A B S T R A C T

FOETAL DISTRESS EQUALS FOETAL ACIDOSIS

Paul Desjardins, M.D.
Investigative Medicine

August 5, 1968

Our main concern as obstetricians has always been to decrease maternal and perinatal mortality. Foetal distress is an obscure term requiring further research towards establishment of a precise definition. The progress in physiology and pathophysiology favours a biochemical definition of foetal distress as being a metabolic or respiratory acidosis.

Acidosis in the foetus may be determined by scalp sampling and measurement of blood gases.

By routine use of this technique in obstetrical units, it is hoped that the stillbirth rate will be decreased, the apgar score at birth will be increased, and mental retardation with or without neurological deficit will also be decreased.

Other areas of research will blossom from the availability of the foetus for study, such as the physiology of the foetal pancreas.

FOETAL DISTRESS EQUALS FOETAL ACIDOSIS

"Thesis submitted to the Faculty of Graduate Studies and Research, Investigative Medicine, McGill University."

Paul Desjardins, M.D.

August 5, 1968

INTRODUCTION

If one looks at the evolution of obstetrics in our modern era it becomes easy to see how obstetrical practice was aimed primarily at maternal survival in the years up to 1955. The great progress that has been made in obstetrics is reflected by a tenfold decrease in maternal mortality due, for the most part, to the organization of blood banks, the practice of cross-matching and having blood available at all times during labour and at the time of delivery, and to the development of anaesthesia and the perfectioning of anaesthesiological techniques aimed at the safety of both mother and foetus. Antibiotics have also played an important role in almost eliminating puerperal sepsis, a great killer of the past.

These factors plus the antenatal care the obstetrician has been giving his patients, the early diagnosis of toxaemia, its prevention by diet, watchfulness for other maternal diseases, and hospitalization at or near term has brought maternal mortality down from 6 per 1,000 births to 0.6 per 1,000 births. Figure 1 (1) illustrates this trend over North America. In Canada, in good obstetrical clinics accepting all forms of complications, the figures may vary from 0 to 0.3 per 1,000 births.

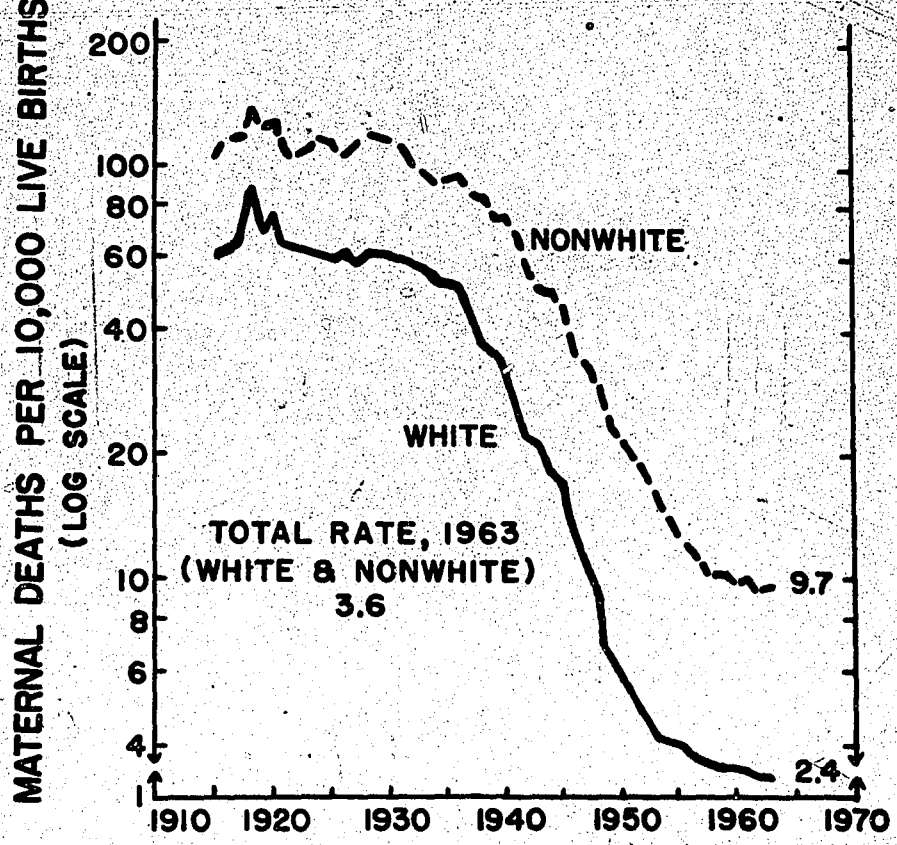
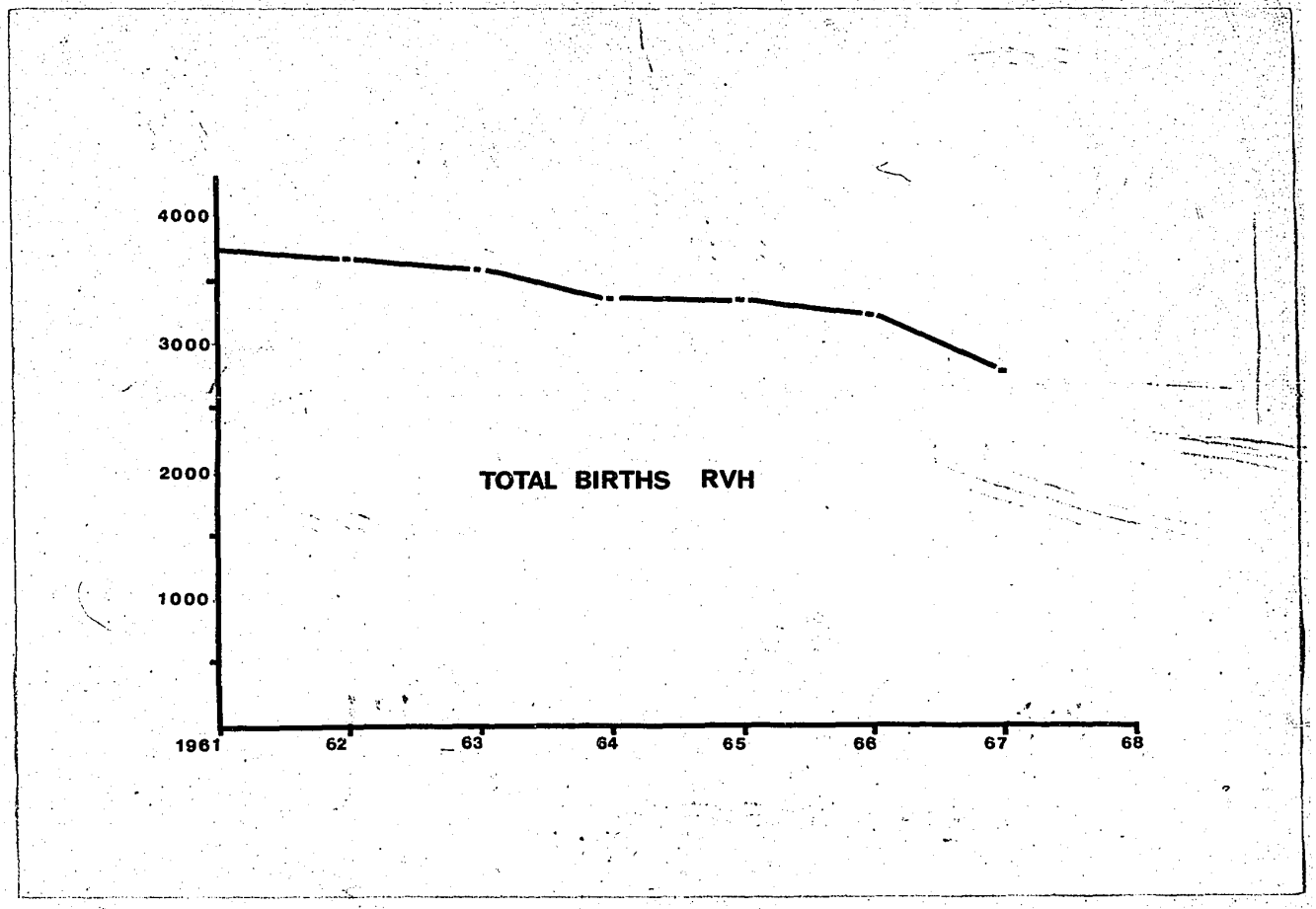


Fig. 2. Maternal mortality, 1915–1963, United States Birth Registration Area. (Department of Health, Education and Welfare. Welfare Administration. Children's Bureau. Based on data from the National Center for Health Statistics, Division of Vital Statistics.) (Rates have been rounded.)

