128		

TABLE IV:10 - COMPARING WRITTEN OBSERVATIONS MADE IN LABOUR WITH THE TRUE CARDIOGRAPHIC APPEARANCES IN THIRTEEN
WRONGLY INTERPRETED TRACES June 1973 - June 1974 U.C.H.

OBSERVATION	TRUE APPEARANCE AT TIME OF OBSERVATION
Early decelerations (2 hours duration)	Variable decelerations, tachycardia, loss of baseline variation.
Tachycardia and irregularity	Loss of baseline variation, profound bradycardia.
Occasional type I	Loss of baseline variation, late and variable decelerations.
Tachycardia	Profound deceleration followed by late decelerations and marked tachycardia.
No comment - assumed normal*	Loss of baseline variation, profound deceleration, tachycardia and unclassified decelerations.
Tachycardia	Transient accelerations. Profound deceleration after lithotomy.
No comment - assumed normal*	Bradycardia becoming a marked bradycardia.
No comment - assumed normal*	Late decelerations over 50 minutes.
Dips with contractions	Variable decelerations.
No comment - assumed normal*	Loss of baseline variation and bradycardia.
Tachycardia	Tachycardia but superimposed on loss of baseline variation and late decelerations.
Normal	Early decelerations with preceding accelerations and rebound accelerations.
Late decelerations and silence	Loss of baseline variation and very unimpressive decelerations.

*The only comment in each of these cases was a normal rate and regularity

The correlation between the low pHs and Apgar scores was poor with only 24 of the 41 acidotic fetuses showing low Apgar scores. Of these, two were breeches which, according to Elliott and Hill (12), can be expected to have a pH of 0.05 less than cephalic presentations in similar circumstances. The corrected incidence of low Apgar score amongst the low-pH neonates was, therefore, 22 out of 39 (56.4 per cent). The question of pH/Apgar correlation is one which is often raised during labour ward practice when obstetricians are accused of having expedited delivery unnecessarily because a low-pH fetus has become a high-Apgar neonate. However, the aim of intrapartum FHR monitoring is to detect hypoxia and acidosis before they become so severe as to jeopardise the respiratory efforts of the neonate. Delivery was expedited in all but five of the low-pH fetuses and a general anaesthetic was required in fifteen of the cases. As mentioned above, wedging was not used and this could well account for the high incidence of low Apgar neonates. Five of the low-pH fetuses were delivered by vacuum extraction because of incomplete cervical dilation; the advisability of this method of delivery in the presence of established fetal acidosis is open to question. The clinical impression of the author is that fetal distress is aggravated by vacuum extraction especially in primigravid patients. If this is the case then there were only two neonates in whom low Apgar scores were explicable purely in terms of low pHs. Against the possibility of the low Apgar scores being related to anaesthesia and vacuum extraction however is the fact that the two factors were observed with similar incidences in the low-pH/high Apgar group as shown in Table IV:11. Neither does the Apgar score seem to depend on the degree of fetal acidosis as there was one very low pH (<7.18) in the high Apgar group compared with only two in the low Apgar group.

It is possible that the immediate delivery of a low-pH fetus, with no intervening anaesthetic, supine position or vacuum extraction, would result in a high Apgar neonate and that low Apgars occur because such insults as these are levelled at fetuses with reduced oxygen reserves over variable lengths of time.

The diagnosis of fetal distress

Fetal distress warranting intervention by Caesarean section was

TABLE IV:11 - DISTRIBUTION OF APGAR SCORES IN 39 LOW pH FETUSES

June 1973 - June 1974 U.C.H.

DELIVERY FACTORS	Low Apgar Low pH	High Apgar Low pH
G.A.	15	11
VACUUM EXTRACTION	5	3
UNCOMPLICATED FORCEPS	2	3
	22	17

diagnosed without a fetal blood sample in 29 patients. In eleven there were sufficient grounds for early intervention in the presence of fetal heart rate irregularities either because of a risk factor or slow progress. In thirteen the presenting part was inaccessible at the time of the fetal heart rate irregularity and, in the majority of these, meconium was also present. In the remaining five there were no stated reasons for the omission of an FBS and, although in each case there were grounds for concern, procrastination may have been of benefit in three of them.

In summary a twelve-month period of monitoring usage has been reviewed. Although there were no preventable intrapartum stillbirths, a survey of defined perinatal morbidity suggests that at the time of the review there was insufficient attention paid to selection of patients for FHR monitoring. This was particularly noticeable in respect to prematurity, breech presentations and second twins. In addition, intrapartum signs of fetal distress were ignored too frequently. Interpretative problems existed in as many as 50-70 per cent of cases and this was taken to imply that teaching methods required considerable improvement and/or simplification. Two-thirds of the interpretative problems were attributable to trained midwifery staff and one third to medical staff. The use of fetal blood sampling was correct in all but a small minority of cases. A high incidence of low Apgar scores in neonates born by elective Caesarean section has been attributed to the lack of routine patient wedging at the time of the review. The relationship between intrapartum acidosis and Apgar score has been discussed especially bearing this factor in mind.

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V: 'FETAL DISTRESS' (FETAL ACIDOSIS)

including an examination of the fetal heart rate traces in 41 acidotic or pre-acidotic fetuses.

DISTRESS : noun : extreme pain : that which causes suffering : calamity : misfortune from Latin, dis-tringere : to pull asunder.

CHAMBERS TWENTIETH CENTURY DICTIONARY
OF ENGLISH LANGUAGE

The term 'fetal distress' has been used loosely for many decades to describe the physical signs which suggest impending danger to the fetus (1). It is a poorly defined term which has been used synonymously with fetal hypoxia, fetal anoxia, fetal asphyxia and fetal acidosis.

The criteria for the diagnosis of so-called 'fetal distress' have been attributed to von Winckel (2) who described early and late slowing of the fetal heart rate, fetal tachycardia and fetal heart rate irregularity as signs suggestive of fetal hypoxia; these signs he attributed to a raised level of carbon dioxide in the fetal blood. Von Winckel also discussed the relevance of meconium, stating that its presence did not spell immediate danger to the fetus although it did imply a raised level of fetal CO₂ causing peristalsis through vagal stimulation.

Von Winckel was unable to agree with other authors who believed that serious danger to the fetus was preceded by increased fetal movements, although his explanation of such a phenomenon, if it did occur, was extremely enlightened. He suggested that the rise in fetal CO₂ levels above a certain trigger point would cause stimulation of fetal respiratory movements via the respiratory centre.

When the subject of fetal distress was reviewed in 1959 by Walker (please see ref. 1) it was obvious that unnecessary obstetric interference could only be avoided by studying the physiological status of a 'distressed' fetus and that the clinical signs constituting the syndrome of 'fetal distress' were too varied to be used with any great accuracy. The same situation prevails today, as the rest of this discourse will demonstrate. Currently the physiological status of the

'distressed' fetus is investigated by sampling fetal scalp blood and determining the pH. Further management then depends on the pH level because unlike 'fetal distress', fetal acidosis is a strictly defined condition which is said to be present when the pH of fetal scalp blood lies below 7.20 (3). It may be caused by fetal hypoxia or it may be secondary to maternal acidosis. A fetus is described as pre-acidotic if its scalp blood pH lies at or above 7.20 but below 7.25.

During the twelve months between June 1973 and June 1974 forty-one acidotic or pre-acidotic fetuses were discovered at University College Hospital, London - an incidence of slightly more than 2 per cent. In order to gain more information concerning FHR patterns which signify probable acidosis, each of the FHR traces preceding a relevant fetal blood sample was examined in detail and classified according to accepted principles described by Huntingford and Pendleton (4) and based on classifications attributable to Hon and Quilligan, Caldeyro-Barcia and colleagues and Hammacher (5, 6 7).

In these classifications the normal fetal heart rate is defined as lying between 120 and 160 beats per minute (B.P.M.), a range which is universally accepted, although, in the past, some writers have questioned it (8). At least one present day researcher (9) has presented evidence which suggests that the range should be 110-150 b.p.m.; the author's clinical experience in intrapartum FHR monitoring would certainly support the lower level of this range.

A new baseline rate is said to be present if an alteration in heart rate persists for ten minutes or more. An alteration of less than ten minutes duration, however, is described as a transient change in rate; such alterations may be accelerations or decelerations. Decelerations are classified according to their temporal relationship with the uterine contractions and according to their shape. A deceleration is said to be late if its onset is 'some 20-30 seconds or more after the beginning of a uterine contraction' and provided that the 'slope of the descending limb is falling at about the same rate that the ascending slope of the associated uterine contraction is rising'. Early decelerations fulfil the second criterion but their 'onset coincides with that of the associated uterine contraction.

Decelerations may be described as variable if

- a) the time relationships between the FHR waveform and the associated uterine contractions are inconstant i.e. a mixture of early and late decelerations
 - b) 'the FHR pattern varies in shape from contraction to contraction'
- or
- c) 'the slope of the descending limb of the FHR pattern is falling faster than the ascending slope of the associated uterine contraction'.

The quoted definitions have been taken from Hon and Quilligan's revised working classification (see ref. 5). Although this is the most frequently used classification, close examination shows that several of the definitions are imprecise. First, there are three unrelated ways in which a deceleration may be described as variable and yet variable decelerations are said to reflect cord compression (10). Experience in the analysis of 950 consecutive routine FHR traces at University College Hospital (11) has shown that, in any series of decelerations, both the shape and the timing of onset are often variable features. Are such variations always attributable to cord compression? It is doubtful that Hon meant either to state this or even to imply such a possibility but for teaching purposes alone the term 'variable' requires to be defined more precisely in a way which clearly distinguishes between significant and insignificant degrees of variation. The classic 'cord-compression' deceleration pattern shown in Figure V:1 is a variable deceleration by virtue of its fast falling descending limb but there is no strong evidence to suggest that it bears a strong aetiological relationship with other types of variable deceleration patterns.

The definition of a late deceleration is equally imprecise with the required delay being 'some 20-30 seconds'. For teaching purposes it is necessary to state a precise length of time which distinguishes between early and late decelerations. Caldeyro-Barcia suggested that the critical peak-to-nadir lag time should be 18 seconds; this, at least, was dogmatic but in the clinical situation on a labour ward it

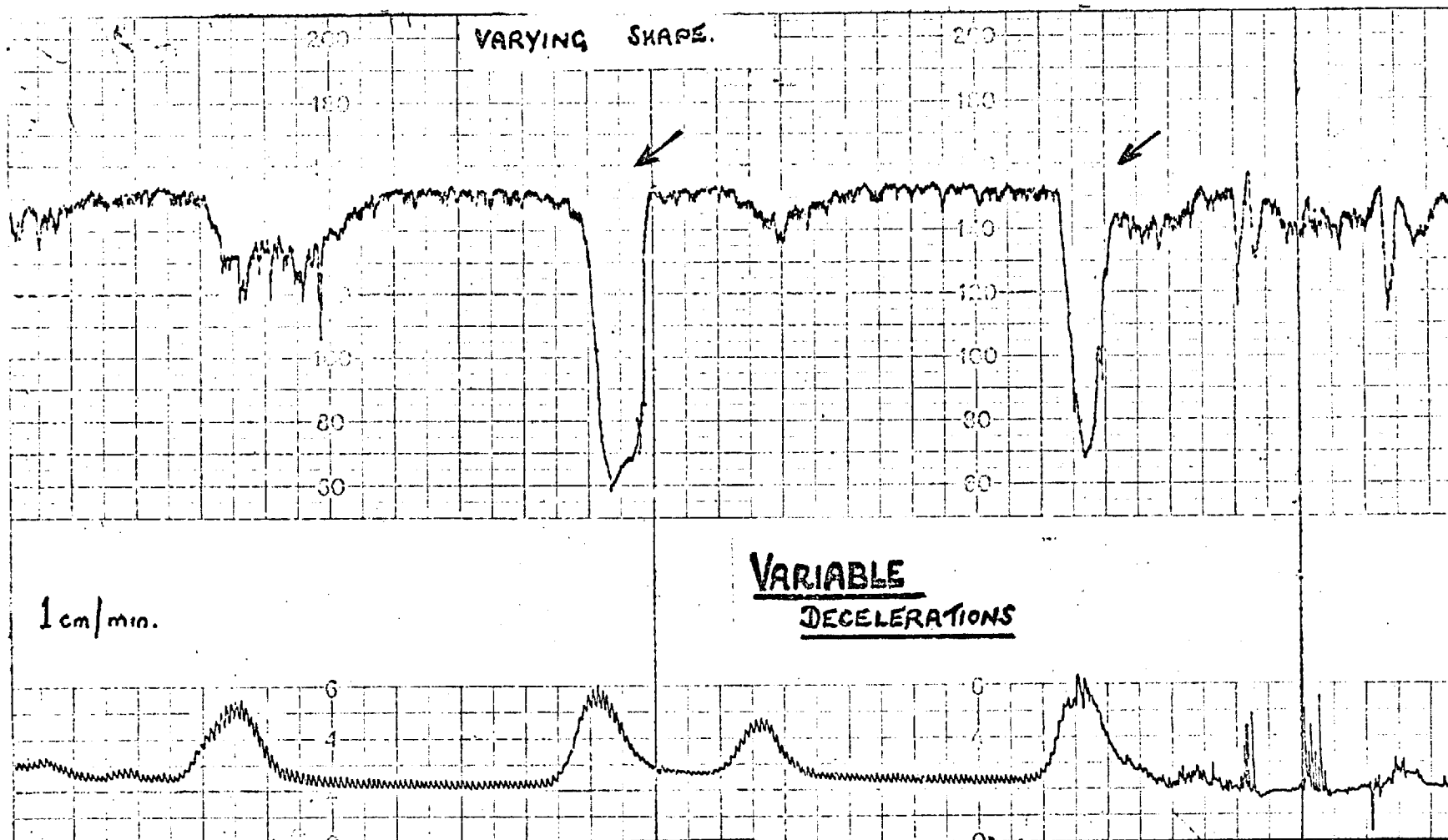


Fig.V:1 Classic 'cord-compression' decelerations

is an unmeasurable time.

The shortcomings of their classification have been recognised by Hon and Quilligan who, with their respective colleagues, have strived to improve and simplify the system (12).

Analysis of the FHR traces preceding the sampling of scalp blood in the aforementioned acidotic and pre-acidotic fetuses at U.C.H. highlighted the shortcomings of Hon's classification.

Two of these forty-one fetuses were discovered as a result of fetal blood sampling indicated only by the presence of meconium. It is not the purpose of this thesis to discuss the significance of meconium in detail but its relationship to the use of fetal heart rate monitoring and fetal blood sampling should be described. Wood and Pinkerton (13) studied the subject carefully at Queen Charlotte's Hospital, London and, although there was no significant difference between the perinatal mortality in the 200 patients with meconium and the perinatal mortality in the 1488 patients with clear liquor, there were many other factors to be considered which might have distorted the figures. The incidence of meconium in anoxic stillbirths was 1:4 compared with 1:7 in all patients delivered at Queen Charlotte's Hospital during the time reviewed. It has been estimated that in ten per cent of cases with meconium its appearance is significant (14) and an increased neonatal mortality rate has been reported in babies born to mothers who have shown meconium during labour (15). Whilst this observation may be related as much to aspiration of meconium by the newborn as it is to intrapartum hypoxia, the need for intensive FHR monitoring and fetal blood sampling in cases where meconium is present has been shown recently by Miller and colleagues (16) and has been suggested by Abramovici who considered meconium to be a sign of compensated fetal distress (17).

In the 1973-1974 U.C.H. Review there were 39 patients whose fetal hearts were being monitored continuously prior to a fetal blood sample being taken. In four of these the traces were too poor for analysis and in a further four there were no tocographic records so that the study of temporal relationships between decelerations and contractions was impossible. In the 31 cases where complete trace analysis was possible, early decelerations were predominant in twelve, late decelera-

tions in three and variable decelerations in thirteen. In three cases there were no decelerations observed. The presence of early decelerations as the predominant pattern in as many as twelve cases was in-keeping with the clinical experience gained through the analysis of 950 routine traces; this experience suggested that deep, symmetrical, early decelerations of 30 to 40 beats per minute depth were of considerable significance in the diagnosis of fetal acidosis. In addition Beard and his colleagues (18) had reported two instances in which early decelerations alone were associated with a fetal pH of less than 7.26, Wood (19) had described a case in which an intrapartum stillbirth had been heralded by deep, early decelerations and Simmons and Lieberman (20) had found that, out of thirteen monitored compromised fetuses (pH <7.20), three showed only early decelerations. O'Gureck and her colleagues (21) went so far as to state that their highest incidence of neonatal depression occurred in conjunction with early deceleration patterns. It seems obvious from these reports and from the findings at U.C.H. that early decelerations are not necessarily innocuous. The impression that early decelerations are of little or no significance has developed over the years since Chung and Hon (22) described the similarity between decelerations caused by pressure on the fetal skull and early decelerations and since Caldeyro-Barcia (23) showed that decelerations with a peak to nadir lag time of more than 18 seconds had much more significance than those with shorter lag times. Of course, early decelerations may form part of a variable pattern either by virtue of a variation in lag time or by virtue of a variation in shape and it must be borne in mind that the depth of an early deceleration may be of considerable importance in deciding whether it should be described as early (because of timing) or variable (because of steep sides). Beard (24) and Wood (25) have suggested critical depths of 40 b.p.m. and 60 b.p.m. respectively. This divergence of opinion leads to further confusion in the art of FHR trace analysis since, in theory, an early deceleration signifies physiological head compression whilst variable decelerations signify cord complications and possible fetal compromise. It seems logical to assume that there is no precise dividing line between the two and that inexplicable early decelerations should always be investigated further by fetal blood sampling.

Even allowing for this area of overlap between early decelerations and steep-sided, deep variable decelerations, there were no obvious,

consistent FHR signs suggestive of fetal acidosis when Hon and Quilligan's or Caldeyro-Barcia's classifications were used.

Assessment of the same traces using Hammacher's classification of baseline variability gave the following results. Nineteen were silent, nine showed narrowed undulatory variability and seven were normal. Saltatory variability was seen for a significant duration in only one trace in which it alternated with episodes of silence. Only in one of the 39 FHR traces was there totally normal baseline variability seen in conjunction with a total absence of decelerations.

It has always been unclear as to why Hammacher chose the level of 5 b.p.m. in order to differentiate silent baseline variability and it has been suggested recently that the level of 10 b.p.m. might be more appropriate at least in antenatal cardiotocography (26). If this were the case then the baseline variability would be labelled as suspicious in 28 out of the 35 analyseable traces (80 per cent). As Table V:1 shows, this compares with 19 out of 35 (54 per cent) if 5 beats per minute were used as the critical level, 16 out of 31 (52 per cent) if late and variable decelerations were used alone to diagnose fetal acidosis and 28 out of 31 (90 per cent) if all decelerations were considered to be significant. If all decelerations and all baseline variability below 10 b.p.m. were considered to be suspicious together with any meconium, then only one of the 41 acidotic or pre-acidotic fetuses would have been missed. This was not the policy at U.C.H. during the study period and many of these affected fetuses were sampled purely because of a poor obstetric history or because of a previous fetal blood sample at the lower end of the normal range.

Whilst the figures in Table V:1 suggest the need for fetal blood sampling in any patient whose FHR trace shows decelerations or baseline variability of less than 10 b.p.m. or in any case where there is meconium, such a policy is barely feasible with current methods of scalp sampling and the low staffing levels encountered in many labour wards.

It is possible that other features of an FHR trace might indicate more readily the provisional diagnosis of fetal acidosis. At first glance at the traces of the 41 acidotic and pre-acidotic fetuses this would not appear to be so but in Section VI there is described a study

TABLE V:1

THE PERCENTAGE INCIDENCE OF CLINICAL AND FHR SIGNS IN THE
41 ACIDOTIC AND PRE-ACIDOTIC FETUSES DETECTED AT U.C.H.
BETWEEN JUNE 1973 AND JUNE 1974.

Clinical sign or FHR Variable	Number of cases*	Percentage incidence
Meconium only (no FHR reading)	2	5
Baseline variability <10 b.p.m./ ²	28/35	80
<5 b.p.m./ ²	19/35	54
Late and variable decelera- tions	16/31**	52
Unspecified decelerations	31/35	89
Baseline variability <10 b.p.m./ ² plus unspecified decelera- tions	34/35	97

*Four traces were not analyseable

**In four traces there was no tocographic recording so that
timing of decelerations was impossible.

which was designed not only to determine the relative importance of the major features of HHR traces but also to examine the importance of lesser features and the dip-area, and to investigate the possibility of measuring baseline variability more accurately in order, perhaps, to improve its diagnostic potential.

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VI: Part 1: THE ANALYSIS OF CONTINUOUS FETAL HEART RATE TRACE
IN THE FIRST AND SECOND STAGES OF LABOUR

Truth is rarely pure, and never simple.

OSCAR WILDE

The foregoing text shows that misinterpretation of FHR traces can be an important problem even in a unit where the teaching is constant and in accordance with the revised working classification of Hon and Quilligan (1) and with Hammacher's classification of baseline variability (2). However, these classifications are not without their shortcomings as described in Section V. It has been shown, both from experience at University College Hospital (U.C.H.) and from the experience of other authors that early decelerations cannot be dismissed as innocuous. Their differentiation from so called variable decelerations can be difficult for persons lacking in experience. Detailed analysis of routine FHR traces shows that there is always a certain degree of variation in the shape of consecutive decelerations and also in the duration of lag time, which is, in physiological terms, an inaccurate measurement when determined from an external tocograph.

Regarding late decelerations, Beard and colleagues (3) found evidence which suggested that late decelerations were indicative of severe fetal asphyxia but they observed that the decelerations were often of small amplitude and accompanied by loss of so-called 'beat-to-beat' variation thus making them difficult to detect except by the experienced eye. It should be stressed that these were relatively early observations in the history of clinical cardiotocography and later experience at U.C.H. suggested that late decelerations were not invariably a sign of fetal compromise. Experimental evidence shows that late decelerations never occur in association with normal fetal oxygenation (4) and they should, therefore, be a reliable variable in the diagnosis of fetal hypoxia. It was reasoned by the author that there might be some detectable difference between those late decelerations associated with hypoxia and those which were not. A large series of FHR traces demonstrating late decelerations is

described in Section VII; it shows without doubt that only 50 per cent of late decelerations are associated with fetal compromise and it indicates that the difference between the observations of Beard and those of the author at U.C.H. may well be attributable, at least in part, to an increasing use of epidural analgesia.

It would appear that cardiotocography, used alone, is not sufficiently accurate to allow the diagnosis of fetal acidosis except, perhaps, in extreme cases although its use as a detector of possible acidosis has been *proved* by a reduction in intrapartum stillbirths (5, 6, 7). The word *proved* is italicised as it has been suggested, quite rightly, that the reduction in stillbirths may be due to an improvement in obstetric care which is secondary to the use of cardiotocography (8).

The diagnostic inaccuracy of cardiotocography is disputed by Shelley and Tipton who devised a method of measuring dip-area, found that the total dip area in the last hour of labour correlated well with neonatal outcome (9) and suggested that it was the only variable required in the diagnosis of fetal acidosis.

Part II of this Section contains a study which was designed to test that hypothesis of Shelley and Tipton. The protocol was extended to facilitate the investigation of the significance of twenty-one other features observed in FHR traces and to allow the assessment of the relative importance of the major features.

In Section VII the diagnostic relevance of late decelerations is discussed and in Section VIII there is described a third original study designed to examine the significance of pattern progression through the first and second stages of labour.

Using the three studies together it is hoped that the following pertinent questions may be answered.

- a) Can intrapartum cardiotocography be used reliably without fetal blood sampling using
 - i) the Hon/Hammacher classifications

- or ii) an adaptation of the Hon/Hammacher Classifications
 - or iii) dip area
 - or iv) any other single variable ?
-
- b) Assuming the necessity to use fetal blood sampling, which single variable or small group of variables could be used to detect all the at-risk fetuses ?
 - c) Is there any prognostic purpose in measuring any of the twenty-one variables listed in addition to total dip area ?
 - d) Is there any prognostic purpose in studying pattern progressions or in noting the first observed fetal heart rate response in early labour ?

The patient population involved in these studies consisted of a broad cross-section of society ranging from the professionals of Highgate and Hampstead through the middle social classes to the cosmopolitan population of Camden and Islington. The population included a relatively large proportion of Greeks and Greek-Cypriots.

The three papers are presented in their entirety as they were published in the British Journal of Obstetrics and Gynaecology. Although each paper contains a discussion, further discussion will be included in Section IX.

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VI Part 2: THE ANALYSIS OF CONTINUOUS FETAL HEART RATE TRACES
IN THE FIRST AND SECOND STAGES OF LABOUR*

SUMMARY

In the search for a simplified method of fetal heart rate trace assessment 50 traces recorded during the first stage of labour and those recorded in the last hour of each of a further 50 labours have been analysed intensively. The importance of baseline variability in predicting possible fetal distress in both the first stage and the last hour of labour is shown and the possible use of an objective measurement of baseline variability in the further screening for fetal acidosis is described. The importance of lag time measurement in the first stage of labour is demonstrated and the relationships between fetal scalp blood pH and other heart rate parameters is discussed. Significant inverse correlations between one-minute Apgar score and maximum amplitude of deceleration and total deceleration area in the last hour of labour are also shown. Temporal relationships between intrauterine pressure rises and transient changes of fetal heart rate failed to give significant correlations in the last hour. These findings in the last hour of labour are compared with those obtained in the first stage of labour and the differences are discussed.

Continuous electronic fetal heart rate monitoring is now a routine labour ward practice. Certain abnormal fetal heart rate patterns were described soon after the introduction of the technique (Hon, 1963; Hon and Quilligan, 1967; Caldeyro-Barcia et al., 1966; Hammacher, 1962) and these have been utilised with little modification since that time.

*Published in the British Journal of Obstetrics and Gynaecology - Aug. 1975. Vol.82. pp.634-642.
 Co-author : R. J. Blackwell, Principal Physicist

TABLE VI:1List of Measured Parameters

Average Basal Heart Rate

Basal Fetal Heart Rate Score

Baseline Variability

Amplitude of Decelerations

- a) Maximum
- b) Mean

Total Deceleration Area

Acceleration Areas*

- a) Total
- b) Initial Acceleration (including Transient Acceleration)
- c) Rebound Acceleration

Ratios

- a) Total Acceleration/Total Deceleration
- b) Initial Acceleration/Total Deceleration
- c) Rebound Acceleration/Total Deceleration
- d) Rebound Acceleration plus Deceleration/Initial Acceleration
- e) Initial Acceleration plus Deceleration/Rebound Acceleration

Lag Times

- a) Onset of Contraction to onset of Deceleration
- b) Onset of Contraction to onset of Transient Change
- c) Peak to Nadir**
- d) End of Contraction to End of Deceleration
- e) Onset of Contraction to Nadir

Slope of Deceleration (Deceleration rate)

Slope of Recovery (Recovery rate)

Recovery Time (Nadir to end of deceleration)*

*First stage analysis only

**Mid-plateau to nadir in second stage

The application of these strictly classified abnormalities to the clinical situation is not without its difficulties and several attempts have been made to simplify the task of interpreting fetal heart rate patterns (Shelley and Tipton, 1971; Goodlin, 1972). Such simplification would enable paramedical staff to interpret traces more easily and might facilitate on-line computerised assessment of fetal heart rate. This study was designed to determine which fetal heart rate parameters bear the most constant relationship to fetal well-being in terms of fetal scalp blood pH and Apgar score.

METHODS

One hundred continuous fetal heart rate records were analysed. Fifty were obtained in the first stage of labour over the course of an hour prior to the taking of a fetal scalp blood sample by an experienced labour ward operator. A further 50 were analysed in the last hour prior to vaginal delivery. The samples were obtained either in the course of routine labour management or for control purposes in another study. Each record was derived from the electro-cardiograph of a singleton fetus using a scalp electrode and a Hewlett Packard monitor (HP 8025A). Contractions were recorded using external tocography and the paper speed used was one centimetre per minute. The traces were analysed retrospectively by one person (G.T.) to whom the pH results and Apgar score were unknown until after completion of the analyses. The parameters which were measured are listed in Table VI:1 and illustrated in Figures VI:1 and VI:2.

Definitions and methods of measurement

Average basal heart rate. Average basal heart rate refers to the rate between contractions and between transient changes in rate. An average rate was obtained from a basal rate assigned to each ten-minute period of recording. If the basal rate in any ten-minute period was not constant then an average for that period was determined by assessing each minute of basal rate.

