Staff Meeting Bulletin
Hospitals of the » » »
University of Minnesota

Premature Separation Of Placenta

Volume XI Friday, May 17, 1940 Number 37

STAFF MEETING BULLETIN
HOSPITALS OF THE . . .
UNIVERSITY OF MINNESOTA

Volume XI | Friday, May 17, 1940 | Number 27

INDEX

Published for the General Staff Meeting each week during the school year, October to May, inclusive.

Financed by the Citizens Aid Society.

William A. O'Brien, M.D.

I. LAST WEEK

Date: May 10, 1940

Place: Recreation Room,
Powell Hall

Time: 12:15 to 1:20 p.m.

Program: Movie: "The Bear That Couldn't Sleep"

Electroencephalography
G. B. Logan

Discussion
Arild Hansen
R. A. Jensen
W. T. Peyton
A. B. Baker

Present: 126

Gertrude Gunn
Record Librarian

- - -

II. MOVIE

Title: "Band Concert"

Released by: United Artists

- - -

III. ANNOUNCEMENTS

1. MINNESOTA PATHOLOGICAL SOCIETY

University of Minnesota Medical School, Institute of Anatomy annual meeting Tuesday, May 21, 1940 at 8:00 p.m.
Address - Hyperinsulinism
By President O. J. Campbell.

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2. CENTER FOR CONTINUATION STUDY

Diseases of Infancy and Childhood - May 20 - 25, 1940

Will be attended by physicians from North and South Dakota, and Iowa.

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Hospital, Medical, and Institutional Library Service, May 22, 23, and 24, 1940.

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Gynecologic Tumors - June 6 - 8, 1940.

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3. UNIVERSITY OF MINNESOTA HOSPITALS

STAFF MEETING PROGRAM ASSIGNMENTS

May 24 - Physical Therapy
May 31 - Laboratory Service (Chemistry)
June 7 - Anesthesia
June 14- Administration

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IV. PREMATURE SEPARATION OF THE NORMALLY IMPLANTED PLACENTA

J. L. McKelvey

The object of this discussion is to present for your consideration two aspects of the problem of premature separation of the normally implanted placenta which are deserving of attention at the present moment. These have to do with etiology and with treatment. Both are far from being settled but there is every possibility that both represent essential advances in our understanding of this condition and the handling of it. No attempt will be made here to present a complete picture of the disease.

It will serve our purposes to consider first a few of the essential features of the condition since any concept of etiology must fit into these and therapy must give attention to the fundamental problem to be attacked.

The outstanding anatomical disturbance is that associated with the escape of blood into the decidua basalis, producing separation of the placenta and uterine walls. The amount of this separation varies widely from areas of a few millimeters to complete separation of the whole placenta. There is nothing in the definition of the disease upon which may be based a decision as to the extent which such hemorrhage must reach to be dignified by the term premature separation. It has been somewhat arbitrarily decided to include under this condition only those in which hemorrhage is sufficient to produce external bleeding, interference with uterine contraction, or interference with the welfare of the child. It has been chosen to call hemorrhages below this level "miniature premature separation." It is important, however, to remember that the fundamental disturbance is the same.

The amount of blood loss apparently represents an interaction of several factors. The size of the vessel involved, the resistanee of the local tissue to infarction and the possibility of escape of the blood into the non-resistant areas between the placental cotyledons, the so-called trophoblastic septa, all seem to play a part.

Blood loss at other levels also occurs. This is seen scattered through the uterine musculature, beneath the peritoneum, in ovaries, tubes, broad ligaments and, to a lesser degree, beneath the pelvic peritoneum. This condition only occurs with the more severe grades of the condition and has been described as the Couvelaire uterus. This hemorrhage is not simply an extension of blood from the decidua basalis. If this were so it might reasonably be expected to assume a fan shape with its apex at the placental site. The tendency is for it to show a major concentration about the uterine vessels lateral to the uterus, with decreasing concentration as the distance from these increases. This again is not the whole story since hemorrhage is seen which is not in continuity with that from the main uterine vessel stems. It may be said, then, that a scattered infarction may involve any part of the pelvic vessels. The source of this or the mechanism of its production is not known.

The third channel for the escape of blood is through the uterus and to the exterior. This is usually not great in terms of what is seen in other forms of hemorrhage. It is interesting to note that the peculiar shock which is the dangerous feature of the condition is severe and without relation to the amount of blood loss.