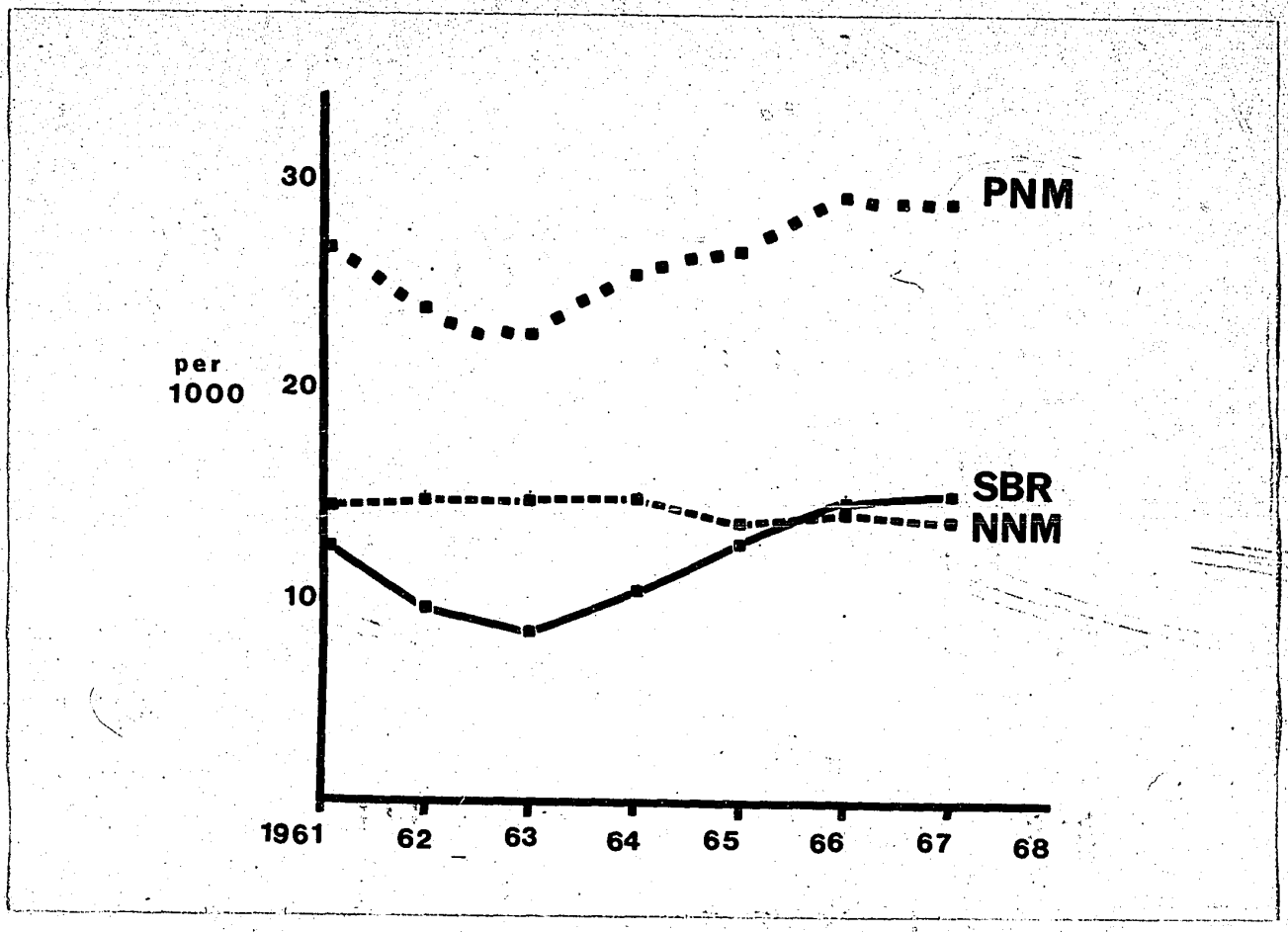
After having succeeded in diminishing maternal mortality the obstetrician then turned toward the perinatal mortality on which he is still working today.

Perinatal mortality is a composite term, made up of the stillbirth rate and the neonatal mortality(infants who die in the first 7 days of extrauterine life). In the years 1930-1960 there has been a fourfold decrease in perinatal mortality due mainly to the obstetrician's and the paediatrician's ability to deal with prematurity, erythro blastosis foetalis, and diabetes in the pregnant mother.

Since 1961 a steady decrease in the birth rate has been noted throughout North America. In Canada the birth rate per 100,000 was in 1967 at its lowest since 1920. This is exemplified by the Royal Victoria Hospital figures in Figure 2.



At the same time perinatal mortality has also been reduced, but if a breakdown is made of the two components, it is noted that the neonatal mortality has been decreasing while the stillbirth rate has been increasing. Because of our interest in decreasing perinatal mortality our attention was directed to the stillbirths which have not decreased proportionally to the neonatal deaths. Figures 3 and 4 show the causes of stillbirths in our hospital for the years 1961 through 1967.



STILLBIRTHS - ROYAL VICTORIA HOSPITAL

1961	1962	1963	1964
a. Total 46 b. Causes: 1. Cong.Abnormalities 4 2. Infection --------- - 3. Iso-Immun.--------- - 5. F. Maln. ---------- 8 7. AB. Pl Pr. -------- 8 8. Labor dyst--------- 3 x Asphyxia ---------- 10 # Unexplained ------- 13 46 12,3 per th.	a. Total 35 b. Causes: 1. --------- 2 2. --------- 2 3. --------- - 5. --------- 5 7. --------- 6 8. --------- 2 x --------- 6 # --------- 12 35 9,5 per th.	a. Total 30 b. Causes: 1. -------- 2 2. -------- 2 3. -------- 2 5. -------- 5 7. -------- 2 8. -------- 1 x -------- 4 # -------- 12 30 8,3 per th.	a. Total 36 b. Causes: 1.---------- 3 2. --------- 2 3. --------- 2 5. --------- 11 7. --------- 3 8. --------- 1 x --------- 5 # --------- 9 36 10,7 per th.

NEONATAL DEATHS

1961	1962	1963	1964
14,2 per th 53	14,4 per th. 53	14,4 per th. 52	14,8 per th. 50

PERINATAL DEATHS

26,5 per th. 99	23,9 per th. 88	22,7 per th. 82	25,6 per th. 86

No. of BIRTHS

3727	3675	3597	3356

STILLBIRTHS - ROYAL VICTORIA HOSPITAL

1965	1966	1967
a. Total 43 b. Causes: 1. Cong. Abnormalities 3 2. Infection --------- 3 3. Iso-Immun. -------- 3 5. F. Maln. ---------- 11 7. AB. PL.Pr.--------- 5 8. Labor dyst. ------- 2 x Asphyxia ---------- 7 # Unexplained ------- 9 43 12,7 per th.	a. Total 48 b. Causes: 1. ---------- 5 2. ---------- 4 3. ---------- 4 5. ---------- 5 7. ---------- 4 8. ---------- 1 x ---------- 6 # ----------19 48 14,8 per th.	a. Total 42 b. Causes: 1. ---------- 5 2. ---------- - 3. ---------- - 5. ---------- 3 7. ---------- 7 8. ---------- 1 x ---------- 7 # ----------19 42 15,per th.

NEONATAL DEATHS

1965	1966	1967
13,9 per th. 47	14,2 per th. 46	13,9 per th.. 39

PERINATAL DEATHS

26,7 per th. 90	29,1 per th. 94	29,0 per th. 81

No. of BIRTHS

3362	3223	2787

It should be noted that in 1966 and 1967 unexplained stillbirths rose to a high of 19 out of 48 and 42 respectively. Attempting to understand and decrease the stillbirth and perinatal mortality rates has brought about a very close link between the obstetrician and the paediatrician. This link has been established through two sub-specialities. In obstetrics has been born the subdivision of foetal physiology and in paediatrics has been born the speciality of neonatology. The dividing line between these two is thin and probably cannot always be identified.

The foetal physiologist is faced with the major problem of the availability of his subject for study. The foetus, until recently, has been a mysterious being floating weightlessly in his amniotic space. Attempts to reach the foetus for study have encountered great difficulty, sometimes even failure, giving rise to many indirect studies on amniotic fluid and audible foetal heart tones as a reflection of foetal well-being. Many great research men turned toward animal experimentation because of the difficulty of studying the human foetus directly.

This great difficulty is reflected in the context of discussing perinatal mortality and stillbirth rate with the definition of foetal distress. Clinicians have difficulty in correlating foetal distress in utero with stillbirths and neonatal deaths. It has been stressed that early diagnosis of foetal distress and its correction will eliminate or at least decrease perinatal mortality and morbidity. But what exactly

is foetal distress? This term is defined in most textbooks as a condition manifested by tachycardia or bradycardia, and irregular foetal heart rate sometimes accompanied by passage of meconium - but what exactly is foetal distress? This is something that still does not remain clear today. The definition is loose, inaccurate, clinical in nature, and completely without measurement of the true status of the foetus in utero.

At a conference on Prenatal Physiopathology Adamson reported a study correlating the incidence of neurological defects at one year of age with birth weight and the apgar score at one minute of age.(2, 3, 4, 5, 6, 7) This is a very interesting study and for an apgar at one minute of age of 0 to 3 for a birth weight of 2,000 grams or less the incidence of neurological defects at age one year is 20.8%. It is obvious that some type of foetal distress will cause permanent neurological defects and if the foetal distress could be diagnosed early during labour or during delivery perhaps the incidence of neurological defects could be decreased. Figure 5 illustrates the apgar scoring method.

Evaluation of a Newborn Infant

SIGN	0	1	2
HEART RATE	ABSENT	SLOW (BELOW 100)	OVER 100
RESPIRATORY EFFORT	ABSENT	SLOW IRREGULAR	GOOD CRYING
MUSCLE TONE	FLACCID	SOME FLEXION OF EXTREMITIES	ACTIVE MOTION
REFLEX IRRITABILITY	NO RESPONSE	GRIMACE	CRY
COLOUR	PALE BLUE	BODY PINK EXTREMITIES BLUE	COMPLETELY PINK

The plan for this thesis includes:-

(1) Some specific aspects of foetal physiology.

(2) A method of investigation.

(3) Material and technique.

(4) Results obtained.

(5) A conclusion with a tentative definition of foetal distress and an outlook for the future.

SPECIFIC ASPECTS OF FOETAL DISTRESS

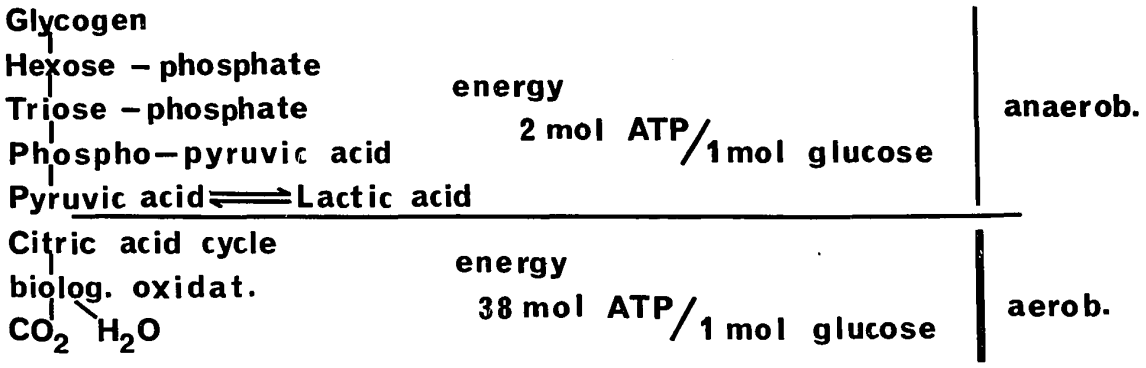
We have already seen that severe neurological deficit at one year of age correlated with birth weights and apgar scores and the unexplained rise in stillbirths has brought about a specific interest in foetal physiology.