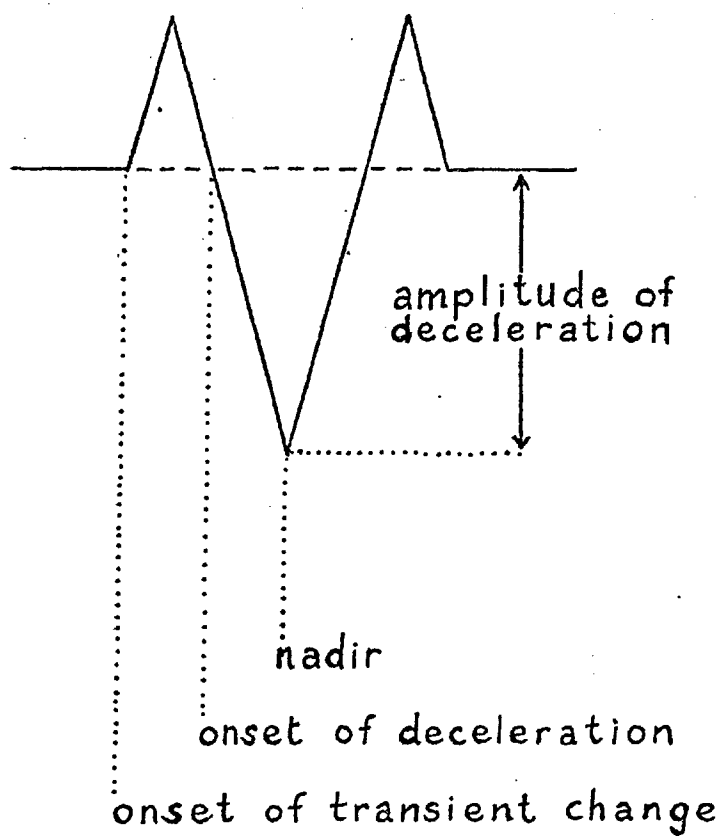
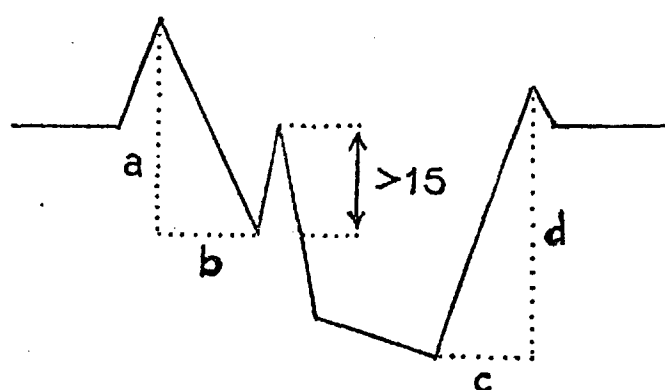


Fig. VI:1

Lag times

slope of deceleration = a/b
 slope of recovery = d/c

Fig.VI:2

Rates of change in fetal heart rate

Basal fetal heart rate score. The fact that both tachycardia and bradycardia may be associated with fetal distress* suggests the possible existence of a subclinical continuum in the relationship between pH and fetal heart rate. This possibility was investigated by devising a score relating the deviation of the heart rate from a central value to the pH. The score was calculated by taking the difference between the average basal heart rate and a standard value (130 beats per minute) and multiplying all negative values by a negative factor (-2.7). The standard value and the factor were derived from a computer trial relating a range of possible values to pH by Spearman's rank correlation co-efficient (Sokal and Rohlf, 1969), the values giving the highest correlation being selected.

Baseline variability. Baseline variability is often referred to as beat-to-beat variation. It may be defined as the range within which the basal heart rate varies in any one minute. Measurements were made using translucent wax paper marked with a grid corresponding to the Hewlett-Packard recording paper markings and the scoring system was based on a modification of Hammacher's classification shown in Table VI:2. A measurement was assigned to each ten-minute period of recording wherever possible but in the event of inconstancy the trace was further subdivided into minutes. Baseline variability during transient changes in rate was not measured except in instances where a new rate was present for more than one minute.

Deceleration size. Deceleration size was assessed in two ways, (i) by determining the difference in beats per minute between the basal rate after the deceleration and the lowest point of that deceleration and (ii) by measuring the actual area of each deceleration using a D-Mac plotting table, a device which records trace co-ordinates in a form suitable for computer input.

Acceleration area. Any acceleration of heart rate exceeding ten beats per minute in size and 20 seconds duration was measured using the D-Mac table. Accelerations occurring immediately prior to a deceleration were termed initial accelerations, those occurring immediately after were termed rebound accelerations. Accelerations which were unrelated to decelerations were labelled transient accele-

*For distress please read acidosis

TABLE VI:2Classification of baseline variability

	Baseline variability in beats per minute	Coarse score in beats per minute
Silent	<5	2.5
Mixed Silent/Undulatory		9.0
Undulatory	5-25	15.0
Mixed undulatory/Saltatory		25.0
Saltatory	>25	35.0

rations. Since only two fetuses demonstrated accelerations in the second stage of labour the measurement of acceleration areas was omitted from that part of the study.

Acceleration/deceleration ratios. As the significance of accelerations is unclear five ratios relating accelerations to decelerations were tested against pH. These ratios are shown in Table VI:1.

Lag times. Five lag times defined in different ways were measured by the non-parallax method using a scale calibrated to 0.5 millimetre or three seconds. In the second stage, when recording limitations caused most contractions to show plateaux, the peak of each contraction was taken as the mid-point of the plateau for purposes of measurement.

Initial rate of deceleration. This was determined from the time taken for the heart rate to fall from basal rate to nadir where nadir was defined as that point below which the rate did not fall without first rising by 15 beats per minute or more.

Recovery time and rate of recovery. The recovery time, from which the rate of recovery was calculated, was defined as the time taken for the heart rate to rise from the true nadir, the lowest point of the deceleration, to the basal rate.

Statistical methods

There is no physiological reason why any of the measured parameters need necessarily be linearly related to fetal condition. Considering the probable threshold effect of hypoxaemia and the unlikely possibility of any parameter size being normally distributed, the trace parameters are likely to be discontinuous and non-linear. These considerations militate against the use of parametric statistics in this study, nevertheless Pearson's coefficient of correlation (r) was calculated in every case. Monotonic trends in the tested parameters were sought by using the non-parametric coefficients of Spearman (R_s) and Kendall (τ) corrected in both cases for ties. The latter was

the preferred test as it facilitated the derivation of partial correlation coefficients and the close approximation of levels of significance by the method described by Siegal (1959).

RESULTS

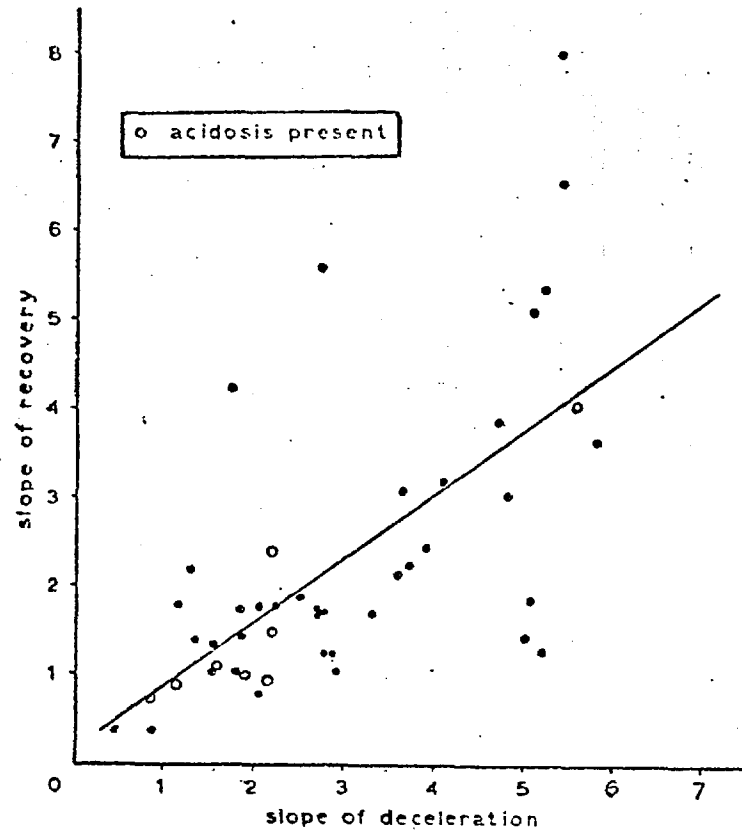
First Stage

No significant linear relationships were demonstrated between pH and any of the measured parameters. The following results refer to ranking correlations.

Lag times. There was a strong negative correlation between the mean lag time (onset of contraction to onset of transient change) and pH ($p = <0.005$ $z = -2.5884$). This correlation existed whether the mean was derived from readings over 60 minutes or over 30 minutes prior to the fetal blood sample. The mean lag times (peak to nadir and onset of contraction to onset of deceleration) also correlated negatively at a significant level with pH ($p = <0.05$; $z = -1.9146$ and -1.8579 respectively). The correlations relating pH to mean lag times (onset of contraction to nadir and end of contraction to end of deceleration) failed to reach significance.

Mean baseline variability. This only just correlated positively with pH at a significant level ($p = <0.05$; $z = 1.6485$) but, in graphical analysis of the results, was shown never to exceed a coarse score of 13 beats per minute per minute (b/m^2) in the presence of a pH value of less than 7.25 (Fig.VI:3). Scores of less than $13b/m^2$ were always associated with at least ten minutes of silent baseline variability. The mean variability was above this value in 26 records and below it in the remaining 24 records including the seven associated with low pH values.

Average fetal heart rate and basal fetal heart rate score. As expected, there was no monotonic relationship between the unprocessed average heart rate and pH. By processing the rate into basal fetal heart score it was possible to obtain a significant negative correla-



tion ($p = <0.05$; $z = -1.9572$).

Shape and size of transient changes of rate. The mean initial rate of deceleration correlated with pH ($p = <0.05$; $z = -1.7339$) as did the time of recovery (nadir to end of deceleration) ($p = <0.05$; $z = -1.9888$). The rate of recovery failed to correlate significantly. Correlations between area measurements and pH were poor except for total rebound acceleration area measured over the last 30 minutes prior to fetal blood sampling which correlated at a significant level ($p = <0.05$; $z = 1.7508$). There appeared to be no relationship between the size or depth of decelerations and the pH nor between the tested acceleration/deceleration ratios and pH.

Interparametric correlations. The mean initial rate of deceleration (slope of deceleration) and the mean rate of recovery showed a significant linear relationship ($p = <0.001$; $r = 0.656$; $n = 46$) (see Fig.VI:4). The baseline variability altered inversely and linearly with average basal heart rate ($p = <0.02$; $r = 0.348$; $n = 50$). Kendal's partial correlation coefficient showed an increase in significance in the correlations between baseline variability and pH when the effect of basal heart rate was partialled out.

Second Stage

Only one of the 50 newborn infants scored less than 8 on Apgar assessment at five minutes so that meaningful correlation between heart rate parameters and that score was impossible. Nine of the newborn scored less than 6 on the one-minute Apgar score. Of the twelve measured parameters, three proved to correlate at a significant level with one minute Apgar scores as shown below. There was no correlation between method of delivery and Apgar score ($\chi^2 = 0.015$ with one degree of freedom $p = <0.9$). All deliveries were vaginal.

Baseline variability. There was a direct linear relationship between this parameter and one-minute Apgar score ($p = <0.001$; $r = 0.445$; $n = 48$). As shown in Fig.VI:5 all the newborn infants scoring less than 6 at one minute demonstrated low baseline variability (less than 13 beats per minute per minute (b/m^2) on the coarse averaging

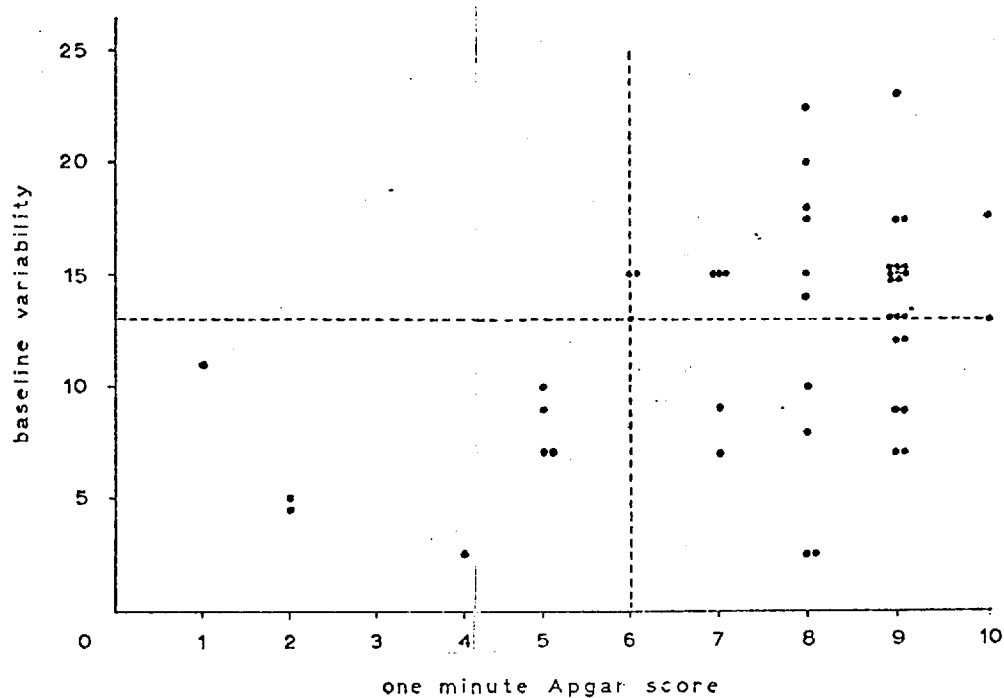


Fig. VI:5

Scatterograph of baseline variability scores in the last hour of each of fifty labours and corresponding one-minute Apgar scores.

scoring system) in the hour prior to delivery.

Deceleration area. There was an inverse linear correlation between total area of deceleration and the one-minute Apgar score ($p = <0.02$; $r = 0.343$; $n = 50$).

Maximum amplitude of deceleration. The inverse linear relationship between this parameter and the one-minute Apgar score failed to reach statistical significance ($p = <0.1$) but there was a significant ranking correlation ($p = <0.001$; $z = -2.3690$) suggesting that there was an inverse monotonic non-linear relationship between the greatest amplitude of deceleration and the Apgar score.

No correlation was shown between Apgar and lag-times, mean depth of decelerations or basal heart rate. Accelerations were seen in only two cases in this second stage study; in both cases the areas were very small and appeared to have no bearing on the outcome.

DISCUSSION

It is generally accepted that the pH of fetal scalp blood is indicative of fetal condition (Beard et al., 1967) and that an abnormal fetal heart rate pattern is an indication for carrying out fetal blood sampling (FBS). Difficulties arise commonly in clinical practice when fetal heart rate abnormalities do not conform to the universally acknowledged criteria. The fifty traces used in the first stage study were first assessed clinically and it was later found that of the seven records associated with low pH values, only five were considered sufficiently abnormal to warrant an FBS. Thus routine screening of the records would have failed to pick up two of the acidotic fetuses. There is clearly a need for a more objective assessment of fetal heart rate traces. The results of this study suggest that the objective measurement of baseline variability may provide an answer to this problem. There was loss of baseline variability for at least ten minutes duration in each of the seven first stage traces associated with low pH values and each of the eight second stage traces associated with one minute Apgar scores of less than six. There were 29 traces with

similar loss of baseline variability but associated with normal pH values and Apgar scores. This means that, in this study, use of baseline variability as the sole criterion would have reduced the group requiring FBS or intervention to approximately half the size of the original monitored group. Loss of baseline variability may be due to hypoxia (Hammacher et al., 1968) or to the administration of drugs to the mother (Scher et al., 1972; Caldeyro-Barcia et al., 1966). An attempt was made to determine the possible aetiology in the 17 non-acidotic fetuses showing this feature in the first stage. It was noted that in six instances in which intravenous chlormethiazole was being administered the mean baseline variability did not exceed 7 b/m^2 on the coarse average scoring system and that, in five of these traces the mean value was 2.5 b/m^2 . Similarly the two premature fetuses in the series demonstrated mean baseline variability values of less than 3.5 b/m^2 . The administration of pethidine or oxytocics to the mother had no significant effect on the mean baseline variability score ($\chi^2 = 2.5682$ with five degrees of freedom $p = <0.8$ and $\chi^2 = 0.031$ with one degree of freedom $p = <0.9$ respectively). It is suggested that the measurement of baseline variability may be used as a screening test to determine which fetuses require further investigation in the form of fetal scalp sampling. It is proposed to test this hypothesis using automated apparatus which will write out the actual baseline variability and give a warning (easily recognised by paramedical staff) when this parameter has been less than a critical value for more than ten minutes.

The fact that the average fetal heart rate can be processed in order to achieve correlation with pH suggests that there is some basis for the time honoured use of abnormal rate as an indicator of possible fetal distress. However, our results suggest that a fetal heart rate within the normal range by no means excludes fetal acidosis.

Little attention has been paid so far to accelerations. Most authors consider them to be benign (Renou et al., 1969; Beard et al., 1971) but rebound accelerations have been related to low Apgar scores in two studies (Coltart et al., 1969; Goodlin and Lowe, 1974a). In addition an association between accelerations and variable decelerations has been reported (Puttler, 1974). Personal experience has

suggested that uncomplicated transient accelerations often progress to become biphasic periodic changes in fetal heart rate, i.e. a transient change in heart rate which commences as an acceleration but alters to a deceleration during a contraction. For this reason it was assumed, perhaps incorrectly, that uncomplicated transient accelerations and initial accelerations have similar significance. Thus, for purposes of measurement, the two were grouped together and labelled as initial accelerations. Rebound accelerations were measured as a separate group. There appeared to be little relationship between either direct area measurements or the tested acceleration/deceleration ratios and pH values. The clinical significance of the findings relating to rebound accelerations is debatable as, in most instances, the measured areas were small compared to the dip area. There were only five cases with total areas greater than 50 beats and none of these were acidotic ($\chi^2 = 2.25$ with two degrees of freedom; $p = <0.5$). Whilst there would be hesitancy to suggest that rebound accelerations are a good prognostic sign the larger rebound areas were seen in traces associated with normal pH values, a finding which does not support the conclusions of the aforementioned authors.

The presence of initial accelerations causes difficulty in assessment and interpretation of lag times. Because of this, part of the study was devoted to measuring the lag time between the apparent onset of contraction and any associated transient change in rate whether it was an acceleration, a deceleration or a combination. It is interesting that this mean lag time correlated slightly better with pH values than did the widely accepted lag times; peak to nadir and onset of contraction to onset of deceleration. This could be due to the fact that these latter two lag times must be relatively elongated in the presence of initial accelerations although there is no evidence to suggest that initial accelerations are associated with acidosis.

It has been shown that the critical lag time is approximately 18 seconds (Chick et al., 1974) but the exact decision level employed clinically is rather arbitrary. The lag times observed in the traces of acidotic fetuses in this study were in the order of 8.5 to 10.5 seconds but it should be noted that the values were derived from

external tocography. A study is in progress to determine the delay, if any, between the onset of intrauterine pressure rise and the onset of contraction determined by external tocography. This is obviously of considerable importance as the majority of clinical units in this country utilise external tocography.

With regard to the size and shape of decelerations the relationship between the slopes of deceleration and recovery shown in Fig.VI:4 suggests that the majority of decelerations are almost uniform in shape with recovery rates which are a little slower than the rates of initial deceleration. As the decelerations become larger the recovery rates become more variable. It is interesting that the traces associated with acidosis all lie in close approximation to the line of regression and all but one lie at the lower end of the line. This suggests that in shape the decelerations seen in acidotic fetuses are indistinguishable from the "normal" and they tend to have slower rates of deceleration and recovery. Unlike previous investigators (Kubli et al., 1969; Wood et al., 1969) we have been unable to demonstrate a significant relationship between the actual amplitude of decelerations and pH. However, this study involved all decelerations irrespective of classifications whereas Kubli et al (1969) demonstrated correlations within each classification of decelerations and Wood et al. (1969) demonstrated correlations using only a coarse assessment for amplitude of deceleration.

The implication drawn from the first stage results that baseline variability is a significant parameter in evaluating fetal distress appears to have been substantiated for the second stage of labour and reinforces the importance placed upon this variable by other investigators (Yeh et al., 1973; Goodlin and Lowe, 1974b).

The presence of a significant linear correlation between total deceleration area in the second stage and one-minute Apgar score supports the findings of previous investigators (Shelley and Tipton, 1971). Together with the lack of correlation between lag times and Apgar score it suggests that the factors affecting the FHR in the last hour of labour are different, either in degree or type, to those in the first stage of labour when there is a significant correlation

between lag times and pH but none between deceleration area and pH.

Deceleration size in the fetus of the rhesus monkey has been shown to correlate with the oxygen saturation of the fetal blood (Myers et al., 1973). It is possible that this correlation exists in the human situation perhaps only below a certain threshold level. In the last hour of labour, the larger part of which is associated with full dilatation, there exist several factors which could be responsible for a lowering of the oxygen saturation to below such a threshold level in the majority of cases. Intra-amniotic pressures far exceed the accepted first-stage maximum of 80 mm. of mercury during the second stage of labour and, as the contractions usually occur more frequently in the second stage the duration of normal tonus is reduced. The blood flow through the placental bed, therefore, is likely to be considerably less than it is during a normal first stage.

The correlation between maximum amplitude and Apgar score merely reflects the correlation between total area and Apgar. The area of each deceleration depends approximately on the square of its amplitude so that the total deceleration area depends strongly on the high amplitude decelerations.

With regard to the lack of correlation between lag time and Apgar score it is possible that, in the second stage, this parameter is masked by the almost invariable presence of immediate or early decelerations occurring either as a result of head compression or mechanical cord occlusion.

In conclusion, the importance in the first stage of labour of lag time measurement has been shown but questions have been raised regarding the critical lag time to be used with external tocography. The relative importance of late initial accelerations has been shown and discussed. The results of this intensive analysis suggest that baseline variability is the most useful of the monitored parameters in the search for possible fetal distress in both first and second stages of labour and that reduction of this parameter to below five beats per minute for more than ten minutes should be an indication for fetal blood sampling or, in the second stage, the consideration

of instrumental intervention. The work of earlier investigators regarding the relationship between the total deceleration area in the last hour of labour and the Apgar score has been endorsed and a possible explanation for this relationship occurring only in the second stage has been suggested.

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VII: THE AETIOLOGY, CHARACTERISTICS AND DIAGNOSTIC
RELEVANCE OF LATE DECELERATION PATTERNS IN ROUTINE
OBSTETRIC PRACTICE*

SUMMARY

A retrospective study is described in which all the late deceleration patterns seen in one obstetric unit over the course of 34 months have been reviewed with regard to their apparent aetiology, their characteristics and associated abnormal fetal heart rate patterns and the incidence of fetal distress.* The results show that, regardless of the aetiology and the characteristics, the incidence of fetal distress (defined as a pH of less than 7.25 or an Apgar score of less than 7 at one minute) lies between 40 and 50 per cent. The implications of this degree of diagnostic inaccuracy are discussed and the importance of fetal blood sampling as the final arbiter in the diagnosis is emphasized. During the course of the study a significant relationship was demonstrated between the use of epidural analgesia and the incidence of late deceleration patterns. This finding is discussed.

The term late deceleration has been applied to transient decelerations of fetal heart rate which commence during a uterine contraction, some 20 to 30 seconds after the onset of that contraction, and persist for 30 to 60 seconds after the end of the contraction (Hon, 1959; Hon and Quilligan, 1967). Clinical observation has shown the frequent association between this type of deceleration and fetal acidosis (Kubli et al., 1969; Beard et al., 1971) and as a result a late deceleration pattern is considered almost diagnostic of fetal distress, especially in the presence of other fetal heart rate abnormalities

*Published in the British Journal of Obstetrics and Gynaecology - February 1975. Vol.82 pp 121 - 125.

**For distress please read acidosis.

(Beard et al., 1971). Unfortunately, the association is not invulnerable and the use of cardiotocography alone, without the aid of fetal blood sampling, is likely to result in some unnecessary operative deliveries.

This retrospective study was carried out to assess the true relevance of late deceleration patterns in clinical practice and to determine whether, by studying more closely the apparent aetiology and the characteristics of each late deceleration pattern and its associated abnormal fetal heart rate patterns, the diagnosis of fetal distress by cardiotocography alone might be made more reliable. In the course of the study an apparently strong relationship between the use of epidural analgesia and the appearance of late deceleration patterns was found and examined.

METHODS

Each routine intrapartum continuous fetal heart trace obtained from the labour ward between July 1971 and May 1974 was analysed by the author. Especial attention was paid to late deceleration patterns which were defined as follows.

A late deceleration pattern was said to have developed if five or more late decelerations occurred consecutively. A late deceleration was defined as any transient deceleration of heart rate exceeding 10 beats per minute in depth, the nadir of which occurred more than 30 seconds after the peak of the contraction.

Labour ward notes and information recorded on the traces were used to determine the sequence of events leading up to and following each late deceleration pattern. An attempt was made to determine the aetiology of the late deceleration pattern in each case. The state of each fetus was assessed using the results of fetal blood sampling and the state of each newborn baby was determined from the one and five-minute Apgar scores and the relevant notes in the labour ward record.

During the 34-month period studied there were 5549 deliveries of which 946 were monitored either because of clinical fetal distress or

as indicated by "at risk" factors. Late deceleration patterns occurred in 79 cases (8.4 per cent). It was necessary to exclude two cases from the study in view of insufficient information regarding the labour.

RESULTS

The relevance of late deceleration patterns in terms of fetal distress

The one-minute Apgar score was less than 7 in 36 cases (47 per cent) and the five-minute Apgar score was less than 8 in 11 of these. Relevant fetal blood samples were taken in 42 patients and the resulting pH was less than 7.26 in 17 (41 per cent). The overall incidence of fetal compromise in terms of either fetal scalp blood pH or the one-minute Apgar score was 47 per cent.

The apparent aetiology of late deceleration patterns

Table VII:1 shows the factors associated with the appearance of late decelerations. The pattern developed within two to fifteen minutes of administration of epidural anaesthetic and/or the adoption of the supine or lithotomy position in 55 patients (71 per cent). There were no obvious aetiological factors in 15 patients (19 per cent). In three patients there was obvious uterine hypertonia, in one the fetus was severely affected by rhesus isoimmunization and in the remaining three patients prematurity was considered as a possible aetiological factor. Nuchal encirclement by the umbilical cord was present in 21 patients. The incidence of fetal compromise lay between 40 and 50 per cent in each of the aetiological groups except where there was no proven factor in which group the incidence was not significantly different at 53 per cent.

The relationship between epidural analgesia, late deceleration patterns and fetal compromise

This was investigated using figures obtained in the last twelve

TABLE VII:1

The factors associated with the appearance of late deceleration

Factor	Total No.	Patients with fetal distress	
		No.	Per cent
Epidural alone	12	5	42
Supine position or lithotomy	24	12	50
Epidural and supine position or lithotomy	19	8	42
Uterine hypertonia	3	1	-
Prematurity	3	1	-
Rhesus isoimmunisation	1	1	-
No obvious cause	15	8	53
Associated factor:			
Nuchal encirclement by cord	21	10	48

months of the study following marked changes in labour ward policy regarding fetal monitoring, epidural analgesia and documentation. During the last twelve months of the study 252 epidural anaesthetics were given and 124 were monitored. Late deceleration patterns were observed in 25.8 per cent of these compared with 5.2 per cent of the 344 monitored labours which were managed without epidural analgesia in the same period. This difference is statistically significant ($p = <0.001$ χ^2 test; 1 degree of freedom).

Despite the significantly higher incidence of late deceleration patterns in the epidural group the incidence of fetal compromise in terms of fetal scalp blood pH and one-minute Apgar score was almost identical in the epidural and non-epidural groups (28.0 and 28.5 per cent respectively).

The characteristics of the late deceleration patterns

The pattern exceeded 60 minutes duration in 35 patients (45.5 per cent) and resolved within one hour in 32 patients (41.5 per cent). In the remaining 10 patients delivery occurred within the 60 minute period. 57 per cent of the persistent patterns were associated with fetal compromise compared with 34 per cent in the short-lived group. This difference fails to reach statistical significance ($p = <0.1$). There was no significant difference between the mean depth of late decelerations in the compromised and uncompromised groups, the values were 40 ± 13 (SE 3.4) and 37 ± 14 (SE 2.9) respectively.

Associated abnormal fetal heart rate patterns

Table VII:2 shows the incidence of other fetal heart rate abnormalities occurring in association with late decelerations in the compromised and uncompromised groups. Tachycardia (more than 160 beats per minute for ten minutes or more) and obvious narrowing of the baseline variability to less than 5 beats per minute were present either individually or together in 20 compromised fetuses (59 per cent) and 14 uncompromised fetuses (41 per cent). This difference is not statistically significant ($p = <0.1$). Bradycardia was seen in six patients and in only one case was the fetus acidotic.

