## INTENSIVE CARE IN LABOUR: A PRELIMINARY APPRAISAL\*

D. R. W. HARTLEY AND D. A. DAVEY, Department of Obstetrics and Gynaecology, University of Cape Town

### SUMMARY

This is a preliminary investigation into the value, function and practicability of an intensive care labour unit for highrisk cases. All the equipment necessary can be mounted on two standard theatre trolleys which can be moved to the patient's bedside, but there is no place at present for this equipment outside a teaching unit.

The investigation reports on continuous foetal heart monitoring of 36 patients and simultaneous foetal scalp pH estimations on 16 of them. A high proportion of babies with low Apgar scores showed one of two (or both) characteristic foetal heart patterns during labour: (a) the well-known type 2 dip, (b) a steppe pattern not previously described as such. The latter is of importance because it is impossible to detect using only clinical methods.

There was poor correlation between foetal pH values and Apgar rating, and between foetal pH values and monitor patterns. Moreover, the pH values of foetal scalp blood and foetal umbilical arterial and venous blood samples bore varying relations to one another. However, for practical purposes, a foetal scalp blood of pH less than 7.20 when associated with a maternofoetal pH difference of 0.250 or more should be regarded with anxiety. The place and value of intensive care are discussed.

'Of 4 200 000 children born annually in the USA, 3% will reach adulthood intellectually lower than a twelve-year-old, 0.3% will be lower than a seven-year-old and 0.1% will be imbeciles."4 This statement is complemented by other equally arresting observations. Norris30 stresses the prenatal and perinatal factors in intellectual and emotional development, and states that the obstetrician is in a position to do preventive psychiatry. Towbin36,37 has demonstrated the changes in the brain following foetal hypoxia. Acute hypoxia at 25 - 35 weeks causes venous stasis, thrombosis and infarction in the paraventricular nuclei; nearer term the damage occurs in the cortex. A slow hypoxia, such as occurs with long-standing placental insufficiency, may produce cerebral damage which is silent, difficult to diagnose and may occur days or even weeks before labour starts. Quite apart from the more obvious effects of retarded brain function following birth, in a large number of cases minor casualties of reproduction may be the cause of emotional instability later in life.19 Equally important, and of more direct concern to the obstetrician, are the number of stillbirths due, directly or otherwise, to undetected foetal hypoxia, and this amounts to approximately 50% of all stillbirths. 45,46 Therefore, if these cases could be diagnosed in time, the stillbirth rate could be much reduced.

For these reasons a search has been in progress during the last decade or so for an early and accurate means of detecting foetal hypoxia and foetal distress. The normally accepted clinical signs of distress—foetal heart irregularities and meconium staining of the liquor—are, as is well known, often inaccurate<sup>22,32,39,44</sup> and many unnecessary caesarean sections have had to be done when relying on \*Date received: 21 September 1970.

these criteria alone. Moreover, foetal death can occasionally occur in the absence of these classical signs.<sup>39</sup>

There have been two approaches to the problem. The first has been continuous foetal heart monitoring which is recorded graphically, usually with a simultaneous recording of the uterine contractions. <sup>13</sup>, <sup>25</sup>, <sup>27</sup> The second has been by the early detection of blood-gas and pH changes in foetal capillary blood, taken from the scalp during labour. <sup>1,5</sup>, <sup>22</sup>, <sup>25</sup>, <sup>33</sup>, <sup>34</sup> Of all the blood-gas levels, it is generally agreed that in practice the pH is the most reliable indicator of foetal well-being <sup>15</sup>, <sup>25</sup> provided the pH of the mother is taken into account simultaneously, and allowances are made for this. PO<sub>2</sub>, for instance, can alter suddenly when oxygen is administered, thus masking a dangerous metabolic acidosis in the foetus.

### MATERIAL AND METHODS

In this small series of patients continuous foetal heart monitoring was performed with a cardiotocograph, the uterine contractions being recorded by an abdominal transducer. The foetal heart rate (which is recorded automatically every 3 seconds) was simultaneously recorded with either an abdominal electrode or a scalp electrode. We used the Michel clip type, 3,4,14 but others have been described, incorporated into a No. 3 ventouse suction cup.26

There were 36 patients in the series who were monitored with the cardiotocograph. Nine of these patients were normal and in the other 27 the foetuses were at greater risk than normal. The length of time that the monitor was applied varied, but as experience progressed the monitoring was continued until delivery. The shortest recording time was 4 minutes and the longest 398 minutes with an average duration of 142 minutes. In 16 of the patients simultaneous measurements were made of foetal and maternal blood pH.<sup>20</sup> Of the 27 abnormal cases, 19 patients had a normal vaginal delivery; 3 an assisted delivery with the ventouse and 5 had a caesarean section. Foetal well-being was assessed by the Apgar rating and by the heart rate one minute after birth (Table I).