The fact that asphyxia neonatorum is always accompanied by acidosis is a well recognized correlation in our neonate nurseries and emerging recognition for the foetus in utero.

It becomes evident that there is a necessary production of energy in utero for foetal survival, development, and metabolism. A study has been reported showing that the transformation of energy in utero is equal to 100 joules per mg. per minute, 1 joule equals 10 million ergs - 1 erg is a unit of force that can move a body 1 centimeter against the force of 1 dyne and 1 dyne is a unit of force which, when acting continuously on a mass of 1 gram, will impart to it an acceleration of 1 centimeter. Thus at term a normal foetus weighing 3,400 grams will require 340 million joules per minute for its necessary production and transformation of energy. This energy requirement is a sine qua non to survival itself.

Energy in the foetus is provided mainly by the aerobic metabolism of glucose. Oxygen deprivation is compensated for by anaerobic glycolysis of the large stores of glycogen in the foetus, principally in his liver and muscles. These will enable the foetus or the newborn infant to survive relatively long periods of anoxia but are a poor source of energy. Furthermore, during anaerobic

glycolysis the foetus will produce somewhere in the neighbourhood of a surplus 10,000 hydrogen ions. Therefore, any condition producing deprivation of energy will affect the concentration of hydrogen ion and the acid base status.

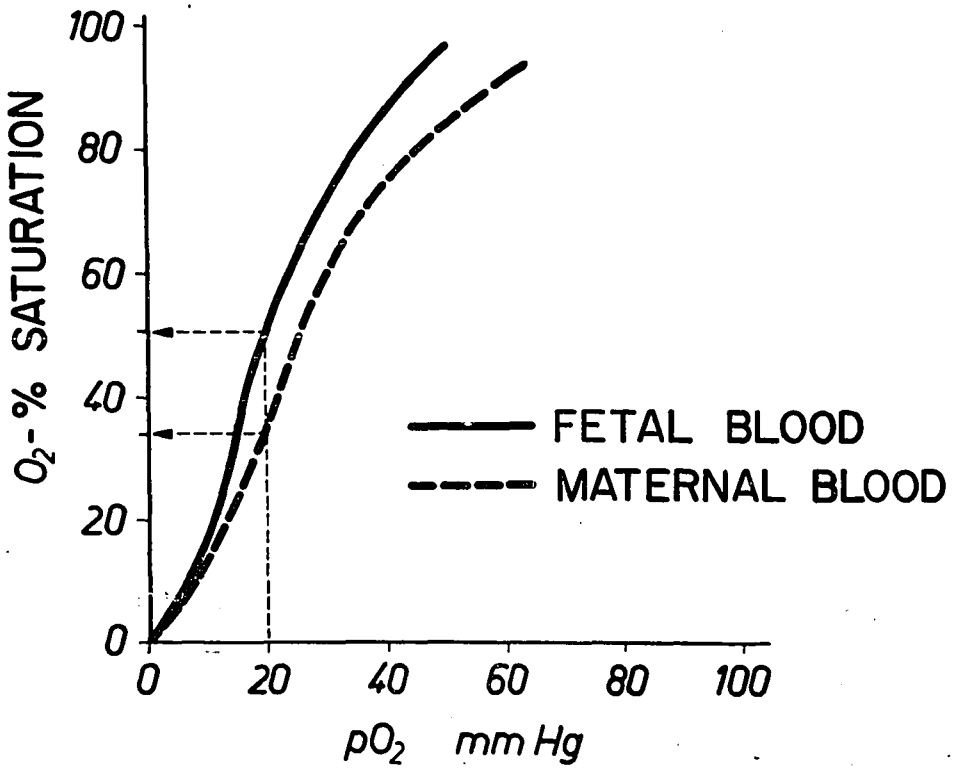


In other words, the change in hydrogen ion is equal to energy availability to the foetus. A search was then conducted for a measurement of some sort which would be simple, accurate, inexpensive, and fast. To determine this energy availability pH represents all of the above criteria. Saling in 1964 described

a technique for obtention of foetal blood from the scalp and measurement of pH to determine foetal acidosis. (references 8-17).

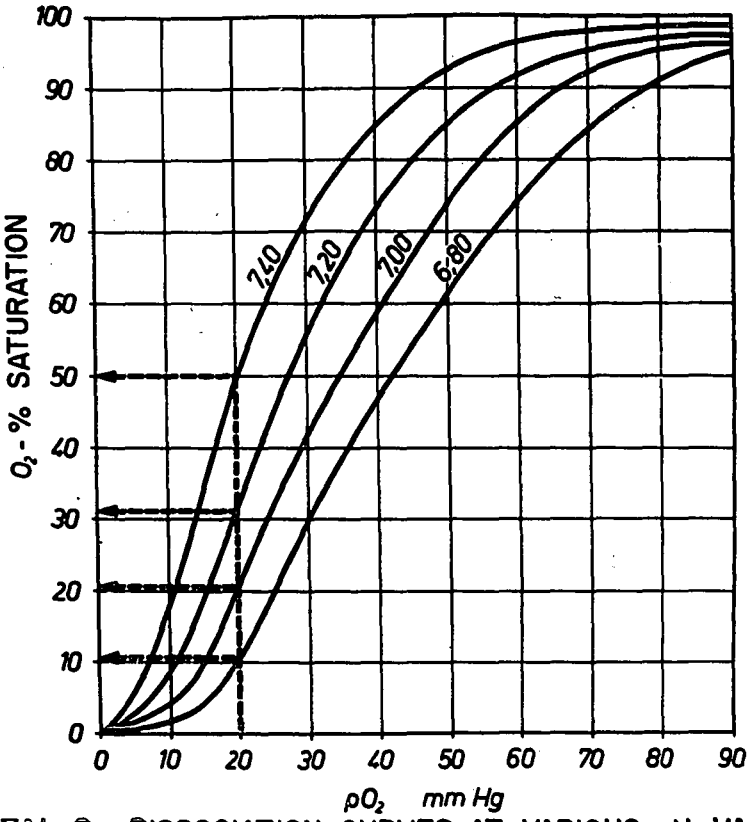
In Figure 6 above the relationship of 19 to 1 moles of ATP in aerobic versus anaerobic states, and the production of carbon dioxide and water versus the production of lactic acid, is worth noting.

The importance of energy availability by glycolysis in the presence of oxygen has stimulated a multitude of studies. Eastman has described the "Everest In Utero" (18) syndrome and has explained how the foetus compensates for this situation. Assali (19) has proven how the foetal cardiac output is greater than adult cardiac output and in Figure 7 the hemoglobin dissociation curves of foetal and maternal blood give us another explanation on how the foetus compensates.



MATERNAL-FETAL O_2-COMBINATION CURVE

It should be noted that at a pO_2 of 20 millimeters of mercury the foetal hemoglobin saturation will be greater than the maternal hemoglobin saturation. At the same pO_2 of 20 millimeters of mercury Figure 8 illustrates how the oxygen saturation will vary according to the pH of the foetus. The lower the pH the lower is the oxygen saturation.