TABLE VII:2

The incidence of associated abnormal fetal heart rate patterns

Abnormal pattern	Total No.	Incidence of fetal distress	
		No.	Per cent
Silent baseline variability	21	13	(62)
Tachycardia	24	15	(62)
Silent baseline variability and tachycardia	11	8	(73)
Silent baseline variability and/or tachycardia	34	20	(59)
Bradycardia	7	1	(14)
Cord occlusion pattern	11	7	(64)

DISCUSSION

Published reports on the use of electronic fetal heart rate monitoring have shown the necessity of using the method as a screening procedure and not as a final arbiter in the diagnosis of fetal distress (Beard et al., 1971; Shenker, 1973). Despite such reports the cardiotocograph is still used alone, without resort to fetal blood sampling, in some obstetric units. This study was set up to determine whether such use of the cardiotocograph is justified and to investigate possible methods of improving the diagnostic accuracy of the fetal heart rate trace. The results show that only half of the fetuses demonstrating late deceleration patterns show any other signs of compromise and that, even by looking at the apparent clinical cause, the size of the decelerations, the duration of the pattern and associated abnormalities of heart rate, there was no way of distinguishing the compromised from the uncompromised fetuses without further investigation in the form of fetal blood sampling.

It may be argued that the criteria used in this study for a late deceleration pattern were not strictly correct according to classical usage. Eleven of the late deceleration patterns were suggestive of cord compression by virtue of the shape and depth of the decelerations and these would therefore have been labelled as "variable" according to the accepted classification (Hon and Quilligan, 1968). This was felt to be unimportant as both variable and late decelerations are associated with fetal distress and it would therefore seem logical to include late, variable decelerations in the clearly defined late deceleration group rather than in the vague variable deceleration group. Seven of the eleven fetuses involved were distressed.

The significant association between epidural analgesia and late deceleration patterns was an interesting finding in the light of previous prospective trials designed to investigate the effect of epidural analgesia on fetal heart rate (Noble et al., 1971; Schifrin, 1972). Noble et al. (1971) found an 18 per cent incidence of maternal hypotension related to epidural analgesia but there was no significant difference between the respective incidences of late decelerations in the epidural group and a non-epidural group. Schifrin (1972) found

that late decelerations occurred in 72 per cent of cases in which similarly defined hypotension occurred and there was an incidence of 23 per cent even in the absence of measurable hypotension. This latter figure agrees closely with the incidence of late decelerations associated with epidural analgesia in this present study. It is not usual, in this unit, to experience large falls in maternal brachial blood pressure associated with epidural analgesia but it is possible, as Schifrin (1972) suggests, that this measurement does not necessarily reflect the uterine artery pressure upon which intervillous perfusion depends. There is evidence to suggest that during normotension the uterine artery pressure as reflected by pressure in the femoral artery, is equal to the brachial blood pressure but that during hypotension, whatever the degree of severity, the fall is 1.4 times greater in the uterine artery than it is in the brachial artery. This effect has been attributed to aortic compression (Bieniarz et al., 1966). The discrepancy between the fetal heart rate findings of Noble et al. (1971) and those of Schifrin (1972) and this present study requires examination. It is probably related to epidural technique and it is possible that speedier turning of the patient from the supine to a lateral position following an epidural top-up might have resulted in a lower incidence of fetal heart rate abnormalities. The three studies are agreed that the use of epidural analgesia makes no difference in terms of perinatal outcome.

The supine and lithotomy positions appeared to be responsible for the appearance of late decelerations in the absence of epidural analgesia in 15.6 per cent of the patients in this study. The supine hypotension syndrome is thought to occur in only 2 per cent of pregnant patients (Holmes, 1960), but as its appearance depends on the inability of the collateral venous circulation to compensate for compression of the inferior vena cava (Kerr et al., 1964) it might reasonably be expected that a spectrum exists between no supine hypotension and the complete syndrome. As described above it is possible for a small fall in brachial blood pressure to be accompanied by a greater fall in uterine artery pressure.

The information accrued in this study suggests that a large proportion of late deceleration patterns occur secondarily to transient

lowering of the uterine artery blood pressure caused either by compression of the abdominal aorta or by a fall in cardiac output secondary to compression of the inferior vena cava or by a combination of the two. Whether or not acidosis develops depends on the state of the fetus at the onset of labour and presumably on the duration of the aetiological insult although it was not possible to prove that in this study. Animal experiments have shown that late decelerations are a reflection purely of fetal hypoxia and that correction of the hypoxia may bring about resolution of the heart rate despite the continued presence of acidosis (James et al., 1972; Myers et al., 1973). It is apparent that, in view of the inconstant relationship between late deceleration patterns and fetal scalp blood pH, serial estimations of pH are necessary in order to determine the true state of fetal well-being.

In conclusion, a review of all the late deceleration patterns occurring in one obstetric unit in a 34-month period has shown the necessity of carrying out fetal blood sampling in addition to cardiotocography and that there is no justification for the use of cardiotocography alone. A significant relationship has been observed between the use of epidural analgesia, the adoption of the supine or lithotomy positions and the appearance of late decelerations.

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VIII: PROGRESSION IN RESPONSE PATTERNS OF FETAL HEART RATE
THROUGHOUT LABOUR*

SUMMARY

A study of 70 fetal heart rate (FHR) traces throughout labour has allowed analysis of FHR responses associated with approximately 7000 individual uterine contractions. The significance of the less emphasized FHR patterns has been assessed and an attempt has been made to identify any trend or progression in the occurrence of these patterns. Transient acceleration was the most frequently occurring response in early labour and was the first observed response in the majority of cases. Decelerations became prevalent as labour progressed. A hypothesis is proposed to explain the observed overall progression in FHR responses and the possible physiological mechanisms underlying the cyclic changes in response patterns which occur throughout the course of labour.

Intrapartum fetal monitoring depends on assessment of the significance of fetal heart rate (FHR) responses during and between uterine contractions. Following the original description of classical response patterns by Hon (1968) there have been additional reports of significant patterns (Beard et al., 1971; Shelley and Tipton, 1971; Goodlin and Lowe, 1974). It is becoming increasingly clear that many different FHR response patterns are present in labour and that the clear interpretation of FHR traces would be easier if it were possible to elucidate the significance of the less common FHR responses. In this study an attempt has been made to determine their significance and to identify any trend or progression in their occur-

*Published in the British Journal of Obstetrics and Gynaecology - December 1975. Vol.82 pp 943 - 951.

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rence which may be of predictive value.

MATERIALS AND METHODS

The 70 patients in this study were delivered at University College Obstetric Hospital between January 1973 and June 1974 and each fulfilled the criterion of having been monitored throughout established labour to within 5 minutes of delivery, using a Hewlett-Packard cardiotocograph (HP 8025A). Monitoring was limited to "at risk" fetuses, as defined by Beard et al. (1971), and to fetuses demonstrating clinical signs of distress. FHR tracings were taken directly from a scalp or spiral electrode and uterine contractions were registered by external tocography or intrauterine pressure measurements.

A code, which is shown in Table VIII:1, was prepared to represent ten major FHR responses to contractions, the delay in response time and the basal rate and baseline variability between responses. The traces were then analysed progressively contraction by contraction. This made it possible to determine the prevalence of each of the response patterns and to determine any pattern progressions which might occur. Infants with a one-minute Apgar score of 6 or below were then compared with the high Apgar group with respect to these prevalences and pattern progressions.

Five minute and 15 minute segments of FHR trace demonstrating constant response patterns were then sought and the respective incidences in the low and high Apgar groups were compared. Similarly, the characteristics of basal fetal heart rate and baseline variability in the two groups were compared.

The respective incidences of early and late decelerations and the timing of their appearance in relation to the time of delivery were compared in the two groups of newborn babies.

TABLE VIII:1

Definition and description of fetal heart rate response patterns observed in the 70 traces

Basal conditions	Types	Description
Baseline variability	Silent	Fluctuates less than five beats interval difference
	Normal	Range between 5 and 25 beats interval difference
	Saltatory	In excess of 25 beats interval difference
Basal fetal heart rate (between contractions)	Bradycardia	Less than 120 beats per minute
	Normal	120 to 160 beats per minute
	Tachycardia	In excess of 160 beats per minute
Patterns of response to contractions	Types	Descriptions
	I Normal	Basal rate of 120 to 160 per minute, baseline variability between 5 and 25 beats interval No change with contractions
	II Transient acceleration	Transient increase in rate of more than 10 beats lasting 30 seconds or more
	III Pre-acceleration dip	Initial acceleration of more than 10 beats followed by deceleration

Continued

TABLE VIII:1

Definition and description of fetal heart rate response patterns observed in the 70 traces

Continued

Patterns of response to contractions	Types	Description
	IV Pre-acceleration deceleration and rebound	Similar to III with a rebound acceleration exceeding 10 beats above baseline
	V Early deceleration	A pure deceleration lasting 30 seconds or more
	Vb Late deceleration	Pattern similar to V except nadir is delayed with regard to peak of uterine contraction by 30 seconds or more
	VI Block deceleration	Large steep deceleration lasting more than 30 seconds and characterized by flat troughs
	VII Prolonged deceleration	Deep deceleration which recovers slowly through a few contractions
	VIII Deceleration and rebound	Rebound of at least 10 beats above baseline after deceleration
	IX Saltatory response pattern	Large acceleration and deceleration differing from saltation in its excursions being spread over 30 seconds or more

RESULTS

Twenty-two of the 70 monitored fetuses (31.5 per cent) had an Apgar score of less than 7 at one minute. Only in two of these was the Apgar score less than 8 at five minutes.

There was no significant difference in the management of the two groups of patients. The number of uterine contractions per half hour was similar in the two groups. There was a mean of 11 contractions per half hour in early labour and this increased to 13 during the second stage.

Transient FHR changes in response to uterine contractions were present in all tracings and early decelerations were recorded at some time during each of the 70 labours in the study.

Distribution of response patterns

In early established labour approximately 60 per cent of traces were without transient FHR responses. This percentage decreased as labour progressed until one hour before delivery when only 12 per cent showed no FHR changes (Fig. VIII:1). The first observed response to contractions in 78 per cent (55/70) of patients was a transient acceleration. The incidence of transient accelerations fell gradually during the course of labour. The incidence of pure decelerations was low in early labour but increased gradually to reach levels equal to transient accelerations (10 to 15 per cent), three to four hours before delivery. There was a sharp increase in the incidence of decelerations in the last 90 minutes of labour. Each of the remaining FHR response patterns occurred in association with between 5 and 10 per cent of contractions but there were no obvious trends in incidence.

The distribution of response patterns in the low Apgar group was similar to the overall distribution shown in Fig. VIII:1, with the exception that the incidence of transient accelerations was marginally lower in the first half of labour and the rise in incidence of pure decelerations tended to occur earlier, i.e. 6½ hours as opposed to 5 hours before delivery (Fig. VIII:1).

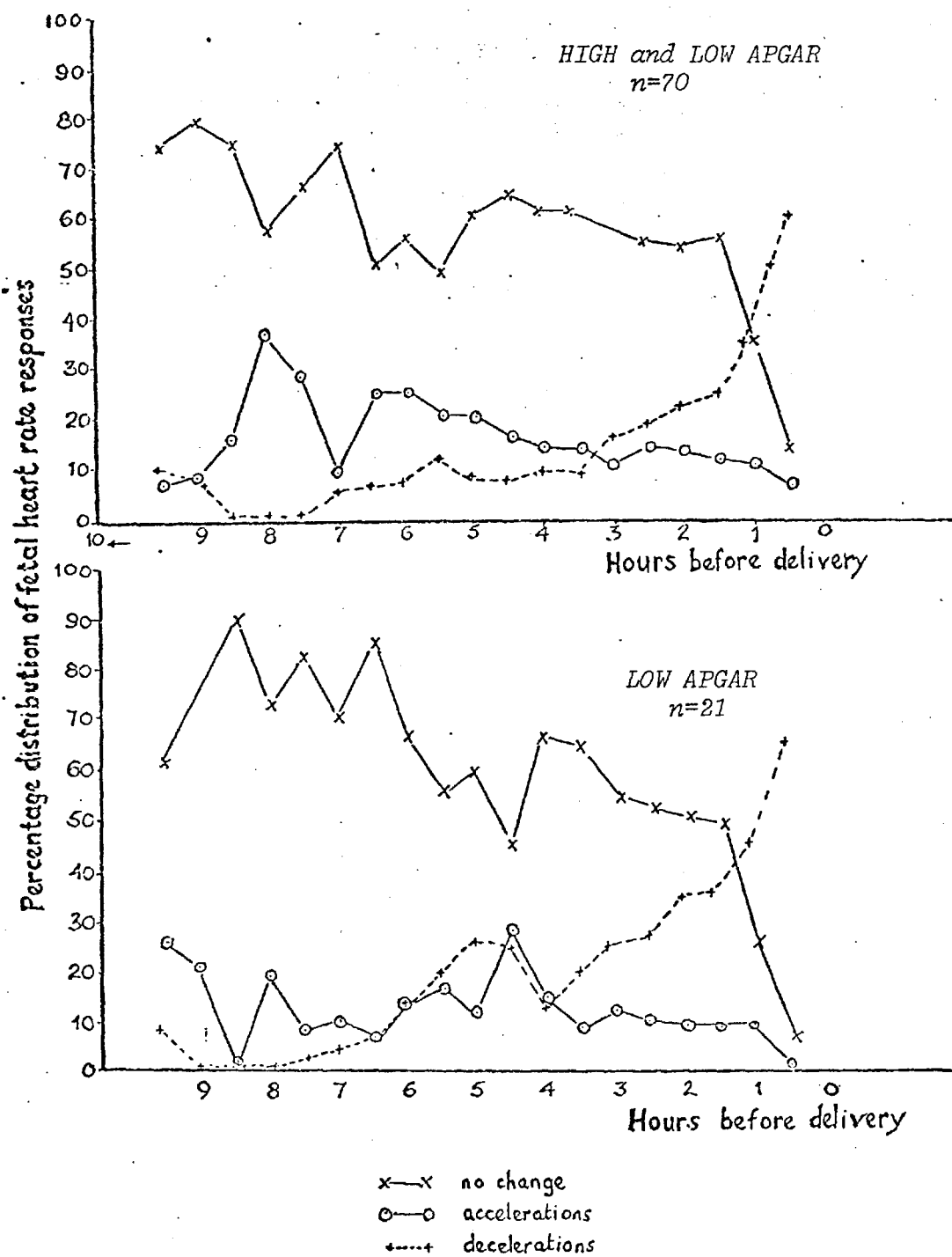


Fig. VIII:1

Showing the percentage distribution of fetal heart rate responses and comparing the low Apgar group with the total group comprising both high and low Apgar neonates.

TABLE VIII:2

The significance of five minute segments of constant response pattern in the two groups of infants

Patterns of fetal heart rate response	Patients in low Apgar group		Patients in high Apgar group		Significance using chi-square
	Number	Per cent	Number	Per cent	
II	18	81.8	47	97.9	$\chi^2 = 3.72$ $p^c = <0.1$
III	18	81.8	35	72.9	-
IV	12	54.5	21	43.8	-
V	22	100.0	48	100.0	-
Vb	10	45.5	10	20.8	$\chi^2 = 3.36$ $p^c = <0.1$
VI	9	40.9	7	14.6	$\chi^2 = 4.53$ $p^c = <0.05$
VIII	9	40.9	19	39.6	-
IX	16	72.7	31	64.6	-

Five minute segment analysis (Table VIII:2)

Five minute segments of FHR trace demonstrating only transient accelerations were more frequent in the high Apgar group of fetuses ($p = <0.1$). Segments of late decelerations and block decelerations (pattern VI) were more often associated with the low Apgar group ($p = <0.1$ and <0.05 respectively). Only one episode of protracted deceleration (pattern VII) occurred in this study.

Fifteen minute segment analysis

Fifteen minute segments of trace in which only one type of response pattern was seen occurred much less frequently than did five minute segments. Fifteen minute segments of patterns other than early decelerations, late decelerations and transient accelerations were very uncommon. Those segments demonstrating early decelerations and those showing transient accelerations occurred with similar frequency in the high and low Apgar groups. In the high Apgar group, 30 fetuses (62.5 per cent) demonstrated fifteen minute segments of early decelerations compared with 15 fetuses (68.1 per cent) in the low Apgar group. Similarly 12 high Apgar fetuses (25 per cent) demonstrated fifteen minute segments of transient accelerations compared with five fetuses (22.7 per cent) in the low Apgar group. The differences between these respective incidences are not significant. Fifteen minute segments of late decelerations were present in 14 high Apgar fetuses (29.2 per cent) and nine low Apgar fetuses (40.9 per cent). This difference is not significant.

Progressions

Table VIII:3 shows the distribution of response patterns which were seen to occur immediately after a normal trace and compares the respective incidences in the high and low Apgar groups. Transient accelerations were the most commonly occurring first response patterns (52.8 per cent). Early decelerations were observed as the first patterns in 20 per cent of fetuses whilst the incidence of each of the remaining patterns as a first response was less than 10 per cent. There was no apparent relationship between the nature of the first

TABLE VIII:3

Types of first response pattern following a normal trace in the two groups of infants

Type of first response pattern following a normal trace		Number of patients		
		Total	Low Apgar	High Apgar
Pattern I	—————→ Pattern II	37	11	26
Pattern I	—————→ Pattern V	14	5	9
Pattern I	—————→ Pattern III	7	1	6
Pattern I	—————→ Pattern IV	5	2	3
Pattern I	—————→ Pattern IX	5	2	3
Pattern I	—————→ Pattern VI	1	1	-
Pattern I	—————→ Pattern VIII	1	-	1

TABLE VIII:4

Progression of response patterns through transient acceleration in the two groups of infants

Progress in patterns of response				Number of patients			Significance by chi-square
				Total	Low Apgar	High Apgar	
Pattern I	→	II	→ V	14	5	9	-
Pattern I	→	II	→ IX	9	0	9	$\chi^2 = 3.2$ $p^c = <0.1$
Pattern I	→	II	→ III	7	5	2	$\chi^2 = 3.9$ $p^c = <0.05$
Pattern I	→	II	→ IV	6	1	5	-
Pattern I	→	II	→ VIII	1	0	1	-

FHR response and one minute Apgar score.

An attempt to assess possible significance in progression of response patterns from normal through transient acceleration to a third pattern is shown in Table VIII:4. Early deceleration, saltatory responses and pre-acceleration dips were seen to follow transient accelerations in decreasing order of incidence. The trend, normal to transient accelerations to pre-acceleration with deceleration was significantly more common in the low Apgar group ($p = <0.05$) whilst the progression of normal to transient acceleration to saltatory response was significantly more common in the high Apgar group ($p = <0.1$).

In view of the number of labours studied, it became impractical to continue this serial method of progression analysis. The distribution of response patterns following pre-acceleration with deceleration, early deceleration and saltatory response was analyzed and tabulated (Table VIII:5). Pre-accelerations with deceleration were most commonly followed by early decelerations as were saltatory response patterns. Early decelerations were most commonly followed by transient accelerations. Frequent alternations between early and late decelerations occurred in both groups of fetuses.

Early and late decelerations

Considering the total number of decelerations in the study, there were 417 early and 245 late decelerations in the low Apgar group of fetuses. In the high Apgar group the respective numbers were 654 and 226. The early to late ratio was therefore 1.6 in the low Apgar group compared with 2.7 in the high Apgar group. Although the incidence of 15 minute segments showing late decelerations was higher in the low Apgar group this was not statistically significant.

The delay between the peak of a contraction and the nadir of a deceleration varied considerably from contraction to contraction so that the prevalence of individual early and late decelerations fluctuated throughout the course of labour regardless of the eventual outcome.

TABLE VIII:5

Distribution of patterns following pre-acceleration dips, early decelerations and saltatory responses

Preceding patterns	Types and total incidence of patterns following preceding patterns in the 70 traces							
	II	III	IV	V	Vb	VI	VIII	IX
III	27	-	10	32	18	2	4	18
V	50	38	11	-	30	12	13	41
IX	30	19	14	39	14	3	9	-

TABLE VIII:6

Distribution of episodes of basal rate and FHR variability recorded in the two groups of infants

	Low Apgar infants			High Apgar infants		
	Below 120	120 to 160	Above 160	Below 120	120 to 160	Above 160
Basal fetal heart rate in beats per minute						
Number of episodes	18	54	27	65	111	20
Baseline variability in beats per minute per minute	Less than 5	5 to 25	More than 25	Less than 5	5 to 25	More than 25
Number of episodes	84	90	11	116	194	70

Baseline variability and basal FHR

Analysis of baseline variability and basal heart rate showed a tendency for these parameters to fluctuate throughout labour with no evidence of progressive trends. However, there was a highly significant difference in the frequencies of abnormal episodes of basal heart rate and baseline variability ($\chi^2 = 17.32$, $p = <0.001$ with 2 df and $\chi^2 = 21.43$, $p = <0.001$ with 2 df respectively), in the low and high Apgar groups (Table VIII:6). Episodes of tachycardia ($\chi^2 = 13.06$, $p = <0.01$) and silent baseline variability ($\chi^2 = 11.4$, $p = <0.01$) were increased significantly in the compromised group of fetuses.

DISCUSSION

The interpretation of fetal heart rate traces is not as straightforward as was implied by the classical descriptions of FHR monitoring (Hon, 1968; Hammacher, 1967; Caldeyro-Barcia et al., 1966). There are several reasons for this :

(a) A persistently unchanging trace throughout labour is uncommon as shown not only in this study but also by others (O'Gureck, 1972). This accounts for difficulty in the definition of normality. The significance of the classical responses is not universally accepted (O'Gureck, 1972; Tipton and Finch, 1972) and other responses such as overshoot or rebound patterns (Goodlin and Lowe, 1974) have been advocated increasingly as prognostic signs.

(b) It is not always clear whether the significance of any response pattern lies in its single occurrence, its repeated occurrence or its temporal relationship to other events of labour. Both basal FHR and the frequency of transient FHR patterns fluctuate throughout labour.

(c) Although certain patterns such as late decelerations are associated with fetal distress this association is not invariable (Thomas, 1975)). In this study five minute segments of late decelerations were seen in 50 per cent of compromised fetuses but they were also observed in 25 per cent of non-compromised fetuses.

All studies so far associate poor prognosis with cumulative

appearance of response patterns which indicate evidence of hypoxia. A small segment of tracing is of little prognostic or predictive value. It is hoped that a postulate based on the findings of this study may lead to better understanding of FHR responses when considered in conjunction with the following physiological facts:

(i) There are, in the fetal medulla, cardio-inhibitor and cardio-accelerator centres controlling the fetal heart via sympathetic (cervical ganglia) and parasympathetic (vagal) nerves (Best and Taylor, 1968). As pregnancy advances a vagal dominance becomes prevalent (Hon et al., 1961; Hon and Lee, 1963).

(ii) Hypoxia depresses the myocardium and thus slows the myocardial response (James et al., 1972, Renou et al., 1968).

(iii) Persistent hypoxic stress affects basal fetal heart characteristics, such as rate and variability (Dawes et al., 1959; Dawes, 1969). Superimposed on the existing level of tissue oxygenation uterine contractions exert a further acute hypoxic episode which may elicit a transient fetal heart rate change via stimulation of the neurological system. The type of response pattern resulting depends on the type of neurological centre affected (sympathetic or parasympathetic) and the strength (amount of hypoxia) of stimuli in conjunction with the physiological condition of the myocardial tissue.

In adults the response to continued hypoxia is initially tachycardia and hypertension followed by bradycardia and hypotension. In human fetuses (Hon and Lee, 1963) and the subhuman primate (James et al., 1972) a similar response to hypoxia pertains. When subject to persistent hypoxic stress the fetal adrenergic and neurological response first produces basal tachycardia but, with continuing hypoxia, bradycardia associated with a failing myocardium is recorded (Hon, 1959; Barden and Stander, 1963). Hypoxia also dampens the fetal medullary control of sympathetic/parasympathetic activity (Gellhorn, 1957), thus diminishing baseline variability, i.e. the picture of silent baseline variability is produced. Baseline variability must, however, be considered against the background of drugs administered, maturity of the fetal medullary centre and the idiosyncrasy of the recording machinery.

The fetal medullary centre mediates the transient FHR changes observed when uterine contractions produce an acute episode of hypoxic stress. It is postulated that in any labour where fetal oxygenation is satisfactory the normal basal FHR recordings remain unchanged during contractions. With the progress of labour basal hypoxia increases as indicated by progressive acidosis (Beard et al., 1967). The threshold level of hypoxic stress is lowered and a stage is reached when additional hypoxia during a contraction will suffice to stimulate a cardio-accelerator response in the form of a transient acceleration. As labour progresses the fetal head is wedged in the pelvis and pressed against a half-dilated cervix, resulting in greater cranial compression, the umbilical cord may be tightened and hypoxia is increased as a result of stronger uterine contractions. This combination of vagal stimuli overrides the sympathetic cardio-accelerator response and a transient bradycardia (Type I or early deceleration) results during the added hypoxic stress associated with a contraction. Results from this survey support this hypothesis, as the primary and most common progression of FHR responses is an unchanging trace progressing through transient accelerations early in labour to early decelerations from the 5 cm cervical dilatation stage onwards. Incomplete vagal dominance results in the expression of combined acceleration and deceleration FHR responses (e.g. patterns III, IV, VIII and IX). The distribution in incidence suggests that these patterns are interposed between transient accelerations and early decelerations (Table VII:5). A pre-acceleration/dip occurs earlier in the progression whilst a rebound pattern may be associated with increasing hypoxia (Goodlin and Lowe, 1973). Since the most common pattern following early deceleration is a transient acceleration, it is possible to depict a cycle of FHR response to an episode of hypoxic stress (Fig. VIII:2). Throughout labour, subjection and recovery from episodes of hypoxia possibly imposed by the effects of position and drugs may result in cycles of FHR response patterns.

The intact myocardium responds to severe hypoxic stress by a protracted transient bradycardia e.g. a deep deceleration. The physiological explanation of lag time and hence its clinical significance is the subject of recent deliberation. It is possible that in the presence of severe hypoxia there is depression of either myocardial or neurological function which results in a delayed fetal heart rate res-

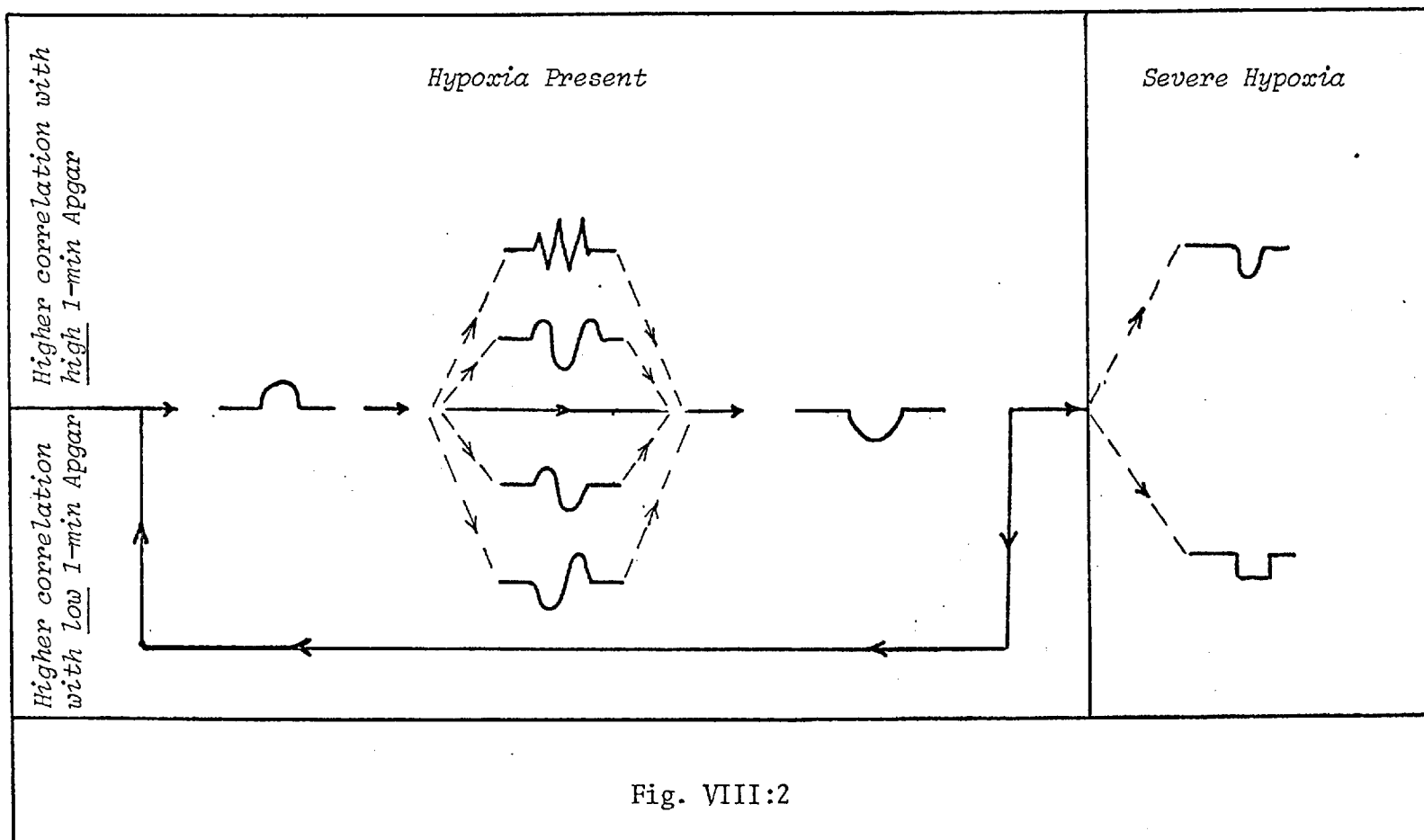


Fig. VIII:2

Postulated cycle of fetal heart rate response patterns during the transient hypoxic stress imposed by uterine contractions

The continuous line shows the most common pathway and the broken lines show possible alternatives: by-pass and reversal occurs.

ponse, i.e. late decelerations. As an alternative to a haemodynamic aetiology for late decelerations (Hon, 1968), it may be suggested that the length of delay before deceleration is related to the amount of hypoxic damage and the myocardial idiosyncrasy of the individual. This subject is discussed further in Section IX page 137 where it is suggested that whilst increasing hypoxia depresses neurological function and thereby delays vagal slowing of the heart rate in response to contractions, further lowering of oxygen saturation causes a diminution of myocardial reserve so that slowing of the heart rate then occurs earlier. In other words, some early decelerations may represent a level of oxygenation which is lower than that represented by late decelerations. Application of the above concept when interpreting FHR traces provides a better understanding of fetal status. No single pattern holds prognostic value; cycles of response patterns correlate with cycles of hypoxic stress and the cumulative evidence of gross hypoxia, e.g. numerous occurrences of silent baseline variation, persistent tachycardia or late decelerations are associated with ultimate fetal compromise. It must be emphasized that FHR patterns depict fetal hypoxia but cannot reliably indicate the amount of fetal reserve to stress. To date the final arbiter of fetal welfare is still fetal blood sampling as it alone draws the demarcation for definitive action.

