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Fetal Cardiac Function and Asphyxia

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William A. O'Brien, M.D.

I. LAST WEEK

Date: November 19, 1943

Place: Recreation Room, Powell Hall

Time: 12:15 to 1:15 p.m.

Program: "Guillain-Barré's Disease"
A. B. Baker

Discussion: J. C. McKinley
Renold Jensen
Alvin Wert

Attendance: 121
Alice Carlson,
Record Librarian

- - -

II. MEETINGS

1. ANATOMY SEMINAR

Saturday, November 27, 1943, 11:30 a.m., Room 226, Institute of Anatomy.

"The Growth of the Human Hypophysis during Childhood"
A. T. Rasmussen

"Secretory Function of Cells in the Hypothalamus"
R. N. Winger

- - -

2. PATHOLOGY SEMINAR

Monday, November 29, 1943, 12:30 p.m., Room 104, Institute of Anatomy.

"Hyperparathyroidism with Special Emphasis on the Biochemical Syndrome"
E. B. Flink

- - -

3. PREVENTIVE MEDICINE AND PUBLIC HEALTH SEMINAR

Monday, November 29, 1943, 4:00 p.m., Room 116, Millard Hall.

"Incidence of Caries"
Alan Treloar

4. PHYSIOLOGY AND PHARMACOLOGY SEMINAR

Tuesday, November 30, 1943, 12:30 p.m., Room 214, Millard Hall.

"Estrogens and Mammary Cancer in Mice"
J. J. Bittner

- - -

5. PHARMACOLOGY SEMINAR

Wednesday, December 1, 1943, 12:30 p.m., Room 105, Millard Hall.

"Trypanosomiasis in the Americas"
H. M. Wright

- - -

6. BACTERIOLOGY SEMINAR

Thursday, December 2, 1943, 4:30 p.m., Room 129, Millard Hall.

"The Psittacosis Virus and Related Viruses."
Mary Muedeking

- - -

7. E. STARR JUDD LECTURESHIP IN SURGERY

Monday, December 6, 1943, 8:15 p.m., in the Museum of Natural History Auditorium.

"Surgery in War"
Major General Norman T. Kirk

- - -

III. FROM THE MAIL BAG

Would certainly appreciate getting the bulletins. Am down here where I see very little medicine. Routines of Navy seem to be of prime importance. Am feeling very well, however. Say hello to my friends at the hospital.

Lt. Comdr. I. M. Goldberg

- - -

I certainly enjoy my bulletin more than ever since I am in the service.

C. E. Miller,
Capt. M.C.

IV. FETAL CARDIAC FUNCTION AND ASPHYXIA

Curtis Lund

For purposes of comparison we may consider the fundamental facts of human developmental anatomy rather complete; by contrast, the facts of fetal physiology are relatively incomplete. These lacunae in our knowledge are now being closed by investigative work which will eventually make fetal function well understood. However, it is unlikely that research upon the human will keep pace with animal research for the human fetus remains almost inaccessible until the moment of delivery when at that same moment it ceases to be a fetus.

The most tragic result of fetal asphyxia is death. That such an end is not uncommon becomes apparent as we review mortality statistics. The Children's Bureau of the United States Department of Labor[1] in their study of infant mortality observed the following trends:

a. Deaths under one year were cut in half from the years 1915 to 1937.

b. Deaths under one month were reduced by 25 per cent.

c. Deaths under one day remained unchanged.

d. From 1922 to 1936 the incidence of still births was reduced only 15 per cent.

In recent years additional improvement has been made; yet, a total of 1,332,444 fetuses and infants under 1 year of age have been lost during the last five year period. Of these deaths 519,349 were still births and 813,005 were infants under one year.[2] Of the latter approximately 225,000 (30 per cent) died during the first 24 hours of life.

It is obvious that abnormalities of the fetus and certain unpreventable complications of pregnancy account for an irreducible portion of these deaths. Nevertheless this does not justify the tendency of some to accept these deaths as something predestined or inevitable. The fact that several large maternity services[3] have reduced their still births and neonatal deaths by one-third is evidence to the contrary. If this improvement could be effected on a national scale about 100,000 lives would be saved yearly.