TABLE L ASSESSMENT OF FOETAL WELL-BEING

Abnormal cases	No.	Apgar ratings
Pre-eclamptic toxaemia	13	10 (7 cases) 6 (1 case) 4 (2 cases)
		2 (1 case) 1 (2 cases)
Pre-eclamptic toxaemia and prolonged		200
rupture of membranes	1	2
Pre-eclamptic toxaemia and prediabetes	1	10
Prediabetes	2	10, 10
Antepartum haemorrhage	2	10, 3
Unexplained foetal distress	1	6
'Small for dates' foetus	1	10
Breech: tachycardia	1	2
Prolonged rupture of membranes	4	10 (all cases)
Postmaturity	1	10
RESULTS		

The foetal heart patterns in Figs. 1-7 were observed in patients who subsequently delivered healthy babies of a high Appar rating; we therefore feel that similar tracings

should, for practical purposes, be regarded as being within normal limits in most cases, provided that they do not change to the abnormal pattern.

The foetal heart patterns in Figs. 8-11 were observed in patients who subsequently delivered hypoxic babies of low Apgar ratings requiring resuscitation.

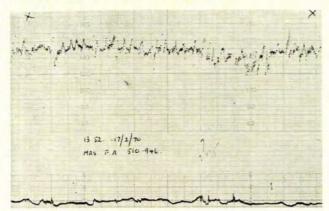


Fig. 1. Oscillations in foetal heart rate of amplitude ± 20 occurring 2-3 times per minute. Probably represents seesaw of autonomic vascular tone in foetus.

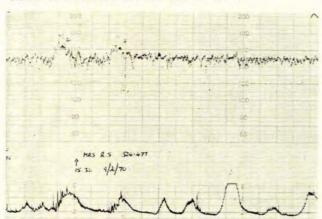


Fig. 2. Normal rises in foetal heart rate. These are associated with Braxton-Hicks contractions in early labour.

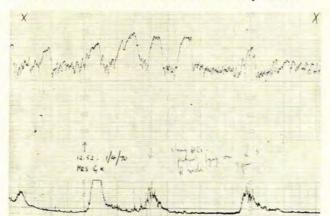


Fig. 3. More severe rises in foetal heart rate. These are not always associated with a contraction, and did not recur later. Possibly due to pressure on the cord.

# Comments on Foetal Heart-Rate Patterns

Our findings agree with those of previous workers. Basal rates between 160 and 120/min (Figs. 1 - 5) and

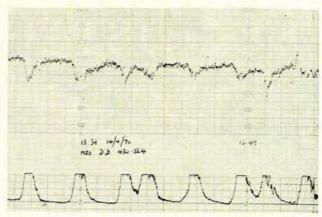


Fig. 4. Typical type 1 'dips'. This is a normal finding. Note the timing of the fall and the recovery in rate in relation to a uterine contraction. Compare with type 2 'dip' (Fig. 9).

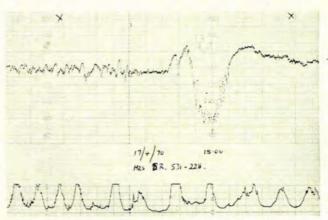


Fig. 5. Severe prolonged bradycardia with subsequent recovery. Not repeated. Probably due to pressure on the cord or on the foetal head descending in the pelvis.