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IX :

GENERAL DISCUSSION

including a critique of Sections VI, VII and VIII and discussion on the pathophysiology underlying fetal heart rate responses, the objectives of intrapartum fetal monitoring and fetal asphyxia.

We must have many new thoughts and feelings to analyse and to discover whether a little more knowledge has not made us more ignorant.

JOHN KEATS

Before discussing further the contents of the three previous sections it would be wise to recap the known physiological facts which relate specifically to fetal heart-rate and fetal acid-base status in utero, and then to consider the prime objectives of intrapartum fetal monitoring.

EARLY DECELERATIONS : The work of Sir Joseph Barcroft and his colleagues (1) has served as a basis for most of the studies of fetal cardiovascular haemodynamics which have taken place in the last thirty years. Their work is not entirely unchallenged, however, particularly with regard to the mechanisms involved in bringing about fetal heart rate decelerations which are related solely to uterine contractions, namely the type I, early or head-compression deceleration (2, 3). As early as 1927, in an extremely interesting paper, Freed (4) had suggested that decelerations associated with contractions were caused by intracranial pressure. Barcroft suggested that these decelerations were brought about by vagal activity which was stimulated as a result of hypoxia caused by a reduction in placental circulation at the height of a contraction, an opinion which was endorsed by Reynolds and Paul (5) following work on fetal lambs. However, in 1958, Prystowsky (6) observed a bradycardia resulting from head compression in the neonate and concluded that hypoxia was not involved in the pathogenesis. Meanwhile, Hon (7) had applied pressure to the fetal skull in utero and achieved fetal heart rate decelerations as a result. Later the experiments were repeated by Chung and Hon (8) using hard-rubber pessaries; the effects of forceps application and forceps traction on fetal heart rate were also documented. The authors described the observed decelerations

as being synonymous with those decelerations seen in labour which start as the contraction commences and return to normal as the contraction finishes. These decelerations were therefore labelled as head-compression bradycardia, the term deceleration not being introduced until later in order to prevent confusion between transient FHR changes and long term changes in rate. Chung and Hon postulated that if there were an hypoxic step in the pathogenesis of these decelerations then there would be a time lag after the onset of contraction and before the start of the associated deceleration; it was therefore concluded that the decelerations were the result of a direct neurological reflex. These decelerations were first described by Naegele the Younger in 1838 (9) and they can be reproduced in the majority of patients in an antenatal clinic merely by applying a particularly powerful Pawlick's grip; their appearance is sudden and this would again support the theory of a direct neurological aetiology. Looking carefully at Hon's work, however, a question must be raised as to the advisability of likening these reflex decelerations to some of those early decelerations seen in labour. In the paper presented by Chung and Hon it is shown and stated that the bradycardia (deceleration) returned to normal before the head compression was released and, similarly, during forceps traction the fetal heart rate recovered to normal whilst the traction was maintained except during the last pull in which case other factors such as perineal pressure or cord compression might have been operating. Except in the case of spikes (decelerations lasting less than twenty seconds) it is common for early, type I decelerations to return to normal as the contraction subsides so that it is not entirely safe to liken them to head-compression decelerations. The decelerations observed at the onset of the second stage tend to commence as spikes, gradually increasing in width and depth as the second stage becomes more established and it is likely that these are related, initially and at least in part, to head compression. However, at any other stage of labour there may be two types of early deceleration :

- a) those which are spike-like and return to normal before the contraction ceases, namely head-compression decelerations, and
- b) those which exactly mirror the shape of the uterine contraction, possibly reflecting hypoxia ⁺ head compression.

Of interest in this respect is the report of Paul and colleagues

(10) on head compression in fetal lambs. There were clearly two groups, one in which a return of FHR to normal was observed before head compression was released and another group of equal size in which the deceleration persisted until 10-30 seconds after removal of the stimulus. In the first group there was a secondary bradycardia of only 10-30 seconds duration at the time of removal of the stimulus. From their studies Paul et al, concluded that cerebral hypoxia, secondary to reduced carotid blood flow, was the cause of the observed bradycardia. The difference between the two groups might be attributable to different basal levels of cerebral oxygenation.

In a paper entitled 'Does uterine contractility cause fetal bradycardia ?' Vasicka and Hutchinson (11) described observations in forty unclassified patients in various stages of labour. They observed that an acceleration of fetal heart rate by 10-20 beats per minute (b.p.m.) was frequently observed in normal labour when the intrauterine pressure during a contraction did not exceed 60 millimetres (mm.) of mercury, the resting tone did not exceed 12 mm. of mercury and contractions lasted no longer than 50-60 seconds. No appreciable bradycardia was seen under these conditions in early labour prior to rupture of the membranes and engagement of the vertex. However, if the contractions were prolonged, especially in the presence of excessive intra-uterine pressure, then conspicuous slowing of the fetal heart was observed and this persisted until the first adequate relaxation. Once the amniotic sac was ruptured and the head had engaged fetal bradycardia (decelerations) was observed often, coinciding with the peak of each contraction. These decelerations were attributed solely to head compression in view of the 'presence of an ideal uterine contractility and absence of umbilical cord incarceration.' A representative FHR trace from that paper is reproduced in Figure IX:1; it is interesting to note that the head-compression decelerations return to baseline well before the tocographic recording, certainly in two out of the three decelerations shown.

Perhaps it is necessary to look more closely at the recovery phase of early onset decelerations in order to discover two types, one type carrying considerably more significance than the other in terms of fetal well-being. There is scope for further investigation here.

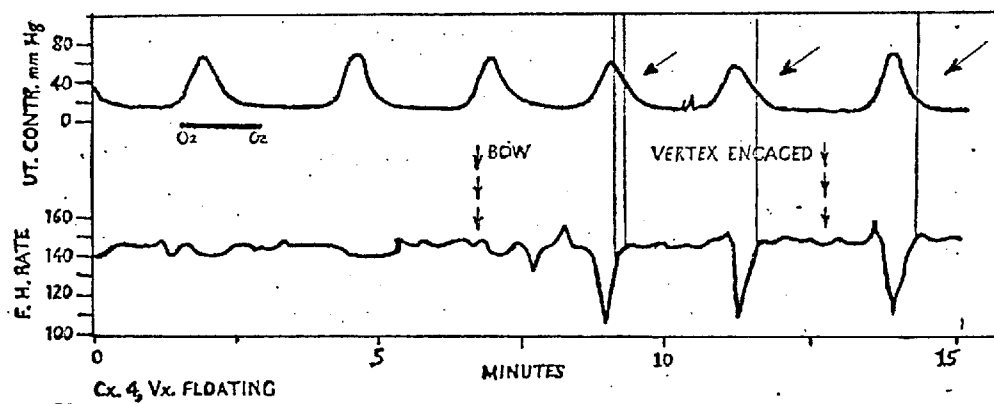


Fig. IX:1

Showing the way in which decelerations attributed to head compression return to baseline well before the tocographic recording

BOW = breaking of waters

*(Trace reproduced from Vasicka, A and Hutchinson, H.T (1963)
Obstetrics and Gynaecology, 22, p 400)*

It is of interest that, in some instances, Chung and Hon observed small accelerations prior to their supposedly innocuous 'head-compression' decelerations. These are the pre-accelerations referred to in Section VI; they have been linked with variable decelerations by Puttler (12) and by O'Gureck (13) and they have actually been described as part of a cord-compression FHR pattern by Goldkrand and Speichinger (14).

VARIABLE DECELERATIONS : The relationship between cord compression and FHR response is inconsistent both in clinical practice and in research models. For that reason the term 'variable' is a very appropriate description for the observed FHR patterns associated with cord occlusion. O'Gureck and her colleagues (please see reference 13), in their attempts to simplify Hon's classification of FHR patterns, found it necessary to subdivide variable decelerations into four groups.

The physiological effects of cord compression were observed by Barcroft in an exteriorised fetal goat in which total occlusion caused an abrupt fall in FHR which, in view of its sudden onset, was attributed to a vagal reflex. Bilateral vagotomy led to a delayed bradycardia (deceleration) which was less severe and was attributed to myocardial hypoxia. Reynolds (15) also demonstrated a vagal role in the abrupt bradycardia observed as a result of complete cord occlusion in fetal lambs. It is likely that the vagal reflex is initiated via sensory depressor receptors in the aorta by rises in fetal systemic blood pressure occurring as a result of the cord occlusion. This assumes complete occlusion but in the clinical situation partial occlusion is probably a more common phenomenon. Lee and Hon (16) showed in human fetuses at Caesarean section that complete cord occlusion caused a rise in umbilical artery pressure associated with an immediate, marked bradycardia but that release of the occlusion led to unpredictable changes in fetal blood pressure and fetal heart rate. The results of partial cord occlusion were less predictable in terms of fetal systemic blood pressure but bradycardia was a consistent finding. Umbilical venous compression alone led to a fetal bradycardia although the change in fetal blood pressure was minimal and slow. In fetal lambs umbilical venous compression led to a fall in blood pressure which was associated with a reduction of fetal heart

rate. Presumably the fall in blood pressure was secondary to a fall in cardiac output, reflecting a reduced 'venous' return, but it is likely that the fetal bradycardia was mediated by a vagal reflex secondary to cerebral hypoxia.

It has been suggested that pre-accelerations are associated with variable decelerations, being a reflex increase in FHR caused by the fall in cardiac output. The FHR response to umbilical cord occlusion is therefore unpredictable, depending on the relative degrees of occlusion of the arteries and vein.

LATE DECELERATIONS : Delayed 'bradycardia' related both to uterine hypertonicity and maternal hypotension was described by Hon in 1959 (17). Later, in the revised working classification (18) these were relabelled as late decelerations, patterns of FHR response attributable to uteroplacental insufficiency. In 1972 Myers (19) reported experimental work carried out in monkeys at term in which the effects of total asphyxia were compared with those of partial asphyxia, the latter being brought about by stimulation of strong uterine contractions by oxytocin injections to the mother. Myers noted that late decelerations or type II dips appeared invariably as the pH of the fetal arterial blood dropped to levels at or below 7.10 to 7.15. Each contraction led to an increasingly deep late deceleration although the delay was regularly between 20 and 30 seconds. Despite relating the onset of late decelerations to a specific level of pH, Myers was careful to stress that the oxygen availability was the prime factor in determining vital organ function reflected by heart rate during asphyxia. This was a conclusion reached by James and colleagues (20) as a result of experiments in monkeys and baboons in which progressive fetal acidosis, hypoxia and hypotension in advancing labour were found to be associated with fetal tachycardia and late decelerations. The latter were abolished by the administration of oxygen to the mother despite the persistence of the fetal acidosis and hypotension. Although both fetal hypoxia and fetal acidosis were present in most instances of late decelerations, the occasional fetus was seen in which late decelerations were associated with severe hypoxia but only moderate acidosis. It would seem, therefore, that severe fetal acidosis is not necessary for the development of late decelerations. This might explain, in part, some of the findings of the

late deceleration study in Section VII; it shows the dangers of confusing the terms hypoxia and acidosis under the label of fetal distress and demonstrates the need for routine assessment of fetal oxygenation in the management of labour. James was unable to demonstrate any correlation between the degree of hypoxia and the depth of the late decelerations; indeed, the response to the contractions was inconsistent with some decelerations even preceded by small accelerations, the so-called pre-accelerations described earlier which are said to be suggestive of partial cord compression.

In 1973 Myers (21) reported further important observations in asphyxiated fetal monkeys near term. Asphyxia was achieved in spontaneous labours by compression of the lower third of the maternal aorta. It was found that late decelerations developed when the oxygen saturation fell below 35 per cent provided the contractions were normal. However, the length of the contraction influenced the response so that even relatively well-oxygenated fetuses responded with late decelerations when contractions were prolonged. This may well explain the phenomenon of the fetus which shows late decelerations but which is delivered in excellent condition. There was no relationship between the appearance of late decelerations and fetal pH nor between the characteristics of the late decelerations, namely lag time, rate of deceleration and depth of deceleration, and pH although the lag time appeared to fall as the hypoxia increased and the rate of deceleration increased with increasing hypoxia.

The reduction of lag time as the hypoxia worsens is an interesting observation suggesting a gradually diminishing fetal oxygen reserve. In the human, Hon (22) has clearly documented one case in which lag time shortened progressively through labour prior to an intrapartum death. The delay in onset of deceleration is attributed to the time taken for the deoxygenated umbilical venous blood to reach the heart. Obviously the higher the level of oxygenation prior to the insult which results in reduced placental perfusion, the longer the delay before the myocardium is subjected to relatively de-oxygenated blood. Barcroft considered myocardial hypoxia to be the major factor in the aetiology of late decelerations but it is possible that a vagal reflex is mediated as a result of cerebral hypoxia.

The lack of correlation between the presence of late decelerations and pH must cause some concern in view of current methods of intrapartum fetal monitoring. Kubli and colleagues (23) stated that late decelerations were not invariably associated with acidosis and this is something which has been re-emphasised by the study described in Section VII. Hon and Khazin (24) maintained that the relationship between the two was more marked when there was no baseline fluctuation present. Both Beard (25) and Wood (26) showed a highly significant clinical association between late decelerations and fetal acidosis. In 1975, Paul and colleagues (27) confirmed their belief that late decelerations were associated with fetal acidosis if there was silent baseline variability but that late decelerations in the presence of normal baseline variability were usually associated with higher levels of pH.

It is possible to debate the late deceleration/fetal pH relationship endlessly depending on whether the work quoted consists of relatively coarse clinical studies or whether the work is purely physiological animal experimentation. This point was brought out by Myers in 1972 (please see reference 19) when he stated that because most clinical situations leading to fetal asphyxia are not readily reversible it is expected that the correlation between FHR and pH would be better than in studies such as his own involving easily reversible asphyxia in animals.

There is, perhaps, a basic philosophical point which warrants discussion at this juncture. Whilst the teams of Hon and Caldeyro-Barcia were investigating the prognostic value of FHR patterns in labour and were presenting physiological and clinical evidence for their hypotheses, Saling, in Germany, was studying the prognostic value of fetal pH (28); all three presented their clinical evidence in terms of neonatal outcome. Both FHR patterns and fetal pH were found to be effective in predicting fetal demise in the majority of cases. Thereafter began the questionable activity of cross correlation between the two methods. Some correlation was certain to exist since both methods were seeking to detect the same type of fetus, the potential low-Apgar neonate, but how much cross-correlation should exist in the individual fetus is open to question especially in the light of animal experiments.

THE OBJECTIVES OF INTRAPARTUM FETAL MONITORING

That the use of intrapartum cardiotocography and fetal blood sampling is associated with the prevention of intrapartum fetal death from asphyxia has been shown both in this discourse and by other authors (29, 30, 31). The main objective, however, is to prevent mental retardation and spasticity attributable either to intrauterine asphyxia or to birth trauma which is particularly prevalent in hypoxaemic or asphyxiated fetuses. This should not be taken to imply that intrapartum cardiotocography and fetal blood sampling should be used merely to improve the potential intelligence of all neonates. Such improvement may well occur as an offshoot of the main objective but whether it is totally desirable, even in modern, automated society, is open to question.

There have been attempts to relate intelligence quotient (I.Q.) to the circumstances surrounding the birth process but, in view of the multitudinous environmental factors which work from the time of birth it is unlikely that I.Q. assessments can be used to determine the role of intrapartum fetal monitoring. Much more important in terms of national and personal economy and in terms of personal emotional distress is to study mental retardation and spastic paralysis and to see if intrapartum monitoring can reduce the incidence of these tragic conditions.

The difficulty in scientifically proving that intrapartum FHR monitoring has a role in preventing handicap has been referred to earlier in this discourse. As Quilligan indicated in 1972, 'such a study is very easy to insist upon and very difficult to do' (32).

The nearest approximation to such a study was described at the Study Group in the Management of Labour at the Royal College of Obstetrics and Gynaecology in 1975 (33). In Melbourne, Australia, the management of high risk patients using intrapartum cardiotocography and fetal blood sampling has been compared with the management of an equal number of similar patients without the aid of intrapartum monitoring. The difference was highly significant with five neurologically damaged babies and one death on the unmonitored side compared with one neurologically damaged baby in the monitored group. (33a)

The study proved the value of intensive care in labour but it must not be assumed that cardiotocography and fetal blood sampling were any more contributory than the intensive observation which necessarily accompanied them.

THE PRE-LABOUR CONDITION OF THE FETUS

In both Sections VII and VIII it is postulated that the pre-labour condition of the fetus is of as much importance as the course of labour in determining early neonatal outcome.

To date the available methods of fetal monitoring in pregnancy have been sadly lacking in precision and reliability and even time-honoured clinical assessment has been shown to leave undetected approximately thirty per cent of fetuses affected by intra-uterine growth retardation (34). Biochemical placental function tests are notoriously inaccurate and fetal movement counts and antenatal cardiotocography have remained unacceptable until very recently, the first because of the lack of objectivity and lack of knowledge regarding fetal motor activity, the second because of limited facilities and, again, lack of documented knowledge. Antepartum cardiotocography involves personnel and equipment which have not been available in most units except for intrapartum monitoring.

Pearson (35) has recently presented evidence which shows the potential value of continuously monitoring the fetal heart rate early in labour, a practice which has not become well established partly because of insufficient space or staff in labour wards, partly because of practical difficulties but mainly through a failure to appreciate the diagnostic potential of this ultimate, natural stress test in picking out the previously undetected, chronically hypoxaemic, growth retarded fetuses.

Fortunately, the increasing use and accuracy of diagnostic ultrasound and a slowly improving understanding of antenatal FHR characteristics (36) and fetal movements (37) are assisting in the drive to detect these at-risk fetuses before they are subjected to the risk of further hypoxia and possible asphyxia during labour.

ASPHYXIA

Windle et al. (38) showed that intrauterine asphyxia led to alterations in brain structure in guinea pigs and much more recently Myers (please see reference 19) showed two patterns of brain damage related respectively to total and partial intrauterine asphyxia in the near term monkey. In his paper Myers presented an argument which suggested that the changes occurring in the fetus as a result of asphyxia depend upon the degree of asphyxia and upon the physiological state of the fetus prior to the causative insult. Total asphyxia causes a type of brainstem damage which is unlike that found in damaged humans and it is suggested that such a degree of asphyxia would normally lead to myocardial failure and death rather than present as brain damage. On the other hand, post-mortem examination of term monkeys which have been subjected to partial asphyxia and then resuscitated prior to sacrifice shows that the cerebral hemispheres may be damaged to a varying degree which may involve the whole of both cerebral hemispheres or be limited to the para-central area or to the basal ganglia. These lesions resemble those seen in cerebral palsy in the human and it is of interest that the partial asphyxia was brought about by excessive syntocinon, drug-induced maternal hypotension, constriction of the abdominal aorta or partial placental ablation, all of which are potential causes of hypoxia during labour in the human.

Myers classified partial asphyxia into three subgroups :

- a) Mild, where the fetal pH is approximately 7.10 but the fetus is in reasonable condition.
- b) Severe, where the fetal pH is less than 7.00 for several hours and myocardial damage leads to fetal death.
- and c) Intermediate, representing a small number of fetuses between the other two groups in which death does not occur but brain damage does.

Myers stated that severe partial asphyxia can lead to brain damage even in the absence of hypotension - a situation which is equi-

valent to severe intra-uterine growth retardation in the non-labouring woman. The added insult of labour, causing further hypoxia and hypotension associated with late decelerations would cause death in such a fetus. The theoretical consequences of delivering such a severely growth-retarded fetus by Caesarean section require close scrutiny. The reader is referred to the work of Emmen et al. (39) which shows that of twelve fetuses delivered by Caesarean section because of 'terminal' antenatal cardiotocograph tracings, two died in the neonatal period and one survivor has severe brain damage. The others are all apparently normal.

It is the intermediate group for which intrapartum cardiotocography has the greatest potential benefit.

Multiple physical handicap and associated mental retardation can occur as a result of anoxic brain damage in the human. By far the most common form of handicap is spastic diplegia but studies in Sweden (40) have shown a fall of 60 per cent in its incidence in the fifteen years from 1954 to 1970. This fall has been attributed to improved perinatal care but which aspect bears the greatest credit is open to question. Scott in 1974 (41) and Steiner and Neligan in 1975 (42) reported the follow-up of apparent stillbirths which had been resuscitated and concluded that prolonged asphyxia was of much more consequence than an episode of acute anoxia in the aetiology of handicap. In the years 1972 to 1975 from 12,000 to 13,000 deliveries in Oxford there were 25 resulting cases of cerebral palsy but in only ten of these was true birth asphyxia (acute) observed (43). Griffiths (44) reported that the incidence of spastic diplegia was greater in first twins than in second twins despite the fact that the latter are known to experience a high incidence of acute asphyxia. This suggested a lack of association between acute asphyxia and spastic diplegia but associated findings indicated that the more sinister but much less common spastic tetraplegia and its accompanying mental manifestations were more likely sequelae of acute asphyxia. As the result of her observations Griffiths proposed that the two types of cerebral palsy were separate conditions rather than degrees of one condition. Tizard (45) has postulated that spastic diplegia is related more to pre-term delivery and hypothermia in the neonate than it is to asphyxia in labour. From the foregoing observations it would seem that prenatal factors such as prematurity and intra-uterine growth retardation and

postnatal factors such as neonatal heat loss carry as much significance, if not more, in the aetiology of cerebral palsy as a whole than does acute anoxia in labour and birth trauma.

Previous discussion has implied that the growth-retarded fetus, and possibly the premature fetus also, is already hypoxaemic when labour starts. The discussion has also shown that, until recently, the plight of up to 30 per cent of these fetuses remained undetected until delivery or disaster and the fetuses were often unmonitored. Because of the reluctance of many obstetricians to carry out a Caesarean section in order to deliver a fetus of less than 32 weeks' gestation, the need for intrapartum monitoring of such fetuses has been questioned. This is ironic since it is these smaller fetuses which contribute to the spasticity figures in greater numbers than any other group. The benefit of intrapartum monitoring in terms of perinatal mortality has been shown to be at its greatest in the 1,000-1,500 G. group of neonates (46) and the figures presented in Section IV show a relatively large number of unmonitored, premature infants who might have benefited from fetal blood sampling and cardiotocography.

CRITIQUE

The papers presented in Sections VI, VII and VIII should be discussed further now that the concept of intrapartum FHR and acid-base monitoring has been placed in perspective and its objectives outlined.

First the papers must be examined critically. They represent clinical studies relating to the art of 'eyeballing' fetal heart rate traces. The word 'eyeballing' has become an accepted term which implies the informed assessment of continuous FHR traces without the aid of measuring instruments. It requires experience. Unfortunately it is often relatively inexperienced attendants who are present throughout the labour but it is they who need to know when to raise the alarm. How, then, can FHR analysis be simplified, without sacrificing information, in order to facilitate teaching and in order to dispense with the need for expertise or to facilitate automated trace analysis ?

Specific questions relating to these problems were raised in

Part I of Section VI. Before attempting to answer them, the methods used in the studies will be examined more carefully. In physiological terms they are inaccurate but they represent a very accurate version of clinical eyeballing. The use of intra-uterine catheters in these studies was specifically avoided as it was considered that external tocography was used more commonly in clinical practice and it was clinical practice which was under scrutiny. The relationship between external and internal tocography in any one patient has been shown to be inconstant in the studies at University College Hospital, London (47), although Beard (48) maintains that the peaks achieved with external tocography correlate very well with those achieved using intra-uterine catheters. Assuming that there is a slightly varying relationship between an external tocograph trace and the true intra-amniotic pressure then the lag-time measurements in these studies are inaccurate but, again, they do represent clinical assessments which are in common use.

Similarly, in the assessment of baseline variability, there is no claim to having measured beat-to-beat variation or even an approximation of it; rather the usual five to ten minute 'window' used for the clinical eyeballing of the FHR variation on the trace has been reduced to a half to one minute window. In Section V it was shown that the use of the normal five to ten minute window was not successful in detecting all acidotic fetuses, the percentage detected being 54-80 per cent depending on whether 5 b.p.m/² or 10 b.p.m/² respectively was used as the critical level. Similar percentages were achieved when the traces which were examined in Section VI were assessed by eyeballing.

The major criticism in relation to the accuracy of data obtained in these studies concerns the analysis of FHR traces in the first stage of labour; the conclusions depend on the comparison of two variables, namely the FHR characteristics under scrutiny and the fetal scalp blood pH. The accuracy of a fetal scalp blood pH cannot be guaranteed but the methodology was uniform and based on the technique of Saling (49) using heparinised glass tubes for collection. The samples were collected by a limited number of operators, each with considerable expertise in the technique. Cases were excluded from the study if there was any doubt at all about the reliability

of the sample. Some of the limitations of fetal blood sampling techniques were discussed by Beard et al. (50) and this discussion was continued in a paper by Lumley et al. in 1971 (51). In the latter paper the authors compared normal values as determined by many authors. They maintained that there was often considerable inaccuracy of measurement and they referred to unpublished data from their own unit which suggested that scalp capillary blood may range from arterial to venous in a single patient in a short period of time, a factor which could account for much of the lack of agreement on normal values and which could be a source of considerable inaccuracy. Of course, the relevance of pH to fetal condition must be questioned but this has already been discussed. To date, the pH is the only biochemical variable which, in the clinical situation, provides any usable information about the fetus other than base deficit, isolated po_2 estimations being even more inaccurate on the average labour ward blood-gas analyser.