Perhaps the most important basic feature of the condition is this shock. It is certainly the most important aspect from the point of view of treatment. There is no adequate explanation of it at present. To this may be added, late in the course of severe forms of the disease, the shock of blood loss.

One other feature of the disease is its association with evidences of what is loosely called pregnancy toxemia. In about 50% of the cases there is albuminuria and/or hypertension. Albuminuria alone is probably not important but the

result of the conditions described above. What is important is the frequent occurrence of premature separation in the presence of an established hypertensive toxemia. This association is almost entirely with the arteriolosclerotic form of the pregnancy toxemias. This may take one of two forms. The classical picture of premature separation may occur and its incidence roughly parallels the degree of the hypertension. The chance of its occurrence shows a curve with an increase in the incidence as pregnancy advances. Premature interruption of such a pregnancy may then be desirable in the interests of the child.

A second form of this is not usually associated with premature separation. In the presence of arteriolosclerosis a relatively large proportion of fetuses die in utero in the last trimester of pregnancy. Careful examination of the placentae of these shows a condition long known as placenta trouffée, in which multiple hemorrhages have occurred into trophoblastic septa. Presenting on the maternal surface are small brown areas which represent small hemorrhages into the decidua basalis and some of these, at least, are directly continuous with the septal blood clots. This is the most fertile source of what has been described as areas of miniature premature separation. These hemorrhages are quite evidently of different ages so that they represent a gradual accumulation of the effects. The intervillous space is gradually diminished. Degenerative changes in the fetal portion of the placenta occur. Finally, the placenta is unable to perform its function and the child dies. There is a very close relationship between the gross findings here and in premature separation and what is more important to the present discussion, the vascular lesions are the same.

Since the first known anatomical description of premature separation at the beginning of the 17th century, interest has been shown in attempts to determine the etiology of the condition. All sorts of explanations have been presented and critically examined. These fall into two large groups. All of the associated disturbances have been examined. These include nephritis, torsion, trauma and the hypertensive toxemias referred to above. They are obviously not acceptable since they are associated with only a small proportion of the cases. Attempts have been made to reproduce the lesion in the experimental animal with a hemochorial placenta. The production of nephritis by chemical and bacteriological means has been shown to produce somewhat similar lesions. The exhibition of large quantities of histamine will likewise produce retroplacental hemorrhage. In all of this, little attention has been given to demonstrating similar circumstances in the pregnant human and less to the question as to why the vascular system broke down at this particular point in the decidua basalis.

At first glance, it might seem strange that the actual source of the hemorrhage, the blood vessels in the decidua basalis, was not examined. A little experience with the premature separation placenta soon explains this. A diffuse infarction is produced at the area of separation. Blood plasma escaping into connective tissue of this sort produces with surprising speed a necrosis of such degree that cell form and fine organ structure are very quickly lost. It is almost impossible to use the premature separation placenta for vascular studies.

For this reason it seemed wise to turn to two other approaches. It would, first of all, be well to know what is happening in the vessels of the decidua basalis in normal pregnancy and this study will be presented for your consideration.

Two peculiar features recently have come to light in regard to the anatomy of the decidua basalis. The main vessels in this area show nothing in their walls to allow a decision as to whether they are afferent or efferent. Spanner has recently investigated this, and, in so doing, re-examined the whole of the vascular anatomy here. His most important finding was that the vessels to the whole of the central portion of the placental area are exclusively arterioles, while the venous return from the intervillous space is entirely at

the margin of the placenta. It is limited to a ring 2 to 3 cm. wide and is made up of great sinuses. The department here has had only one specimen suitable for examination of this point but the findings in this seem to confirm those of Spanner. It seems reasonable to conclude, then, that vessels taken from the center of the placenta are arterioles.

A word as to further vascular anatomy here is perhaps in order. The afferent vessels to the intervillous space are made up only of endothelium and basement membrane. This lies directly against decidua or chorionic cells. They are enormously tortuous and coiled. They are much larger than one might expect and open by a bottle-neck constriction into the intervillous space. Two other types of vessels are found in this area. The afferent arterioles to the intervillous space give off nutrient branches to the decidua just after leaving the uterine musculature. These are comparatively straight, have a muscular media and they possess a lumen very much smaller than the main afferent stem. Secondly, fetal vessels enter the decidua. These come down the main anchoring villus stems, enter the decidua, and leave to join villous branches. These vessels, too, are small and have a musculature.