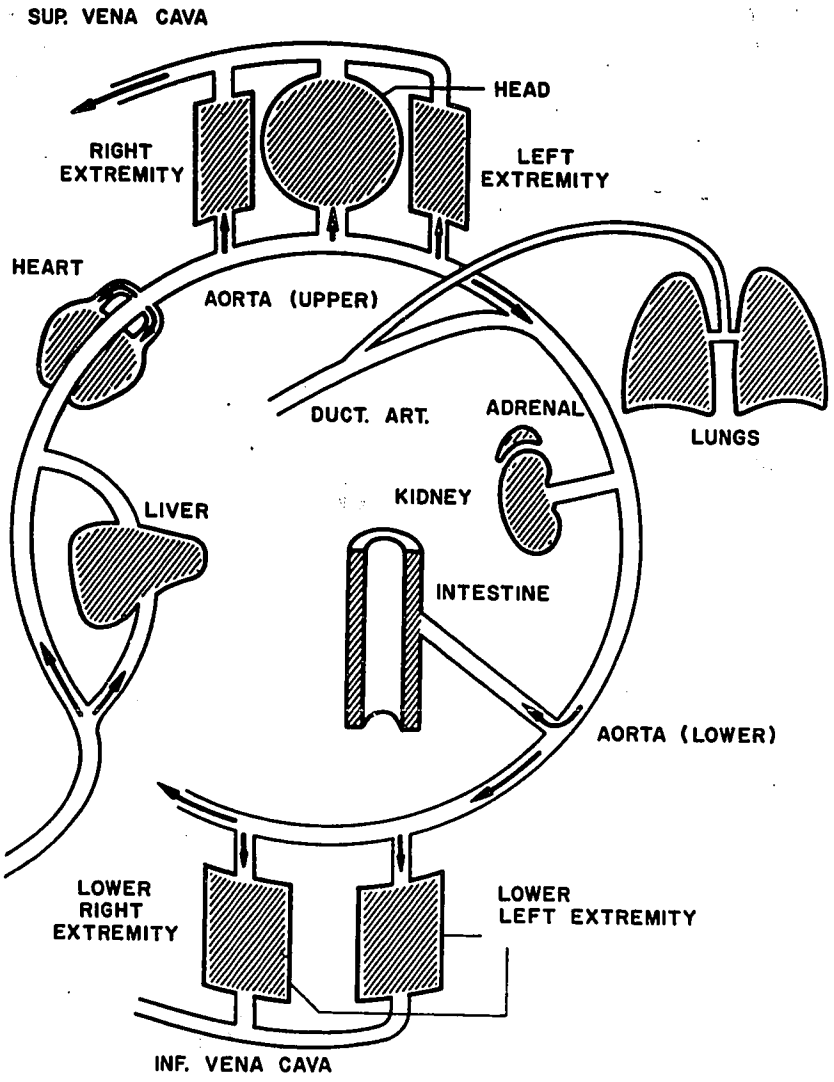


FETAL O_2-DISSOCIATION CURVES AT VARIOUS pH VALUES

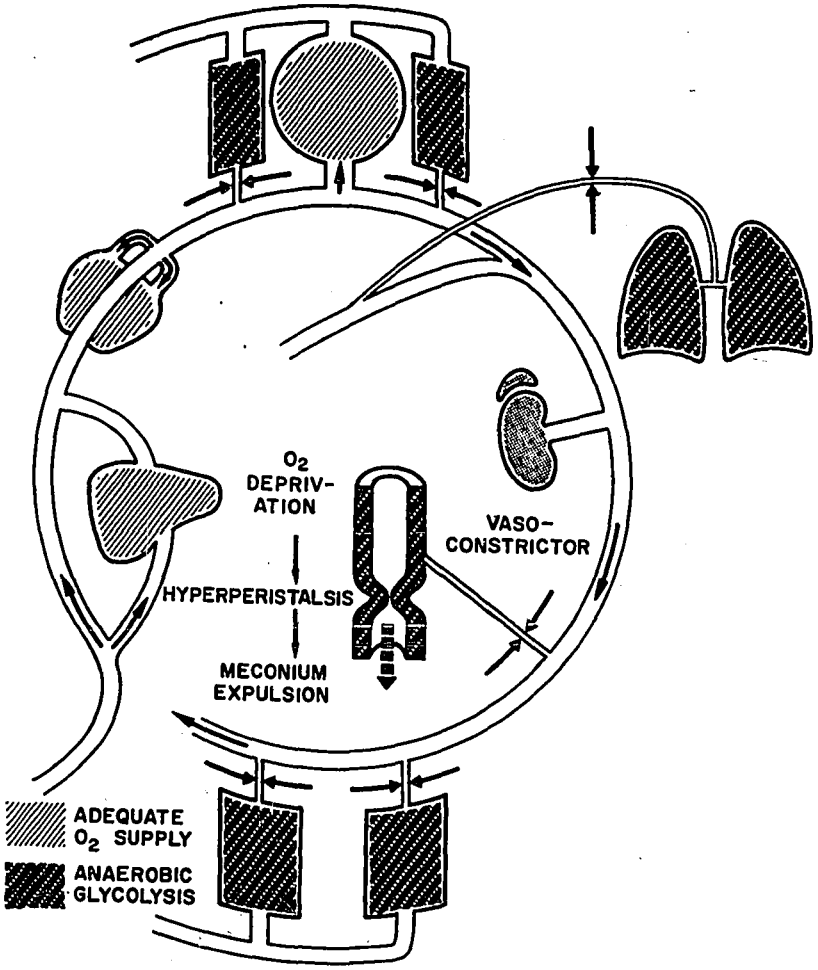
This brings about a chain reaction from hypoxia to anoxia, to diminution of energy availability, anaerobic glycolysis, over-production of hydrogen ions, accumulation of lactic acid, acidosis,

and finally to a diminution of oxygen saturation. (See references 18 - 30).

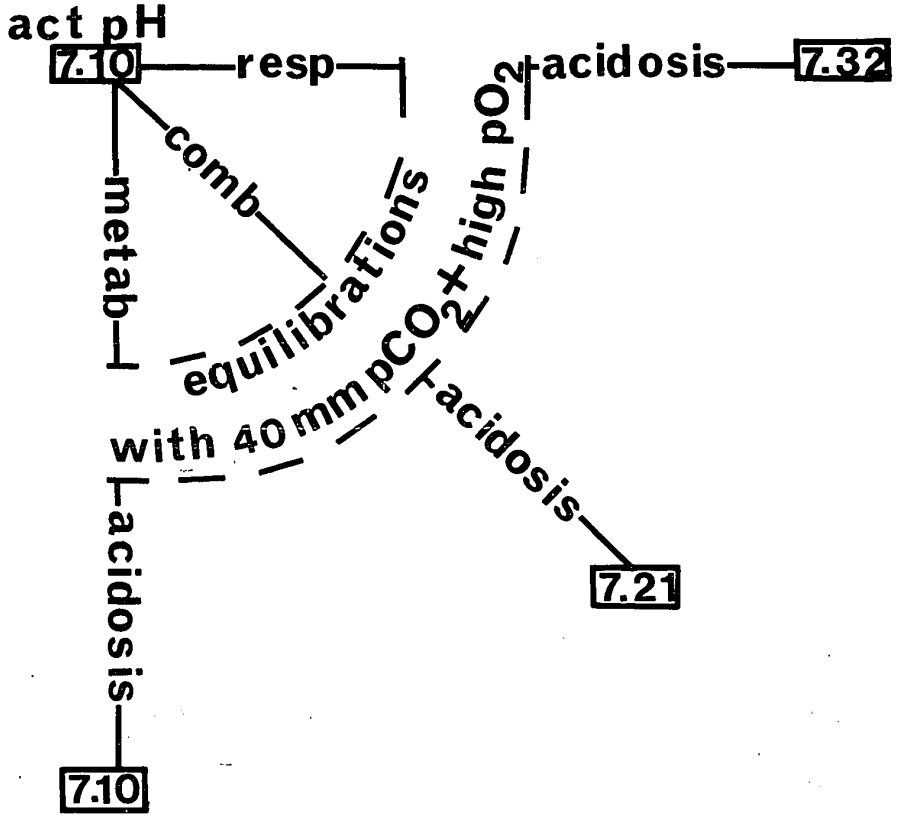
One other method of compensation is available to the foetus in utero in the presence of deprivation of oxygen. This is illustrated in Figure 9 which is a simplified drawing of foetal circulation under normal conditions.



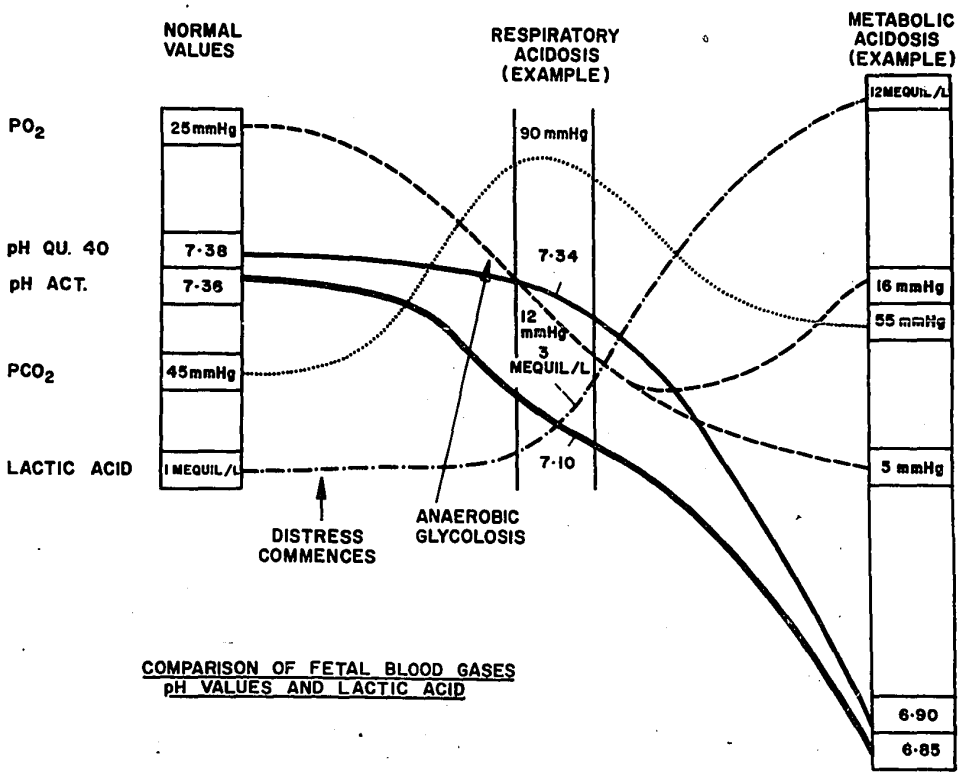
In a situation of oxygen deprivation, the foetus compensates by pooling his resources. He does this by decreasing circulation to areas such as the limbs and the lungs and eventually, as the situation worsens, to the bowel. This is accomplished by vasoconstriction, producing hyperperistalsis and an expulsion of meconium. (See Figure 10). Expulsion of meconium is a kind of foetal distress and should attract the clinician's attention. (31, 32, 33, 34, 35, 36)



Acidosis in the foetus can be metabolic when lactic acid, the end product of anaerobic glycolysis, accumulates. Or, in an acute type of anoxia where there has been insufficient time for the metabolic acidosis to develop, carbon dioxide may rapidly accumulate and a respiratory acidosis ensue. This is typically seen in cases of umbilical cord compression. A combination of both types of acidosis may be present in more gradual hypoxia, for example, that due to a failing placenta where a combined respiratory and metabolic acidosis is usually present. The various types may be distinguished by equilibration at a high pO_2 at 40 millimeters of pCO_2. This is illustrated in Figure 11.



A normal foetus with normal biochemical and acid base components may change, for instance, to a case of acute cord compression producing an acute respiratory acidosis, illustrated by an increase in pCO_2, a decrease in pO_2, a decrease in pH and a very slight increase in lactic acid. If the situation is not remedied quickly the next step is a secondary metabolic acidosis illustrated by a high production of lactic acid, a slight lowering of pCO_2, a continuous lowering of pO_2, and a gradual fall in pH. This is illustrated in Figure 12. Thus, conditions producing acidosis may vary from a chronic foetal distress such as that which exists in foetal malnutrition producing metabolic acidosis to an acute foetal distress such as that produced by cord compression giving rise to respiratory acidosis and eventually to a combined acidosis and we must differentiate among the different types to determine energy availability. (37, 38, 39, 40, 41)



COMPARISON OF FETAL BLOOD GASES
pH VALUES AND LACTIC ACID

MATERIALS AND METHODS

The importance, accuracy, and reliability of uterine monitoring with regard to pressure and contractility during labour has been well established by Caldeyro-Barcia. The same comments can be made for foetal heart rate monitoring during labour, its variations and interpretation as described by Hon. In recent years the value of scalp vein sampling as described by Saling et al has gained world recognition. During the spring of 1967 Caldeyro-Barcia reported the fusion of both techniques in the assessment of both foetus and mother during labour. This present paper will deal only with the scalp sampling aspects of our method of investigation.