The findings of the study on pattern progressions might have been different had much larger numbers of patients been used. The number of patients whose traces were studied was seventy; in none of these was the first observed response a late deceleration. The appearance of such a transient change in FHR in early labour before the use of oxytocics or epidural analgesia would almost certainly imply the presence of fetal hypoxia. Anecdotal evidence has been avoided as much as possible in this discourse but in Figure IX:2 there is reproduced an FHR trace which was obtained from a fetus during early labour in a mother who was shown eventually to have anti- $\bar{c}$ antibodies. The fetus was anaemic as a result of haemolysis. The decelerations are shallow and late in onset and there is minimal baseline variability which also suggests hypoxia. There was old meconium at artificial rupture of the membranes and the pH taken thirty minutes later was 7.14, one of the lowest obtained at U.C.H. during the three year period in which these observations were made. The fetus was delivered by Caesarean section when the theatre was free some thirty minutes later but the neonate was in poor condition and succumbed. This case has been outlined because it demonstrates three contentions :

- 1) the importance of continuously monitoring the fetal heart

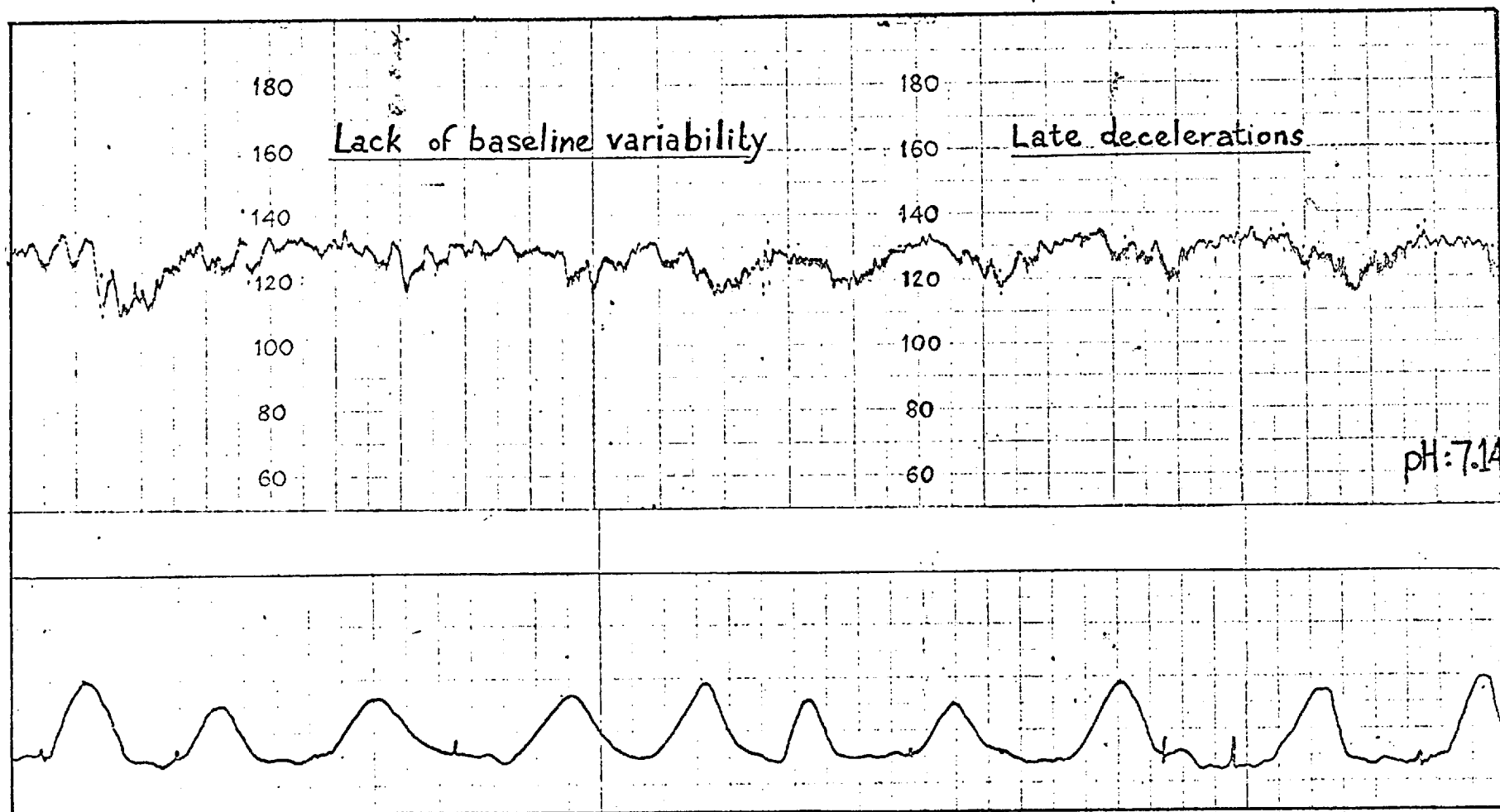


Fig. IX:2

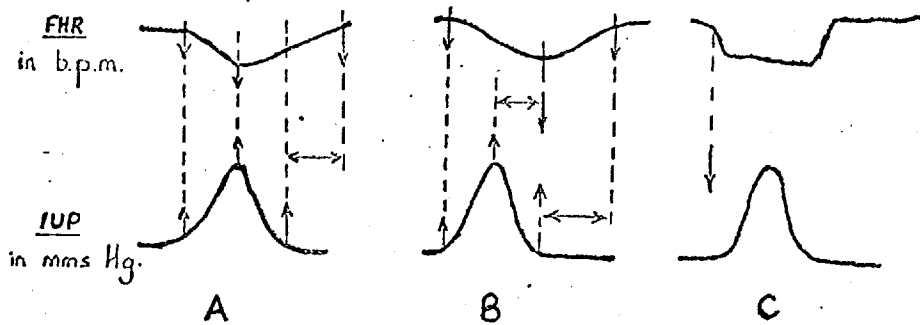
FHR trace from an anaemic fetus during early labour (pH:7.14). Note the shallow late decelerations and the lack of baseline variability.

rate early in labour in order to detect previously unsuspected intra-uterine hypoxia.

- 2) the inadequacy of dip-area measurement in the prediction of fetal state in the first stage of labour. Tipton (52) has stated that transient bradycardia occurring with contractions in early labour is an ominous sign and early intervention is indicated whatever the dip area but this immediately detracts from the advocated value of the measurement since it requires a strict definition of early labour.
- 3) that in the absence of oxytocics, epidural analgesia or the supine position, late decelerations imply the presence of hypoxia especially if there is silent baseline variability. There can be some prognostic significance, therefore, in the first observed FHR response despite the statistical findings of the paper on pattern progressions. Oddly, Caldeyro-Barcia et al. (please see reference 2) postulated that if type II (late) dips appeared early in labour but disappeared at least two hours before delivery then the newborn might be vigorous despite the fact that type II dips were usually ominous. Wood and colleagues (please see reference 26) could not confirm this observation; on the contrary, they observed an intrapartum stillbirth some three hours after the disappearance of type II decelerations.

THE SIMPLIFICATION OF TRACE ANALYSIS

On this question one needs to look no further than the manuals which accompany some makes of cardiotocographic equipment (53, 54) in order to see that an FHR classification would be much simpler were it not for the concept of variable decelerations. The foregoing papers and discussion have indicated the difficulties encountered in trying to distinguish early decelerations from both variable and late decelerations. These difficulties have been described by others including Hon (55, 56) and have been well demonstrated by O'Gureck and her colleagues (please see reference 13) to whom part of Figure IX:3 is attributable. That figure demonstrates some of the more confusing



A, B, and C reproduced from O'Gureck (1972)

Fig. A - early by onset but
late by recovery time.
No peak to nadir delay

Fig. B - early by onset but
late by peak to nadir delay
and by recovery time.

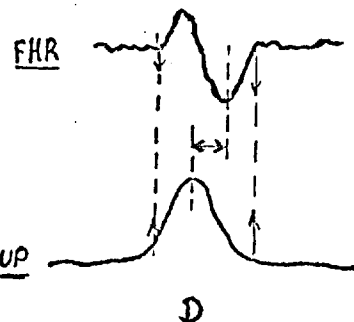


Fig. C - early by onset but
variable by shape in view of precipitous onset and
recovery.

Fig. D - early by onset of transient change but late by
onset of deceleration and by peak to nadir delay.
Early by recovery time.

Fig. E - transient accelerations or late decelerations?

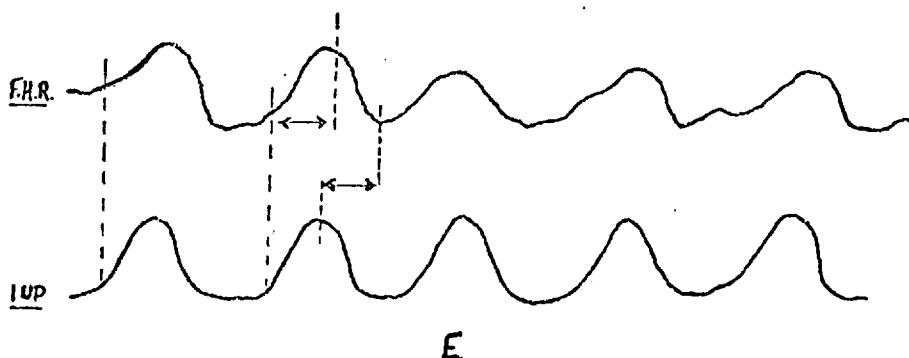


Fig. IX:3 Showing some of the more confusing aspects of FHR classification

aspects of FHR classification.

The difficulties can be overcome by experience but whilst that experience is being gained by the individual operator, that person must have available a simple fail-safe method of using intrapartum monitoring techniques.

Wood et al. (in reference 26) were the first group to point out the difficulties of the original FHR classifications; they created a new classification, which is shown in Table IX:1, for use in conjunction with fetal blood sampling. In that classification the basal heart rate was examined along with early and late slowing of the fetal heart rate in relation to contractions. An 18-second peak to nadir time was used as the critical differentiating lag-time and the depths of the decelerations were used to distinguish between significant and insignificant early decelerations, a drop in excess of 60 b.p.m. being considered as significant. The 'new' classification appeared to have promise but the fact that 'a small number of records was excluded because the FHR changes showed the features of both early and late slowing' again stresses the problem of variable patterns. The authors indicated the dangers of applying statistical information to individual cases and also the risk of applying to one obstetric population information gained from another.

In their 1972 paper (please see reference 13) O'Gureck and her colleagues devised what they proposed as an international classification which took into account fetal heart rate, fetal heart reactivity and combined and isolated accelerations and decelerations. As Table IX:2 shows, the decelerations were classified into Type I or early, Type II or late (using a 20 second peak to nadir lag time) and Type III or variable but, again, the variable decelerations were divided into four subdivisions based entirely on waveform. Early decelerations were said to be ominous if the depth exceeded 50 b.p.m. and isolated accelerations were similarly considered as ominous if their height exceeded 50 b.p.m. Whilst this classification appears perfectly straightforward in print, its application to the clinical situation, particularly, with regard to variable decelerations, is far from easy. Again, the authors stress the need for fetal blood sampling to be used in conjunction with FHR monitoring in labour.

TABLE IX:1

CLASSIFICATION OF FHR CHANGES IN RELATION TO FETAL SCALP
BLOOD pH AND APGAR SCORE

Wood, C. et al. (1969)
American Journal of Obstetrics and
Gynecology, 105, 942

FHR change in relation to contractions	Fetal scalp blood pH	Apgar score
30-60 b.p.m. drop	7.25*	7.07
>60 b.p.m. drop	7.16**	5.82*
early (<18 second delay) deceleration	7.17*	7.10
late deceleration	7.14**	5.40**

*Significantly different ($p = <0.05$) from normal group (pH = 7.32)

**Highly significant difference ($p = <0.001$) from normal.

These observations imply that any early deceleration of >60 b.p.m. and any late deceleration are of significance in the search for fetal acidosis, although the authors stress the danger of applying such statistical information to an individual case.

TABLE IX:2 : O'GURECK'S PROPOSED CLASSIFICATION DESIGNED TO ASSIST INTERNATIONAL COMMUNICATION

O'Gureck, J.E. et al (1972) Obstetrics and Gynecology, 40, 356.

FHR Variable	Abnormality	Characteristics and Significance
<u>Baseline Rate</u>	<u>Tachycardia</u> (for >5 mins)	Moderate : 160-179 b.p.m. : } Severe : >180 b.p.m. : } significant when associated with decelerations >50 b.p.m.
	<u>Bradycardia</u> (for >5 mins)	Moderate : 90-119 b.p.m. : not significant Marked : 70-89 b.p.m. : suggests progressive acidosis Severe : <70 b.p.m. : suggests rapidly developing acidosis
	<u>Non-reactivity</u>	Silent : fluctuations <5 b.p.m. - may be associated with placental insufficiency
<u>Fluctuations in FHR</u>	<u>Acceleration</u> (>15 b.p.m.)	Type I : precedes or follows variable deceleration - unknown significance Type II : moderate : <50 b.p.m. : not significant severe : >50 b.p.m. : suggests progressive acidosis
	<u>Deceleration</u> (>15 b.p.m.)	Moderate to 90-119 b.p.m. : not significant Marked to 70-89 b.p.m. : suggests progressive acidosis Type I : early : peak to nadir time = <21 seconds gradual onset and recovery smooth waveform recovery within 15 secs of end of contraction Type II : late : peak to nadir time = >21 seconds gradual onset and recovery smooth waveform recovery more than 15 secs after end of contraction Type III : 1st degree: precipitous onset & recovery, jagged waveform, short duration:mild (variable) cord involvement 2nd degree: precipitous onset & recovery, smooth waveform, duration=contraction: some tension in cord - no compromise 3rd degree: precipitous onset, gradual & late recovery, smooth waveform; extreme tension in cord - progressive compromise 4th degree: precipitous onset & recovery, biphasic, smooth waveform:cord prolapse, knot in cord - serious compromise.

Also in 1972, Goodlin and Schmidt (57) presented an entirely new concept in an attempt to simplify FHR monitoring. Their suggestion was that the state of the fetus might be predicted from the reactivity of the heart rate occurring in response to contractions and/or external stimuli. The evidence implies that transient accelerations are a good prognostic sign, but the classification of these periodic FHR changes is both cumbersome and complicated and their assessment is purely qualitative whereas quantitative systems are required if trace analysis is to be made easier for paramedical personnel.

The concept of dip-area measurement has already been introduced. Shelley and Tipton (58) were the first to test the correlation between the manual measurement of total deceleration area in the last hour before delivery and the Apgar score at one minute, an exercise which they began because they were 'unable to apply accepted classifications to the observed FHR changes'. The total deceleration area/Apgar score correlation was highly significant ($p = <0.001$); it will be discussed in further detail later in the text. Regrettably it is not fail-safe and its possible clinical use is therefore in doubt.

The four questions raised in Section VI and referred to earlier in this Section will now be considered in turn, remembering that there is always a danger in applying findings within groups of patients to the clinical situation in individual cases.

1) Can intrapartum cardiotocography be used reliably without fetal blood sampling using the original classifications or adaptations of those classifications, using deceleration area measurements or using any other single, measurable variable ?

The simple answer to this is 'no'. In the U.C.H. studies neither a single variable nor a small group of variables was found to correlate closely with acidosis and pre-acidosis and the work of other authors already discussed would support this. The dip-area concept of Shelley and Tipton was found to correlate closely with perinatal condition but only when measured in the second stage.

2) Assuming the necessity to use fetal blood sampling, which single variable or small group of variables might be used to screen out all at-risk fetuses ?

The assessment of baseline variability using half to one minute 'windows' as shown in Figure IX:4 appears to be of value in screening out a group of fetuses including all acidotic and preacidotic fetuses. Unfortunately, the baseline variability is probably affected by other factors such as fetal sleep state and circadian rhythms (59) and is also affected by drugs (60, 61) so that the screened group is a relatively large one, constituting approximately 50 per cent of the monitored population.

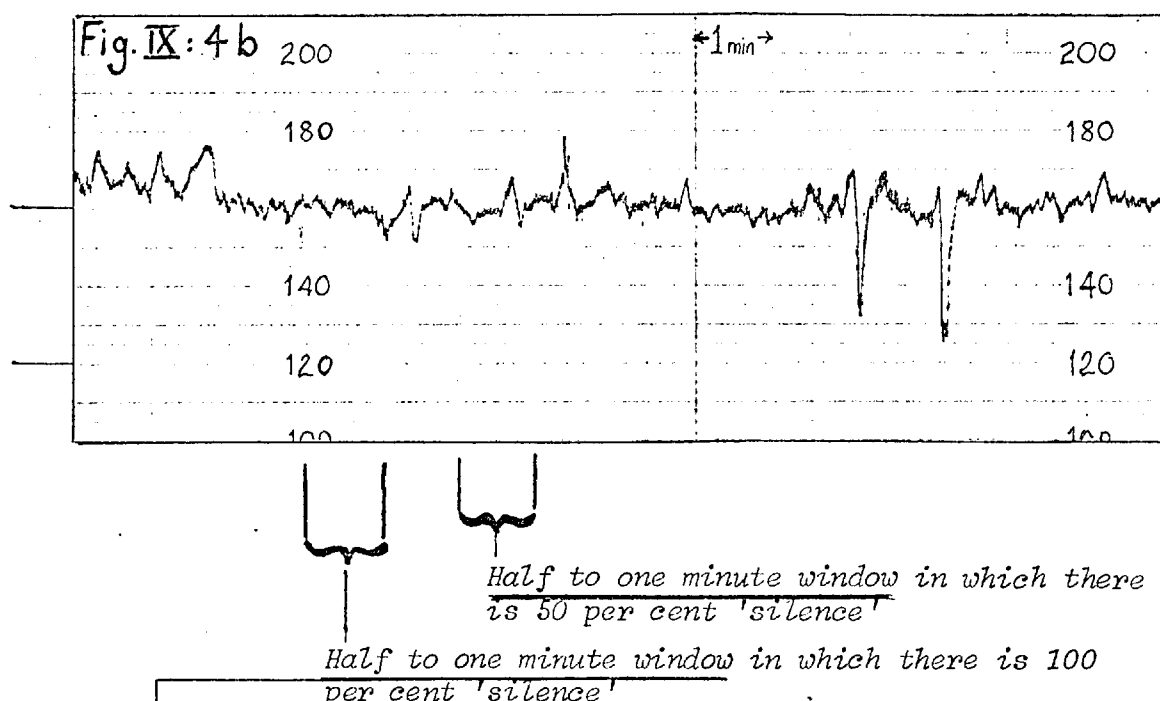
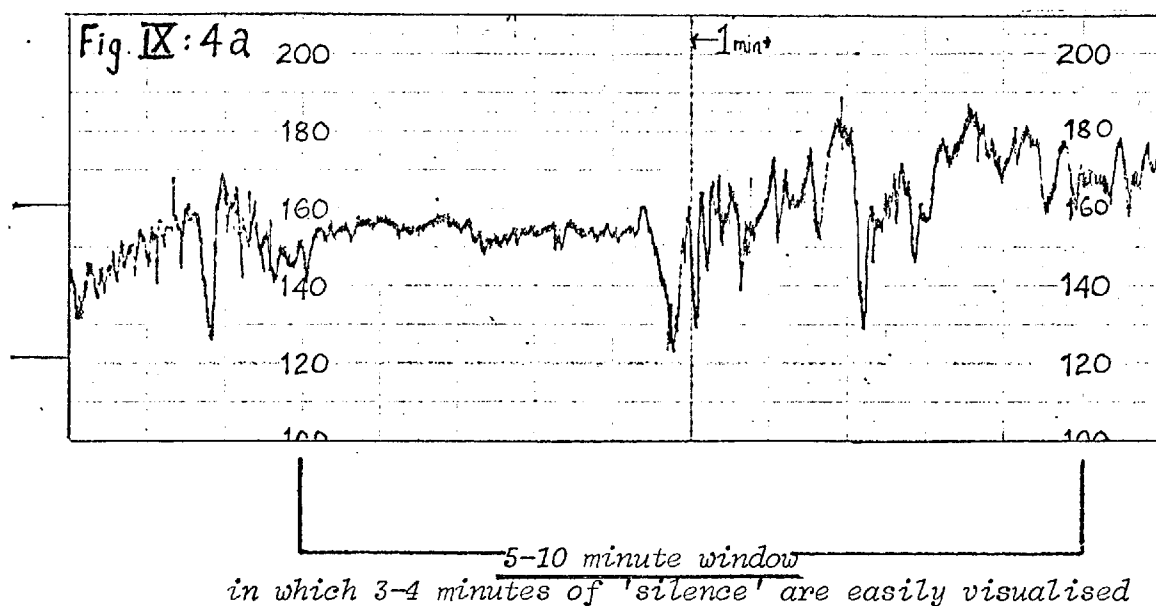
3) Is there any prognostic purpose in the measurement of total deceleration area, baseline variability or any of the other twenty listed variables ?

In the absence of highly significant findings in the trace analysis study described in Section VI few significant clinical conclusions can be drawn from that study alone.

The strongest correlation(*negative*) in the first stage was between lag-time (onset of contraction to onset of transient change) and fetal pH. The more commonly used lag-time (onset of contraction to onset of deceleration) correlated(*negative*) with the same degree of significance as the baseline variability(*positive*), fetal heart rate score(*negative*), initial rate of deceleration(*positive*), time of recovery (*negative*) and area of rebound accelerations(*positive*). In the last hour of labour including the second stage both baseline variability and total deceleration area correlated with one minute Apgar score both significantly and linearly, the first positively, the second negatively. The correlation between baseline variability and Apgar score was greater than it was between total dip area and Apgar score. The negative ranking correlation between maximum amplitude of deceleration and one-minute Apgar score was thought to be related to the total deceleration area/Apgar correlation. The relatively strong linear correlations observed in the last hour of labour may be due to the rather insensitive nature of the Apgar score whereby neonates are really being classed into three groups; vigorous, unresponsive and intermediate. Alternatively, the

Fig. IX:4

Showing the use of different-sized observation 'windows' in the assessment of baseline variability



This method is particularly useful for traces in which the degree of baseline variability changes frequently

findings may indicate that there is a totally different physiological state pertaining in the last hour of labour or, more likely, in the second stage.

It seems reasonable to postulate that head compression is of such a degree in the second stage that early onset decelerations occur invariably so that there is no lag time. The shape of many decelerations in the second stage suggests a degree of cord occlusion, a phenomenon which might well be expected at that time either directly as a result of increased intraamniotic pressure or secondary to compression between the uterine wall and fetal parts or as a result of tightening of a nuchal cord. It is likely, therefore, that second stage decelerations are due to a combination of factors - head compression and partial, or even total, cord occlusion. In addition there is an increased hypoxic threat to the fetus during the second stage because of reduced placental perfusion caused by an increased intra-uterine pressure, an increased frequency of contractions, partial placental separation as postulated by Pearson (62) and the adoption of an almost supine position or even the lithotomy position for delivery. These added hypoxic threats may be reflected to a highly significant degree towards the end of labour by reduced baseline variability and a prolongation of deep decelerations which, in its turn, would be reflected by total deceleration area.

It is interesting that Beguin et al. (63) showed a significant correlation between total deceleration area during the last hour of labour and the one-minute Apgar score but a correlation between deceleration area and pH in the first stage of labour was less easily demonstrated. The results indicated that the pH correlated reasonably well with events taking place during the twenty minutes preceding the pH determination but less well with events occurring 20-30 minutes before sampling. The measurement within the last twenty minutes which correlated most with pH was the area of deceleration occurring between 80 and 140 seconds after the onset of the causative contraction. This particular measurement of dip area is a reflection either of 'lateness' or of very slow recovery both of which correlated within the hour prior to sampling in the U.C.H. study described in Section VI. The fact that this was not reflected by a correlation with total

dip area in that study is probably related to a difference in proportion of early/[variable + late]decelerations i.e. insignificant $\left[\frac{+}{-} \right]$ area/significant area between the two studies. Certainly the incidence of early or Type I decelerations has been shown to reflect obstetric practice in that it appears to be greater in induced labours (64). Presumably such factors as epidural usage, choice of analgesia and maternal position during labour could also affect the proportion. Despite demonstrating a 20 minute dip-area v pH correlation Beguin concluded that the correlation was too poor to be utilized alone for clinical purposes. In two false negatives the authors found that consideration of baseline variability was a better indicator of fetal condition.

4) Is there any prognostic purpose in studying pattern progressions or in noting the first observed FHR response in early labour ?

This has already been discussed in part. Whilst an average type of progression in FHR patterns through labour appears to be normal trace $\longrightarrow$ transient accelerations $\longrightarrow$ combined acceleration/deceleration $\longrightarrow$ early deceleration no deviation from this could be related to low Apgar score so that a study of progression cannot be used prognostically, although any change in FHR or FHR response in clinical practice should always be viewed in the light of previous FHR characteristics. Decelerations seemed to appear earlier in labour in fetuses with eventual low Apgar scores but no consistent relationship between the first observed response and the Apgar score was demonstrable. It is likely that using much greater numbers of patients and so providing a small number of fetuses with intra-uterine growth retardation some relationship between responses in early labour and the neonatal outcome might have been observed.

The studies described in this discourse have revealed no new single variable which is reliable and fail-safe in the diagnosis of fetal acidosis. They have indicated, however, that in order that the inexperienced observer may reliably detect all acidotic and pre-acidotic fetuses, all deceleration patterns which cannot be reversed by a) the cessation of oxytocin, b) a change in maternal po-

sition or c) the correction of maternal hypotension should be investigated by fetal blood sampling. This rule should apply even to early decelerations provided that full dilatation has been excluded. Similarly all patients with evidence of meconium should be investigated further together with fetuses demonstrating heart rates greater than 160 b.p.m. or less than 100 b.p.m. for more than ten minutes. Silent baseline variability ($<5 \text{ b.p.m./}^2$) persisting for more than ten minutes should also be investigated unless external stimuli can be used to arouse the fetus and thus encourage an increase in baseline variability. Such an investigatory régime is presented in Appendix D; it would result in a need for much more fetal blood sampling than is used currently in many units but this need would diminish considerably as the experience of each individual increased.

The findings relating to baseline variability may be of some assistance in this respect. It has been shown that a score achieved by measuring baseline variability through half to one minute 'windows' over the course of an hour will indicate whether or not a fetal blood sample is required to exclude acidosis or pre-acidosis. This type of assessment is feasible at an automatic level were its usefulness to be proved by confirmatory evidence. This topic will be discussed in the next Section. Based on the findings already presented fetal blood sampling would be indicated in 50 per cent of all high-risk monitored cases but this is probably a lower percentage than would be indicated by a régime such as that shown in Appendix D, since decelerations alone have been observed in 60 per cent of first stage traces, not to mention bradycardia, tachycardia and silent baseline variability. As discussion in the next Section will show, the percentage of traces by which fetal blood sampling is indicated should fall as obstetric practice continues to improve.

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X : BASELINE VARIABILITY

Baseline variability is like beauty in that it cannot be assigned numbers, it can only be described.

ROBERT C. GOODLIN

The analysis of fetal heart rate traces described in Section VI was set up originally to investigate the relationship between total deceleration area in the last hour of labour and the one-minute Apgar score and between deceleration area and fetal scalp blood pH in the first stage of labour. Other fetal heart rate (FHR) variables were studied and the most significant finding was that baseline variability, assessed and scored as it was in that study, was the only FHR characteristic which could have been used to screen out all the acidotic or pre-acidotic fetuses and all the low Apgar neonates. This is demonstrated by the graphs shown in figures VI:3 and VI:5 to which the reader is referred.

A review of the literature concerning the interpretation of intrapartum FHR traces shows that Hon and Lee (1) were the first to draw attention to the importance of so called 'beat-to-beat' variation, although Hammacher (2) was the first author to attempt a quantitative classification. Since then many authors have shown or stated that baseline variability or variation is of importance both in labour (3-11) and in the antenatal period (12, 13) when trying to diagnose fetal acidosis and/or hypoxia.

Before embarking on further discussion it is necessary to refer to terminology. When the concept of continuous fetal heart rate monitoring was first introduced the write-out of rate was derived directly, with very little processing, from measurement of beat-to-beat interval, the 'R' wave of each fetal ECG complex being the trigger point. The observed fluctuation in FHR was therefore labelled 'beat-to-beat variation'. The commercial apparatus used in most labour wards, however, includes logic circuits which are designed to exclude artifacts and the influence of maternal ECG; unfortunately some of the genuine information is also excluded so that an effect is

achieved by which extreme changes of rate which are not part of a trend may not appear on the record. The final record is also affected by the response time of the recorder pen so that the write-out may differ remarkably in different makes of equipment. Suffice it to say that the FHR trace which is assessed by the clinician on the labour ward is not a true record of beat-to-beat variation and the term should be reserved for observations in which the equipment is capable of measuring R-R intervals to an accuracy of 200 microseconds (14). The phenomenon which is observed in routine FHR traces has been referred to as baseline fluctuation, baseline irregularity, and baseline variation or variability. The latter is the preferred term as the word variability means the 'tendency to depart in any direction from the mean character' in a biological sense (15).

In addition to ascribing a name to the observed phenomenon it is necessary to describe it. Baseline variability is said to consist of two variations of fetal heart rate (16, 17). The first is a short term variation attributable mainly to the processed beat-to-beat variation but also to cardiac arrhythmias. The normal magnitude of this variation is ± 8 b.p.m. (18). Its presence depends on an intact medulla oblongata and autonomic nervous system as well as good oxygenation of the fetus and the absence of the influence of central depressant sedative drugs. The second is a long term variation which de Haan describes as the gradual changing of R-R interval length associated with at least ten intervals. This variation gives rise to undulating fluctuations of heart rate which occur with a frequency of 2-6 cycles per minute and a range of 6-25 b.p.m. These two types of variation are shown in Figure X:1.

All the records analysed in Section VI were obtained using scalp electrodes so that there was an element of both short term and long term variation in the baseline variability assessment. When phonocardiography or Doppler ultrasound methods are used to record fetal heart rate, the nature of the signals is so imprecise as to preclude the measurement of beat-to-beat intervals so that short term variation cannot be assessed; indeed, artificial or ultrasound noise is sometimes so great that even the long term variations are impossible to assess.