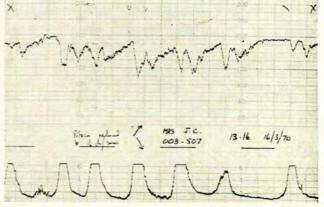


Fig. 6. Disturbed foetal heart rate when an oxytocin infusion was run in too rapidly. Heart rate becomes steady when the drip is slowed.

early acceleration (Figs. 2-3) or early deceleration with rapid recovery (type 1 dip or 'V' dip—Fig. 4) are all normal variations. They may be due to reflex vagal tone caused by temporary pressure on the umbilical cord. 3,4,15,29,31 They may be abolished by atropine and are not corrected by oxygen. They may also be caused by squeezing of the head. 13,16 In addition, they are frequently inconsistent in relation to uterine contractions, and may be abolished by turning the patient on her side. 12

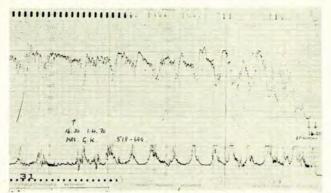


Fig. 7. Typical foetal heart-rate pattern during the second stage of labour. Note the severe bradycardia of 50 as the head was crowning.

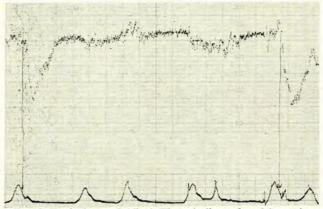


Fig. 8. Typical type 2 'dips'. These indicate foetal hypoxia. Note the timing of the fall and the recovery in rate in relation to a uterine contraction. Compare with type 1 'dip'.

Type 2 or 'U' dips (Figs. 8 and 9) and a steppe-like pattern we have observed (Fig. 11) have been associated in our experience with hypoxic babies of low Apgar rating. Nine babies were born with Appar ratings of 6 or less. Seven of these had type 2 dips or steppe pattern or both present. In the other two no tracing was performed. There were two cases exhibiting a steppe pattern which after 30 minutes reverted to normal. These babies subsequently were born with an Apgar rating of 10. These changes probably reflect foetal hypoxia due to insufficiency of the placenta16 which may be aggravated if the relaxation period between uterine contractions is too short (Fig. 6). The type 2 dip begins as the uterus is relaxing, and takes a long while (over 60 seconds) to recover. It may be severe, causing a bradycardia of 60 or less. It can be abolished by oxygen (Fig. 10).1,2 If it is accompanied by a gradually

rising basal rate it is of especially serious prognosis and a persistent bradycardia following this heralds impending foetal death.<sup>17</sup>

The steppe pattern is particularly interesting. It has a quite characteristic shape, consisting of a sharp drop and a slow recovery, taking 2-3 minutes, until the next drop occurs. We feel that this is an important observation which has not been described previously. In this type of

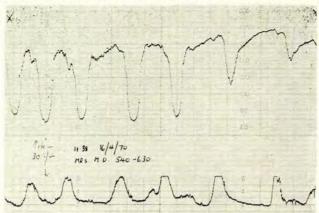


Fig. 9. More marked type 2 'dips' in a patient who underwent induction of labour for severe pre-eclampsia.

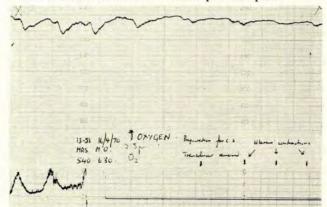


Fig. 10. In the same patient a 'steppe' pattern is abolished by oxygen.

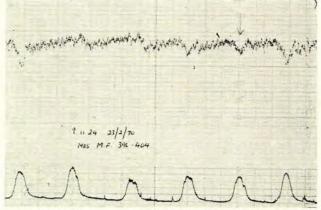


Fig. 11. 'Steppe' pattern probably indicates foetal hypoxia. There is only a slight fall in rate with contraction but recovery takes at least 3 minutes. At no time is the rate clinically abnormal.

foetal heart pattern the rate at any given time is within normal limits and the range of variability is difficult to detect by ear, so that clinically it would be regarded as a normal foetal heart. This type may well explain some of those cases where, despite close observation, the foetal heart stopped 'without warning'. This pattern is really a modified type 2 curve with a small amplitude.

A normal foetal heart rate undergoes a rhythmical oscillation of 5-20 beats/min about 2 to 4 times/min. This is well demonstrated in Fig. 1, and it probably represents a seesaw effect produced by the tone of the sympathetic and parasympathetic autonomic system. Lack of this, giving a flat line (Fig. 10), is abnormal and carries a poor prognosis.