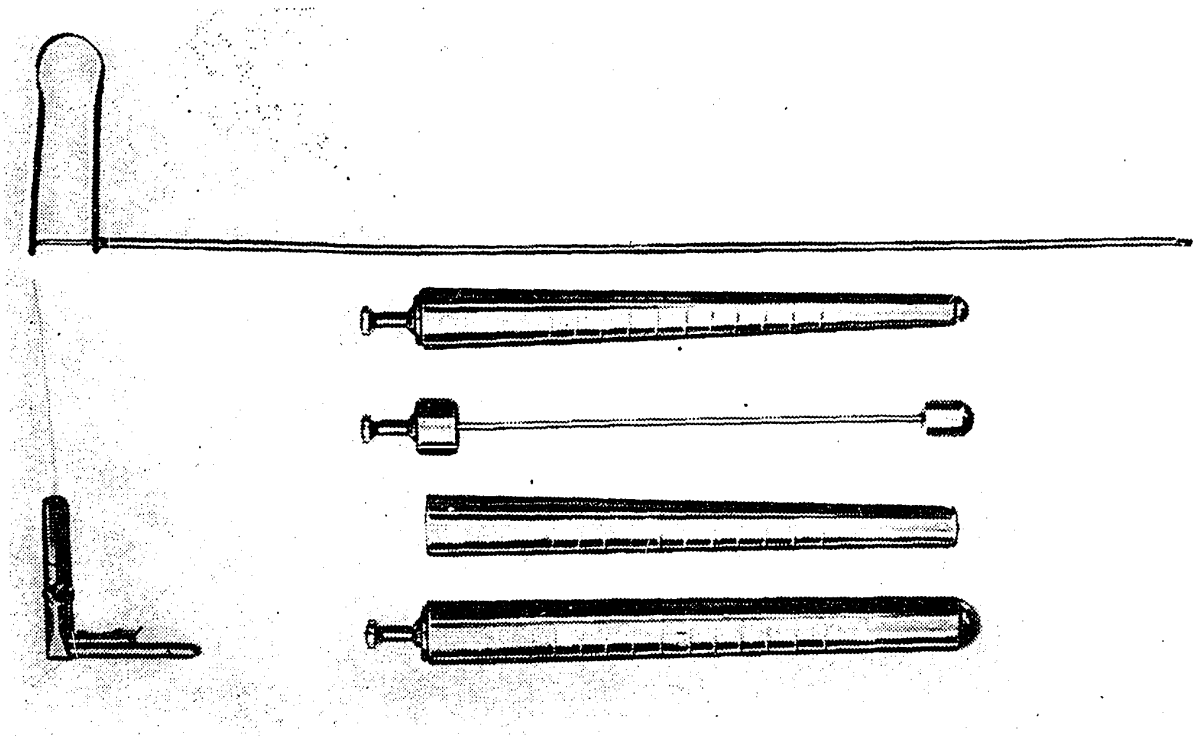
The following background data is given for the understanding of our Tocology Unit where the work was carried out. Our Unit is adjacent to our Case Room and is fully equipped for the care of the pregnant woman and her newborn child. Furthermore we have added the equipment for scalp vein sampling and determination of acid base balance in the foetus. Patients were first selected for the purpose of setting standard curves of acid base balance for the foetus in utero and at the same time to gain control data for uterine monitoring. They were therefore selected from three groups: 1) normal pregnant women from the Obstetrical Clinic at term and readily inducible, who were then admitted for elective induction; 2) similar patients already admitted to the Antepartum Wards; and 3) women already in spontaneous labour in the Case Room. In this manner we have produced what we feel are normal curves from which further work can be done and further conclusions drawn.

TECHNIQUES

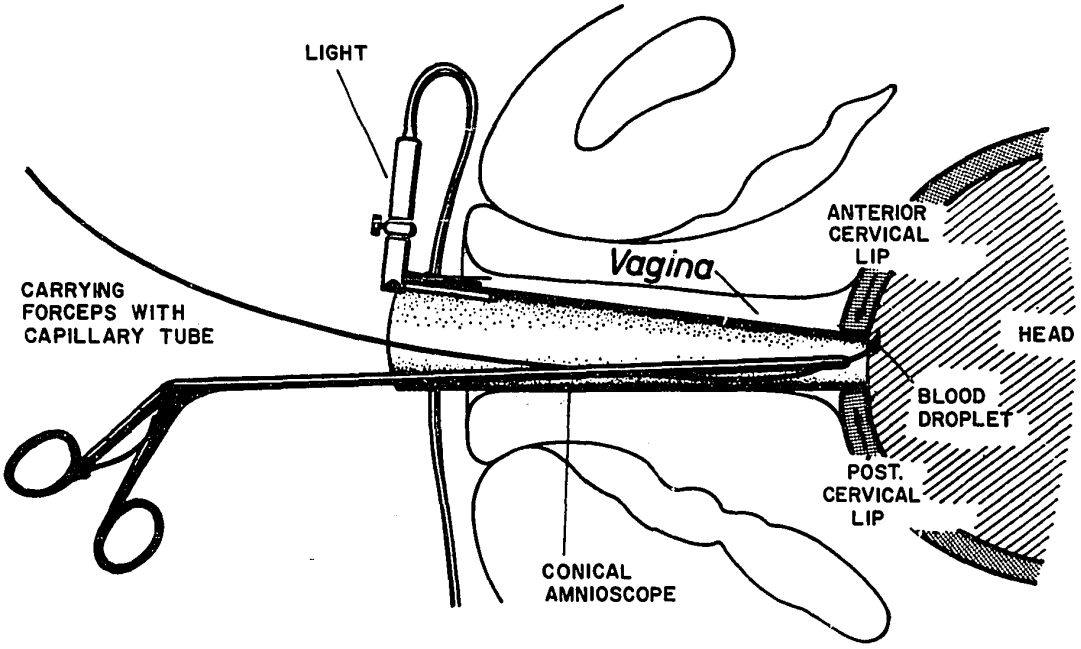
Preferably our patients were chosen either in the Clinic or on the Antepartum Ward at which point a placento gram was done to exclude patients with anterior location of the placenta. The patient was then scheduled for elective induction in our Tocology Suite the following morning. Prior to coming to our Tocology Unit the patient was given an enema, a minor prep, and was not given breakfast. She was then moved into our Tocology Unit where administration of an elaborate cocktail containing Demerol, Largactil, and glucose and water was set up intravenously with a steady and slow diffusion rate measured with a Harvard pump. A foetal electrode was then inserted into the foetal buttock using an amniocentesis approach and this served to record foetal heart rate at all times. This was fed into one channel of our six channel tocometer and was integrated to give us a foetal heart rate reading. Two manometers were then introduced into the amniotic cavity, again using an amniocentesis approach, and these were hooked up to two channels of our tocometer. A Syntocinon drip was then administered intravenously, again using a Harvard pump, starting at 1 or 2 milliunits of Syntocinon and rising as necessary to establish good contractions in the neighbourhood of about 8 to 16 milliunits. As soon as labour became well established and the patient had dilated her cervix to somewhere around 4 centimeters she was put in stirrups, in the lithotomy position, an artifical rupture of membranes was carried

out at the same time as a first scalp sampling (as will be described below), and an epidural anaesthesia was initiated. Disinfection of the skin was carried out with Germamedica and the patient was appropriately draped.

A vaginal examination was then performed to determine cervical dilatation and the level of the presenting part at which point the correct size of amnioscope was inserted through the external os of the cervix to lean against the presenting part. The amnioscope is illustrated in Figure 13.



An appropriate light source with a Rheostat was then attached to the amnioscope. The foetal scalp was then dried with a cotton swab and sprayed with ethyl chloride to produce a hyperemic effect. The scalp was then dried once again and a thin layer of white petrolatum jelly applied. An incision was made using the Queen Charlotte's Hospital modification (41) of Saling's technique with a Wilkinson's Sword blade. A drop of blood collecting at the site of the incision was removed anaerobically either through polyethylene or glass capillary tubing. Figure 14 shows this stage in cross-section with the foetal scalp on the right, the cervix dilating in front of it, the amnioscope in the vagina, and the light source attached to the amnioscope. The incision has already been made and the forceps carrying the capillary tube is introduced to the drop of blood for collection.



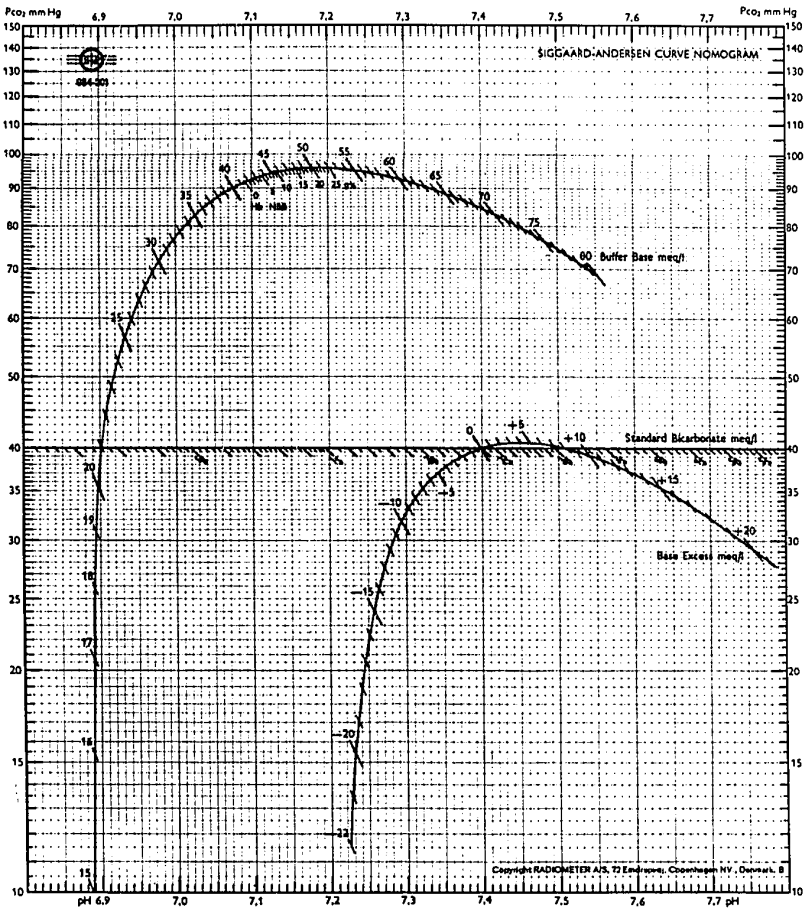
The specimen is then analyzed. Care must be taken at this point to ascertain that the scalp bleeding has ceased; the recommended technique is to apply pressure with a cotton swab to the site of the incision on the foetal scalp for approximately five minutes or until bleeding has completely ceased.