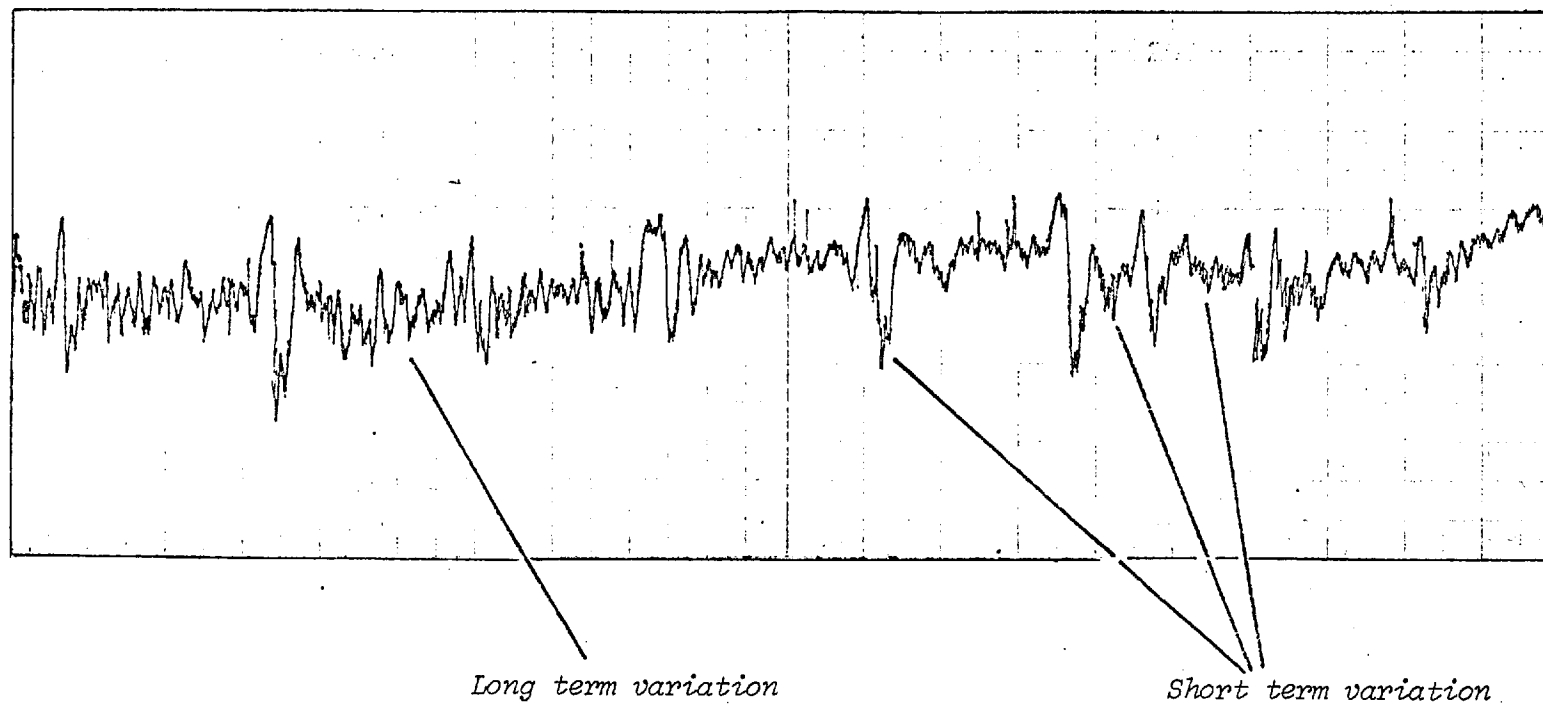


Fig. X:1

Showing the two types of variation seen in baseline fetal heart rate variability

Apart from Curran and Macgregor, the authors of reference articles numbers 1-14 all describe a reduction of baseline variability in cases where there is fetal acidosis and/or hypoxia and/or neonatal depression. Curran and Macgregor (please see reference 12) describe an increase in the coefficient of variation (SD mean beat-to-beat interval/mean beat-to-beat interval as a percentage) associated with poor neonatal outcome. This is not such an easy concept to grasp in relation to heart rate variability and there is one important tabulated value which appears erroneous; however, their findings appear to be in contradistinction to those of the other quoted authors and the U.C.H. study described in Section VI. There may be good reasons for this in that the fetal heart rate records were derived almost directly from the original R-wave triggers thus making the observed variability much closer to true beat-to-beat than it would be using any commercial monitor. In addition, the records were obtained in the last two weeks of pregnancy and not in labour. The only other quoted paper describing baseline variability in antenatal traces is that of Emmen et al (please see reference 13) which looks in retrospect at so-called terminal traces using phonocardiography.

Observations in fetal lambs show that true beat-to-beat variation is increased initially when the animal becomes hypoxic (19), and only terminally does the variation fall. This observation applies whether the hypoxia is acute or chronic (20). Similar observations have been made in the fetuses of Rhesus monkeys (21). There is obviously a time scale of variable length during which hypoxia worsens and FHR baseline variability changes, the direction of change (increase or decrease) depending on the level of oxygenation. Wheeler (22) has shown that in a group of fetuses between 38 and 40 weeks gestation there is a significantly greater amount of saltatory baseline variability than there is in a similarly sized group of fetuses between 30 and 36 weeks gestation. The U.C.H. study on FHR pattern progression, described in Section VIII, shows saltatory responses to uterine contractions occurring in a sequence between transient accelerations and combined accelerations/decelerations or decelerations. In accordance with the author's view that most decelerations are reflections of some degree of hypoxia it seems reasonable to postulate that saltation in the human fetus is a reflection of mild hypoxia, as it is in the fetal

lamb. Although the observation of increased saltatory baseline variability in late pregnancy may be related to changing autonomic balance (vide infra) it may be equally a reflection of mild hypoxia, the incidence of which is certainly likely to be higher in the older fetuses.

The suggestion is, therefore, that saltatory baseline variability is seen in a fetus when there is mild hypoxia which is tolerated perfectly well by that fetus but that once the fetus is sufficiently hypoxic to become preacidotic or acidotic the baseline variability will, by then, have become 'silent'.

Unfortunately, hypoxia is not the only factor which affects baseline variability. Atropine (23) abolishes normal baseline variability by changing the autonomic balance. Such drugs as diazepam (24), thiopentone (25), pethidine (26), phenobarbitone (27) and probably other central depressants reduce the variability to levels of silence (<6 b.p.m.)

The picture is further confused by the fact that variability alters with gestation. Barcroft described vagotonia increasing with gestational age in fetal lambs (28) and this has been demonstrated in the human by Schifferli and Caldeyro-Barcia (29). The presence of sympathicotonia in fetal lambs has been shown by Reynolds (30) and confirmed in humans by Caldeyro-Barcia (31) but whether the parasympathetic and sympathetic nervous systems develop functionally in parallel or whether their respective degrees of influence vary at various gestational ages is not known. James and colleagues (32) have recently postulated the use of an intrapartum scoring system in which FHR patterns are attributed weighted scores with a view to correlating the total with the neurological status of the neonate at the time of discharge from hospital; the correlation was high but it was noted that the scoring system was not applicable to fetuses of less than 32 weeks gestation. It was suggested that the most likely reason for that would be the inability of those younger fetuses to respond to hypoxic stress in the same way as older fetuses because of the relative immaturity of their autonomic nervous systems.

The relevance of baseline variability observations to premature

fetuses during labour is therefore unknown.

As one might expect from observations in the neonate and the adult and from the presence in the fetal chest of at least four pressure receptor areas (33), there appears to be a relationship between fetal heart rate variability and fetal chest wall movements (34). The latter have been shown to be related to the state of sleep in fetal lambs, the movements occurring when the lambs are in a state of rapid-eye-movement sleep (35); it is likely, therefore, that baseline variability is similarly affected, variability being greater when the chest wall movements are taking place. Baseline variability in fetal lambs also shows both a considerable diurnal rhythm and a 2-4 hour periodicity and preliminary studies suggest that these rhythms also occur in the human fetus (please see reference 34).

There are, then, several truly physiological variations in baseline variability and, as Dalton indicates in reference 34, these may well prevent much objective assessment of variability until the 'silence' associated with the greater degrees of hypoxia is seen.

Despite the influence of many factors other than hypoxia there does seem to be a usable relationship between normal baseline variability and fetal well-being in terms of fetal pH, or neonatal well-being in terms of one-minute Apgar score. It remains to determine how this relationship may be used in clinical practice and whether it is necessary to study the phenomenon of variability in greater detail or less in order to achieve optimum prediction.

Most authors who have referred to the clinical importance of baseline variability have assessed it by 'eyeballing' (please see Section IX), using a 5-10 minute observation 'window' as shown in Fig. IX:4a on page 154. By this is meant that the observer looks for ten-minute episodes of 'silent' baseline variability but, in reality, he will take into account shorter episodes between 5 and 10 minutes long provided they recur frequently with only short episodes (< 5 mins) of normal variability between them. The graph in Figure X:2 shows the predictive capability of this method when applied to the first-stage traces analysed in Section VI. Although, in theory, an observation

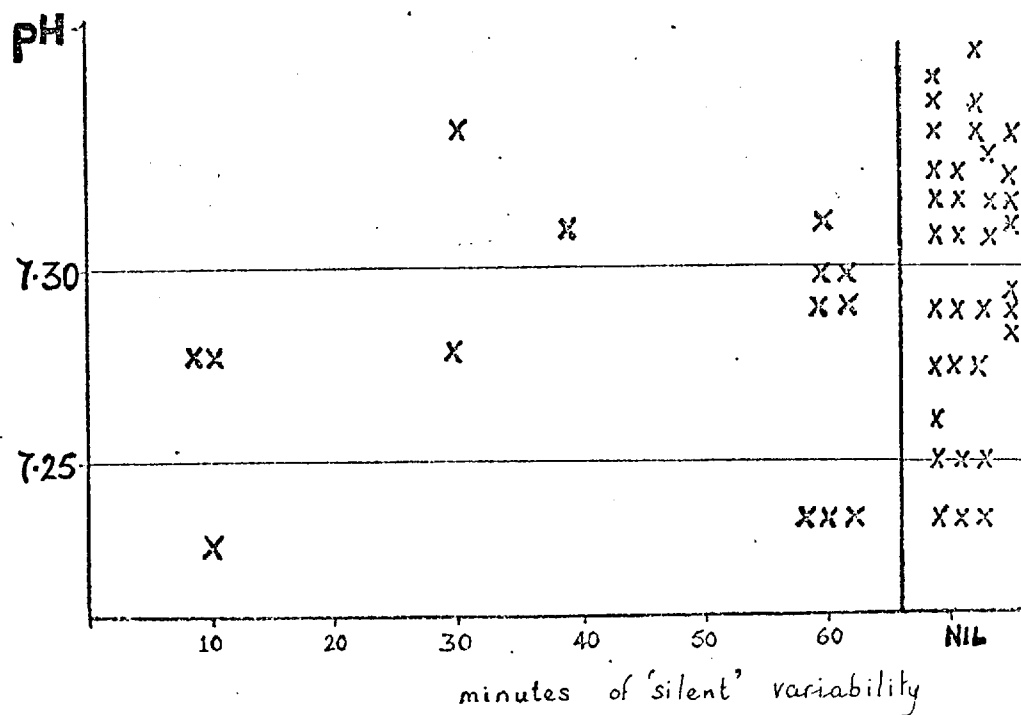


Fig. X:2

Showing the predictive capability, in terms of pH, of a 5-10 minute observation window when applied to the traces analysed in Section VI.

It may be observed that of the seven instances of pH less than 7.25 only four were preceded by 10 or more minutes of 'silent' baseline variability as assessed by 'eyeballing' using a 5-10 minute observation 'window'.

'window' of that size or greater should be highly predictive, since only acidotic or drugged fetuses should show such long episodes of silent baseline variability, this would appear not to be the case.

The window-size used in Section VI was one half to one minute as shown in Fig. IX:4b (page 154). This was the smallest size of window which facilitated physical assessment of long term variation occurring at 2 cycles per minute. Since one of the objectives was to test the possibility of automated screening the question has to be asked whether electronic assessment through a smaller window might give more accurate information. Figure X:3a represents an enlargement of one minute of a fetal heart rate trace. Using the half to one minute window the baseline variability in the first thirty seconds would be 'x' whereas, using a ten to twenty-second window the baseline variability would be 'z', 'y-a' and 'x-b'. The window size determines the degree of low pass filtering so that the larger the window, the lower the low pass filter and the less accurate the information as shown in Figure X:3b. Certainly a smaller window-size would provide more accuracy but whether that would prove more useful in the detection of acidotic or pre-acidotic fetuses, only prospective testing will show. The problem is not so much the improved detection of affected fetuses as the exclusion of unaffected fetuses and in this respect the smaller window-size may be advantageous.

The use of a 'static window' gives only a sample of low pass waveform as shown in Figure X:3b. The use of a 'sliding window', shifting every second, would give a total low pass waveform and thereby provide an immense amount of detail. Since there are 2-6 long term cycles per minute the size of such a window would need to be between 10 and 30 seconds in order to detect as many of the fluctuations as possible.

In view of the need for prospective testing there is in use at University College Hospital at the present time a system of recording supposed beat-to-beat information from human fetuses during labour on FM magnetic tape. The eventual computer analysis will take several forms as outlined above in order to test the different methods and correlate them with neonatal outcome. Although the equipment is designed to accept raw beat-to-beat information the quality of the

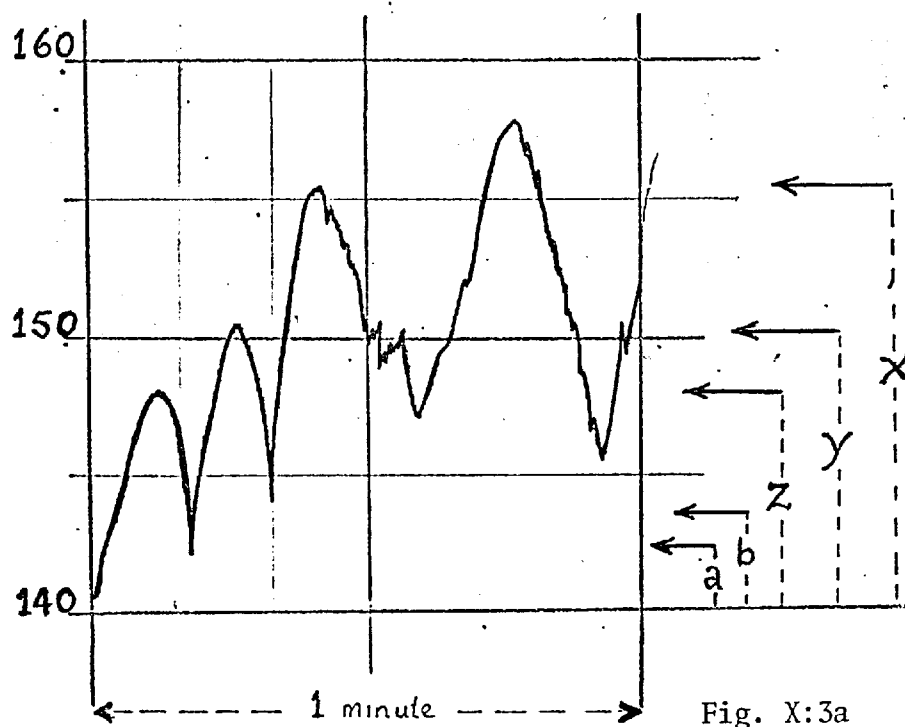


Fig. X:3a

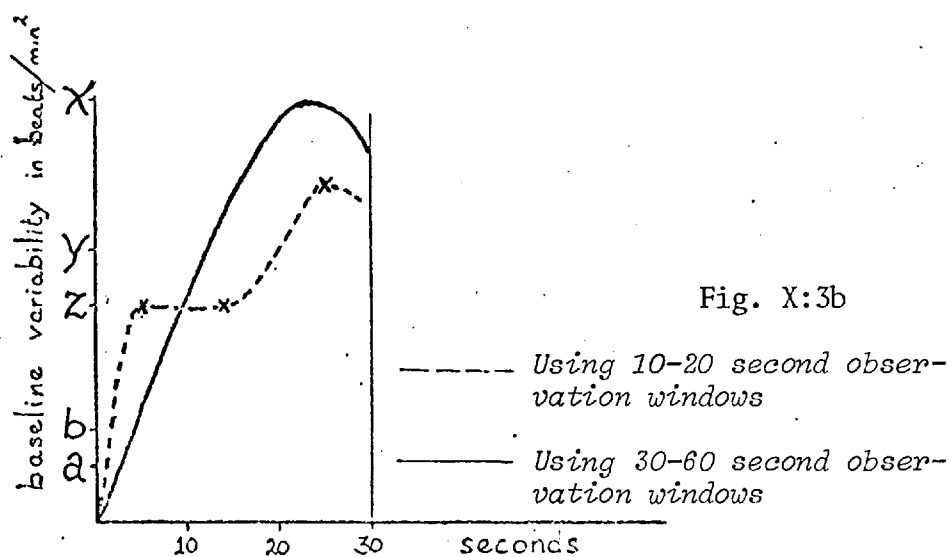


Fig. X:3b

Fig. X:3

Showing on enlargement of FHR trace over one minute (X:3a) and comparing the degree of information obtained regarding variability using a 10-20 second observation window and a 30-60 second observation window respectively (X:3b).

signals may be suspect as they are achieved using routine spiral electrodes (36) and, therefore, may not represent true beat-to-beat data. This will be of little consequence in trying to confirm the results achieved in Section VI since 'eyeballing' using half to one minute windows results in an assessment of long term variation more than it does in an assessment of short term variation, for which more accurate beat-to-beat information is required. A larger number of patients will be required than were available for trace analysis in Section VI. Amongst them there will need to be a significant number of acidotic or pre-acidotic fetuses. These are becoming increasingly difficult to find partly because of improvements in antenatal care resulting in less growth retarded fetuses, less rhesus-affected fetuses and less pre-term neonates and partly because of the intensification of intrapartum care which has taken place in recent years.

It is important that the data collection continues but the role of intensive FHR monitoring in labour may well have a limited future as the next Section shows.

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XI: FUTURE CONSIDERATIONS

Time present and time past
Are both perhaps present in time future
And time future contained in time past.

GEORGE ELIOT

FETAL BIOCHEMICAL STUDIES

Throughout the foregoing discourse the well-being of the fetus has been described in terms of fetal heart rate patterns, fetal scalp blood pH and the neonatal Apgar score. The underlying pathophysiology, however, has been described in terms of fetal oxygenation. Whilst under research laboratory conditions it is possible to measure the oxygen tension and oxygen saturation of fetal scalp blood, in routine clinical circumstances such measurements are limited both by time and by the availability of suitable equipment. Indeed, only three years ago the value of fetal blood sampling even for simple pH measurement was questioned largely because of the delay in obtaining results (1). Fortunately, as an increasing number of labour wards are provided with blood-gas analysers, that excuse for failing to carry out fetal blood sampling is becoming less tenable. The foregoing text has shown that fetal blood sampling for pH measurement is an essential diagnostic tool which warrants the existence on the labour ward of a well maintained and accurate pH meter.

For a long time it has been thought that a continuous assessment of fetal oxygenation would be of considerable value both in research and in clinical obstetrics. Whilst the clinical studies described in this discourse were in progress members of the Department of Medical Physics at University College Hospital (U.C.H.) were investigating methods of continuously monitoring the PO_2 in neonates and it was hoped to apply a similar technique to the fetus provided, of course, that the chosen method was non-invasive. Eventually a method was devised in which intravascular electrodes were utilised (2) so that it was not a method which was applicable to the fetus.

In 1971 Walker and colleagues (3) had described a method of con-

tinuously monitoring fetal scalp tissue oxygen tension throughout labour but they had concluded that simpler, more stable electrodes were required.

In 1973, Murayama and colleagues (4) described the technique of reflection oximetry, a method of assessing the oxygen saturation of surface capillary blood by a fibre-optical reflection technique. Prototype equipment was set up and calibrated in the Department of Medical Physics at U.C.H. It was then tested on the scalps of newly-born infants and eventually used by the author on fetal scalps during labour. Unfortunately, the equipment proved too cumbersome to be acceptable in the limited labour ward space. In addition, difficulties were encountered in maintaining adequate contact between the rather heavy fibre-optic light guide and the fetal scalp for a time of sufficient duration to permit stabilization.

Recently Huch et al. (5) published well-documented but preliminary findings relating to the use of transcutaneous measurements of oxygen tension ($tcPo_2$) in fetal blood using a heated Clark electrode (6). The relationship between fetal heart rate characteristics (using a score) and $tcPo_2$ was examined in detail. There was an overall correlation between the two but it was weak and was considered to be of no clinical usefulness. The FHR/ $tcPo_2$ correlation in individual patients was much stronger which suggests individual idiosyncrasies, perhaps related to acid-base status either at the onset of labour or during labour when it depends very much on maternal breathing patterns, analgesia, nutrition, etc. The authors were able to show a total cause and effect relationship between the reduction of $tcPo_2$ and late and severe variable decelerations. A similar relationship was shown in 83 per cent of the assessed ten-minute periods in which there were slight or moderate variable decelerations; during these periods the $tcPo_2$ levels were less than 15 mm. of mercury. Of particular interest is that similarly low levels of $tcPo_2$ were observed in 11 per cent of the ten-minute periods in which there was either no abnormality or there were early decelerations.

Although there was no demonstrable relationship between $tcPo_2$ and the 'eyeballed' amplitude of baseline variability, there was some

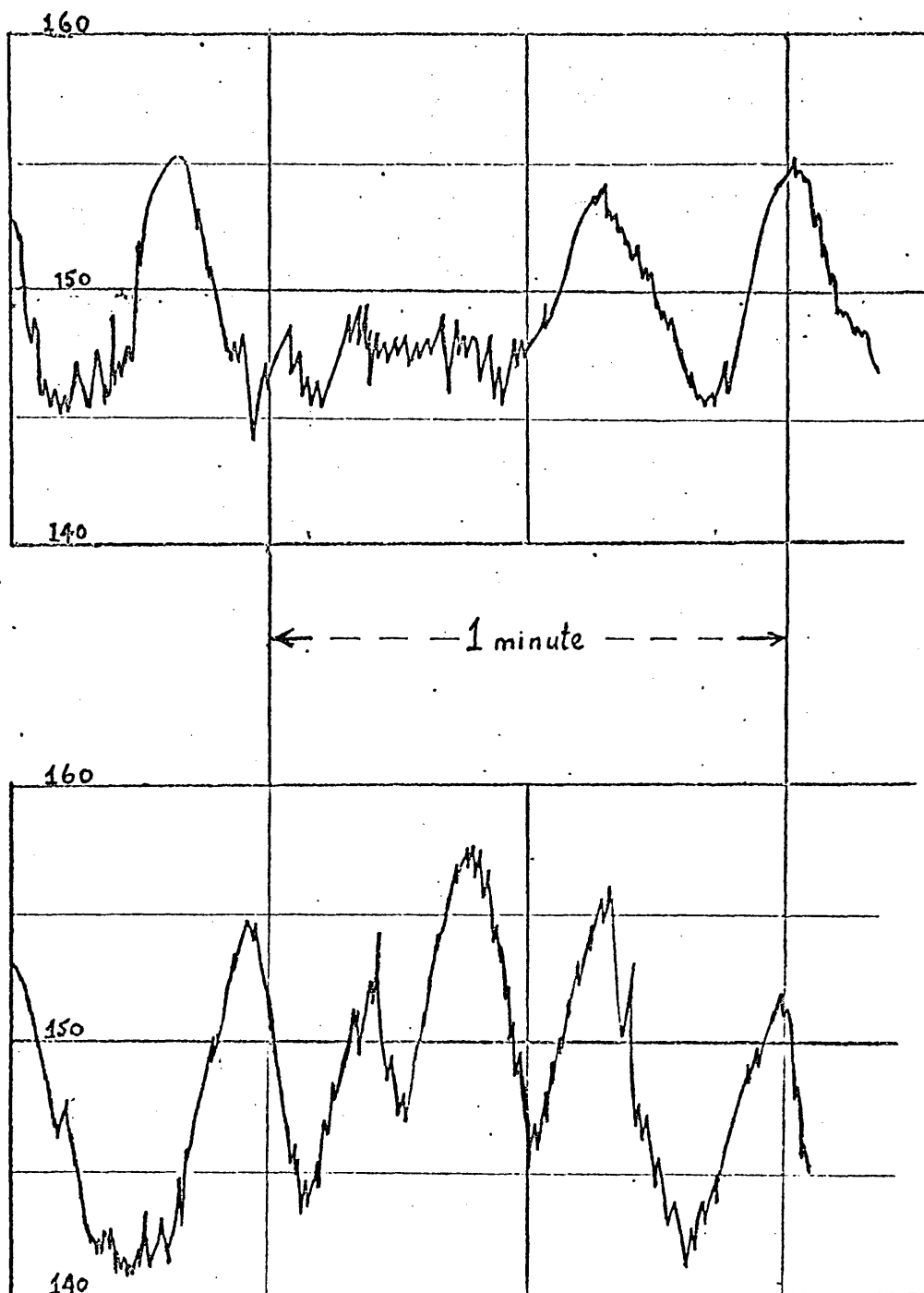
correlation between the $tcPo_2$ and the frequency of long term variability. This is another interesting finding since the half to one minute window used for assessment of variability in the U.C.H. studies is particularly sensitive in the detection of reduced frequency as Figure XI:1 demonstrates. Huch and colleagues quoted several example cases which supposedly demonstrated the lack of correlation between $tcPo_2$ and baseline variability. In most of the quoted examples there was silent baseline variability in the presence of good oxygenation. In other examples there was an increasing amplitude of baseline variability despite falling levels of $tcPo_2$. Previous discussion in Section X has shown that there are many factors which may account for loss of variability in the presence of good oxygenation. The increasing variability seen in some traces whilst the $tcPo_2$ was falling may have been a reflection of an initial response to hypoxia or a response to increasing hypoxia in an already-hypoxaemic but well-compensated fetus. In all but one of the quoted examples there were transient accelerations present so that, in the light of clinical experience, the fetuses either were not hypoxic or they were hypoxic but well-compensated. The relationship between $tcPo_2$ and FHR characteristics obviously warrants very much more investigation. Meanwhile Huch and colleagues suggest, as do the previous Sections of this discourse, that in the event of a suspect FHR trace in terms of variability, a fetal scalp blood pH should be taken or continuous $tcPo_2$ monitoring should be commenced.

The application of the necessary electrode is not without its clinical problems as a small area of fetal scalp has to be shaved prior to application. Nevertheless, the technique has great potential and, although it is unlikely to replace FHR monitoring, it may well augment it in the future. The ideal would be a single electrode complex which could detect the necessary signals to record either FHR or $tcPo_2$, or both simultaneously.

STUDIES OF FETAL ELECTROPHYSICAL PROPERTIES OTHER THAN FHR

Fetal electrocardiography : In 1961 Caughey (7) reviewed the literature on the subject of electronic detection of fetal life; he concluded that the fetal EKG (FEKG) would have great value in the early

A: Reduced frequency - 1 to 2 cycles of long term b.v. per minute reflected by an increase in the number of 30-second episodes of silent variability



B: Normal frequency - 4 to 5 cycles of long term b.v. per minute. No 30-second episodes of silent variability.

Fig. XI:1

Showing how the use of a 30-60 second observation window for the assessment of baseline variability facilitates particularly the detection of a reduced frequency of long term baseline variability

diagnosis of fetal death in utero and of fetal distress during labour. However, he quoted the work of Hon and Hess (8) in which it had been stated that electrocardiographic changes due to anoxia were obtained only minutes before death whereas pathological fetal heart rate changes were seen some hours before death. There have been many papers from a limited number of authors on the subject of FECG changes and many of their observations have been discussed in detail by Lee and Blackwell (9) who also presented their own observations. There is considerable variance between observers and, in view of this, together with the need for sophisticated equipment and highly trained personnel for analysis, the technique has not proved a viable method of monitoring the fetus during labour. However, Symonds (10) is hopeful that fractionation of the various variables and the continuous recording of them will expand the whole concept of fetal ECG monitoring.

The Cardiac cycle : There have been references to the possibility of using the characteristics of the fetal cardiac cycle as a means of diagnosing fetal compromise (11, 12). The technique involves both fetal ECG recordings and accurately placed ultrasonic (Doppler) recordings of fetal heart activity together with complicated equipment so that clinical usefulness is unlikely.

Fetal electroencephalography : Whilst this is a feasible technique (13, 14) it is unlikely to achieve clinical usefulness in the near future mainly because of analytical difficulties. On a research basis, however, it is an important development as it may be used to assess the effect of a drug upon a fetus and once that effect has been determined, the time taken for it to develop may give an assessment of placental transfer and, possibly, of placental function. Similarly, it is likely that fetal EEG changes are commonly related to both fetal chest wall movements and fetal heart rate variability so that fetal electroencephalography may well provide information about these two variables.

ANTENATAL FETAL ASSESSMENT

Much of the material in this discourse has pointed to the importance of the condition of the fetus prior to labour. Antenatal car-

diotocography has been the centre of discussion for over ten years, in which time its clinical acceptance in the United Kingdom has turned full circle. The recent upward trend in its use has been attributed to a better understanding of the observed FHR characteristics together with an increase in monitoring facilities. The antenatal FHR is achieved using Doppler in most instances so that some traces are technically very poor. Electrocardiographic derivation of the antenatal fetal heart rate may prove to be more useful and work is being carried out on this at the present time (15).