### pH Values of Foetal Scalp Capillary Blood

In 16 out of the 36 cases monitored, foetal and maternal capillary blood pH determinations were made to see if there was any correlation between the pH values and the foetal heart patterns obtained. Attempts were made to obtain foetal scalp capillary blood in early labour and just before delivery. The blood from the umbilical cord was also analysed (Tables II and III). Note the large variation in pH of umbilical (venous) blood in babies with a low Apgar rating (Table III), and particularly those associated with the patients who had type 2 dips and steppe patterns (No. 17, 22, 24 and 33). Note also low pH values found in patients with babies born in good health (No. 23, 26 and 29).

Conclusions on the value of pH readings. It has been shown over a large series that increasing foetal hypoxia is associated with a lowered foetal scalp blood pH. However, there are enough exceptions on each side of mean values to make a pH reading in any particular case sometimes difficult to interpret. If hypoxia is suspected a pH value of over 7.25 will allay anxiety temporarily, perhaps until vaginal delivery occurs. In this way unnecessary caesarean sections may be avoided. This appears to be the most useful feature of this measurement.42.12 The greater the difference between the maternal pH and the foetal pH, the more significant is a low foetal pH. This is especially so if the patient is in a high-risk category, and particularly if hypertension and proteinuria are present.23 For practical purposes a maternofoetal difference of more than 0.250 might be regarded as abnormal, especially if the foetal pH is below 7.20.

There is disagreement over the value of this investigation as a routine procedure. Saling claims a 4-fold lowering in perinatal mortality with its routine use, 23,34 On the other hand, Farr 22 estimates that had it been used in Aberdeen as a routine over a whole year it might have saved only two foetal lives.

The measurement of the pH of foetal scalp blood is a task which takes some practice, is time-consuming and is uncomfortable for a patient already exhausted in labour. Our feeling is that there is no place for it as a routine procedure, nor is there any place for it in the second stage

TABLE II. pH VALUES IN PATIENTS WITH HIGH APGAR BABIES

	Early labour			At or near delivery					
Case No.	Mat.	Foetal scalp	Diff-	Mat.	Foetal pH (vein) (V)	Foetal pH (scalp)	Foetal pH (art.)	Diff- erence	Inaa
28	pH 7:404	<i>pH</i> 7⋅275	0·129	<i>pH</i> 7·525	7-365	7·180	(A) 7·343	(D) 0·182	Apgai 10
26	7.374	7.264	0.110	7.336	7.300	7-077	7.290	0.046	10
38	7-434	7.290	0.144	7.385	7.348		7.270	0.115	10
38 37	7.384	7.169	0.215	7.419	7.413	7.310	7.246	0.173	10
30	7.410	7.160	0.250	7	7.330	120	7.210	_	10
32 35	_	_		7-355	7.220	7.225	7.219	0.136	10
35	7.350	7.291	0.059	7-319	7.272	7.269	7.177	0.142	10
34	7-422	7.300	0.122	7.370	7.280	-	7.138	0.232	10
23	7.461	_	_	7-290	-	7:130		0.160	8
29	7.401	7.260	0.141	7.301	7-175	7.030	7.120	0.181	9
31	7.432	7-202	0.230	7.368	7.257	7.225	7.096	0.272	10

This table is arranged so that the pH of foetal umbilical arterial blood at delivery decreases progressively from top to bottom (column A). The range is wide, from 7.343 to 7.096. Column V is the pH of umbilical venous blood at delivery and column S is the pH of scalp blood just before delivery. Column D shows the difference in pH between maternal blood and foetal umbilical arterial blood at delivery (or foetal scalp blood in case 23).

Correlation between foetal scalp and umbilical vessel pH readings is poor. In cases 26, 28 and 29 the scalp pH is lower than both the other two; in case 32 it is higher than both.

TABLE III. pH VALUES IN PATIENTS WITH LOW APGAR BABIES

Case No.	Early labour			Delivery					
	Mat.	Foetal scalp pH	Diff.	Mat. pH	Foetal pH (V)	Foetal pH (S)	Foetal pH (A)	Diff.	Apgar
27	7.400	7.330	0.070	7.365	7.340			0.025	3
22	7-360	_		7.360	7-295	-		0.065	5
24	7.335	7.255	0.080	7.320	7.240	_		0.080	3
33	7.398	7.170	0.228	7.430	7.230	7.180	7.135	0.295	1
17	7.2			7-340	7-120	-	_	0.220	6
36	7-319	6.970*	0.349	_	_	-	_	70.00	2

Compare column V with column V in Table II. Comments on these patients are in the text. \*This foetus was believed to be dead but was delivered alive unexpectedly (para 2 with pre-eclampsia)

of labour when it merely delays active intervention if required.

