Obstetric Clinic

THE woman about to become a mother or with a new-born infant upon her bosom, should be the object of trembling care and sympathy wherever she bears her tender burden, or stretches her aching limbs. The very outcast of the streets has pity upon her sister in degradation, when the seal of promised maternity is impressed upon her. The remorseless vengeance of the law brought down upon its victims by a machinery as sure as destiny, is arrested in its fall at a word which reveals her transient claim for mercy. The solemn prayer of the liturgy singles out her sorrows from the multiplied trials of life, to plead for her in her hour of peril. God forbid that any member of the profession to which she trusts her life, doubly precious at that eventful period, should hazard it negligently, unadvisedly or selfishly.

OLIVER WENDELL HOLMES

11th of the Series

ABRUPTIO PLACENTAE*

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ONE of the greatest hazards of childbirth for both mother and baby is abruptio placentae. The major determinants of mortality associated with separation of the placenta are still hemorrhage and shock even though the availability and early administration of sufficient amounts of blood have resulted in some lowering of the death rate. New problems have arisen. Although hemorrhage and shock are adequately treated, death may occur from acute renal failure or afibrinogenemia.

CASE I. A twenty-seven year old primipara with vaginal bleeding in the eighth month of pregnancy was admitted to the hospital in shock. Her antepartum course had been uneventful except for mild toxemia. Immediate shock treatment consisted of morphine, intravenous glucose and Trendelenburg position. After considerable delay she was given 1,000 cc. of blood and she recovered from shock. Surgery was performed about five hours after admission. Examination of the abdomen revealed a tender, board-like uterus characteristic of complete separation of the placenta. No fetal heart sounds were heard.

At operation under local anesthesia the uterus was enlarged to the xiphoid process and of a dark purplish hue with ecchymotic areas in the broad ligaments. A small amount of bloody peritoneal fluid was present. A low vertical incision was made and a dead fetus delivered. The placenta was found completely separated with numerous large retroplacental blood clots. Bleeding was not unusual.

The patient's condition was fair at the end of operation. Her blood pressure was 90/50 and her pulse 130. Another 500 cc. of blood with glucose infusions were given but shock became deeper and death occurred six hours later.

CASE II. A multipara with mild toxemia

* Cases are from the Committee on Maternal Welfare of the Medical Society of the County of Kings, Brooklyn, N. Y. The text of the case reports is essentially as submitted to the Committee. The views expressed are those of the authors.

was admitted to the hospital after sharp bleeding during the seventh month of pregnancy. Her blood pressure was 142/87. The uterus was of normal size, the fetal heart sounds were good and the cervix undilated. Vaginal spotting occurred for a few days. Cystogram was negative for placenta previa, so the diagnosis was separation of the placenta and observation was continued.

One week later the patient had occasional pain and passed a few blood clots. She was then taken to the operating room which was prepared for cesarean section and examined vaginally. When the cervix was found 3 cm. dilated and the presentation vertex, the membranes were ruptured. Labor progressed rapidly with delivery of a 4 pound fetus within two hours. The placenta with about 1,000 cc. of blood promptly followed. Intravenous ergotrate controlled hemorrhage but the patient was in shock. She was given 500 cc. of blood which had been cross matched before delivery, and 2 units of plasma were administered while awaiting more blood. There was no further bleeding but death occurred one hour later.

Examination of the placenta revealed a dark blood clot covering about one-half the maternal surface.

Questions. (1) How common is abruptio placentae? (2) What are the symptoms? (3) How is it produced? (4) Is the diagnosis difficult? What is the differential diagnosis?

Answers. The incidence of premature separation of the normally implanted placenta is not known. Reports show a varying incidence of from 1:80 to 1:250. Discrepancies depend upon the different criteria accepted for diagnosis. The degree of abruptio placentae may range from the simple evidence of a clot found upon the placenta at delivery to complete separation resulting in the death of the fetus. Classification of cases by the severity of the maternal symptoms serves us best in outlining treatment. Patients with a ligneous uterus, absent fetal heart sounds and some evidence of shock may be classified as having severe conditions; the remainder are either mild or moderate depending upon the amount of bleeding and degree of fetal embarrassment. It is clear that abruptio placentae is not a well defined entity like placenta previa. In mild cases the frequency of diagnosis will depend to a great extent upon the obstetrician.