As a result of an article by the medical correspondent of the Sunday Times in February 1974 the general public (and some obstetricians) were led dangerously close to believing in the measurement of fetal breathing movements as a totally reliable test in the search for hypoxic fetuses (16). The article was a report on the work of Boddy and Dawes (17) who had recorded fetal chest wall movements using A-mode ultrasonic equipment and had related the observed patterns to the state of the fetus. Using similar equipment to that of Boddy and Dawes, Farman, Thomas (this author) and Blackwell (18, 19, 20) found that the method required such expertise that it was not of practical use. This was not to deny that the movements could be observed or that they might reflect fetal well-being. Since then, other methods have been devised, namely real-time B-scanning (21) and the Doppler shift principle using simple Sonicaid D205 fetal heart detectors (22). Whilst this is still much in the field of research, it is believed that, given an easily used and reliable method of observing fetal chest wall movements, they, together with cardiotocography and observation of fetal movements, will provide valuable information regarding the state of the fetus in the late antenatal stages.

INTRAPARTUM TOCOGRAPHY

This subject was raised in Section IX when the findings of Section VI were being discussed. Currently, in the majority of labour wards uterine activity in labour is monitored using external tocomography. This technique is easy to use but it is uncomfortable for many patients and unbearable for a few. An internal technique would be preferable, therefore. This would also provide more accurate

information regarding lag-times should that information continue to be required. The 'routine' internal method currently used is a saline-filled, open-ended, intra-uterine catheter but this requires a lot of attention and frequent re-calibration as the work in Appendix C suggests. A closed, air-filled system was tested by the author at U.C.H. but problems were encountered with hysteresis and the project was discontinued, at least temporarily. The ideal would be an electronic transducer which is either very cheap and therefore disposable, or is easily sterilised and of a size suitable for insertion into the pregnant uterus.

TELEMETRY AND CENTRAL CONSOLES

The maternal problems of intrapartum fetal monitoring have been alluded to in Section II. There are very few physical or medical complications but there are problems of acceptance by an outspoken minority group. Some of these problems may be overcome by the use of radio-telemetry as described by Flynn and Kelly (23). The technique involves the transmission of signals from a 'maternal' transmitter to a receiver on the cardiotocograph machine or a central console. The system allows patient mobility and facilitates the siting of the receiver and cardiotocograph at any place of convenience within 200 metres. The application of this technique to the recording of intra-uterine pressures has yet to be carried out but the method holds promise.

The advisability of trace assessment being carried out at a place removed from the patient is debatable; there is good argument for all FHR traces being assessed in conjunction with a clinical assessment of the patient and not at central consoles except, perhaps, where the attendant/patient ratio is less than one. Even then, the use of central consoles should not be considered as the answer to such staffing problems.

ALARM SYSTEMS

In 1972 it was stated that alarm systems, designed to attract the attention of an observer to a suspicious trace, had promise (24) but experience with those that have been marketed since that time should be heeded as a warning against them. Satisfactory alarm systems rely on precise definitions and, as the previous text has shown, these do not exist in FHR monitoring. Alarm systems may range from the very simple bradycardia/tachycardia alert to the more sophisticated type which is designed to alert the attention of the observer to both abnormal rates and decelerations of rate. To the author's knowledge there is no commercial alarm system which detects abnormal characteristics of baseline variability. Obviously, in the light of previous discussion, the isolated rate alert is of little use except to detect profound decelerations such as that shown in Figure XI:2 or to detect persistent marked tachycardia which may reflect hypoxia. The normal range of fetal heart rate is not known so that the FHR levels above which and below which the alarm should trigger are difficult to set meaningfully.

One of the more sophisticated alarm systems was tested by the author at U.C.H. The first observation made on this American equipment was that there was no tachycardia alert; evidently this was excluded because fetal tachycardia is so common in the United States (25). Such an arrangement would be unacceptable in the United Kingdom where fetal tachycardia is taken to imply the presence of hypoxia until proved otherwise. The deceleration alert system was designed in such a way that a regular baseline was assumed so that, if the basal rate were 150 beats per minute (b.p.m.) the depth of deceleration required to trigger the alarm would be 30 b.p.m. deeper than the depth required if the basal rate were 120 b.p.m. Obviously the basal rate requires to be tracked by the apparatus so that decelerations of 15 b.p.m. will trigger the alarm regardless of the basal rate. Such tracking may prove to be an expensive electronic exercise but without it the alarm system is next to useless.

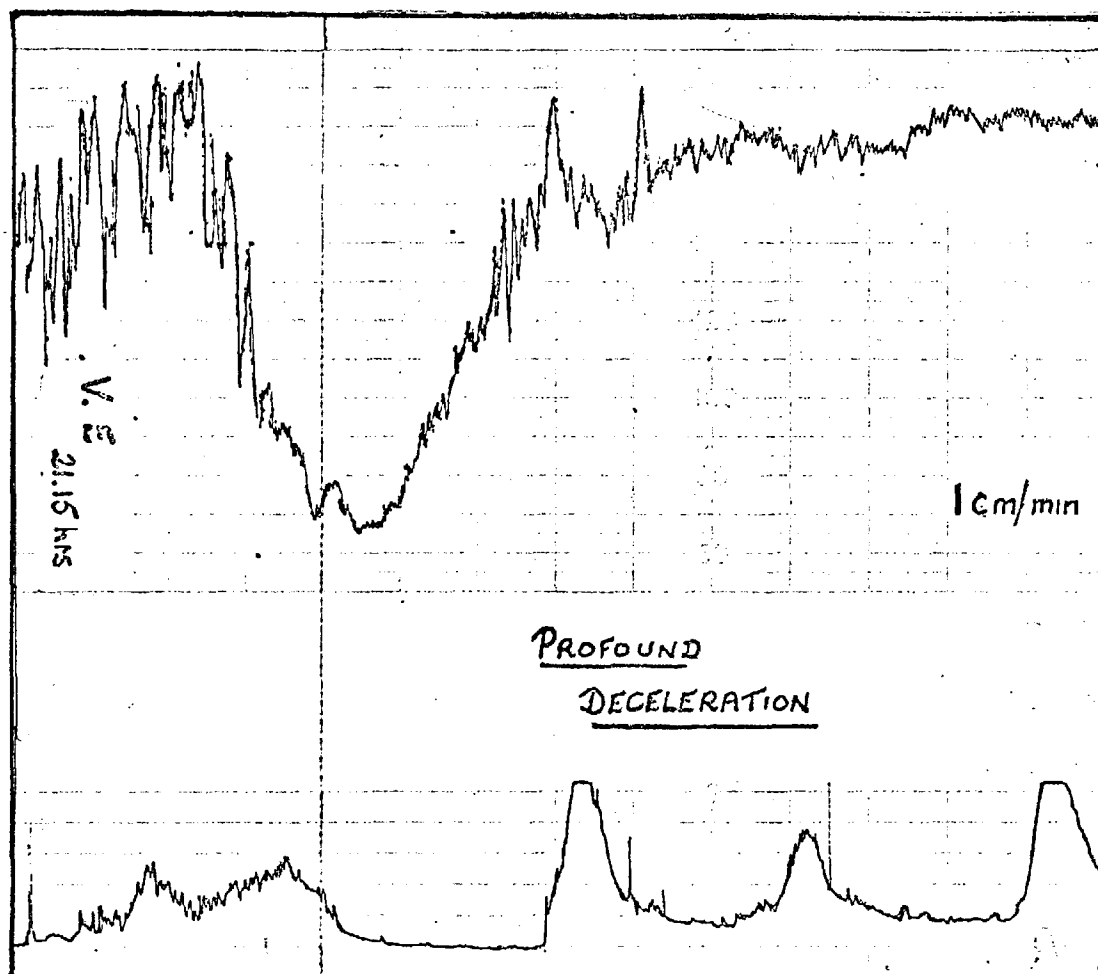


Fig. XI:2

Showing a profound deceleration of fetal heart rate caused, in this instance, by supine hypotension.

TEACHING

Experience is the best teacher of intrapartum cardiotocography and far exceeds the usefulness of alarm systems, once the basic facts have been taught. Experience cannot be imparted so that case discussion with members of the team who are more experienced should take place. Both midwives and doctors should be involved in the 'teach-ins' and both groups should be taught to undertake the necessary technical procedures.

In addition to an 'in-service' teaching programme for staff there should be some reference made at parentcraft classes to the rationale and techniques of intrapartum fetal monitoring. In this way patient acceptance can be improved considerably.

THE IMMEDIATE FUTURE

The most desirable and likely developments in the immediate future are the increasing use of antenatal fetal heart rate and fetal movement monitoring and the increasing availability of accurate diagnostic ultrasound in order to reduce the incidence of undiagnosed growth retardation and its consequences. The use of an intrauterine transducer to record uterine contractions and the use of telemetry to transmit those recordings and the FHR signals to a recorder, removed from the bedside but within the labour room, would improve maternal acceptance of the techniques. There should be facilities on all labour wards for the taking of fetal blood samples in the lateral position and for measuring the pH. It may not be long before continuous $tcPo_2$ monitoring will be available for 'at risk' cases in which a doubtful FHR indicates biochemical investigation of the fetus. The diagnosis of a doubtful FHR has been discussed in great detail in this discourse. The author believes that for inexperienced personnel the régime shown in Appendix D should be used in order to ensure absolute efficiency in the search for acidotic and/or hypoxic fetuses. As the experience of each individual increases so then will the incidence of biochemical investigation by that individual be reduced. The author is hopeful that automated analysis of baseline variability of heart rate may be used to reduce the initial high incidence of biochemical

investigation.

WHO SHOULD BE MONITORED

Certainly all patients included in any of the categories listed in Table IV:1 should be monitored as they are at greatest risk of delivering acidotic or hypoxic babies. In view of the high incidence of undiagnosed growth retardation there is an argument for monitoring all patients. Premature fetuses should be monitored as the results suggest that they show greatest benefit as a group.

Continuous fetal heart rate monitoring for all would improve the acceptance of the technique; it is likely that a patient of Lejumeau would have been as astonished at the sight of a stethoscope upon her abdomen as some patients are today at the sight of a cardiotocograph!

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CONCLUSIONS

The major conclusions from the original work and the literature review are here summarised.

Many authors have found difficulty in applying accepted classifications of fetal heart rate (FHR). The aim of this discourse was to simplify these classifications, to seek alternative diagnostic information, to determine the role of fetal blood sampling and to ensure justification for the continued use of intrapartum cardiotocography.

Either intrapartum cardiotocography or the associated intensive care has brought about a reduction in intrapartum stillbirths and it seems that only one instance of severe spasticity need be prevented in 10,000 deliveries in order to provide economic justification. The incidence of complications is much less than one per cent and may be reduced further by careful postnatal observations when infection is suspected. The importance of checking clinically both the fetal heart rate and the state of uterine contractions has been stressed; this will help prevent complications due to misunderstanding.

The safety of long term Doppler monitoring of the fetal heart has yet to be completely proved in view of the risk of damaged transducers. Nevertheless the risk is so small as to warrant the monitoring of all premature fetuses so that they may be delivered in an optimal state. All breeches and all second twins should be monitored. Cardiotocography should be started as early as possible in labour.

Maternal supine and lithotomy positions should be avoided whenever possible; if unavoidable, then a wedge should be used.

A significant relationship between the use of epidural analgesia and the appearance of late decelerations has been shown. However there is no evidence that epidurals are harmful. In animals late decelerations are always due to hypoxia but in humans only 50 per cent of late decelerations are associated with fetal acidosis. There is no method of distinguishing the acidotic from the non-acidotic so that fetal blood sampling is essential as a final arbiter.

Not all early decelerations are innocuous. Some may represent levels of oxygenation which are lower than those associated with late

decelerations. Lag time has been shown to fall as hypoxia worsens. A normal fetal heart rate does not exclude fetal acidosis.

Cyclic changes in response patterns occur through labour. The major responses show trends with transient accelerations tending to occur in early labour. Early decelerations occur at some time in almost all labours. A sharp increase in the appearance of decelerations occurs about 90 minutes before delivery. In low Apgar fetuses transient accelerations are less prevalent and decelerations tend to occur earlier, episodes of silent baseline variability and tachycardia are significantly more common as are the respective incidences of late decelerations, block decelerations and the progression: transient acceleration to pre-acceleration. Fifteen minute segments of transient accelerations, early decelerations and late decelerations occur with equal frequency in the high and low Apgar groups. Rebound accelerations seem to carry no significance.

Although dip area in the second stage correlated well with Apgar score there was no demonstrable dip area/pH relationship in the first stage; similarly lag time appeared important in the first stage but not in the second. Different physiological conditions may pertain in the second stage.

Baseline variability may be used to screen out low pH fetuses. However, because it is affected by drugs, physiological rhythms and prematurity, it provides only a crude screen. Its relevance to fetuses of less than 32 weeks gestation is unknown. It seems that reduced frequency of long term variability may be indicative of acidosis so that fractionation of baseline variability information may be important. Saltatory baseline variability probably reflects mild hypoxia.

There is increasing evidence that the ante-natal condition of the fetus is ultra-important. FHR patterns may depict hypoxia but they cannot reliably indicate the degree of fetal reserve.

APPENDIX ATHE COST-EFFECTIVENESS OF INTRAPARTUM FETAL HEART
RATE MONITORING

In Section II reference was made to the relatively negligible cost of setting up and maintaining an intrapartum fetal heart rate monitoring service when compared with the cost of caring for a handicapped person. This was implied by Beard in 1974 (1) and shown by Quilligan and Paul in 1975 (2). A costing exercise carried out by the author at University College Hospital, London in 1974 gave similar results to those of Quilligan and Paul. Since that time, in Great Britain, inflation rates of between 10 and 20 per cent have led to a near-doubling of the cost of caring for handicapped persons. Official statistics now suggest that the total, lifetime cost of severe handicap in one person is between 150,000 and 200,000 pounds sterling (3).

The increasing acceptance of fetal heart rate monitoring during the last four years has enabled the manufacturers of monitoring equipment to maintain stabilised machinery costs so that the overall cost of intrapartum fetal heart rate monitoring has risen at a much slower rate than that of handicap.

Table I is an updated summary of the approximate cost of an intrapartum fetal heart rate monitoring service in a maternity unit in which there are 2,000 deliveries per year. It assumes a monitor requirement of 2.5 per thousand patients for a high-risk monitoring policy (U.C.H. statistics) and 3 per thousand patients for a total monitoring policy in which an attempt is made to monitor all fetuses during labour (St. Mary's Hospital, London statistics) (4). The costs of the two policies are compared. Although in a high risk policy only 30-50 per cent of the patients are monitored, the cost of a total monitoring policy does not rise in proportion, mainly because the majority of high risk labours are planned to take place during the day, leaving the monitors idle at night.

Obviously the cost of a 'monitoring' service depends on many local factors and divisional policies. The costing in Table I is based

TABLE I : QUINQUENNIAL COSTS OF AN INTRAPARTUM FETAL HEART RATE MONITORING SERVICE FOR 2,000 DELIVERIES PER YEAR.
December 1977.

Item	Cost per Unit	HIGH RISK CASES ONLY say 50 per cent		ALL CASES	
		No. of Units	Total Cost/ 5 Years	No. of Units	Total Cost/ 5 Years
	in pounds sterling		in pounds sterling		in pounds sterling
<u>FHR</u>					
CARDIOTOCOGRAPH	4,000	5	20,000	6	24,000
REPLACEMENT CTG.	4,000	1/Year	20,000	1/Year	20,000
ELECTRODE APPLICATORS	13.50	25	338	50	675
REPLACEMENT APPLICATORS	13.50	5/Year	338	10/Year	675
ELECTRODES	1.00	1/Patient	5,000	1/Patient	10,000
PAPER	1.95	1/2 Patient	4,875	1/2 Patient	9,750
SUNDRIES	1.00	1/Patient	5,000	1/Patient	10,000
<u>FBS</u>					
pH METER	upto 4,000	1	4,000	1	4,000
FIBRELIGHT SYSTEM	upto 500	1	500	1	500
FBS KIT	20.00	25	500	25	500
REPLACEMENT KITS	20.00	5/Year	500	5/Year	500
<u>STAFF</u>					
TECHNICIAN	upto 4,000/yr	1	20,000	1	20,000
MIDWIFERY SISTER	2,000/yr	Part-time	10,000	Part-time	10,000
<u>COMMERCIAL SERVICING</u>					
CARDIOTOCGRAPHS	40.00	26/Year	5,200	26/Year	5,200
pH METER	40.00	12/Year	2,400	12/Year	2,400
		TOTAL:	98,651	TOTAL:	118,200

on the assumption that a scalp electrode is applied to each monitored fetus and that one cardiotocograph in five is replaced each year. The costing also allows for the monthly maintenance of a pH meter and for two-weekly visits by commercial technicians to solve machinery problems, although the provision of a medical physics technician should minimise such commercial costs. In addition to such a technician there should be, in each maternity unit, one midwifery sister whose duties include the supervision of the monitoring service.

The Spastics Society, London estimates that the incidence of spasticity is approximately 2.5 per thousand live births, so that twenty-five such affected babies would be expected from an obstetric unit in which there were 10,000 monitored deliveries in five years. Table I shows the cost of setting-up and maintaining an intrapartum 'monitoring' service for five years in such a unit is approximately 118,000 pounds sterling, less than the cost of caring for just one handicapped person through a lifetime. Therefore, despite the unknown relationship between the use of intrapartum fetal heart rate monitoring and the prevention of spasticity, there appears to be economic justification for such a monitoring service even if it is responsible for preventing only one instance of handicap in 10,000 babies.

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APPENDIX B

The Safety of Diagnostic Ultrasound

The following paper is an updated and re-edited version of a paper submitted to the Ethics Committee at University College Hospital, London in 1973, prior to a proposed ultrasonic investigation into fetal breathing movements and their relation to fetal heart rate. At that time the submission represented a reasonable review of the literature concerned with the safety of diagnostic ultrasound but there has been an increasing interest in the subject and there are, currently, at least 1,027 known references (1). Despite this, there exist very few certain facts regarding the safety of diagnostic ultrasound in pregnancy and, particularly, in early pregnancy.

Ultrasound, as applied in the medical field, is a form of longitudinal wave motion of a frequency above the range of normal animal hearing. 'Therapeutic' ultrasonic energy intensities are known to have biophysical properties which can cause damage to animal tissues. 'Diagnostic' ultrasonic equipment generates a very low energy intensity, the safety of which has been the subject of many thorough investigations over the last decade.

Two types of ultrasonic wave fields are used for diagnostic purposes :

- 1) Pulsed fields, in which the energy is emitted in very short, regular bursts. The energy is reflected from tissue interfaces and the resulting echoes are displayed on an oscilloscope screen either as an 'A-scan', which may be used for measurements, or as a compound 'B-scan' which gives a two-dimensional representation of the structure under investigation.
- 2) Continuous fields which may be used to detect movements such as the flow of blood within a vessel, by the Doppler frequency shift principle.

The biophysical effects of high intensity ultrasound may be a) thermal, due to energy absorption, b) cavitational, caused by resonance phenomena in homogeneous tissues, or c) 'direct' action, causing enzyme inactivation, degradation of macromolecules or cell membrane damage by mechanisms which, at present, are not completely understood. The aim of relevant research during the last five to ten years has been to ensure that low intensity ultrasound does not have these biophysical effects and to define the threshold below which insonation may be considered safe.

The amount of damage caused by ultrasonic irradiation at therapeutic intensities is inversely proportional to the frequency of the wave and probably directly proportional to the time of exposure and to the mean intensity of the wave-field, which is expressed in Watts per square centimetre.

The intensity of a continuous wave field is constant but the exposure is continuous and of a duration limited only by the operator. Pulse-echo systems utilise higher peak intensities but the duration of each pulse is of the order of a microsecond and is determined by the design of the generating equipment. The average intensity of the 'diagnostic' pulsed field is of the same order as the constant intensity of the diagnostic continuous field system and is determined by the pulse repetition frequency, the pulse duration and the peak intensity.

These observations relate to levels of intensity at the body surface but the amount of energy dissipated in the tissues being studied depends on absorption and attenuation, the degree of focusing of the ultrasonic beam and the amount of movement of the target organ.

Table I outlines the field intensities produced by diagnostic equipment in routine use at University College Hospital in 1973/1974.

Investigations into the safety of diagnostic insonation have taken the form of animal experiments, in vitro experiments with cell cultures and prospective and retrospective studies in pregnant women.

TABLE I

Field intensities produced by diagnostic equipment
in routine use at University College Hospital in 1973/1974

EQUIPMENT pulsed/ continuous	USE	MEAN INTENSITY in mWatts per sq. cm.	PEAK INTENSITY in Watts per sq. cm.	PULSE REPETI- TION FREQUENCY	TRANS- DUCER RESONANT FREQUENCY in MHz
DIASONOGRAPHY pulsed	Cephalometry A & B scan- ning in preg-	approx 2.0* (max)	40* (max)	600 (fixed)	2.50 (selec- ted)
EKOLINE pulsed	A-scanning of fetal breath- ing movements	10.0** (max)	unknown	1,000 (fixed)	2.25 (fixed)
MODIFIED SMITHS INDUSTRIES pulsed	A-scanning of fetal breath- ing movements	approx 0.1*	20* (max)	50 selected	2.50 selected
SONICAID FM ₂ continuous	Continuous fetal heart rate monito- ring (Doppler)	5.00	-	-	2.00 (fixed)
SONICAID D205 continuous	Fetal heart detection in early preg- nancy (Doppler)	16.0	-	-	2.00 (fixed)

*The figures asterisked thus are based on experimental, electrical measurements and do not take into account the inefficiencies of the transducer and coupling arrangements. Acoustic measurements are difficult to perform and, as a rule, are not supplied by manufacturers.

**At the time of writing this figure is in doubt. The value is certainly no more than 10 mWatts per square centimetre. The English representative was unable to supply the correct value. The figure of 10 mWatts per square centimetre is quoted from Boddy, K. and Mantell, C.D. (The Lancet 2, 1219, Dec. 1972).

The earliest studies were directed toward detecting gross structural damage to organisms, genetic damage and behavioural changes. Smyth exposed rats and mice to routine diagnostic insonation and found no deleterious effects (2,3), whilst Andrew insonated perch and frog spawn and found no evidence of teratogenesis in the progeny (4). Woodward and Warwick insonated pregnant mice with intensities far in excess of those encountered in diagnostic applications; they found no evidence of damage either in the mother or in the first and second generations (5). A combined clinical trial carried out in New York, Glasgow and Lund and involving 1,114 pregnancies did not show any increase in fetal abnormality following the use of diagnostic ultrasonic equipment with a maximum average intensity of 10 mW per square centimetre (6). Until 1970, then, no adverse effects of diagnostic insonation had been observed.

A report by Macintosh and Davey in 1970 suddenly shattered the sense of security regarding diagnostic ultrasound. Using 'Doptone' fetal heart detectors with continuous intensities of 17 mW and 8 mW respectively to insonate lymphocyte cultures in Tc199, these authors were able to produce a significant number of chromosomal breakages (7). In the same year Serr reported chromosomal breakages in insonated fibroblasts from amniotic fluid (8). By 1971 Coakley and Hughes had repeated the experiments of Macintosh and Davey and had obtained similar results, noting, however, that the chromosomal breakages were not observed if the lymphocytes were cultured in whole blood nor if the container material was changed from the original polyethylene to metal foil. The authors felt that the persistence of the effect made ultrasonic energy an unlikely cause unless the first mitosis had been delayed. The possibility was raised of a chemical agent being released from the polyethylene container (9). Macintosh and Davey continued their in vitro experimentation and in 1972 they reported an ultrasound-dose/response curve for chromosomal damage with a threshold intensity of 8.2 mW per square centimetre. The experiments showed that prolonged exposure of the lymphocyte cultures to polyethylene, without insonation, led to a significant number of chromosomal breakages but the authors were unable to demonstrate the release of toxic factors from the polyethylene during a short exposure to ultrasound (10). In 1972 Coakley reported that he and his colleagues had found

no reason to change their original conclusions (11). They had noted, however, that there was a high incidence of chromosomal aberrations even in the control series and that the aberrations visualised were achromatic gaps which, according to studies on plant chromosomes, are temporary, reparable lesions and not true chromosomal breakages (12). Looby Watts and Hall have pointed out that an insult which causes damage to the genetic structure of a cell brings about chromatid damage only if it is applied after D.N.A. Synthesis (13). Close inspection of Macintosh and Davey's results shows that some of the aberrations involved chromatids despite the fact that insonation was carried out before culturing and hence before D.N.A. Synthesis. This again suggests the role of another more persistent factor than ultrasonic energy as a cause of damage.

Looby Watts used a wide variety of routine ultrasonic energy generators to insonate lymphocytes from healthy volunteers during incubation in polythene containers. There was no difference between the insonated and the control cultures. Bobrow and colleagues insonated lymphocyte cultures with energy intensities varying from 14 mW to 2.77 Watts per square centimetre and found no adverse effects (14) whilst Boyd's team failed to demonstrate any genetic damage after 4-13 hours of continuous insonation at 20 mW per square centimetre or after pulsed insonation with a 1.5 MHz Diasonograph transducer. Both the continuous and the pulsed fields were applied at varying stages of the cell cycle (15). Boyd also studied the blood cultures of six infants who had been exposed to diagnostic ultrasound in utero and, like Fischer, who studied cultures of insonated cord blood (16), was unable to demonstrate any chromosomal damage. The contradiction between these results and those of Macintosh and Davey together with the findings of Coakley point increasingly toward there being an experimental factor involved.

The results of Serr have been disputed by Abdulla and his colleagues (17) and by Looby Watts and Stewart (18); indeed, Serr has only followed up his initial report with contradictory results (19). In order to detect chromosomal damage in insonated cells it is necessary to study them after the first mitosis, otherwise artefacts may occur. Cells from amniotic fluid need to be cultured through several

generations before they can be studied and Serr's original findings, therefore, are invalid.

By exposing the spinal cords of adult rats and subjecting them to pulsed ultrasound with peak intensities of 25 or 50 Watts per square centimetre and output frequencies between 0.5 MHz and 6 MHz it is possible to produce gross haemorrhage and, in some cases, paraplegia (20). The damage observed is maximal at the lowest output frequency and decreases in amount as the frequency increases. No damage is observed when the frequency exceeds 5 MHz. Taylor and Pond, who have made these observations, consider them to be of no significance in diagnostic applications except, perhaps, in the event of technical malfunction or adventitious focussing in bony cavities. The latter is thought not to apply to fetal monitoring as there are no bony cavities between the transducer and the fetus; however it is possible that the curve of the sacrum may exercise a focussing effect on reflected beams.

There are phenomena other than chromosomal aberrations and direct damage which have been observed as a result of insonation. Selman and Counce first reported the effect of streaming in developing embryos and suggested that this may cause fetal abnormality (21). More recently Dyson and her colleagues have observed that particulate matter, suspended in saline, aggregates in vertical planes at right angles to the direction of insonation. Similarly red blood corpuscles in the blood vessels of a chick embryo form sharp-edged aggregates which are temporary and disperse when insonation ceases (22). Streaming phenomena have only been observed as a result of standing wave formation due to the interference of forward and reflected components of high intensity continuous fields. Dyson used an intensity of continuous insonation in the order of 40 Watts per square centimetre. However, using pulsed ultrasonic energy with a peak intensity of only 500 mW/cm², she demonstrated tissue regeneration in experimentally produced defects in rabbits' ears (23). This effect was paralleled by an increased uptake of tritiated thymidine which indicates increased nucleic acid synthesis. Subsequent ultrastructural examination of material subjected to this treatment showed damaged lysosomes.

A review of the literature in 1973 showed that, according to circumstantial evidence, there were no apparent dangers associated with diagnostic ultrasound but there were theoretical dangers demonstrated by animal and 'in vitro' experiments. These possible dangers included chromosomal aberrations caused by continuous ultrasonic fields, direct damage to tissues caused by technical malfunction, lysosomal damage caused by pulsed ultrasound and streaming phenomena, including red cell aggregation, in developing embryos under accidental high intensity, continuous standing-wave conditions.