We started off with polyethylene tubing and since then have changed over to glass capillary tubing. Preparation is as follows: Forty centimeter lengths of polyethylene tubing are prepared by disinfecting them in a solution of Germamedica and then rinsing them in sterile distilled water. The tubes are then heparinized with 2,000 USP units per millimeter of Heparin, after the tubes have dried by gravitational methods. Each tube is then individually packaged and sterilization is done by gas autoclaving. Glass capillaries of 0.7 to 1.0 millimeter bores are prepared by washing 35 centimeter lengths in concentrated sulphuric acid and then rinsing them in sterile distilled water. The tubes are then dried at 120 degrees centigrade. Each tube is filled with a solution of Heparin again with 2,000 USP units per millimeter and dried at 120 degrees centigrade for about 10 days. Three sterile steel mixing wires are placed in each tube to mix the blood before analysis - the tubes are then packaged at five per pack since some of the tubes become blocked by crystals of Heparin and cannot be used.

Determination of the acid base balance was done with an Astrup apparatus at 38 degrees centigrade. pO_2 was measured in duplicate with a direct reading electrode on a total sample of

50 microliters of blood, repeatable with an error of plus or minus 1.0 millimeter of mercury. An oxygen free solution of borax containing sodium sulphate was used as the zero standard. Oxygen and nitrous gas mixtures with water saturated with oxygen were used as positive standards against barometric pressure in millimeters of mercury with an accuracy of 0.05%. pCO_2 was measured with a direct reading electrode using 3 CO_2O_2 gas mixtures as standard against barometric pressure with an accuracy of 0.05%. The pCO_2 was measured in duplicate indirectly with the equilibration technique employing the Astrup Micro Tonometer using the low and the medium of the above-mentioned CO_2O_2 gas mixtures as standard.

The pH determination required 25 microliters of blood with an accuracy of plus or minus 0.005 pH units after standardizing against radiometer (Copenhagen) phosphate buffers at pH 6.840 and pH 7.381 at 38 degrees centigrade. On no occasion was tonometered blood used for the actual calibration of the electrodes. The acid base status of all blood samples taken was obtained by plotting actual blood pH and tonometered blood pH's on a Siggaard-Andersen Nomogram (42, 43) as is illustrated in Figure 15.



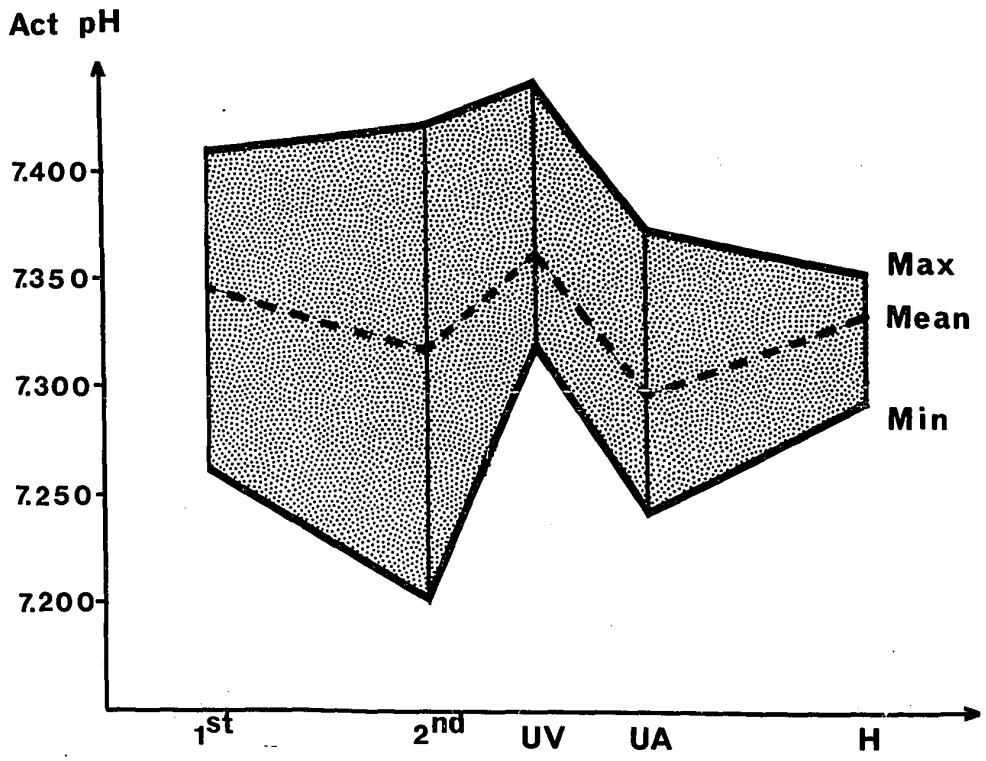
Patient's name:		Barometric pressure		mm Hg	READINGS			RESULTS	
		CO_2 percentage	Cylinder No 1:	%	Before equilibration	Actual pH:		Actual P_{CO_2}	mm Hg
Dept:	Sample No.:		Cylinder No 2:	%				Base Excess	meq/l blood
Date:		CO_2 partial pressure	Cylinder No 1:	mm Hg	After equilibration	high P_{CO_2}	pH:	Buffer Base	meq/l blood
Hour of Sampling:			Cylinder No 2:	mm Hg		low P_{CO_2}	pH:	Standard Bicarb.	meq/l plasma
Remarks:		Hemoglobin:		g/100 ml	Readings made by:			Actual Bicarb.	meq/l plasma
		Oxygen Saturation:		percent	Signature:			Total CO_2	meq/l plasma

References: Siggaard-Andersen, O. and Engel, K.: Scand. J. clin. Lab. Invest. 12, 177, 1960
Siggaard-Andersen, O.: Scand. J. clin. Lab. Invest. 15, 598, 1963

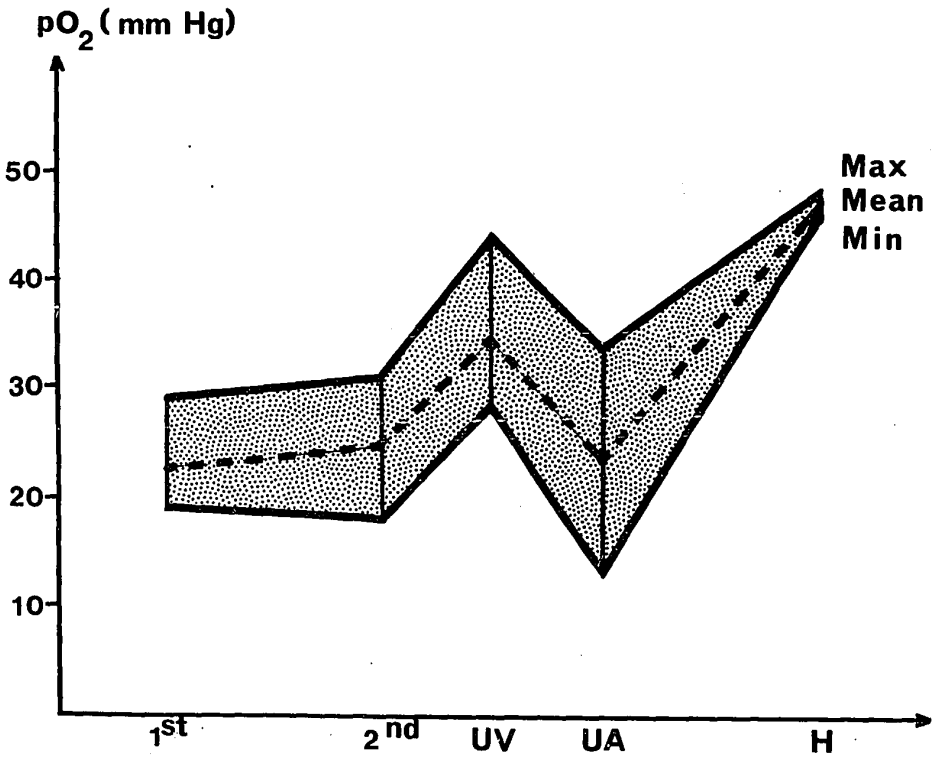
Such sampling was usually carried out during the first and second stages of labour, then on umbilical vein and artery, and at one-half hour of life on the newborn child by heel prick, so that essentially five determinations were done in each case. A total of 24 patients were examined in this manner.

RESULTS

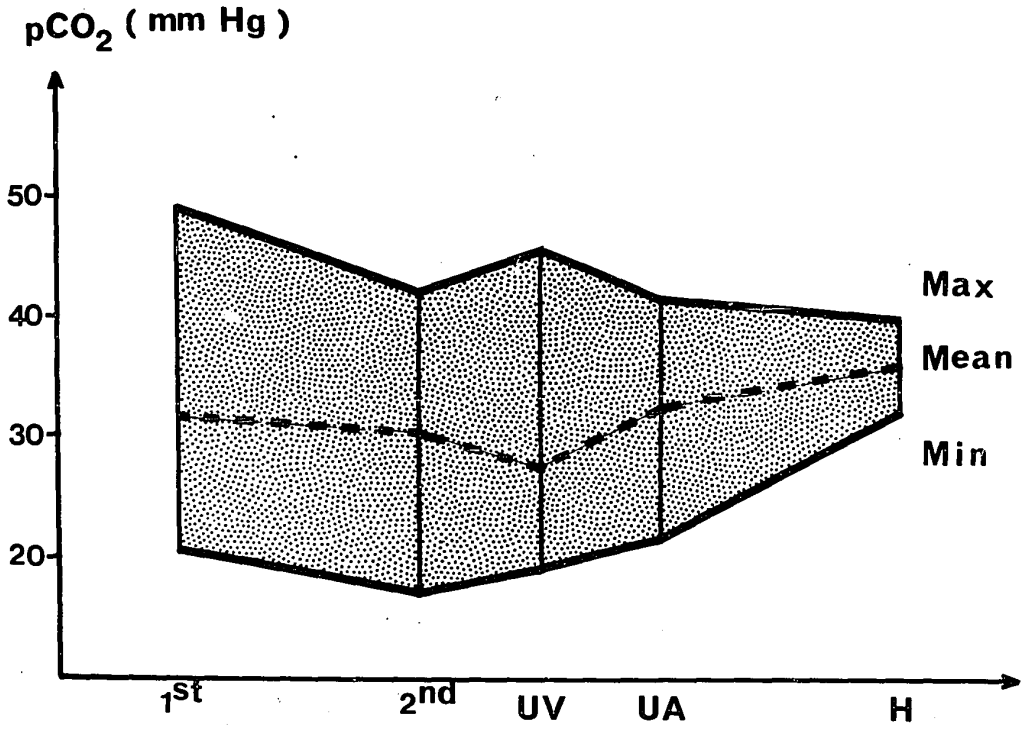
The results are shown graphically in Figure 16. Variation in pH was from a low value of 7.260 to a high value of 7.410 with a mean value of 7.346 during the first stage of labour. During the second stage of labour our mean value is 7.317 with a low value of 7.200 and a high value of 7.420. In the umbilical vein our mean value is 7.360 with a low value of 7.315 and a high value of 7.440. In umbilical artery our mean value is 7.294 with a low value of 7.240 and a high value of 7.370. The heel prick samples had a mean value of 7.330, a low value of 7.290 and a high value of 7.350.



