Suid-Afrikaanse Tydskrif vir Obstetrie en Ginekologie South African Journal of Obstetrics and Gynaecology

Kaapstad, 4 Desember 1965 Deel 3 No. 4 Volume 3 Cape Town, 4 December 1965

THE DIAGNOSIS OF INTRA-UTERINE ASPHYXIA BY MICRO-BLOOD ANALYSIS OF THE FOETUS IN UTERO*

PETER V. WESTON, M.B., B.CH., B.SC., DIP.PHARM., F.C.O.G. (S.A.), M.R.C.O.G., *Senior Lecturer and Senior Gynaecologist and Obstetrician at the University of Natal and the King Edward VIII Hospital, Durban;* AND DERK CRICHTON, M.B., B.CH. (CAPE TOWN), D.PHIL. (OXON.), F.R.C.S.E., F.R.C.O.G., *Professor of Gynaecology and Obstetrics at the University of Natal, and Head of the Division and University Unit of Gynaecology and Obstetrics at the King Edward VIII and Addington Hospitals, Durban*

The traditional signs of foetal distress (such as variation in the foetal heart rate and the appearance of meconium in the liquor amnii) are notoriously unreliable. Similarly, the cries of a baby at birth and the normality of Apgar rating in no way exclude asphyxia.[3] The blood chemistry of the foetus alone constitutes an accurate index of the foetal state.

After birth the blood gas chemistry of babies can be determined with ease from umbilical cord samples or from within the baby. This is not a reliable reflection, however, of the baby's state *in utero,* for it is common knowledge that contractions impair the placental transfer of gases, especially during the second stage of labour.

Even at caesarean section under optimal conditions, the estimation of foetal blood chemistry is unreliable. For instance, obstruction to the inferior vena cava owing to the pressure of the gravid uterus, when the patient lies on her back for any length of time (called the supine-hypotensive syndrome, when of severe degree) impairs blood-flow through the intervillous space. Epidural or spinal anaesthesia may add to the hypotension, and general anaesthesia may affect the foetal blood-gas chemistry. Manipulation of the uterus during abdominal delivery may alter the uterine circulation and affect the maternal and foetal gas exchange. Finally, the incision into the uterus and escape of liquor may cause an immediate contraction, and even at this stage it may take upwards of 60 seconds to deliver the foetus which may add to an asphyxia if anoxia is already present.

Blood taken from the intervillous space (as described by Prystowsky[4]) by transabdominal puncture is unreliable because there is uncertainty as to the whereabouts of the needle in relation to arterioles and venules.[2]

It is knowledge of the chemistry of the blood going to the foetal tissues that is required, and this information must be readily available before birth if it is to be of value in reducing perinatal mortality.

THE BLOOD CHEMISTRY OF HYPOXIA

When hypoxic, the foetus substitutes anaerobic for aerobic (oxidative) metabolism in variable degree in an attempt to meet its energy requirements. The inefficiency and consequences of this process are these: 'Anaerobic' metabolism of a molecule of glucose results in the production of only 2 molecules of ATP, whereas 'aerobic' (oxidative) metabolism contributes no less than a further 36 molecules of ATP. Furthermore, anaerobic metabolism results in the accumulation, not only of carbon dioxide but also of acid metabolites (which reduce the buffer base levels in the blood); whereas aerobic metabolism produces *only* carbon dioxide which is capable of easy disposal. These are the changes of asphyxia. Thus asphyxia includes hypoxia, CO_2 retention, acidosis, a rise in serum-potassium levels and a fall in buffer bases.

Two kinds of acidosis, 'respiratory' and 'metabolic' require distinction:

Respiratory acidosis results from short-lived inadequate ventilation in which CO_2 accumulates and the pH drops, but there is no alteration in the buffering substances of the blood unless inadequate ventilation is prolonged. Respiratory acidosis is readily reversible by ventilation. Thus, if arterial blood, taken from a patient with respiratory acidosis, is agitated with an oxygen/carbon dioxide gas mixture, comparable with that found in the alveolar air, the depressed pH readily returns to normal, because this is simply simulating the process of ventilation and liberating CO_2. The above gas mixture has a PCO_2 (or partial pressure of CO_2) of 40 mm.Hg.