The situation is much the same in 1977. The circumstantial evidence in favour of diagnostic ultrasound is obviously much greater and it is of considerable interest that Macintosh has reported his inability to reproduce his original alarming results now that he has altered his staining technique and has used a blind culture scoring system (24). However, there has come to light the work of Japanese investigators who report an increased incidence of congenital abnormalities in inbred white mice after insonation over 6 hours with a Doppler device (25). Not unnaturally it is sensational results such as these which are quoted, without critical appraisal, by the lay-press (26), thus leaving clinicians and physicists with some very difficult questions to answer. The work of Mannor and Serr (see ref. 19) was similar to that of the Japanese group but the results were dissimilar. An increased incidence of abnormality was noted but only at intensities in excess of 1500 mW/cm^2 when they were adequately explained in terms of thermal changes. Nevertheless the Japanese results still cause concern as they are inexplicable; they could be due to excessive inbreeding of the mice or to relative hypoxia in the insonated embryos but this remains to be proved. The concern was barely counteracted by the general statement made in 1976 by the Committee on Bioeffects of the American Institute of Ultrasound in Medicine (27). It read as follows :

"In the low megahertz frequency range there have been, as of this date, no demonstrated significant biological effects in mammalian tissues exposed to intensities (spatial peak, temporal average, measured in free field of water) below 100 mW/cm^2 . Furthermore, for ultrasonic exposure times (including off-time as well as on-time for a repeated pulse regime) less than 500 seconds and greater than 1

second, such effects have not been demonstrated even at higher intensities, when the product of intensity and exposure time is less than 50 J/cm^2 ."

In parenthesis the committee observed that most of the data applied to mammals other than man and that the information concerning the effects of low intensities of ultrasonic energy with long exposure times and also repeated short pulses was scant. As it is these latter types of ultrasonic energy which are most used in obstetric diagnosis there remains a need for more information.

Taylor reviewed the current status of toxicity investigations in 1975 (28); he re-iterated the difficulty of applying results of animal experiments to human populations and stated that the ultimate criteria must be an extensive epidemiological survey of a scale similar to the one required to quantitate the long-term hazards of ionising radiation (29). He stated that the unique results obtained with diagnostic ultrasound demand its use before toxicity data are complete, a view expressed more recently by Wells (30).

One of the major problems with extensive epidemiological surveys is the standardisation of machine specifications. Currently manufacturers quote specifications as field intensities which are averaged over both space and time. The distribution of intensity may be modified by tissue attenuation, deliberate focussing within a transducer or accidental focussing by tissue planes and the possibility of a major redistribution of intensity within a damaged transducer, resulting in a modified beam profile or an ultrasonic hot spot, cannot be discounted (31).

An additional problem in the research field is the difference between 'in vitro' and 'in vivo' conditions as brought out by the recent discussion concerning the insonation of D.N.A. in solution. It appears that therapeutic intensities (1.5 W/cm^2 , 1.0 W/cm^2 and 200 mW/cm^2) cause breakdown of D.N.A. molecules in solution (32). Although these intensities are not used for diagnostic purposes, 200 mW/cm^2 is only ten times the maximum diagnostic intensity and the

margin of safety, therefore, is quite small. However, Thacker points out that these effects are caused by cavitational phenomena and that reproduction of these phenomena within tissues would require much higher intensities which would cause cell death as well as D.N.A. degradation (33). This contention is disputed by Galperin-Lemaître (34) who quotes Hill when he suggests that ultrasound can produce biological responses directly (35). Prasad et al maintain that insonation inhibits D.N.A. synthesis directly and they express the need for further investigation (36).

It is impossible to draw conclusions from the aforementioned information. The current situation has been summarised concisely by P. N. T. Wells who states that "clearly there are insufficient data to state with any certainty that ultrasound diagnosis is 'safe', but it is certainly justifiable when the information which it might provide could help in the care of the patient" (30).

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Appendix C

A CONTROLLED TRIAL OF THE CARDIFF AUTOMATIC INFUSION SYSTEM IN THE MANAGEMENT OF INDUCED LABOUR*

The concept of oxytocin titration in the stimulation of labour was introduced by Turnbull and Anderson in 1968,¹ after which time a method of automated, controlled infusion was sought. The Cardiff automatic infusion pump was originally reported in 1970²; it was described as an accurate, simple-to-use piece of equipment which left the attendant free to observe and care for the patient. The low volume of fluid required to deliver the necessary quantity of oxytocin renders the equipment especially safe for patients with hypertension, preeclampsia or cardiac disease. The results suggested that the induction-delivery interval was shortened by the use of the automatic pump. Since that time an automatic system has been developed in which the pump is regulated by the amniotic fluid pressure by means of a closed loop feedback control unit.

The Cardiff automatic infusion system consists of two units. Both of these, the infusion pump and the control unit, have been assessed by obstetric personnel and medical physicists at University College Hospital at the request of the Department of Health and Social Security and full reports were submitted there in 1972 and 1973. Since these reports several features have been altered in order to improve the safety of the system.

During its initial trials at University College Hospital the fully automatic system failed to impress the midwifery staff partly because of a high false alarm rate, partly because of an apparent relationship between the use of the system and fetal distress and partly because of an apparent prolongation of labour. A small controlled trial was set up to compare the use of the automated infusion system with a conventional infusion system utilizing an Ivac 501 drip counter.³

*Published in the British Journal of Clinical Practice Vol.28 No.6 p.203, June 1974. Co-author : R. J. Blackwell M.Sc., M.Inst.P.

The Trial

The trial involved 36 patients in their first or second pregnancies who were delivered at University College Hospital between January 1973 and August 1973. In each case labour was induced by forewater rupture of the membranes followed by the administration of oxytocin within 2 hours of amniotomy. Before amniotomy each patient was allocated at random to either the study group or a control group by the selection of an unmarked envelope. At the time of amniotomy a saline filled Portex intra-amniotic catheter was inserted and a scalp electrode applied to the fetal scalp. In the study group the intra-uterine catheter was connected to a Sanborn 267AC pressure transducer which was adjusted to the level of the symphysis pubis and connected to the Cardiff automated infusion system, which, with the system open to the atmosphere, was adjusted to read zero pressure. Pressure readings and the continuous fetal heart rate, derived from the fetal ECG, were recorded on a Sonicaid FM₂ monitor. The infusion was started at 2mU/min and the system was then allowed to operate automatically. In the control group the intra-uterine catheter was connected to a Hewlett-Packard pressure transducer (HP 1280) and the pressure readings and fetal heart rate were recorded on a Hewlett-Packard monitor (HP 8025A). Oxytocin ('Syntocinon') was infused at 2mU/min using an Ivac T.M.501 electronic drip counter. The infusion rate was increased by 2mU/min every 15 minutes. In both groups one litre of 5% dextrose was kept in reserve and connected to the intravenous infusion system via a Y connector. Fig.1 compares the respective rates of increase of oxytocin dosage in the 2 groups.

In the event of possible fetal distress being indicated by an abnormal fetal heart trace, fetal blood sampling was carried out. The state of each newborn infant was assessed using the Apgar scoring system.⁴

Minor modification to the Cardiff infusion system

Before commencing this trial the system had been used as it was supplied by the manufacturer. The spasm alert level of 30mm. Hg. was considered too high and was therefore adjusted to the

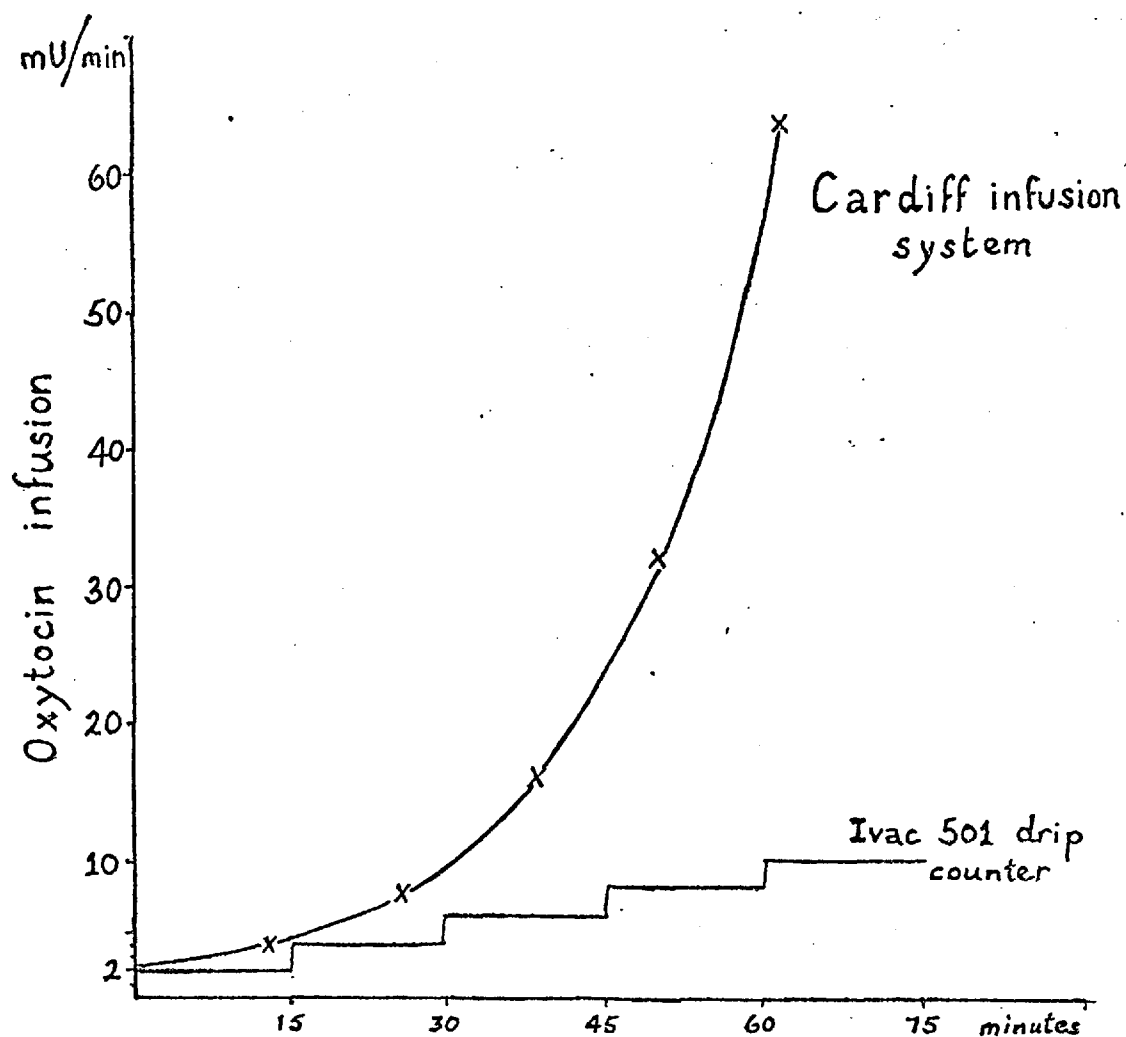


Fig. I (Appendix C)

In which are compared the respective rates of increase of oxytocin dosage using the Cardiff automatic infusion system and an Ivac 501 drip counter operated manually.

lowest possible level of 23mm.Hg. No other adjustments were made.

Results

There were 25 primigravid patients and 11 patients with one previous viable pregnancy. The Cardiff system was used for 17 patients and the control system for 19 patients. For the purpose of more meaningful comparison it was possible to match 15 pairs of patients with regard to age, parity, indication for induction and, in 9 pairs, the state of the cervix at induction using centimetres of dilation and degree of effacement.

Duration of ruptured membranes and duration of labour :

Of the 15 matched pairs, there were 5 in which one or both labours were terminated by Caesarean section. In the remaining 10 pairs the mean length of ruptured membranes in the Cardiff group was 10.6 ± 4.1 hours compared with 7.8 ± 2.0 hours in the controls. Taking the onset of labour as the time at which a rise in the intra-uterine pressure occurred at least once every 5 minutes, the mean length of labour in the Cardiff group was 9.3 ± 4.1 hours compared with 6.1 ± 2.2 hours in the controls. The differences between these 2 parameters both reach statistical significance ($P < 0.05$) using "t" tests for both matched and unmatched data. If the onset of labour was taken as the time at which an intra-uterine pressure rise of 35mm.Hg. or more occurred at least once every 5 minutes, then the respective mean lengths of labour in the Cardiff and control groups were 7.8 ± 3.6 hours and 5.4 ± 2.9 hours. This difference does not quite reach statistical significance ($P < 0.10$). Stricter matching of the pairs for absence of either an epidural or vibration in the management of labour, in addition to the factors mentioned previously, reduced the comparison to 4 pairs in which the duration of ruptured membranes was longer by a mean of 5.1 hours (S.D.= 3.3) and the duration of labour was longer by a mean of 5.6 hours (S.D.=3.9) in the Cardiff group.

The mean time interval between amniotomy and the commencement of infusion was similar in both groups of 15 patients, being 1.3 ± 0.5 hours in the Cardiff group and 1.2 ± 0.4 hours in the controls.

Similarly, the time interval between the commencement of infusion and the onset of labour was hardly different, being respectively 0.3 ± 0.4 hours and 0.4 ± 0.7 hours in the Cardiff group and the controls.

Method of delivery :

There were 6 forceps deliveries in the Cardiff group, of which only one was for apparent fetal distress and 4 were for persistent malposition. Four of the controls were delivered by forceps and vacuum extraction, none for fetal distress and one for persistent malposition. There were 3 Caesarean sections in each group. All 6 were indicated primarily by the failure of the cervix to dilate. In the Cardiff group one was an elderly primigravida and 2 were young primigravida. All 3 control patients were elderly primigravidae. There was evidence suggestive of fetal distress in each of the 6 cases but the lowest pH measured was 7.25. Table I indicates the details of labour and delivery of the 30 matched patients and gives an assessment of the most efficient method in each pair based on the length of labour, the method of delivery (Caesarean section versus vaginal delivery), and the influence of other variables.

Fetal wellbeing :

Fetal heart rate patterns indicated possible hypoxia in 4 cases in each group of 15 patients. Distress was proved in one instance in the Cardiff group and was borderline in a second (7.25). There was no proven distress in the control group.

There were 4 low Apgar scores in the Cardiff group, 2 following Caesarean section, one following forceps delivery of a distressed fetus and one in a case of unproven distress but variable decelerations. The 3 low Apgar scores seen in the control group all followed Caesarean sections.

Uterine activity :

Three episodes of hypertonia sufficient to cause fetal heart rate changes were seen, 2 in the Cardiff group and one in the controls. In one of the Cardiff patients the pattern was seen at the commencement of infusion when the incorrect infusion set had been used in setting up the system.

TABLE I

Comparison of the Age, Parity, Management of Labour, Length of
Labour and Method of Delivery in the 15 Matched Pairs

Pair No.	Age group (years)	Parity	More favourable cervix	Epidural used	Vibrator used	More efficient method	Length of Labour Cardiff	Control	Method of Delivery Cardiff	Control
1	15-19	Primip.	Cardiff	Both	Both	Control	15.30	6.69	Forceps	Forceps
2	20-24	Primip.	Control	-	Cardiff	Control	LSCS	(5.13)	LSCS	SVD
3	20-24	Primip.	-	-	-	Control	11.78	8.08	Forceps	SVD
4	20-24	Primip.	-	-	-	Control	LSCS	(12.50)	LSCS	SVD
5	25-29	Primip.	-	Cardiff	-	Cardiff	8.15	8.67	Forceps	Forceps
6	25-29	Primip.	-	Cardiff	-	Cardiff	4.17	10.18	SVD	Ventouse
7	30-34	Primip.	Cardiff	-	-	Cardiff	3.48	5.00	SVD	SVD
8	30-34	Primip.	-	Control	Control	Cardiff	(6.22)	LSCS	Forceps	LSCS
9	35-39	Primip.	Control	Control	-	Cardiff	(4.37)	LSCS	Forceps	LSCS
10	40+	Primip.	-	Control	Cardiff	Cardiff	LSCS	LSCS	LSCS	LSCS
11	25-29	1+0	-	-	-	Control	15.92	4.45	SVD	SVD
12	25-29	1+0	-	-	-	Control	7.85	4.02	SVD	SVD
13	30-34	1+0	Control	-	Cardiff	Control	9.50	5.13	SVD	SVD
14	30-34	1+0	Control	-	-	Control	8.25	4.55	SVD	Forceps
15	30-34	1+0	-	-	-	Control	8.12	4.56	Forceps	SVD

TABLE II

Relative Frequencies of the Alarms and Situations Responsible for
the Triggering of False Alarms in the Cardiff System

Alarm type	Excess pressure	Blockage	Spasm	Supply empty or mains	Total
True	12	11	12	3 (mains)	38
False	+++ (in 1 case)	9	11	1	22
Situations precipitating false alarms	Unknown cause	Patient movement. Use of bedpan. Accidental turning off of pressure measurement. (1) Unknown cause. (1)	Patient movement. Vomiting. Use of bedpan.	Old type bottle hook moved on trigger switch arm. Faulty fuse caused 3 mains off alarms.	

Practicability :

In the 17 labours managed with the aid of the Cardiff pump there were 22 false alarms and 38 true alarm situations. Only 3 labours were completely free of alarms. The highest number of alarms in the course of one labour was 15, of which 7 were false. Table II shows the alarms involved and the precipitating factors. In the 19 control patients there were 43 apparent abnormalities of uterine action or recording which would have caused an alarm to trigger in the Cardiff system. Of these 23 were artefactual, due either to inaccurate adjustment of the recording or to maternal position change. There were 20 true alarm situations due either to blockage of the catheter, a resting tone in excess of 20mm.Hg. or excess pressure during contractions.

Discussion

Although this was a small trial, it does suggest that neither fetal distress nor operative intervention is increased in frequency when the Cardiff automatic infusion system is used in the management of induced labours. It is thought that the apparent increase in frequency, noted before the onset of this trial, may have been due to the biased selection of high risk cases and patients with unfavourable cervixes.

Use of the automatic system does appear to increase significantly the induction delivery interval and the length of labour. This is probably related to the all-or-nothing method by which the infusion is regulated; each time the alarm is triggered the pump is stopped and the input of oxytocin ceases. As oxytocin has a short half-life even a delay of 30-60 seconds, during which time the abnormality is corrected and the system reset, is sufficient for the plasma level of oxytocin to fall to a suboptimal value. If the alarm has been triggered by an abnormally high resting tone or by an excessive rise in intrauterine pressure during a contraction then a fall in the plasma oxytocin level is advantageous. A fall in the level following a blockage alarm or a false alarm, however, may only serve to delay the labour process. If a feedback control unit is to be used in the management of oxytocin infusion then, ideally, the system should be

capable of downward titration as well as upward, but such a system might prove too complicated for routine labour ward use. The majority of false alarms occurred when the patients turned over, sat up or used a bedpan and there seems to be little that can be done to prevent such triggering, short of manually overriding it each time a change of position is contemplated.

It is interesting to note from Table I that the Cardiff system appeared to be most efficient in the elderly primigravidae. This could be related to the comparative rates of increase of oxytocin infusion shown in Fig.1. It can be seen that, provided no alarms are triggered, the Cardiff system infuses at twice the rate of control at 25 minutes after commencement so that the patients most likely to benefit from the Cardiff system are those requiring larger doses of oxytocin. Of the 12 elderly primigravidae in this trial 8 (4 in the Cardiff group and 4 controls) required a maximum dose of 16 mU/min or more, compared with 5 of the 18 remaining patients.

The practicability of the system is questionable in the light of our experience. The trial was designed so that the system could be operated by the regular ward staff but with one of the authors either present or immediately obtainable. Despite regular demonstrations, the midwifery staff were not satisfied that they were able to operate the system competently. The equipment is advertised as capable of being "left with minimal attention and easing the burden on the staff"⁵; the infusion pump satisfied those claims but the control unit was found to demand the almost constant attention either of a medical physicist or of a doctor well acquainted with its functions. The only dangerous problem which was encountered in this trial was the use on 2 occasions of a normal intravenous giving set in the peristaltic pump instead of the Avon A23 set. On the first occasion, early in the trial, this gave rise to hypertonia and profound fetal brachycardia. Thereafter the entire system was checked by one of the authors before connecting the patient's intravenous cannula.

Conclusion

Whilst the Cardiff automatic infusion pump appears to be an

accurate, valuable item of equipment the feedback control unit has not proved its value, at least in this trial. The frequencies of fetal distress and operative intervention were not affected but the induction delivery interval and the length of labour were prolonged by a significant length of time. The "automatic" unit demanded almost constant attention from an otherwise unoccupied member of the medical staff.

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APPENDIX DA DIAGNOSTIC REGIMEN FOR THE INEXPERIENCED OBSERVER

Experience is the best tutor in the safe and efficient use of intrapartum fetal heart rate monitoring. Currently there is no alternative means of learning nor, as yet, are there any electronic aids to diagnosis beyond the fetal heart rate trace. The following regimen is proposed as a means of preventing faulty diagnosis during the period of self-tuition.

ABNORMALITIES OF BASELINE RATE

Normal rate:	120-150 b.p.m.	uncomplicated		no action
Bradycardia:	100-119 b.p.m.	uncomplicated	>10 mins	no action
Marked bradycardia:	<100 b.p.m.	uncomplicated	>10 mins	—————> F.B.S.
Tachycardia:	151-160 b.p.m.	uncomplicated	>10 mins	check ma- ternal temperature
Marked tachycardia:	>160 b.p.m.	uncomplicated	>10 mins	check ma- F.B.S. ternal temperature

ABNORMALITIES OF BASELINE VARIABILITY

Normal undulatory:	10-25 b.p.m. ²	uncomplicated		no action
Narrowed undulatory:	5-9 b.p.m. ²	uncomplicated		no action
Silent:	<5 b.p.m. ²	uncomplicated	>10 mins	
			Check: a) response to external stimuli b) maternal position c) drug status	
				no action
		uncomplicated	>20 mins	F.B.S.
		complicated	>10 mins	F.B.S.
Saltatory:	>25 b.p.m. ²	uncomplicated	>10 mins	observe
		complicated	>10 mins	F.B.S.

ABNORMALITIES OF FHR RESPONSE TO CONTRACTIONS

No response :	uncomplicated	no action
Transient acceleration:	>10 b.p.m. uncomplicated	early no action
		delayed observe
Transient deceleration:	>10 b.p.m. uncomplicated	>5 consecutive
	Check: a) uterine tonicity:reduce	
	b) maternal position:change	
	c) cervical dilation	
	Not fully dilated and)	
	No response to a) & b))	
	give O ₂)	
		F.B.S.
Combined accelerations and decelerations:	>10 b.p.m. uncomplicated*	early } observe
		delayed }
	manage as transient decelerations	
Profound deceleration:	>40 b.p.m. uncomplicated	>3 mins
	Check: a) uterine tonicity	
	b) maternal position	
	c) epidural status	
	No obvious cause }	
	Recurring }	
		F.B.S.
Spikes:	>10 b.p.m. but	
	<20 seconds uncomplicated	no action

Transient decelerations in the second stage are normal but if they are >60 b.p.m. deep, wider than the contraction time and slow to recover then delivery should be expedited. Similarly, should there be other complications of fetal heart rate in the second stage, then delivery should be expedited.

The reader is referred to Section IV for the suggested regimen referring to the use of fetal blood sampling. All fetal blood samples should be taken when the mother is lying on her side.

*i.e. no silent variability or abnormal basal rate

APPENDIX E

PUBLICATIONS BY GARETH THOMAS, M.B.,BS.,M.R.C.O.G.

AN OPEN TRIAL OF INDOMETHACIN THERAPY IN EXACERBATED PHASES OF LEPRO- MATOUS LEPROSY

Thomas, G., Karat, A.B.A., Karat, S. and Rao, P.S.S. (1969)
Indian Journal of Medical Sciences, 23, 68.

INDOMETHACIN IN THE MANAGEMENT OF ERYTHEMA NODOSUM LEPROSUM - A DOUBLE-BLIND CONTROLLED TRIAL

Karat, A.B.A., Thomas, G. and Rao, P.S.S. (1969)
Leprosy Review, 40, 153.

CHANGES IN RENAL FUNCTION DURING REACTIVE PHASES OF LEPROMATOUS LEP- ROSY

Thomas, G., Karat, A.B.A., Rao, P.S.S. and Prathapkumar, C.
(1970)
International Journal of Leprosy, 38, 170

also read by Thomas, G. at :

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FETAL INTRAPERITONEAL PRESSURE CHANGES DURING INTRA-UTERINE TRANSFU- SION

Holt, E.M., Boyd, I.D., Gillmer, M.A., Naylor, C.J. and Thomas,
G. (1972)
Journal of Obstetrics and Gynaecology of the British Commonwealth,
79, 255.

A CONTROLLED TRIAL OF THE CARDIFF AUTOMATIC INFUSION SYSTEM IN THE MANAGEMENT OF INDUCED LABOUR

Thomas, G. and Blackwell, R.J. (1974)
British Journal of Clinical Practice, 28, 203
Please see Appendix C.

THE AETIOLOGY, CHARACTERISTICS AND DIAGNOSTIC RELEVANCE OF LATE DECELERATION PATTERNS IN ROUTINE OBSTETRIC PRACTICE

Thomas, G. (1975)

British Journal of Obstetrics and Gynaecology, 82, 121

Please see Section VII.

THE ANALYSIS OF CONTINUOUS FETAL HEART RATE TRACES IN THE FIRST AND SECOND STAGES OF LABOUR

Thomas, G. and Blackwell, R.J. (1975)

British Journal of Obstetrics and Gynaecology, 82, 634

Please see Section VI - Part 2.

A HAZARD ASSOCIATED WITH THE USE OF SPIRAL FETAL SCALP ELECTRODES

Thomas, G. and Blackwell, R.J. (1975)

American Journal of Obstetrics and Gynecology, 121, 1118.

Please see Section III.

PROGRESSION IN RESPONSE PATTERNS OF FETAL HEART RATE THROUGHOUT LABOUR

Liu, D.T.Y., Thomas, G. and Blackwell, R.J. (1975)

British Journal of Obstetrics and Gynaecology, 82, 943.

Please see Section VIII.

INTERPRETATION OF DATA DERIVED FROM FETAL MONITORING

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in *The Management of Labour. Proceedings of the Third Study Group of the Royal College of Obstetricians and Gynaecologists* (1975).

Edited by Beard, R.W., Brudenell, M., Dunn, P. and Fairweather, D.V.I. p.181.

THE USE OF ULTRASOUND FOR MONITORING FOETAL BREATHING MOVEMENTS

Farman, D.J. and Thomas, G. (1975)

Biomedical Engineering, 10, 172.

ERRORS AND ARTIFACTS ENCOUNTERED IN THE MONITORING OF FETAL RESPIRATORY MOVEMENTS USING ULTRASOUND

Farman, D.J., Thomas, G. and Blackwell, R.J. (1975)

Ultrasound in Medicine and Biology, 2, 31.

SOME FACTORS AFFECTING THE VALIDITY OF A-MODE ULTRASONIC RECORDINGS
OF FETAL RESPIRATORY MOVEMENTS

Farman, D.J., Thomas, G. and Blackwell, R.J. (1975)

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ALPHA-FETOPROTEIN LEVELS IN MATERNAL PLASMA AND AMNIOTIC FLUID DURING
PROSTAGLANDIN-INDUCED MID-TRIMESTER ABORTIONS : THE RELATION
TO FETAL DISTRESS AND DEATH

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Chard, T. (1976)

British Journal of Obstetrics and Gynaecology, 83, 299.

ACKNOWLEDGEMENTS

I am deeply indebted to Professor D. V. I. Fairweather for his guidance and encouragement and to the Consultants in Obstetrics at University College Hospital, London for allowing me unlimited access to their patients. I am also grateful to the midwifery staff, the obstetric junior staff and the students at U.C.H. who made these observations on fetal heart rate monitoring possible by documenting the traces.

None of the work would have been possible without the assistance of David T. Y. Liu, Obstetric Research Fellow and the advice and assistance of Roland J. Blackwell and David J. Farman, medical physicists at University College Hospital, and we four, as a group, are grateful to both the Obstetric Unit secretarial staff and the Medical Physics Department secretaries for their unfailing efforts in producing type-written manuscripts as and when required. Mary Krasu, M.R.C.O.G. kindly checked the consistency of the baseline variability measurements in Section VI and to her I extend my thanks.

I have been particularly fortunate during the final preparation of this thesis in having close at hand a most efficient secretary, Mrs. Rosalind Gilbert, who has typed the entire manuscript and prepared the tables in their final form. I am extremely grateful to her and also to my wife, Alison, who has been a constant source of encouragement and a willing proof-reader.

I am indebted to several librarians for their expert assistance but, in particular, I would like to thank Mrs. U. Spanner, Librarian at the Royal Berkshire Hospital Postgraduate Centre, Reading, for her enthusiastic help in searching some of the earlier references. I am also grateful to Patricia Want at the Library of the Royal College of Obstetricians and Gynaecologists in London.

I have been particularly fortunate in having at hand the linguistic talents of Dr. Serge Six and his wife, Dr. Veronique Moens, who have translated some of the earlier references from French and German into English. I am grateful to them and also to Professor dr. M.

Thiery of Gent who assisted in the search for an early German reference.

I would like to express my thanks to the National Fund for Research into Crippling Diseases, to the Central University Grants Committee of London and to the Birth Defects Trust, University College Hospital Medical School for providing the funds and equipment grants which were necessary in order to pursue the described investigations. I am grateful, also, to the Oxford Regional Health Authority for financing two weeks of study leave in which to complete the manuscript.

Last, but certainly not least, I would like to thank all the patients who made these investigations possible.

Gareth Thomas