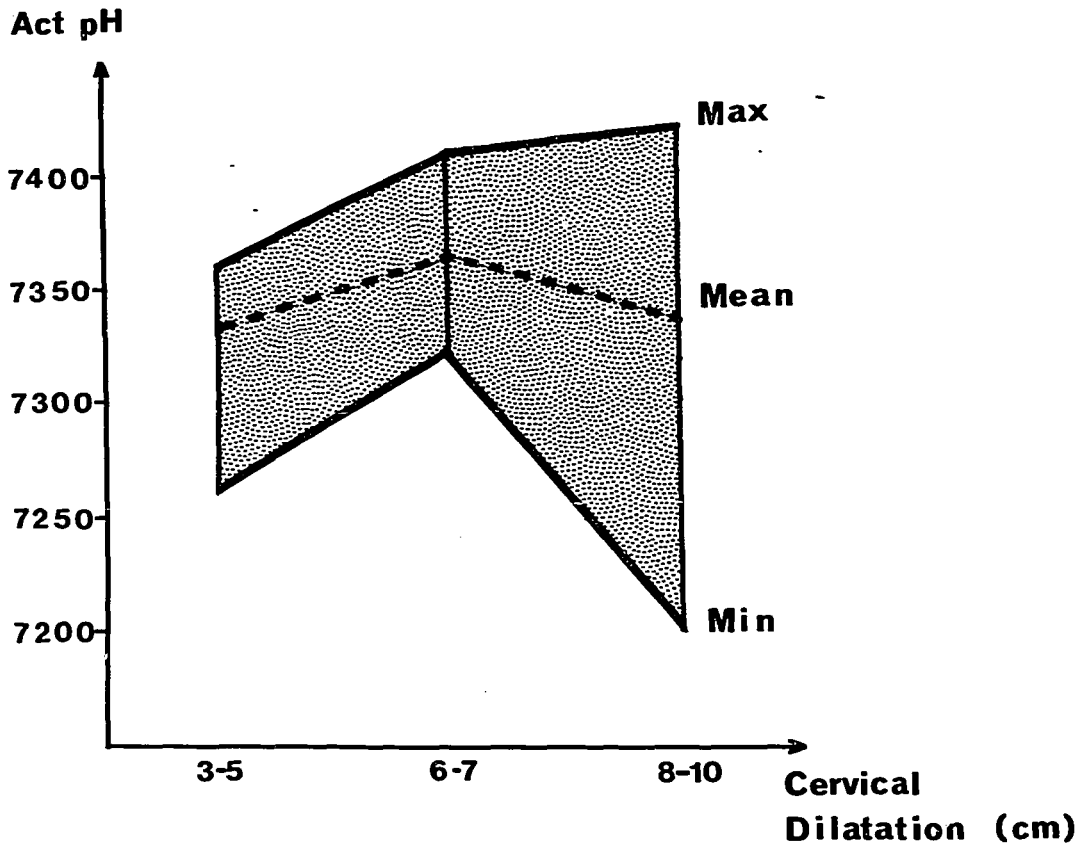
In looking at pO_2 in millimeters of mercury in the first stage of labour our mean was established at 22.4 millimeters of mercury with a low of 19 and a high of 29. In the second stage of labour our mean value was 24.8 millimeters of mercury with a low of 13 and a high of 31. In umbilical vein our mean value was 34.3 millimeters of mercury with a low of 28 and a high of 44. In umbilical artery our mean value was 23.4 millimeters of mercury with a low of 13 and a high of 34. In heel prick our mean value was 47.3 with a low of 46 and a high of 48.5. These values are shown in Figure 17.



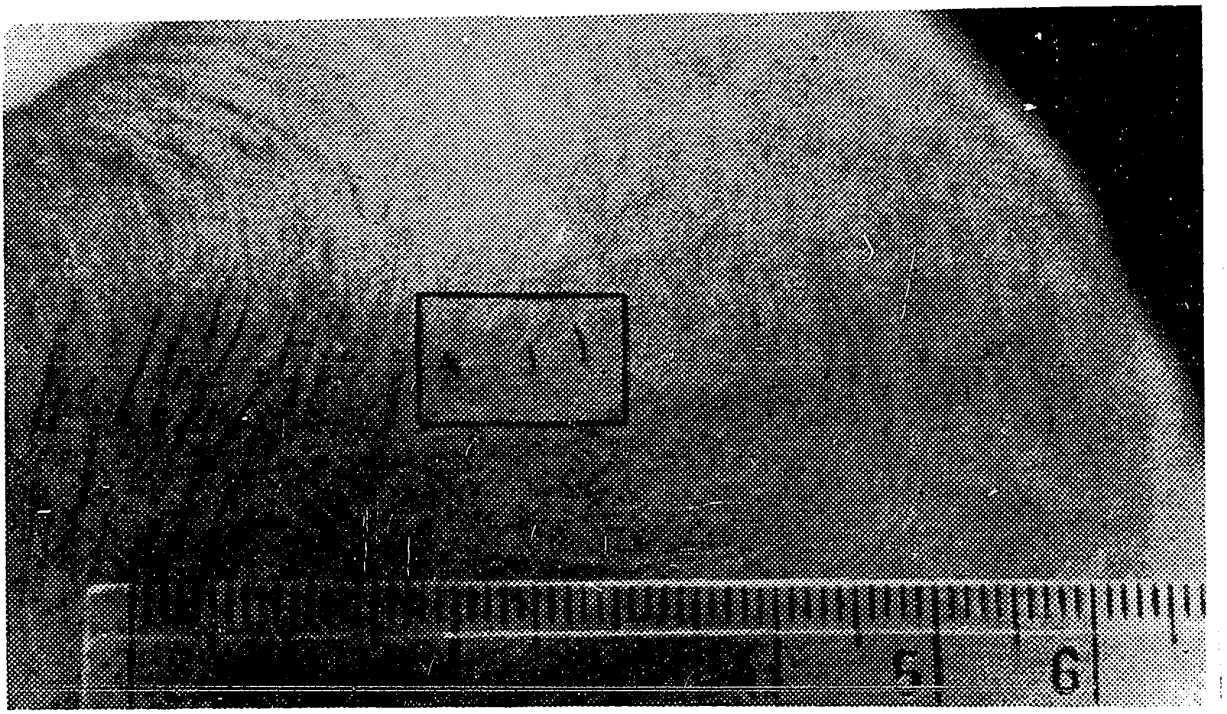
In looking at our results for pCO_2 determinations in the first stage of labour our mean value was 41.5 millimeters of mercury with a low of 30.5 and a high of 59.0. In the second stage of labour our mean value is 40.4 millimeters of mercury with a low of 27 and a high of 52. In umbilical vein our mean value is 37.5 millimeters of mercury with a low of 29 and a high of 45.5. In umbilical artery our mean is 42.3 millimeters of mercury with a low of 31.5 and a high of 61.5. In heel prick determination our mean value is 46.0 millimeters of mercury with a low of 42 and a high of 50. This again is illustrated in Figure 18.



We then correlated measurements of pH with cervical dilatation and we put together cervical dilatations of 3-5, 6-7, and 8-10 and found that our mean value for dilatation 3-5 centimeters was 7.332 with a low of 7.260 and a high of 7.360. At dilatation 6-7 centimeters our mean value was 7.363 with a low of 7.320 and a high of 7.410. At dilatation 8-10 centimeters our mean value was 7.334 with a low value of 7.200 and a high value of 7.420. This is again illustrated in Figure 19.



It should be noted that no serious injury is caused to the foetal scalp. Figure 20 shows the area on the foetal scalp where three scalp samplings were performed. These usually heal within 24 hours of life and are not recognized by the sharp eye of the examining mother. (8)



In looking over our results it becomes evident that the pH is lower in the second stage than it is in the first stage, that the pH is lower in umbilical artery than it is in umbilical vein and that it tends to come back to normal in the heel prick sample.

The values for pO_2 are practically identical in the first and second stages but, as expected, the pO_2 is much lower in the umbilical artery than it is in the umbilical vein and, again as expected, it is much higher in the heel prick sample. Correlating pH with cervical dilatation we find that pH rises during the first part of labour up to 6-7 centimeters and then decreases slightly to its original value at 8-10 centimeters of cervical dilatation. All babies had apgar scores of 8-10 at one minute of age.

It is our contention that these curves represent normal values for a foetus in utero and are within the range of world-wide accepted values.

CONCLUSION

Foetal distress equals foetal acidosis. By the fusion of two techniques, i.e., uterine monitoring, foetal heart monitoring and scalp vein sampling in the assessment of both foetus and mother during labour, Caldeyro-Barcia showed that if the pO_2 drops to below 18 mm. Hg., late deceleration will appear in the foetal heart rate and at the same time the pH will drop to 7.20 or below, so that foetal distress can be equated with foetal acidosis and also with foetal hypoxia and foetal anoxia. (44, 45, 46, 47, 48)

It is our thinking that all these proven techniques should be carried a step further in the assessment of mother and foetus at risk in an effort to further reduce maternal mortality and perinatal mortality and to gain more insight into the normal and abnormal uterine dystocias. Since it is the privilege of our decade to penetrate the inner sanctum of the foetus at the same time some knowledge of foetal physiology and physiopathology will certainly be uncovered.

It is felt that these techniques in routine obstetrical practice will eventually succeed in diminishing neurological deficits, will increase the apgar score at birth, will decrease the stillbirth rate and will decrease the Caesarean section rate or will succeed in justifying intervention by Caesarean section.