### DISCUSSION

The equipment required for this type of intensive care can be accommodated on one standard theatre trolley. All that is necessary are two electric plugs somewhere near the patient's bed, one for the cardiotocograph and one for the light source for use with the endoscope when taking foetal scalp samples. A second trolley is needed for the foetal scalp sampling equipment. An Astrup machine near the labour ward is essential.

Indications are those patients in whom the foetus is at risk due to suspected or proved placental insufficiency. These include:

Pre-eclamptic toxaemia and hypertension, especially if proteinuria is present.40,41

Postmaturity—especially in the primigravida. 8-10

The 'small-for-dates' foetus.

Diabetes and prediabetes.

Antepartum haemorrhage not due to placenta praevia. Unexplained foetal distress.

Any patient on an oxytocin infusion.

The elderly primigravida.10

Membranes ruptured for more than 24 hours.

Rhesus iso-immunization.

Previous history of stillbirth.

Pregnancy following drug-induced ovulation.

In a busy unit catering for abnormal cases there may be simultaneous demands for this care and priorities must be clearly sorted out. The low parity patient, for instance, should have precedence over the high parity and the more intelligent over the less intelligent if only because the former is likely to co-operate better. A high degree of rapport and sympathy between doctor and patient is essential.

Midwives who have to attend such patients find that it saves considerable effort. The monitor is especially useful when using intravenous oxytocin, for the drip rate can be accurately adjusted to simulate a physiological labour with the aid of the graphic record of contractions, and ensure reasonable uterine relaxation between contractions. Occasionally the monitor may help in the diagnosis of twins. It has been suggested that it can be used in the diagnosis of foetal death and even in the mundane task of the diagnosis of the presentation of a foetus.

It is, of course, very important that a correct perspective of the place of the monitor and the foetal pH sampling should be maintained, and that they should never be allowed to be anything more than aids to clinical judgement.

The monitor provides the closest constant observation of the foetal heart rate, something which is impossible to do clinically using the ordinary foetal stethoscope, and in this lies its value. When foetal heart-rate changes occur the timing of these changes in relation to a uterine contraction can be observed accurately. Moreover, a permanent graphic record of labour is obtained, which can be studied later if required.

Normal patterns are type 1 dips, or any irregularity which is not repeated or maintained later. Abnormal patterns due to foetal hypoxia are type 2 dips and the steppe pattern.

In these cases early delivery is indicated, and we feel that these foetal heart patterns are of more value than foetal pH changes, and there is evidence that they occur earlier than pH changes.47 Moreover, they involve less discomfort to the patient than the taking of foetal scalp samples.

What is required is a light, portable machine with two very light receptors placed on the mother's abdomen, one to record uterine contractions and the other to record the foetal heart rate. The latter receptor could be moved to the spot where the foetal heart rate is heard loudest without prejudicing the recording of uterine contractions as at present occurs in the combined transducer, when the foetal back is lateral or posterior. It would also avoid the use of a scalp electrode which injures the baby's scalp with a risk of subsequent infection.

We wish to thank the sisters and staff of the Labour Ward. Groote Schuur Hospital, for their helpful co-operation; Mrs V. Baynham for the blood analyses; and Miss M. Welch for the photographs.

### REFERENCES

- Althabe, O., Schwarcz, R. L., Pose, S. V., Escarcena, L. and Caldeyro-Barcia, R. (1967): Amer. J. Obstet. Gynec., 85, 1033.
   Idem (1968): Obstet. Gynec. Surv., 98, 858.
   Barden, T. P. and Stander, R. W. (1963): J. Amer. Med. Assoc., 186,