If Spanner's work is substantiated, as it seems likely to be, a method is available for recognizing the origin of the vessel concerned. This is important because of the fact that if these are arterioles with blood flow in the direction of the intervillous space, changes occurring in them cannot be due to placental products in the absence of demonstrated backflow.

The second feature of recent acquisition has been brought to attention by Burwell and his co-workers. It is surprising that the significance of so simple a feature should have been so long overlooked. They pointed out that no capillary bed is interposed between the arterial and venous circulations at the intervillous space. Thus, an arteriovenous fistual-like arrangement is set up. It is not the purpose of this paper to go at length into the effects of this. It might be pointed out, however, that numerous similarities exist between pregnancy changes and the results of the naturally occurring or experimentally produced arteriovenous fistula. Among these are the increased blood volume, increased cardiac output, bruit and increased venous pressure in the neighborhood. The feature of interest to us here is the dilatation, not only of the neighboring vein but, as Halsted pointed out, of the neighboring artery. No information can be found as to the effect of the arteriovenous fistula on the surrounding arterioles. Holman who has recently summarized the literature and his own findings in regard to arteriovenous fistula, has nothing to say in regard to vascular histology. One specimen has been examined here and no demonstrable arteriolar lesions found. This is, however, far from conclusive. Under any circumstances, the arterioles of the decidua basalis represent the main arterial supply involved in the fistula and would be more comparable to the main artery of the arteriovenous fistula.

It should be pointed out here that one important feature frequently seen in the classical arteriovenous fistula is missing in its pregnancy counterpart. Separation of the placenta with removal of the arteriovenous fistula mechanism does not show the expected sudden elevation of blood pressure. More work is required in this regard.

There is reason to believe, then, that the afferent vessels of the decidua basalis may be recognized and that they have been influenced by forces which might be expected to produce disturbances. The bleeding of premature separation characteristically begins in the central portion of the decidua basalis and is maternal blood. It arises then from arterioles and, as has been pointed out above, these are only of two types. Examination of these vessels leads to the conclusion that the hemorrhage can only arise from the afferent arterioles.

With this in mind, sections were obtained from these vessels in normal placentae. Details of the findings will be condensed here and the reader referred to

the quoted literature (McKelvey) for fuller description.

Afferent vessels are not frequently encountered in this area. All show the absence of a muscular wall. All are tortuous. Many show varying degrees of dilatation. Fewer show a peculiar fibrinoid material concentrically arranged around the lumen, replacing the decidua. In some cases necrosis of the decidua may be seen in progress but in most, the decidual cells seem to be pushed aside by the fibrinoid material. In one case, extensive round cell infiltration enclosed the coils of the arteriole. Occasionally, the material surrounding the vessel could be recognized as plasma. Transitional stages may be followed to enormous dilatation, extensive destruction of the surrounding decidua, aneurysmal dilation of the vessels and finally to the extrusion of blood. All of these are seen occasionally in the placentae of clinically normal patients. These changes blend imperceptibly into those described as miniature premature separation. Here, rupture of the afferent vessel, gross extrusion of blood and gross destruction of the decidua may be seen. This is premature separation in miniature.

It is suggested that this is the mechanism by which premature separation is produced. If this be so then every pregnancy carries the potentialities of the disease. The mechanism of the vascular changes is not certain but it is suggested that there is an association with the arteriovenous fistula-like arrangement at this area. Further investigation is clearly indicated.

The <u>therapy of premature separation</u> is also undergoing scrutiny. Cesarean section has been the method of choice when clinical symptoms of any degree of severity were present. This was accompanied by treatment of the shock. This is a logical evolution. The mortality rate before blood transfusion was available, or cesarean section safe, must have been severe. The improvement noted after delivery led to a natural desire to interrupt the pregnancy as soon as possible. Forceful delivery from below added seriously to the shock and was given up in favor of the somewhat less shocking cesarean section. The use of ether as an anesthetic agent interfered with an already poorly contracting uterus and many uteri were removed on this basis. While blood transfusion improved the results, the fear of the disease persisted and a dramatic disease process called for a dramatic attack.

A somewhat more calculating consideration of the problem has dictated a different approach. These patients are destroyed by shock and hemorrhage. Cesarean section is not logical treatment for either of these and indeed only increases them.