In the broad technical sense it might be said that all of these infants died of asphyxia, which by definition implies interference with oxygen supply and carbon-dioxide removal. In the more specifi sense, which excludes trauma and certain other abnormalities, primary asphyxia is said to be responsible for 25 to 50 per cent of all still births.[3]

But death is not the only serious result of intrauterine asphyxia. Indeed in some respects the permanent damage to the central nervous system which may result from asphyxia is worse for the infant and the parents as well. Although such tragedies are not common one need not look far for examples. The ease wich which the accident may happen is well illustrated by the following experience. A patient, not in labor, was being delivered by elective cesarean section because of a contracted pelvis. During the procedure certain technical difficulties of anesthesia resulted in maternal respiratory obstruction. Although this existed for no more than 10 minutes before delivery, the infant was severely asphyxiated. After 30 minutes of artificial resuscitation the infant breathed spontaneously. Within a few hours signs of intracranial hemorrhage appeared and bloody spinal fluid was obtained following diagnostic lumbar puncture--a sign of the characteristic petechial hemorrhages of anoxia. Obviously, delivery was without trauma. Signs of mental deterioration appeared later, an encephalogram demonstrated the presence of extensive cortical atrophy. The child is now a permanent resident of an institution for the feebleminded.

A third dangerous result of fetal asphyxia is the production of neonatal asphyxia. In such cases the infant has apnea at birth because of a direct depression of the respiratory center by

anoxia. Oxygen want in the milder degrees stimulates respiration activity but severe anoxia depresses. As a rule these infants do not die or become permanently damaged if the true state of affairs is recognized and promptly treated. Nevertheless, asphyxia neonatorum is never to be taken lightly. While we are not discussing the treatment of asphyxia neonatorum today, a word concerning the treatment of this condition as related to fetal anoxia is of practical importance. When intrauterine oxygen want has reached the point where the respiratory center is depressed, the infant fails to breathe at birth. Apnea increases the anoxia which is already too great. When dealing with this type of asphyxia neonatorum it is wise to administer oxygen to the infant without delay by means of artificial resuscitation. This is one time when early intervention is essential.

The fetus in utero leads what might be called a circulatory existence. The placenta is the organ for respiration and as long as its function and that of the cardio-vascular system are intact, even in the presence of gross developmental anomalies, the fetus usually survives until extra-uterine life begins. The respiratory system is of little or no importance for intrauterine survival even though it is active or capable of activation. Yet, at the moment of birth respiratory function becomes vital.

Time does not permit a review of the details, still incomplete, of transfer of oxygen from mother to infant. The presence of a special type of fetal hemoglobin permits the fetus to take up oxygen at partial pressures which are physiologic in the placenta and which causes the mother's blood to lose oxygen [4,5]. Such a shift of the fetal oxygen dissociation curve to the left also makes the giving up of oxygen to the tissues less easily effected. This is not a serious obstacle, for the fetus in utero is relatively inactive and is well insulated at a constant temperature and hence can get along with a slower tissue respiration. Many other factors add to the complexity of the situation. For example, anaerobic oxidation, of which we know little, must be considered as an important source of oxygen for fetal metabolism. Furthermore the experimental fetal animal can survive far more anoxia than can its mother.[6,7] These then are illustrations of some of the problems which complicate a study of normal fetal respiration.

Admittedly, we know neither the amount nor the duration of anoxia necessary to produce undesirable sequelae in the human fetus or newlyborn infant. As was previously stated, the fetus, at least in the experimental animal, can withstand more anoxia than the mother. On the other hand clinical evidence suggests that this might not be true in the human. The following clinical incident is illustrative: A primigravida, ner term, was hospitalized because of a severe hypertensive heart disease and toxemia of pregnancy. One evening this patient developed acute pulmonary edema. The fetal heart tones, which had been normal just before the attack, were absent within 12 minutes in spite of almost immediate therapy. In this instance the anoxia was sufficient to destroy the fetus but not the mother--the macerated stillborn infant was delivered some time later.

Anoxia is a term which is loosely applied to indicate oxygen deficiency of an unspecified degree and might be better known by the more appropriate term hypoxia. Our existing classifications have been made using the term as defined above and this practice will be continued here. There are four types of anoxia commonly recognized:

1. Anoxic anoxia, which indicates a deficiency of oxygen saturation of the blood, usually due to failing or inefficient respiration.