The symptoms of abruptio placentae vary with the degree of separation. In the mild forms the only finding may be a somewhat increased bloody show as labor progresses. Variation in fetal heart rate will be noticeable only when larger areas of the placenta separate. A history of toxemia is often elicited, or the blood pressure elevation and proteinuria characteristic of toxemia may be observed at the time of bleeding. The greater the separation the easier the diagnosis. The board-like rigidity of the uterus with areas of tenderness is pathognomonic; absence of the fetal heart tones is confirmatory. Vaginal bleeding usually occurs and may be so profuse as to suggest placenta previa. The amount of external bleeding, however, is no index of the severity of separation. Complete separation with massive bleeding may have occurred and yet hemorrhage be concealed.

Concealed hemorrhage is more often than not associated with hypertension and proteinuria. These patients are so often in shock that maternal hazards are greatly increased. It is in the management of these patients that there are differences of opinion.

The etiology of abruptio placentae is still obscure. It is quite likely that there is a link to toxemia. There may be a history of repeated episodes of premature separation of the placenta in successive pregnancies. There is little doubt that the precipitating cause of separation is degenerative changes in the maternal vessels at the placental site.

Traction on a short cord and external trauma have long been mentioned as causes of abruptio placentae. Their occurrence seems improbable or at best quite rare.

In both of the aforementioned cases placental separation occurred prior to the onset of labor. Blood pressure determinations are misleading; once separation begins and internal bleeding occurs, blood pressure evidence of hypertension may no longer be present. Maternal mortality is high in this type of case.

In differential diagnosis placenta previa is most often confused with abruptio placentae. Obviously there will be no difficulty when the uterus is hard and tender and fetal heart tones are absent. When most of the bleeding appears externally, there will be no tenderness or spasticity of the uterine muscle. On vaginal examination placental tissue will not be found and the diagnosis of separation becomes more

apparent. Cystograms are of very little clinical value. Rupture of the marginal sinus of the placenta cannot be differentiated from partial separation of the placenta until after delivery, and even then with difficulty; bleeding is usually not profuse and in either case treatment is the same. Vasa previa, fortunately a rare complication, may be confused with separation of the placenta; only postpartum examination of the placenta and membranes will make this diagnosis. Spontaneous rupture of the uterus, which is very rare, should present no problem in diagnosis.

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CASE III. The patient was a quadripara whose first pregnancy had been complicated by severe toxemia ten years before. Her blood pressure had remained elevated. When registered in the clinic during the eighth month of pregnancy she had proteinuria and her blood pressure was 180/110. Hospitalization was rejected by the patient. She was placed on a salt-poor diet, given sedation and advised as to necessity for rest. During the next month she was observed at weekly intervals. Her blood pressure dropped to 160/100 and proteinuria diminished. A few days after her last prenatal visit she was admitted to the hospital with severe abdominal pain and scant vaginal bleeding. Her blood pressure was 80/40; pallor was marked with some cyanosis. The uterus was described as ligneous with definite areas of tenderness. No fetal heart sounds could be heard. Shock treatment consisted of morphine, oxygen and transfusion of 1,000 cc. of blood.

Five hours later she recovered from shock, so the membranes were ruptured and a Voorhees bag introduced. Pituitrin was administered in ½ minim doses at thirty-minute intervals. After the third dose she went into active labor and delivered spontaneously two hours later. Her condition during labor was fair, the blood pressure being 190/130 and the pulse 80. Following delivery of a stillborn fetus the placenta was expressed with numerous large blood clots. Bleeding was so brisk for several minutes that the patient lost another 800 cc. of blood. Bleeding was controlled by intravenous ergotrate and uterine massage. The patient relapsed into shock. Her blood pressure was not obtainable and death occurred two hours later.