- 3. Barden, T. P. and Stander, R. W. (1963): J. Amer. Med. Assoc., 186, 923.
  4. Idem (1964): Obstet. Gynec. Surv., 19, 242.
  5. Beard, R. W., Morris, E. D. and Clayton, S. G. (1966): J. Obstet. Gynacc. Brit. Cwlth, 73, 562.
  6. Idem (1967): Obstet. Gynec. Surv., 22, 42.
  7. Beard, R. W. and Morris, E. D. (1965): J. Obstet. Gynacc. Brit. Cwlth, 72, 496.
  8. Cushner, I. M. (1964): Sinai Hosp. J. (Baltimore), 12, 39.
  9. Idem (1965): Obstet. Gynec. Surv., 20, 269.
  10. Editorial Comment (1965): Ibid., 20, 272.
  11. Editorial Comment (1969): Ibid., 24, 610.
  12. Farr, V. (1970): J. Obstet. Gynacc. Brit. Cwlth, 77, 294.
  13. Hon, E. H. and Quilligan, E. J. (1968): Clin. Obstet. Gynec., 11, 145.
  14. Hon, E. H. (1963): Amer. J. Obstet. Gynec., 86, 772.
  15. Idem (1959): Ibid., 78, 47.
  16. Idem (1959): Ibid., 77, 1084.
  17. Idem (1962): Ibid., 78, 83.
  18. James, L. S., Weisbrot, I. M., Prince, C. E., Holaday, D. A. and Apgar, V. (1958): J. Pediat., 52, 379.
  19. Knobloch, H. and Pasamanick, B. (1958): Amer. J. Obstet. Gynec., 104.
  10. Kubli, F. W. and Hon, E. H. (1969): Amer. J. Obstet. Gynec., 104.
- Kubli, F. W. and Hon, E. H. (1969): Amer. J. Obstet. Gynec., 104, 1190.
- 23.
- 1190.

  Kubli, F. W. (1968): Clin. Obstet. Gynec., 11, 168.

  Leslie, D. W. (1959): Brit. Med. J., 2, 612.

  Lumley, J., Hammond, J. and Wood, C. (1969): J. Obstet. Gynaec.

  Brit. Cwlth, 76, 512.

  Masland, R. L., Sarason, S. B. and Gladwin, T. (1958): Mental Subnormality: Biological, Psychological and Cultural Factors. New York:

  Basic Books.

  Mayers, B. T. Bradfield, A. and Smyth, E. J. (1963): Med. J.
- Mayes, B. T., Bradfield, A. and Smyth, E. J. (1963): Med. J. Aust., 2, 905.
- McRae, D. J., Bekhit, S. M. and Kundu, G. (1969): J. Obstet. Gynaec. Brit. Cw'th, 76, 419.
   Mendez-Bauer, C., Poseiro, J. J., Arellano-Hernandez, G., Zambrana, M. A. and Caldeyro-Barcia, R. (1963): Amer. J. Obstet. Gynec., 85, 1033.

- 1033.

  Morris, E. D. and Beard, R. W. (1965): J. Obstet. Gynaec. Brit. Cwith, 72, 489.

  Newman, W. (1963): Med. J. Aust., 2, 912.

  Norris, A. S. (1960): J. Amer. Med. Assoc., 172, 413.

  Reynolds, S. R. M. (1962): Amer. J. Obstet. Gynec., 83, 800.

  Saling, E. (1968): Foetal and Neonatal Hypoxia. London: Edward Arnold.

  Idem. (1966): Arch. Disc. China.

- 40.
- Saling, E. (1968): Foetal and Neonatal Hypoxia. London: Edward Arnold.

  Idem (1966): Arch. Dis. Childh., 41, 472.

  Idem (1966): Obstet. Gynec. Surv., 22, 413.

  Shenker, L. (1966): Ibid., 21, 367.

  Towbin, A. (1969): Arch. Neurol. (Chic.), 20, 35.

  Idem (1969): Obstet. Gynec. Surv., 24, 628.

  Weisbrot, I. M. (1958): J. Pediat., 58, 395.

  Wood, C. and Pinkerton, J. H. M. (1961): J. Obstet. Gynaec. Brit Cwith. 68, 427.

  Wood, C., Lumley, J., Hammond, J. and Newman, W. (1968): Med. J. Aust., 2, 707.

  Idem (1969): Obstet. Gynec. Surv., 24, 401.

  Wood, C., Lumley, J. and Renon, P. (1967): J. Obstet. Gynaec. Brit. Cwith. 74, 823.

  Idem (1968): Obstet. Gynec. Surv., 23, 643.

  Wood, C., Ferguson, R., Leeton, J., Newman, W. and Walker, A. (1967): Amer. J. Obstet. Gynec., 98, 62.

  Wren, B. G. (1960): Med. J. Aust., 2, 180.

  Idem (1961): Obstet. Gynec. Surv., 16, 71.

  British Perinatal Mortality Report (1963): Perinatal Mortality. London: E. & S. Livingstone.