2. Anemic anoxia, which means the oxygen carrying capacity of the blood is diminished.

3. Stagnant anoxia, which means there is inefficient circulation of normally oxygenated blood.

4. Histotoxic anoxia, which implies that a given cell cannot utilize

oxygen though it is present in normal amounts.

Whatever the type of maternal anoxia, it will produce an anoxic anoxia of the fetus. Maternal anoxia may be produced in several ways. The simplest form follows a deficiency of oxygen in the atmosphere. Clinically this is not common during administration of inhalation analgesic or anesthetic agents; more specifically with the improper use of certain agents such as nitrous oxide and ethylene in high concentrations without adequate amounts of oxygen. Eastman[8] has reported marked unsaturation of fetal hemoglobin when the mother was given nitrous oxide in concentrations above 90 per cent. Cullon has recently reported[10] nitrous oxide anesthesia without anoxia while using 80 per cent nitrous oxide and 20 per cent oxygen. His patients were well sedated with premedication before induction of anesthesia. Some of our experiences will be mentioned later.

Obstruction of the respiratory passages is a frequent cause of maternal anoxia. The relaxation of pharyngeal muscles during deep narcosis or anesthesia may cause partial obstruction and evidence of snoring or sterterous respiration should be considered a danger signal. Metabolic diseases and increased metabolic activity contribute to anoxia: hyperthyroidism, hyperpyrexia, diabetes, excessive excitement and bearing down efforts during second stage of labor are good examples. Anemia, hemorrhage and shock are common causes of anemic anoxia. Maternal heart disease may readily produce stagnant anoxia. Our knowledge of histotoxic anoxia is limited so discussion of it is not warranted.

Localized maternal anoxia may be present when the uterine contractions are too forceful or frequent, or when there are placental abnormalities such as premature separation, placenta previa or excessive infarction.

All of these maternal complications and many others may produce anoxic fetal anoxia. Partial or complete obstruction of umbilical cord acts similarly. The other types of fetal anoxia are exceedingly rare; erythroblastosis produces anemic anoxia as does rupture of the vessels of the umbilical cord; congenital heart disease occasionally produces stagnant anoxia. More important in this respect is the interrelationship between anoxic anoxia and stagnant anoxia which, as we shall see later, act together in the form of a vicious cycle.

The rate and rhythm of the fetal heart gives us the only direct information for continuous study of the fetus during pregnancy and labor. Other method of study in the human, whether clinical or experimental, give data which are self-limited. By this I mean a single observation which cannot be repeated or re-checked in the same individual as, for example, the presence of meconium or the determination of blood oxygen and carbon-dioxide at the time of birth. By far the best animal experimentation has been done by Sir Joseph Barcroft[10,11] whose studies of sheep have been most valuable. Yet, we cannot apply his research directly to the human fetus because of radical differences in type of placentation, uterine physiology and morphology.

The three signs which clinicians commonly use to diagnose fetal asphyxia are as follows:

1. Excessive fetal movement or convulsions. At best this is an elusive if not delusive, sign. In the first place it is very difficult to determine which movements are excessive and which are normal. Our studies suggest that the fetus is active during early labor but becomes less active as labor progresses. Convulsive movements are usually agonal in nature and are of little practical importance.

2. Evidence of Meconium. This sign is widely used for the diagnosis of fetal distress but we have discarded it as having little value. To be worthy of consideration the following conditions must be fulfilled:

   a. The membranes, obviously, must be ruptured.

b. The presentation must be cephalic. If breech, the meconium is squeezed out of the infant.

c. The meconium must be fresh. If the meconium stained fluid escapes when the membranes rupture can we say when fetal distress was present- - a minute or an hour before?

d. The mother should not have had drugs, such as quinine, for induction of labor.

In the study to be reported today we noted 24 (10%) of 250 patients exhibited meconium during labor. Only two infants were asphyxiated at delivery and in neither case could the asphyxia be related to the meconium; one was asphyxiated by analgesics and a difficult forceps delivery, the other by anesthesia at the time of cesarean section. Six of these 24 women had medical induction of labor with castor oil and quinine. On the other hand, there is little doubt that an asphyxiated infant may pass meconium and we observed slowing of the fetal heart rate in 12 (50%) of the above cases at some time during labor.