CASE IV. A young primipara received good prenatal care from the early months of pregnancy. Her course had been uneventful except for mild toxemia during the last month.

She was admitted to the hospital at term with nausea, vomiting, pain in the lower abdomen and scant vaginal bleeding. The pain was not severe but was persistent. Her blood pressure was 110/70 and pulse varied from 80 to 110. The uterus was in a state of tonic contraction. No areas of tenderness were noted and no fetal heart sounds were heard. The patient's blood was immediately typed but blood was not on hand so blood plasma was administered. When rectal examination showed the cervix to be 3 cm. dilated, a tight abdominal binder was applied and pituitrin given in ½ minim doses. Seven hours later the patient was delivered by low forceps under general anesthesia. Delivery was effected easily and the placenta followed immediately with about 2,000 cc. of blood, mostly in the form of old clots. The fundus contracted down well and there was no further bleeding until twenty minutes later when the uterus relaxed. A uterine pack was then inserted with apparent control of bleeding. Following this the blood pressure was 110/66 and the pulse 150. Another 500 cc. of blood and a second unit of plasma were then given. The patient became very restless, shock deepened and death occurred two hours later. Bleeding had continued in spite of the uterine pack.

Questions. (1) Is the primipara or multipara more likely to develop premature separation of the placenta? (2) How is abruptio placentae best managed?

Answers. It is not surprising that abruptio placentae is more frequently observed in the multiparous patient. With few exceptions, notably pre-eclampsia, repeated childbirth is associated with an increased number of obstetric complications. Multiparity and hypertensive vascular disease occur more often in the older age groups as does premature separation of the placenta.

Proper management of abruptio placentae is a real challenge today. Death rates for mother and baby are still high despite availability of blood and improved operative and anesthetic technics.

Prevention is not feasible inasmuch as the underlying cause is not known; nor can the solution be found in better prenatal care, as the

incidence of abruptio placentae seems to vary little whether prenatal care has been good or otherwise. Earlier induction of labor in some patients with chronic hypertensive disease complicated by toxemia may be indicated.

If our results are to improve, we must direct our attention to better management of the patient once separation has occurred. Most separations which occur during labor are mild or moderate in type and the prognosis for mother and baby is good. In these cases mechanical factors probably play a large role, such as traction on the placental edge by intact membranes or rupture of maternal decidual vessels secondary to uterine contractions. Early recognition is important from the standpoint of fetal salvage. Routine amniotomy should be performed whenever separation is suspected during labor. The fetal heart tones should be checked more frequently and pitocin stimulation of labor will be indicated if uterine inertia is present. In this type of case we are concerned primarily with the welfare of the baby. An occasional indication for cesarean section may arise in the presence of persistent or increasing evidence of fetal distress. Good judgment will be necessary. It is principally in the concealed hemorrhage or so-called toxic group that the mortality is so high, being close to 10 per cent for the mother and 100 per cent for the fetus.

Cases III and IV illustrate well the usual symptoms and course of the toxic type of abruptio placentae. Onset of symptoms began prior to the onset of labor and consisted of abdominal pain, tenderness and spasticity of the uterus, absent fetal heart tones and evidence of shock. Vaginal bleeding was scant or absent at the onset and shock was out of all proportion to the amount of external bleeding. Shock therapy was delayed or ineffectual due to inadequate amounts of blood given too late. *Usually no less than 1,500 cc. of blood and sometimes greater amounts are required to combat initial shock.* Glucose solutions should be administered while awaiting blood. A venoclysis should be functioning at all times with a needle no smaller than 17 or 18 gauge.