Metabolic acidosis follows respiratory acidosis when oxygen deprivation is prolonged. Energy is then obtained in a variable degree from anaerobic glycolysis, and the low pH of arterial blood is not readily reversible. Neither adequate respiration nor equilibration of this blood with oxygen with a PCO_2 of 40 will return this low pH to normal. Here the low pH or acidosis persists because anaerobic glycolysis has resulted in the liberation of lactic acid and other acidic metabolites. Metabolic acidosis leads to death if uncorrected.

In the foetus metabolic acidosis usually follows respiratory acidosis, because oxygen deprivation is frequently prolonged; but naturally the early phase of an acute complication, such as umbilical-cord compression or abruptio placentae, is associated with a respiratory acidosis.

The tolerance of the foetus or neonate to anoxia is inversely proportional to the glycogen content of cardiac muscle. Premature babies are particularly vulnerable to asphyxia, because the glycogen content of their cardiac muscle is directly proportional to maturity.

CLINICAL EVALUATION OF HYPOXIA

From a clinical standpoint it is worth emphasizing that the only value of auscultating the foetal heart before labour is to determine whether the baby is alive or dead; as a practical index of foetal distress it is useless.

Walker,[11] in his analysis of 700 cases of foetal distress in our unit, found that when there are alterations in the foetal heart rate there is no increased perinatal loss in uncomplicated pregnancies; but the perinatal loss was increased threefold if there was meconium-staining of the amniotic fluid.

If the membranes are ruptured the passage of meconium will give an indication of asphyxia in many cases, but usually the membranes are still intact when meconium is first passed.

The presence of meconium before rupture of the membranes has been detected by Saling,[5] from whom one of us (P.V.W.) received personal instruction last year. In the investigation (called amnioscopy) amniotic fluid is screened before the rupture of the membranes.

*Paper presented at the 45th South African Medical Congress (M.A.S.A.), Port Elizabeth, June - July 1965.

The procedure is carried out with the patient in the lithotomy position and, following a sterile vaginal examination (whereby the state, dilatation and position of the cervix is noted), an instrument (Fig. 1) is guided into the

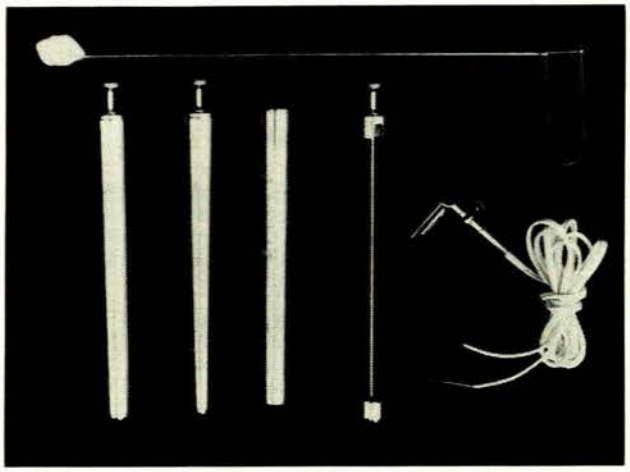

Fig. 1. Amnioscopy instruments.

cervix with the vaginal fingers. The obturator is then removed and, after attaching a light, the amniotic fluid between the forewaters and the presenting part can be visualized.

The possible findings are a clear or milky solution with vernix flakes, a green fluid denoting meconium-staining of the liquor, yellow fluid denoting Rhesus incompatibility, or a pink-brown fluid indicating intra-uterine death of the foetus.

The size of the instrument used depends upon the dilatation of the cervix. If a finger cannot be inserted through the cervix, a 12 mm. diameter tube is passed; if a finger can be introduced, a 16 mm. tube is used, and if it is still further dilated, a 20 mm. tube is passed.