3. Irregularities of Fetal Heart Rate.

Without a doubt changes of cardiac rate are the best criteria we have, at the present time, for determination of fetal asphyxia.

Credit for the first description of the fetal heart sounds is usually given to Mayor, a Geneva surgeon, who published his results in 1818, although the first recognition of the best has been credited to Le Goust (1650) by Fasbender.[12] Kergaradec, an obstetrician and associate of Laennec, presented a complete monograph of his observations to the Royal Academy of Science of Paris in 1822: a treatise so thorough and accurate that mary of its concepts are tenable today. Some of the early investigators, notably Kennedy, Naegle, Kohl and later Cummings, observed that the fetal heart became irregular when the uterus was tetanically contracted or when there was disproportion between the fetal head and bony pelvis. Prognostic significance was attached to variations in heart rate by von Winckel in 1893.[13] He thought that asphyxia was imminent when the rate exceeded 160 or decreased below 100 and advised immediate delivery if possible. This principle was widely accepted and has become a standard rule of obstetric teaching. In 1917 Baumm[14] upheld von Winckel's opinion as regards fetal tachycardia and even went so far as to say that efforts toward delivery were useless once tachycardia appeared. It should be noted that Baumm's opinions were based on the postmortem observation of intracranial hemorrhage in 11 babies. After the autopsy had disclosed the cause of death the obstetrical records were examined and tachycardia was found to have been present in every case. The report also mentioned, without emphasis, that most of the labors were arduous and that two of them were terminated by difficult forceps delivery. More recent reports[15,16,18] suggest that tachycardia is not as dangerous as was formerly believed. On the other hand all agree that slow fetal heart rates are serious and frequently foretell subsequent disaster for the fetus.

The chief hindrance to the study of fetal cardiac function has been the lack of suitable recording apparatus. True, the sounds can be heard and counted by usual methods of auscultation, and such methods are adequate for ordinary use but they do not detect rapid changes with accuracy and do not provide permanent records. The development of electronic research in the last decade has made possible the auscultation, amplification and recording of heart sounds by the phonocardiograph. We used an instrument of this type.[17] The sounds were picked up by a crystal microphone placed over the abdomen, the sounds were amplified and then could be heard at room volume over a loudspeaker or could be recorded on a strip of paper which moved at a constant speed of one or two inches per second.

During the early part of labor the heart rate was recorded every fifteen minutes or more often if indicated. As labor progressed the observations were made at more frequent intervals and during the late first and entire second stage of labor continuous auscultation was carried out. The basic rate was obtained

from 30 second counts made during the late interval between contractions. Shorter periods of counting or counts made during the contraction or immediately thereafter are frequently inaccurate. These seemingly unimportant points have been mentioned in some detail as far too many conclusions have been drawn from inaccurate and incomplete observations. As we shall see, the fetal heart rate at best is quite labile and because of this fact isolated observations mean very little.

Before attempting to discuss abnormalities of fetal heart rate it is necessary to define and understand the normal. It is customary to accept any rate between 120 and 160 as normal. If the rate falls below or exceeds these numbers the fetus is said to be in potential danger, and rates below 100 or above 180 are usually considered to be indicative of fetal asphyxia.

Much of this research had to do with the establishment of normal function and even that is not yet clearly established. Very early one fact became increasingly apparent: persistent fetal tachycardia (rate above 160) was neither common nor dangerous.[18] Transient tachycardia was common and without jeopardy. A review of the first 250 protocols showed that 58 (23.2%) infants had rates in excess of 160 at some time during labor.

Incidence of Fetal Tachycardia in 250 Deliveries

	No. of Cases	Per Cent
* Persistent (Rates maintained for 20 min.)		
Minimum Rate above 160	6	2.4
Minimum Rate above 170	5	2.0
Minimum Rate above 180	3	1.2
Minimum Rate above 190	0	0.0
Total	14	5.6
Transient		
Maximum rate between 160-169	29	11.6
Maximum rate between 170-179	9	3.6
Maximum rate between 180-189	6	2.4
Maximum rate over 190	0	0.0
Total	44	17.6
Grand Total	58	23.2

*Rates may have been higher for a short time but never lower. Thus, two infants exhibiting rates of 210 and 214 were included in this group.