Treatment is instituted as soon as the patient is admitted to the hospital. One does not procrastinate while waiting for transfusion to be completed or for operating rooms to be set up. Intravenous pitocin, 5 minims in 500 cc. of glucose, is given by slow intravenous drip. If the diagnosis is clear, the patient may be examined vaginally in bed, the membranes ruptured and the ripeness of the cervix determined. This can best be discovered by vaginal examination. Rectal examination may be misleading. Has the cervix been so conditioned by hormonal and vascular changes, and Braxton Hicks contractions that one can anticipate that the uterine contractions of labor will result in a rapid progressive dilatation of the cervix? The completely effaced, soft and dilatable cervix is obviously ripe, just as the closed, firm and long cervix is obviously unripe. However, in most cases the degree of ripeness of the cervix will be somewhere between these two extremes. Just as the experienced housewife chooses the ripe melon by palpation, so does the obstetrician recognize the cervix which lends itself to vaginal deliverability.

The ripe cervix will be recognized by a certain softness which involves the entire organ, the so-called "dilatable feel." The amount of dilatation is not important, yet this type of cervix will always admit one finger with ease. In the primigravida effacement will almost always be complete and the cervical canal will be short. In the multipara effacement is neither necessary or common. The cervix may be ripe yet a good inch in length. The shape of the cervical canal, however, is important. If the internal os is closed and the cervical canal cone-shaped, the cervix is not ripe no matter how soft it may be. It is difficult to describe the cervix which is fully prepared for the onset of labor. Actually there is no substitute for experience.

Following vaginal examination conservative treatment is continued or the patient is prepared for cesarean section. Occasionally a patient will deliver or so progress in labor that delivery will become imminent while the operating room is being prepared. An impressive feature of abruptio placentae is the rapidity with which delivery is completed in the majority of cases. It would appear as though nature, realizing the error of her ways, attempts to save face.

An important complication of abruptio placentae is the oliguria and anuria which result from renal spasm and ischemia. The obstetrician may be so busy combating shock and accelerating delivery that no attention is paid to urinary output. We may rescue the patient from hemorrhage and shock yet permit

her kidneys to die. A retention catheter should be placed in the bladder at the time of vaginal examination and the urinary output checked at 30-minute intervals. Persistence of anuria or marked oliguria despite adequate shock therapy and fluid replacement indicates severe renal spasm. Blood pressures should be determined at half-hour intervals. Of themselves, however, they may not be relied upon, for apparently normal readings may be shock levels for patients previously hypertensive. Proteinuria is secondary to shock and renal ischemia. It may not be due to toxemia.

Dilating bags have no place in treatment; nor is there any good reason for the cervical pack which may further traumatize an already shocked patient and interfere with observation on the progress of labor. It serves little purpose.

The time-honored abdominal binder and Spanish windlass is condemned on mechanical and physiologic grounds. It may injure an already edematous and perhaps hemorrhagic uterine wall. The binder cannot limit placental site bleeding and it interferes with respiratory movements, increasing anoxia. Most important, it severely harrasses an already acutely ill patient.

The essentials of treatment are: (1) morphine for pain, (2) oxygen to combat anoxia, (3) early and vigorous shock therapy, (4) careful check of blood pressure and urinary output, (5) rupture of membranes and stimulation of labor in all cases and (6) cesarean section for some patients.

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Case v. A twenty-six year old gravida II, para I entered the hospital with a history of severe lower abdominal pain and scant vaginal bleeding. Her antenatal course had been normal as had her previous pregnancy and delivery. The uterus was tender and firm and no fetal heart sound could be heard. Her blood pressure was 118/60 and urinalysis showed proteinuria. On vaginal examination the cervix was found 1 cm. dilated; the vertex was above the brim. The membranes were then ruptured and 1 minim of pituitrin administered. After three hours of active labor a stillborn fetus was delivered. During labor the patient's blood pressure rose to 160/90 following blood transfusion. The placenta was promptly expressed with about 1,000 cc. of old blood and clots; the uterus contracted well and there was no further bleeding.