OUTLOOK TO THE FUTURE

The technique and its use for detection and/or diagnosis of foetal distress is just one aspect of its usefulness. It gives us food for thought since it represents the availability of the foetus to the foetal physiologist by making unknown material now accessible in a relatively easy fashion. The technique has already been used by Wood in elucidating the physiology of the foetal pancreas, the insulin response of the foetus to over-loading of the mother by intravenous glucose, and the response of the foetal pancreas to injection of glucagon into the foetal scalp.(49) It has been used in certain centres in erythro blastosis foetalis in determining the foetal hemoglobin and making blood available for cross-matching prior to birth. This of course is of very limited use and does not necessarily represent a world-wide consensus.

One last application for this technique which comes to mind is that recent work has shown the importance of human placental lactogen determinations in assessing chronic foetal distress or metabolic acidosis. The barrier from placenta to mother and from placenta to foetus has not been well determined. Possibly this barrier can be side-stepped by simultaneous maternal and foetal sampling for HPL determinations.

The technique is now easily available. Its application in any specific area requires only a researcher's interest.

REFERENCES

1. Eastman, N.J., and Hellman, L.M. Williams Obstetrics. 13th ed. N.Y., Appleton-Century-Crofts, 1966, p. 1.

2. Prechtl, H. Neurologic Sequelae of Prenatal and Perinatal Complications. Brit. Med. J. 4: 763, 1967.

3. Rosen, M.G. Effects of Asphyxia on the Fetal Brain. Obstet. Gynec. 29: 687, 1967.

4. Dawes, G.S., Hibbard, E., and Windle, W.F. Asphyxia at Birth and Brain Damage. J. Physiol. 171: 8P, 1964.

5. Krokfors, E., Wist, A., and Hirvensalo, M. Effect of Duration of Labour, Age and Parity on the Apgar Score of the Newborn Infant. Ann. Chir. Gynaec. Fenn. 52: supp. 118, 1963.

6. Windle, W.F. Neuropathology of Certain Forms of Mental Retardation. Science 140: 1186, 1963.

7. Burnard, E.D. The Relative Dangers of Asphyxia and Mechanical Trauma at Birth. Med. J. Aust. 2: 487, 1962.

8. Saling, E. Das Kind im Bereich der Geburtshilfe. Stuttgart, Georg Thieme Verlag, 1966.

9. Saling, E. Amnioscopy. Triangle 7: 122, 1965.

10. Saling, E. A New Method of Safeguarding the Life of the Foetus Before and During Labour. J. Int. Fed. Gynaec. Obstet. 3: 100, 1965.

11. Saling, E. Amnioscopy and Foetal Blood Sampling: Observations on Foetal Acidosis. Arch. Dis. Child 41: 472, 1966.

12. Saling, E. Amnioscopy. Clin. Obstet. Gynec. 9: 472, 1966.

13. Kubli, F., and Berg, D. The Early Diagnosis of Foetal Distress. J. Obstet. Gynaec. Brit. Comm. 72: 507, 1965.

14. Kubli, F.W. Influence of Labor on Foetal Acid-Base Balance. Clin. Obstet. Gynec. 11: 168, 1968.

15. Kornacki, Z., Biczysko, R., and Jakubowski, A. Amnioscopy as a Routine Obstetric Examination in the Later Stages of Pregnancy and at the Beginning of Labor. Amer. J. Obstet. Gynec. 101: 539, 1968.

16. Bretscher, J., and Saling, E. pH Values in the Human Fetus During Labor. Amer. J. Obstet. Gynec. 97: 906, 1967.

17. Detecting Foetal Hypoxia. Brit. Med. J. 2: 421, 1966.

18. Eastman, N.J., and Hellman, L.M. Williams Obstetrics. 13th ed. N.Y., Appleton-Century-Crofts, 1966, p. 190.

19. Assali, N.S., Holm, L.W., and Sehgal, N. Hemodynamic Changes in Fetal Lamb in Utero in Response to Asphyxia, Hypoxia, and Hypercapnia. Circ. Res. 11: 423, 1962.

20. James, L.S., and Adamson, K., Jr. Respiratory Physiology of the Fetus and Newborn. New Eng. J. Med. 271: 1352 and 1403, 1964.

21. Weston, P.V., and Crichton, D. The Diagnosis of Intra-uterine Asphyxia by Micro-blood Analysis of the Foetus in Utero. South Afr. J. Obstet. Gynaec. 3: 61, 1965.

22. Paul, W.M., Gare, D.J., and Whetham, J.C. Assessment of Foetal Scalp Sampling in Labor. Amer. J. Obstet. Gynec. 99: 745, 1967.

23. Newman, W., McKinnin, L., Phillips, L., Paterson, P., and Wood, C. Oxygen Transfer from Mother to Fetus During Labor. Amer. J. Obstet. Gynec. 99: 61, 1967.

24. Newman, W., Braid, D., and Wood, C. Fetal Acid-Base Status. Amer. J. Obstet. Gynec. 97: 43, 1967.

25. Macdonald, R.R., and Kelman, G.R. Problems of Foetal Blood Sampling. J. Obstet. Gynaec. Brit. Comm. 74: 826, 1967.

26. McDonald, J.S. Evaluation of Fetal Blood pH as a Reflection of Fetal Well-being. Amer. J. Obstet. Gynec. 97: 912, 1967.

27. Huntingford, P.J. A Direct Approach to the Study of the Foetus: Report of a New Technique from West Berlin for the Early Detection of Intrauterine Asphyxia. Lancet 1: 95, 1964.

28. Gare, D.J., Whetham, J.C.G., and Henry, J.D. The Validity of Scalp Sampling. Amer. J. Obstet. Gynec. 99: 722, 1967.

29. Bowe, E.T. The Assessment of Fetal Acid-Base Status During Labor: A Preliminary Report on a Method for the Early Detection of Fetal Distress. Bull Sloane Hosp. 13: 6, 1967.

30. Sampling of Foetal Blood. Lancet 1: 798, 1968.

31. Leonard, J.L. The Significance of Meconium-Stained Amniotic Fluid in Cephalic Presentation. Obstet. Gynec. 20: 320, 1962.

32. Edelstein, H. The Significance of Meconium in Foetal Distress. South Afr. Med. J. 35: 622, 1961.

33. Bailey, N. Amnioscopy. South Afr. J. Obstet. Gynaec. 5: 12, 1967.

34. Barham, K.A. The Use of Amnioscopy in the Detection of Meconium. Aust. New Zealand J. Obstet. Gynaec. 8: 9, 1968.

35. Bowe, E.T. Fetal Distress. Bull Sloane Hosp. 14: 4, 1968.

36. Kumar, D. Intrauterine Diagnosis, Indices of Fetal Jeopardy and Intrauterine Therapy. In Barnes, A.C., ed. Intrauterine Development. Phila., Lea & Febiger, 1968, p. 477.

37. Symposium on Intrauterine Foetal Distress. Prague, Oct. 11-14, 1966. J. Int. Fed. Gynaec. Obstet. 5: 39, 1967.

38. Wood, C., Lumley, J., and Renou, P. A Clinical Assessment of Foetal Diagnostic Methods. J. Obstet. Gynaec. Brit. Comm. 74: 823, 1967.

39. Morris, E.D., and Beard, R.W. The Rationale and Technique of Foetal Blood Sampling and Amnioscopy. J. Obstet. Gynaec. 72: 489, 1965.

40. Beard, R.W., Morris, E.D., and Clayton, S.G. Haemorrhage Following Foetal Blood Sampling. J. Obstet. Gynaec. 73: 860, 1966.

41. Beard, R.W., Morris, E.D., and Clayton, S.G. Foetal Blood Sampling in Clinical Obstetrics. J. Obstet. Gynaec. Brit. Comm. 73: 562, 1966.

42. Beard, R.W., Morris, E.D., and Clayton, S.G. pH of Foetal Capillary Blood as an Indicator of the Condition of the Foetus. J. Obstet. Gynaec. Brit. Comm. 74: 812, 1967.

43. Adamsons, K., Jr., Beard, R.W., Bowe, E.T., and James, L.S. Standard Practices at Sloane Hospital: Fetal Blood Sampling. Bull Sloane Hosp. 13: 11, 1967.

44. Siggaard-Andersen, O., and Engel, K. A New Acid-Base Nomogram: An Improved Method for the Calculation of the Relevent Blood Acid-Base Data. Scand. J. Clin. Lab. Invest. 12: 177, 1960.

45. Siggaard-Andersen, O. The pH-log pCO_2 blood Acid-Base Nomogram Revised. Scand. J. Clin. Lab. Invest. 14: 598, 1962.

46. Walker, A., Phillips, L., Powe, L., and Wood, C. A New Instrument for the Measurement of Tissue pO_2 of Human Fetal Scalp. Amer. J. Obstet. Gynec. 100: 63, 1968.

47. Wood, C., Ferguson, R., Leeton, J., Newman, W., and Walker, A. Fetal Heart Rate and Acid-Base Status in the Assessment of Fetal Hypoxia. Amer. J. Obstet. Gynec. 98: 62, 1967.

48. Mendez-Bauer, C., Arnt, I.C., Gulin, L., Escarcena, L., and Caldeyro-Barcia, R. Relationship Between Blood pH and Heart Rate in the Human Fetus During Labor. Amer. J. Obstet. Gynec. 97: 530, 1967.

49. Althabe, O., Jr., Schwarcz, R.L., Pose, S.V., Escarcena, L., and Caldeyro-Barcia, R. Effects of Fetal Heart Rate and Fetal pO_2 of Oxygen Administered to the Mother. Amer. J. Obstet. Gynec. 98: 858, 1967.

50. Lee, S.T., and Hon, E.H. Fetal Hemodynamic Response to Umbilical Cord Compression. Obstet. Gynec. 22: 553, 1963.

51. Hon, E.H. The Classification of Fetal Heart Rate. I. A Working Classification. Obstet. Gynec. 22: 137, 1963.

52. Graham, F.K., Ernhart, C.B., and Thurston, D. The Relationship of Neonatal Apnea to Development at Three Years. Res. Publ. Ass. Res. Nerv. Ment. Dis. 39: 159, 1962.

53. Paterson, P., Phillips, L., and Wood, C. Relationship Between Maternal and Fetal Blood Glucose During Labor. Amer. J. Obstet. Gynec. 98: 938, 1967.