The mild cases, good operative risks, were frequently seen to deliver spontaneously and to do well. The severe cases, very poor operative risks, were exposed to a form of treatment which added to their difficulties. This scarcely seems logical if the uterus can be made to empty itself in reasonable time without addition to the shock or hemorrhage, if the increased duration of the condition when normal delivery is accomplished can be compensated for by blood transfusion and if the tetanically contracted uterus can be made to bring about cervical dilatation.

The author has had occasion elsewhere to observe a relatively large number of patients with very severe premature separation. Many of these patients were obviously not in a condition to withstand surgical interference. It was surprising to note how well they seemed to do under conservative treatment with the liberal and speedy use of blood transfusion. If the worst cases could be handled by this means, the question naturally arose as to whether it could not be applied to all. If the shock and effects of hemorrhage can be controlled by blood transfusion, much might be gained by the avoidance of surgery.

This type of procedure is being carried out in the department at the present time. The necessary diagnostic undertakings are specifically arranged to avoid infection. Immediate and repeated blood transfusions keep blood pressure

levels as close to normal as possible. The pain associated with the condition is controlled in the usual way. The uterus is given a reasonable time, usually 24 hours, to accomplish dilatation. If progress toward this end is not evident by this time, dilatation is aided by version or the application of a bag. The use of ether and even of other inhalation anesthetics is avoided. Delivery is accomplished when sufficient dilatation of the cervix is reached and then often by craniotomy. The patient is watched carefully for postpartum bleeding and the uterus packed and transfusion of blood given as seems desirable.

The one exception to this rule is interference carried out in the interests of the child. Occasionally in mild premature separation, a viable child in good condition may be an indication for cesarean section. These patients are universally good operative risks since the child is destroyed before the disease advances markedly. Occasionally, premature separation may appear during labor. The duration of labor, the condition of the child and the degree of cervical dilatation will dictate the type of procedure to be followed.

Local anesthesia is undoubtedly the agent of choice in any surgical interference in this condition. It is evident that the use of this type of anesthetic has obviated the necessity of hysterectomy in most if not all circumstances where it was previously undertaken to avoid the danger of or to treat postpartum hemorrhage. Small amounts of blood are almost always lost post partum from the premature separation uterus but this may be readily replaced by transfusion.

It cannot be too strongly pointed out that if this form of therapy is to be given a reasonable opportunity to produce results, speedy blood transfusion must be available at all times. Premature separation destroys by the peculiar combination of shock and hemorrhage and these may produce their manifestations with great rapidity. The administration of whole blood can control these effectively, but it is important that no time be lost between the appearance of the indication for such treatment and its exhibition.

Premature separation is a comparatively rare condition and the department has not yet a sufficient number of cases handled by this method to justify a statistical report. A typical case history is appended. Results will be reported later when a significant series is obtained. The object of this report is to acquaint the staff with the background of the experimental approach.

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CASE REPORT

Premature separation of the Placenta

Patient, age 20, para 0-0-0-0, O.W. L.M.P. May 22, 1938, expected date of confinement March 1, 1939. Patient was living with relatives in small town 40 miles from Minneapolis and had had no prenatal care. There apparently had been no symptoms of toxemia. Approximately ten hours of intermittent lower abdominal cramps occurred 48 hours prior to admission to U. Hospitals.

Serious symptoms appeared first early in the afternoon of 1-29-39; following defecation, patient was seized with

severe, constant lower abdominal pain which put her to bed. Three hours later there was a sudden vaginal hemorrhage sufficient to soak through patient's clothing, heavy mattress pad, and soil the mattress over an area one foot in diameter; patient estimated that 1 pint of blood was lost in a very short time. Slow but constant vaginal bleeding continued until hospitalization 6 hours later.

Following the bleeding episode, patient was taken to office of referring physician and was ordered to go at once to Minneapolis for therapy. No examination was made by the home physician. For reasons unknown, patient was taken first in Minneapolis to the Lutheran Girls' Home, thence to Fairview Hospital (where blood grouping was carried out), and finally was admitted here at 10:20 P.M. on 1-29-39 because of financial ineligibility at the private institution. Morphine sulphate gr. 1/4 had been given at 8:30 P.M. in Fairview Hospital.

Admission examination

Very pale, drowsy, well developed white female age 20, apparently not in pain. Blood Pressure 130/84, temp. 99.2°F, pulse 94, resp. 20. Pupils constricted. Old dried bloody material on lips (had vomited once at Fairview). Complained of thirst. Heart and lungs normal. Gravid uterus rising 28.5 cm. above the symphysis pubis, very firm and tender to palpation, apparently in tetanic contraction. Fetal position not made out. No fetal heart tones audible. External pelvic measurements normal. Presenting part at station -1, cervix 50% effaced with 1-2 cm. dilatation; no placental tissue palpable per rectum. Constant small trickle of blood from vagina.