During the first twelve hours postpartum the patient did not void and but 4 cc. of bloody urine were obtained by catheter. Treatment consisted of hot packs over the lumbar area, hot enemas, potassium citrate, fluids by mouth and 500 cc. of 10 per cent glucose by vein. Oliguria persisted. Continuous high spinal anesthesia for twelve hours did not increase urinary output. No improvement in urinary output followed decapsulation of both kidneys. The patient's hemoglobin was now found to be 36 per cent and the non-protein nitrogen 104 mg. A blood transfusion of 500 cc. was administered. The blood pressure fluctuated between 150/70 and 180/70. Vomiting, dyspnea and pulmonary edema followed and the patient died on the fourth postpartum day. Autopsy showed pulmonary edema and cortical necrosis of both kidneys.

Case vi. A gravida II, para I reported to her obstetrician when about eight weeks pregnant. Total weight gain during pregnancy was 24 pounds and the patient's blood pressure was 130/80. In the fortieth week of pregnancy she entered the hospital with profuse vaginal bleeding. Her blood pressure then was 150/90. The uterus was hard and in tonic contraction and no fetal heart sounds were heard. Vaginal bleeding continued and one hour after admission the patient was in shock. She was given plasma and morphine sulfate while awaiting blood. Three hours later she was examined vaginally. The cervix was found 3 cm. dilated and the vertex engaged. The membranes were then ruptured and the patient was given 4 minims of pituitrin. Spontaneous delivery occurred within three hours. The first blood transfusion had been given just prior to delivery. The placenta showed evidence of retroplacental hemorrhage.

The next day the patient's general condition appeared to be good. Her temperature was normal, the pulse 80 and the blood pressure 150/90. Only 40 cc. of urine were passed after delivery and this showed heavy proteinuria and many casts. Another 500 cc. of blood and 1,000 cc. of sodium lactate solution were then administered.

Oliguria persisted and but 10 cc. of urine were passed on the second day. Urinary output from the third to the ninth postpartum days varied from 125 to 300 cc. For a rather marked anemia, blood transfusion of 1,000 cc. was administered on the fourth postpartum day

and 500 cc. of blood on the sixth and seventh days. On the eighth day the hemoglobin was 8.2 gm. Non-protein nitrogen, urea and creatinine were rising. The CO_2 combining power of the blood was 67 per cent. Fluid intake by mouth was estimated at 2,000 cc. daily. On the ninth day the patient became comatose and died during a convulsion. Autopsy showed pulmonary edema and lower nephron nephrosis.

CASE VII. A primipara in the eighth month of pregnancy was admitted to the hospital with abdominal pain and vaginal bleeding. Abdominal pain was severe and continuous, pallor was marked and she complained of blurred vision. The blood pressure was 156/130, and her pulse regular and of good quality. There was no edema. The uterus was tender and tense. No fetal heart sounds were heard. On rectal examination the vertex was at the spines, the cervix 1 cm. dilated and the membranes intact. No urine was obtained on catheterization.

Blood was transfused and cesarean section performed. The peritoneal cavity contained free blood. The uterine serosa was covered with large scattered purplish areas. After removal of placenta and large dark blood clots the uterus contracted well and was not removed. The patient did well. On the second postpartum day the hemoglobin was 48 per cent. Repeated blood transfusions with washed erythrocytes were administered.

During the first forty-eight hours but fifteen drops of urine were obtained. On the second day, after consultation with the anesthesia department, epidural block anesthesia was given for twenty-four hours without success. There was no restriction of fluids and ⅙ molar lactate solution was administered by vein. The blood pressure was 165/90. On the third day the kidney pelves were irrigated with saline and 15 cc. of bloody urine obtained. On the fourth day the patient had loose, watery stools. Efforts were made to induce diaphoresis. The temperature ranged between 99° and 101.8°F. Anuria was finally complete. On the sixth day after two convulsions the patient died.

Questions. (1) Are kidney complications common sequelae of abruptio placentae? (2) What is the cause of acute renal failure? How is it best managed?