The indications for amnioscopy (according to Saling) are pre-eclamptic toxaemia, suspected intra-uterine asphyxia, and postmaturity. Saling recommends that amnioscopy be performed on alternate days from the tenth post-term day onwards. Labour is only induced if meconium-staining of the amniotic fluid is noted. It remains to be seen, however, to what extent it is justifiable to assume that there is no foetal distress if the foetus has not passed meconium, for we know that meconium is not invariably passed when asphyxia is present.

Possible complications of the procedure are accidental rupture of the membranes (which occurs in about 1% of the cases) and induction of labour (which occurs in 25% of the cases at or beyond term).

The discovery of meconium-staining of the amniotic fluid necessitates artificial rupture of the membranes to enable blood to be taken from the foetus for analysis. Alterations in the foetal heart rate or the presence of pre-eclampsia in labour are additional indications for micro-blood analysis.[8,9]

The endoscopes employed for this procedure are conical in shape (Fig. 2). After the liquor ceases to drain, the membranes are stripped back to the edges of the endoscope to uncover the presenting part of the foetus. The endoscope is now held firmly against the presenting part, while the latter is mopped dry and sprayed with ethyl chloride until the skin becomes white from cooling.

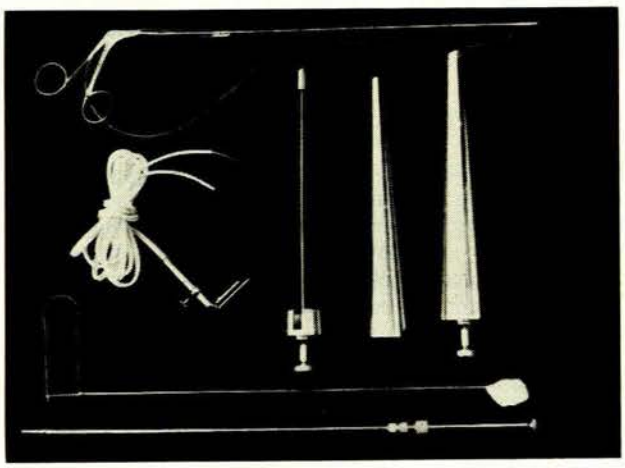

Fig. 2. Endoscopy instruments for obtaining blood by puncture of the presenting part.

A phase of hyperaemia soon follows. By wiping the dried skin (which must be hyperaemic) with a swab soaked in sterile silicone, a thin covering film is created through which a 2 mm. stab incision is made. The blood that flows out of the incision collects as a thick drop which is collected in a thin heparinized capillary tube mounted on a special holder.

The end of the capillary tube for collecting blood (which should project 1 - 1·5 cm. beyond the end of the forceps used to introduce it into the endoscope) is immersed into the drop of blood, and carefully controlled oral suction draws this up into the tube. In order to ensure the aspiration of blood free of air bubbles, the free end of the capillary tube must be visualized during aspiration, and therefore the introducing forceps are kept as near to the posterior wall of the endoscope as possible. The occasional small air bubbles (less than 1 mm. in diameter) affect actual values very little, and they can be eliminated when the blood is transferred to the measuring chamber for analysis. Yet, to maintain maximum accuracy, the estimation should always be made on samples that are free of air bubbles. The blood collected in the heparinized capillary tube is examined immediately for PCO_2, PO_2, pH and for pH after equilibration with oxygen with a PCO_2 of 40 mm. of mercury. It is valuable to appreciate that if an Astrup machine is at hand the full blood-gas picture of the foetus can be available within 5 minutes.

If the cervix is more than 2 fingerbreadths dilated, the presenting part can be visualized with speculae[6] (Fig. 3).