The incidence of transient tachycardia is undoubtedly greater than these figures indicate and would have been recorded had it been possible to auscultate continuously during early labor. We have found that heart rates tend to be more rapid at the onset of labor. Sontag and Newberry[19] recorded rates above 160 in 75% of all fetuses at some time during the last two months of pregnancy.

There was no evidence that tachycardia was associated with asphyxia neonatorum. Of these 58 babies one was mildly asphyxiated, a 10½ lb. first-born whose delivery was delayed 5 minutes because of dystocia due to large shoulders. Two were moderately asphyxiated, one had received a large amount of pentobarbital and the other was delivered by a difficult forceps extraction. There was no severe asphyxia. There was one stillbirth-- a craniotomy because of cephalopelvic disproportion in a breech presen-

tation. Six of the fetuses had heart rates in excess of 160 just before birth and none was asphyxiated.

The cause of persistent tachycardia remains obscure. On the other hand it is possible to discover the cause of temporary tachycardia at least half of the time. The following were observed:

Causes of Transient Tachycardia

Fetal movements	5
Forceps Application	2
Pressure against perineum	7
Uterine contractions	6
Rectal or Vaginal examination	2
Unexplained	22
Total	44

At no time during tachycardia was there evidence of maternal or fetal oxygen want. On many occasions oxygen was administered to the mother but it was without effect on the fetal heart rate. From these results the author cannot subscribe to the notion that persistent rapid fetal heart rates are either common or hazardous. Dangerous and unjustified obstetric intervention has been largely responsible for the unfortunate results commonly and erroneously attributed to fetal tachycardia. Transient tachycardia is relatively common, the cause is more than likely evident, and it is without danger.

On the other hand, a slowing of the fetal heart rate has long been considered a sign of fetal asphyxia and our studies confirm this. In the adult the first sign of oxygen want is usually an acceleration of the pulse. If, in the same individual, the deficiency persists or deepens an "oxygen crisis" is reached which is followed by reversal. Reversal is characterized by a marked slowing of the pulse. It is a common belief that a similar situation exists in the fetus. After many observations of anoxia we have been unable to show that a preliminary period of tachycardia precedes the bradycardia. The usual fetal cardiac response to oxygen want takes place in one of two manners:

1. A rather sudden decrease in rate, usually from 30 to 50 beats per minute, which persists throughout uterine contractions and the subsequent interval. After the immediate drop a further gradual decline may be noted. This type of slowing is similar to the one described for the sheep by Barcroft[10]-- sudden clamping of the cord produced an immediate slowing of the fetal heart rate due to reflex changes in blood flow and blood pressure. This bradycardia blended into the true anoxic slowing which came 45 seconds later. He proved this by cutting the vagi before clamping the cord; under these circumstances the initial slowing did not occur but 45 seconds after occlusion of the cord the true anoxic response was present.

2. A gradual or irregular slowing of the rate which is first noted during the late period of contraction or early interval. It is likely to happen when the contractions are very strong and lon and is due to impairment of placental exchange. (The same situation exists in a tetanically contracted uterus). At first this slowing lasts for a few seconds only; however, if these strong and long contractions are frequently repeated the period of bradycardia becomes longer and longer. Finally, bradycardia superimposes a stagnant anoxia on the pre-existing anoxic anoxia which caused the original break. Once this vicious cycle is completely established the heart remains continuously slow.

There are other factors which produce a temporary slowing and irregularity of the heart rate. Sometimes they are clearly identified and sometimes they cannot be distinguished from the second type of anoxic slowing described above, at least not early in the stages of development. One type of temporary slowing occurs with the onset of a contraction. This, obviously, cannot be due to anoxia. More often than not it is due to coiling of the umbilical cord about the next and it is possible to make this diagnosis with a fair degree of accuracy. The contraction, which causes some degree of occlusion of the cord, produces an immediate reflex bradycardia

similar to the one described by Barcroft for the sheep. As soon as the contraction ceases the normal rate resumes.

A second type of slowing occurring during the later part of the contraction is indistinguishable from the anoxic slowing and is due to pressure of the head against the perineum. This occurs late in the second stage of labor as the head crowns and is of no great practical importance as delivery can be effected immediately if necessary.