Answers. Acute renal failure as a cause of death is definitely on the increase. It is estimated to occur in 6 to 10 per cent of the more serious cases of premature placental separation. In Brooklyn nephron nephroses and cortical necroses are responsible for an increasing percentage of the maternal deaths due to abruptio placentae. Prompt treatment and greater availability of blood have reduced the immediate mortality due to hemorrhage and shock.

Shock associated with abruptio placentae is even more important than was previously realized. It is now believed that the kidney lesions are the result of anoxia resulting from renal spasm. The type of lesion and its extent will depend upon the amount of vascular spasm and, what is more significant, its duration. A continued low blood pressure means increasing tissue anoxia with the ever present possibility of irreversible changes in the kidneys. Prolonged or extensive renal spasm will result in cortical necrosis. Less extensive spasm causes a lesion more commonly known as lower nephron nephrosis. In many cases, however, the entire nephron is involved. Frequently patchy lesions of both types will be found in the same kidney due to the varying degrees of arterial spasm.

From the standpoint of therapy the significant factor is the duration of ischemia. The onset of renal spasm appears to coincide with the onset of the abruptio placentae, or occurs shortly afterward. Fortunately in most cases, regardless of treatment, spasm lasts but a short time and results only in temporary proteinuria and oliguria.

Abruptio placentae is a grave obstetric emergency in which the patient's life depends as much upon the speed with which treatment is initiated as upon the adequacy of that treatment. Every effort must be made to shorten the period of ischemia. As we have pointed out, the obstetrician treating a patient dangerously ill of shock and hemorrhage may easily lose sight of anuria. Frequently no consideration is given to renal ischemia until the patient has suffered severe or irreparable damage to her kidneys.

Blood replacement in these cases was inadequate. It may be truly said of abruptio placentae that one cannot appreciate the amount of hemorrhage by the blood that escapes from the vagina. Blood pressure determinations may give a false sense of security. Blood replacement of less than 1,500 cc. is usually insufficient. If shock therapy is adequate and oliguria should persist, relief of arterial spasm may be attempted with low spinal anesthesia. To be successful it must of course be done before

ischemia has resulted in necrosis. When used twenty-four and forty-eight hours postpartum results are bound to be unsatisfactory.

Confronted with the problem of oliguria or anuria, many are seized with an uncontrollable urge to stimulate kidney function. All of the following methods were used, and as might be expected, to no avail: hot packs, hot enemas, frequent blood transfusions (too late), irrigation of kidney pelves, lumbar diathermy, diuretics, renal decapsulation and, lastly, forcing of fluids. The horse which is being whipped is not only tired but also gravely ill.

Rational treatment must be based on the knowledge that persistent oliguria following abruptio placentae means either cortical necrosis or nephron nephrosis. Differential clinical diagnosis is not possible although occurrence of frank hematuria points to the more serious cortical necrosis. If renal failure should be due to the more common nephron reversible lesion, careful control of the patient during the first eight to twelve days will lower mortality. *Attempts to stimulate renal function will be futile and may result in further damage.*

Fluid balance must be maintained in order to prevent pulmonary edema which occurred in these three cases. Intake of fluids should never exceed the insensible loss plus the equivalent of the previous day's urinary output. Anemia is best treated by the use of packed red cells rather than whole blood. Sodium chloride is contraindicated because of renal inability to excrete salt, the risk of edema and necrotizing arteriolitis. *Azotemia appears early but is not to be feared; blood urea and non-protein nitrogen levels rise markedly but they have little bearing upon the outcome.* Acidosis is important. High carbohydrate diet is advised as liver damage may occur. If the patient is successfully carried through the oliguric stage of eight to twelve days, regeneration of tubular epithelium can take place and marked diuresis begin. Administration of large amounts of fluid and salt will then be indicated. Careful daily determination of the volume and salt content of urine are necessary in estimating replacement.