Laboratory

Admission - urine: 4+ albumin, few gran. casts. Hemoglobin 53%, Leukocytes 13,500 (87% pmn's)

Subsequent examinations:

Urinary albumin: 1+ to trace for 12 days, then none.
Hemoglobin: Drop to 38% 36 hours after delivery, then rise to 80%.
Erythrocytes: 1,800,000 after delivery, then rise to normal.
Leukocytes: Slight elevation continued for 10 days postpartum.
Urea nitrogen: 30.6 mg.% on 1/31/39, 13.3 mg.% on 2-10-39.
PSP: 58% in 2 hours on 2-10-39.

Procedure

Sterile vaginal examination revealed cervix as noted above, frank breech presenting. It was possible to dilate the cervix sufficiently to admit two fingers and with these the anterior fetal leg was brought down by the Pinard technique. A two-pound weight was attached to the delivered foot (right) and the patient returned to bed. In the interim, blood had been grouped, cross-matched and drawn for transfusion; the latter was started at the conclusion of the vaginal manipulation. After an uneventful labor of four and one-half hours, a stillborn female infant weighing 2,380 grams was delivered by breech extraction. The intact placenta followed immediately after the infant. Approximately 600 cc. of clotted blood and 300 cc. of fluid blood was then expressed from the uterus. The latter contracted well following the administration intramuscularly of Pitocin and Ergotrate. A minor first degree laceration at the posterior angle of the introitus was repaired with several interrupted catgut sutures. Blood pressure immediately postpartum was 90/40, but rose to 108/70 within a few minutes after return to bed and the beginning of intravenous administration of 5% glucose in saline.

Postpartum course

Three hours postpartum the blood pressure suddenly dropped to 60/0; a second blood transfusion started at that time brought the patient out of shock promptly. Two subsequent blood transfusions were given (500 cc. each) on the second and fourth postpartum days, bringing the hemoglobin to 80%. There were afternoon temperature elevations to 100-101°F. during the first 8 postpartum days, with a subsequent decline to normal levels. On the sixth day the ophthalmology consultant thought that the retinal arterioles, which were spastic on admission, showed early evidence of permanent pro-

liferative lesions, and the blood pressure was elevated to an average level of 140-150/90-100 during the major portion of the hospital stay. However, six weeks postpartum the B.P. was 120/66 and six months postpartum it was 120/60; follow-up examination of the fundi showed no pathologic changes in the arterioles.

Examination of the fresh placenta showed that at least four-fifths of the maternal surface had been depressed by a massive blood clot, some of which was still adherent along one margin. The umbilical cord was inserted eccentrically, but well within the area depressed by the clot.

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V. GOSSIP

The following letter was received by air-mail last week:

"Mexico City, April 26, 1940

Dear Sir:

A person who knows you and who has spoken very highly about you has made me trust you a very delicate matter on which depends the entire future of my dear daughter as well as my very existence.

I am in prison, sentenced for bankruptcy, and I wish to know if you are willing to help me save the sum of $285,000.00 U.S.Cy. which I have in bank bills hidden in a secret compartment of a trunk that is now deposited in a custom house in the United States.

As soon as I send you some undeniable evidence, it is necessary for you to come here and pay the expenses incurred in connection with my process so the embargo on my suitcases can be lifted. One of these suitcases contains a baggage check that was given to me at the time of checking my trunk for North America; this trunk contains the sum above mentioned.

To compensate all your troubles I will give you the THIRD PART OF THE SAID SUM.

Fearing that this letter may not come to your hands, I will not sign my name until I hear from you and then I will entrust you with my whole secret. For the time being, I am only signing "A".

Due to serious reasons of which you will know later, please reply via AIR-MAIL or WIRE. I beg you to treat this matter with the most absolute reserve and discretion.

Due to the fact that I am in charge of the prison school I can write you like this and entirely at liberty.

I cannot receive your reply directly in this prison, so in case you accept my proposition, please air-mail your letter to a person of my entire trust who will deliver it to me safely and rapidly. This is his name and his address:

Leon Soria.
Chauhtemotzin 229 - Depto 154.
Mexico City.

"A"