A third type of temporary slowing occurs immediately after the contraction has ceased. It is too closely related to the relaxation of the uterine musculature to be anoxic bradycardia. It is not associated with pressure against the perineum for we have observed it in twins. It is of no practical importance as it lasts for a few seconds only.

We do not know the relation of bradycardia to the level of blood oxygen. More investigative work must be done concerning this aspect of the problem. In an indirect manner we have observed that a mixture of 90% nitrous oxide and 10% oxygen, given to the mother for analgesia, will produce fetal bradycardia if continued for any length of time. We have also observed bradycardia from 85-15% mixtures when the mother was bearing down with contractions which were frequent and well sustained. We have not found a slowing of the rate with 80-20% mixtures nor would they be expected under ordinary circumstances.[10]

The customary therapy of fetal asphyxia is still based on the doctrine of von Winckel and may be found in most textbooks in this or a similar form: "....a pulse rate of 100 or less for any great length of time is incompatible with life of the fetus, and under such circumstances rapid delivery is indicated, provided it can be accomplished without too great risk for the mother." Note that nothing is said about the risk for the baby which follows difficult operative deliveries because the tenet is based on the assumption that the situation is always irrevocable. These beliefs have led some obstetricians to attempt unwarranted methods of delivery such as version and extraction, mid- or high forceps extraction, sometimes through an undilated or incised cervix and on occasions even cesarean section has been advised. Many asphyxiated babies have been delivered by these methods convincing the physician of the soundness of his judgment. It seems to me that many of the infants have survived in spite of the procedure rather than because of it, and the asphyxia has been attributed to the conditions associated with the slow fetal heart rather than the conditions associated with delivery, or a combination of the two.

To Waters and Harris[20] must go the credit for emphasizing the fact that anoxia of the fetus can be recognized by changes in the fetal heart rate and successfully treated by administration of oxygen to the mother. This observation was never widely known or fully studied; therefore, in this study of fetal heart rates particular attention was focused on the effect of oxygen therapy on anoxic fetal bradycardia. A description of a single clinical experience clearly illustrates this response which is characteristic:

, a very obese multipara, 40 years of age, weighed 256 pounds. Hypertensive heart disease was present with a blood pressure of 180/110. At bed rest she was neither dyspneic nor cyanotic. However, on very slight exertion dyspnea became marked. Labor was slow and difficult because of breech presentation and marked angulation of the uterus through a ventral hernia. Onset of slight dyspnea was noted after four hours of labor; however, there was no change in fetal heart rate which persisted at 150 to 160 for the following five hours. At this time dyspnea became marked and slight cyanosis could be seen. The fetal heart rate decreased to 135 and then to 70. This rate persisted constantly for over ten minutes. Cervical dilatation was 6 cm. and the breech was not engaged. At this time the patient was allowed to breathe a high concentration of oxygen through a face mask. Within three minutes the rate had increased to 95, after five minutes the fetal heart rate was 120, af-

ter ten minutes was 135, and after twenty-five minutes 148. Similarly there was improvement of maternal dyspnea and cyanosis after ten minutes of oxygen therapy. After twenty minutes respirations were normal, and after thirty minutes the maternal pulse rate had decreased from 120 to 100. Good pain relief was obtained from nitrous oxide oxygen and cyclopropane analgesia and no further slowing of the fetal heart rate occurred. The cervix was allowed to dilate, and delivery was accomplished two hours after the original onset of fetal heart slowing. The infant was in good condition at birth.

The equipment necessary for administration of oxygen need not be elaborate. The minimal needs are a tank of oxygen, a reducing valve and a rubber tube. The addition of a face mask and a breathing bag are desirable. All are a part of the anesthetist's armamentarium and are usually available in any delivery room.

Once the diagnosis of a persistent bradycardia is made, oxygen should be administered. The success of therapy may be measured by the improvement of the heart rate and response is usually rapid. Unless notable improvement is present within 5 to 10 minutes we may assume that oxygen therapy will be of no value. It is apparent that such a procedure does not increase the hazard for the infant inasmuch as at least five or more minutes will elapse during the preparation of any patient for delivery. If, during the period of preparation, the fetal heart fails to improve, exigent delivery is indicated whenever possible. If the heart improves, labor may be allowed to proceed in a normal manner.