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CASE VIII. A thirty-six year old multipara was admitted to the hospital at term with a heavy bloody show and irregular uterine contractions. The cervix was found to be 2 cm. dilated and the vertex at the spines. The fetal heart sounds were regular and strong. The uterus was somewhat tense but this was thought to be due to irritability associated with the onset of labor. Her antenatal course had been normal except for excessive weight gain. Her blood pressure was 130/80.

Six hours later the patient complained of severe backache but did not appear to have labor pains. Her blood pressure was 110/70 and the pulse 80. Rectal examination showed no change so an enema was ordered, plus two doses of quinine to be given at hourly intervals. Two hours later she had a hemorrhage of about 500 cc. She grew pale and her pulse was rapid. Fetal heart sounds could not be heard. She was given morphine sulfate, 1,000 cc. of glucose and then 500 cc. of plasma while blood was being cross matched.

When the cervix was found to be fully dilated, the membranes were ruptured and a stillborn fetus delivered by low forceps. The placenta and large clots immediately followed birth of the baby. Ergotrate was given intravenously and pituitrin intramuscularly, and a blood transfusion of 500 cc. was begun at this time. When bleeding continued, the uterus and vagina were packed. The patient was in profound shock when a second transfusion was begun. One hour later blood was seeping through the packing. While preparing for hysterectomy shock deepened and death occurred two hours postpartum.

CASE IX. A thirty-three year old woman whose first pregnancy had been interrupted for toxemia was admitted to the hospital in the eighth month of her second pregnancy, complaining of severe abdominal pain and scant vaginal bleeding. During the previous month her systolic blood pressure had risen from 130 to 170. The uterus was very hard and tender and fetal heart sounds were not heard.

The membranes were ruptured, pituitrin was administered and spontaneous delivery of a stillborn fetus quickly followed. Local anesthesia was used during repair of the perineum. Many blood clots were extruded with the placenta. While bleeding continued, the vagina was packed then repacked and blood transfusion of 1,000 cc. administered. It was noted that the blood showed no tendency to clot.

Four hours after delivery a supravaginal hysterectomy was performed without difficulty but all cut surfaces bled freely and good

hemostasis could not be obtained. Following operation the patient continued to bleed from the vagina and the abdominal incision. Protamine sulfate, vitamin K and thromboplastin were given without effect. Vaginal bleeding continued for ten hours after hysterectomy. All told, 6 pints of blood were administered, yet the patient gradually sank deeper into shock and expired.

Questions. (1) Is postpartum hemorrhage common in abruptio placentae? (2) May the blood-clotting mechanism be disturbed? (3) If so, how may this be diagnosed and treated? (4) How may the high fetal mortality be lowered?

Answers. Contrary to what might be expected, postpartum hemorrhage following abruptio placentae is not commonly encountered. If general anesthesia is avoided, prompt and adequate treatment instituted, postpartum hemorrhage as a cause of death should be eliminated. True enough third stage bleeding will occur more often than in uncomplicated delivery and preparations should be made for its control. As stressed repeatedly in these Obstetric Clinics, too much emphasis cannot be placed upon the value of a functioning venoclysis through a large bore needle with cross matched blood available. Where blood must be given rapidly, the addition of small amounts of procaine will overcome venous spasm and facilitate administration. Increased pressure by the use of high levels of the blood container or the addition of a hand pressure bulb are also advised.

In some severe cases of abruptio a grave but fortunately rare disturbance in the blood-clotting mechanism is observed. As the result of the absorption of a placental or decidual substance, probably thromboplastin, a rapid and disseminated intravascular coagulation occurs involving chiefly the arterioles and capillaries. This may account for the liver and pituitary necrosis occasionally associated with abruptio.

When this intravascular clotting process is extensive, afibrinogenemia results, i.e., hemorrhagic diathesis develops and bleeding becomes uncontrollable from the uterus or cervical stump, vaginal walls, episiotomy or other body tissues. This hemorrhage is characterized by the absence or marked paucity of clots. Hope for these patients rests upon the early detection of the hemorrhagic tendency. Fresh bank blood because of its increased clotting ability should be administered in large amounts. It should be routine to order this type of blood for all patients with abruptio placentae whether afibrinogenemia is suspected or not; ordinary bank blood should of course be administered in the meantime.