If the pH is low and returns to normal after equilibration, respiratory acidosis is present, and the test is then repeated on another blood sample to ascertain whether a cause such as an acute cord complication has corrected itself spontaneously. If acidosis persists, however, or if metabolic acidosis is present, delivery must be effected

forthwith. To impress upon readers that many urgent caesarean sections for foetal distress may be unnecessary, Saling's[10] results are worth citing:

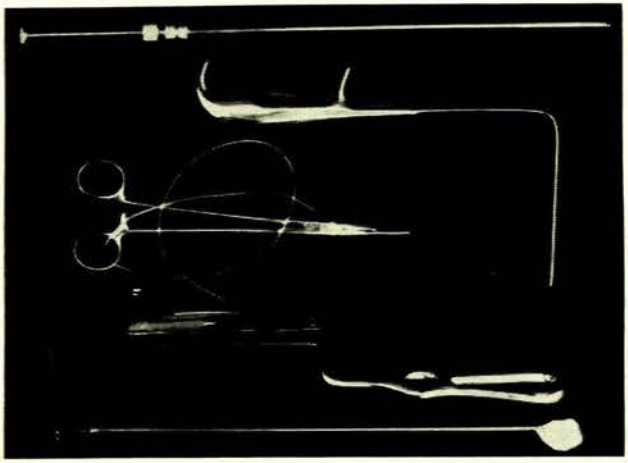

Fig. 3. Instruments for obtaining blood after exposing the presenting part with speculae.

He found abnormal blood-gas chemistry in only 18% of cases where there was meconium-staining of the amniotic fluid, in only 40% of cases where the foetal heart rate was below 100, and in only 33% of cases with meconium-staining of the liquor associated with any alteration in the foetal heart rate.

Saling[7] has also found a close correlation between the oxygen level of hyperaemic capillary scalp blood taken from the foetus *in utero* and that of cord blood at delivery. The oxygen saturation level of blood samples taken from the cephalic vascular system lies between the oxygen content of the umbilical vein and that of the artery. In babies with tetanus neonatorum at King Edward VIII Hospital, Durban, arterialized scalp capillary blood was found to be identical to arterial blood by Desai *et al.*[1] Thus, micro-blood analysis of capillary scalp blood of the foetus *in utero* gives an accurate indication of the blood perfusing the tissues of the foetus, particularly that supplying the brain.

It is of interest to mention that after delivery in normal and depressed babies there is a fall in buffer bases, and PCO_2 and lactic acid levels rise; but with good respiration these results become normal within about an hour. These changes are probably due to re-establishment of normal circulation in areas previously inadequately perfused.

SUMMARY

A method of diagnosing intra-uterine asphyxia with accuracy before and during labour has been described.

Only an Astrup machine and the few instruments described are required for the accurate diagnosis of foetal distress.

This novel method of taking blood from the foetus holds forth hope of an improved foetal salvage in practice and offers exciting new spheres of research.

We wish to acknowledge our admiration of Dr. Erich Saling's work in pioneering this discovery and to indicate our appreciation of the trouble he takes to demonstrate his method to visitors. The hospitality shown to one of us (P.V.W.) by Dr. Saling and his staff made a most memorable occasion of a visit to West Berlin.

We must also thank Dr. H. R. J. Wannenburg, Superintendent of King Edward VIII Hospital, Durban, for permission to publish this paper, and Mr. K. F. Birch for the photography.

REFERENCES

1. Desai, S. D., Holloway, R. and Thambiran, A. K. (1965): S. Afr. Med. J. (in the press).
2. Fuchs, F., Spackman, T. and Assali, N. S. (1963): Amer. J. Obstet. Gynec., **86**, 226.
3. James, S. and Burnard, E., eds. (1961): *Ciba Foundation Symposium on Somatic Stability in the Newly Born.* London: J. & A. Churchill.
4. Prystowsky, H. (1957): Bull. Johns Hopk. Hosp., **101**, 48.
5. Saling, E. (1962): Geburtsh. u. Frauenheilk, **22**, 830.
6. *Idem* (1962): Arch. Gynäk., **197**, 108.
7. *Idem* (1963): Z. Geburtsh. Gynäk., **161**, 262.
8. *Idem* (1964): Geburtsh. u. Frauenheilk., **24**, 464.
9. *Idem* (1964): Germ. Med. Mth., **9**, 449.
10. *Idem* (1964): Personal communication.
11. Walker, N. (1959): Brit. Med. J., **2**, 1221.