In my opinion it is very hazardous to perform any type of operative delivery while mother and fetus are in acute oxygen want. If the anoxia is reversible, as a rule it is unless there has been some damage to the placental site or an occlusion of the umbilical cord, then normal oxygenation should be restored before proceeding with delivery. If the anoxia is irreversible, then there is no justification for unnecessary delay in delivery. Neither should we delay if fetal anoxia is impending or developing at the moment when delivery is about to be completed.

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V. GOSSIP

Major John Whitaker, Marine Corps was a visitor this week. He finished his sophomore medical year 6 years ago intending to take a short fling at flying in the marine corps. Circumstances altered his program and now he is back with a well decorated chest which includes the silver star and the first marine corps presidential citation for good work at Guadalcanal. He is married and has a baby but doesn't get home often to see them. He was one of the group which flew out wounded from Guadalcanal for over 5 months. He is impressed with the possibility of air ambulance service but is still anxious to come back and complete his medical education. The service has changed him little for he is still the same quiet, powerful fellow who was captain of our wrestling team and one of the outstanding athletes in the Big Ten. ..It's Captain Albert M. Snell of the Navy Medical Corps now. Al was back on a recent visit and he was so glad to be on solid ground he had difficulty going indoors. He had been on hospital ship duty for over a year. Captains in the Navy are at a slight disadvantage as far as appreciation of their rank is concerned. Most people know it is the same as a full Colonel in the Army, but not as many know the equivalent rank in the United States Public Health Service is Medical Director.. One Medical Director in order to place his rank properly had this additional note printed on his calling cards - "Medical Director, same as Colonel in the Army". ..Senior Surgeon Herman Ertresvaag Hilleboe was a recent Minneapolis visitor in connection with the opening of the Christmas Seal Campaign. This rank is the same as Lt. Col. in the Army...Richard Brown, Assistant Laboratory Attendant, drug room is a retired city mail carrier. He is doing his bit by coming out of retirement and helping the hospital. He keeps a daily record of solutions made in our drug room and I believe the size of this service will be of interest to you. In October we used the following: 5% dextrose, 168 units of 500cc capacity and 287 of 1,000cc capacity; 10% dextrose-298 of 500 cc and 463 of 1,000cc; 20% dextrose, 6 of 500cc and 19 of 1,000cc. All of these were made with distilled water. Of 5% dextrose in saline we used 133 of 500cc units and 339 of 1,000cc units. Of 10% dextrose in saline we used 79 of 500 cc. and 125 of 1,000cc. Of distilled water we used 103 of 500cc units and 135 of 1,000cc units. Also 335 of 500cc saline and 228 of 1,000cc. Mr. Brown, who has two sons in service, feels everyone shoul do his bit at this time...Thanksgiving Day is the most disabling of all American holidays. There is a greater loss of efficiency the day following than after any other event. Probably because we eat too much...At a business college recently I learned that bad spellers are being trained to spell on the typewriter with their fingers. Mistake tendencies are eliminated by teaching them to type with five carbons. They go just a little slower when they have to erase 5 pages. This training is necessary because of the large number of government forms which must be filled out in multiples....The second War-Time Medical Graduate Meeting was held on Tuesday of this week at the Wold-Chamberlain Naval Aviation Dispensary. Hospital rounds were made at Fort Snelling in the morning and at Wold-Chamberlain in the afternoon by Dr. Charles H. Krusen of the Mayo Foundation. In the late afternoon he addressed Army and Navy medical men on "Use of Physical Medicine in Injuries and Disease." Dr. Miland E. Knapp gave the second talk in the evening on "Management of Infantile Paralysis." Dinner was served at the bachelor officers' mess. These programs are apparently as popular with the service men as with the civilian faculty members. Next program will be at the Fort Snelling on Tuesday, December 7, when Dr. Spink and Dr. Rigler will speak on respiratory infections. Rounds will be made at Navy in the morning and at Army in the afternoon.The Center for Continuation Study program in medicine will have several choice offerings for winter quarter. As we have been crowded out of the building by n Army group, meeting space must be arranged elsewhere. Our advanced students liked the idea of living and going to school in the same place so well they have asked us to arrange courses at a hotel if possible. Up to June 30, 1943 registrations in all fields was 16,387. Of these more than 40% were in medical, hospital and public health courses....

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