When conservative treatment is carried on, a 5 cc. sample of the patient's blood should be taken at hourly intervals, labeled and allowed to stand at room temperature. The formation of a poor clot or subsequent dissolution of the clot should point to a disturbed clotting mechanism; and unless delivery is imminent, cesarean section may be indicated to lessen the hazards of further fibrinogen depletion. It goes without saying that adequate blood replacement must be available before attempting delivery, abdominal or otherwise. It is hoped that in the near future virus-free fibrinogen for intravenous use will become commercially available for treatment.

Recent advances in obstetrics have resulted in marked lowering of cesarean section mortality rates, thus broadening indications for the operation. Purely fetal indications are now being accepted as valid reasons for cesarean section. In fact, even implied fetal risk such as may be associated with breech presentation in the primipara, or toxemia, has been accepted as sufficient reason for cesarean section in well chosen cases. Certainly in abruptio placentae the risk is more than implied; and if we are to lower its exceedingly high fetal mortality, we must concern ourselves not only with early diagnosis but also with earlier cesarean section.

In evaluating treatment and the fetal prognosis, the presence or absence of associated toxemia may be of more importance than the amount of bleeding. Often in toxemia the placental reserve has been so diminished that the loss of a small additional part of its effective area of exchange may result in fetal death.

The immediate management of the baby during its first minutes of life is of prime importance. More often than not, and rightly so, this care will be delegated to the obstetrician rather than the pediatrician. Anoxia and shock must be combated. Resuscitation procedures for the treatment of anoxia, i.e., the maintenance of an adequate airway with the administration of oxygen, are well known; yet shock is rarely recognized and less often adequately

treated. Failure to recognize shock in the newborn is probably due to our inability to obtain blood pressure determinations or observe changes in pulse volume. These observations, however, are not necessary for diagnosis since *asphyxia pallida of itself means shock and demands immediate antishock measures.* Proper management of the cord and placenta is important. Immediate clamping of the cord should never be practiced. If possible the placenta should be delivered with the cord uncut and then hung by a clamp on its membranes or wrapped in a towel several inches above the baby, who is warmly wrapped in a crib or incubator. The additional 40 to 80 cc. of blood which these babies receive in this manner will be important in combating shock. If after the five to ten minutes necessary to drain the placenta have elapsed evidence of shock is still present, immediate transfusion of 50 to 80 cc. of blood should be administered via the umbilical vein. This is a relatively simple procedure requiring only a syringe and a small catheter which is threaded into the umbilical vein. If the mother is Rh positive, her blood may be used or Rh negative type O blood should be given. Cross matching is not necessary. If this type of blood is not immediately available, plasma may be substituted. Subsequent blood examinations will indicate the further course of treatment.

With marked lowering of maternal mortality, interest should be focused on similar lowering of fetal and neonatal mortality.

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CASE X. A thirty-nine year old gravida IX para VI attended prenatal clinic regularly from the fourth month of pregnancy. Her past history was irrelevant and her antepartum course normal.

At term the membranes ruptured. Rectal examination showed the cervix to be 2 cm. dilated, rather thick, and the presenting part was unengaged vertex. No fetal heart sounds could be heard. Since uterine contractions were weak and irregular, no therapy was instituted.

Three hours later the patient bled profusely and was examined vaginally. A No. 6 Voorhees bag was inserted into the lower uterine segment and the vagina tightly packed. Glucose was given intravenously followed by 500 cc. of blood. Two hours later the packing and bag were expelled and followed very shortly by fetus, placenta and large quantities of clots. Postpartum bleeding was profuse for a few minutes but was controlled by ergotrate and pituitrin. Shortly after returning to bed the patient again bled profusely and shock followed. Vaginal packing and counterpressure over the fundus did not control bleeding. The patient was then taken to the operating room and the vaginal packing removed, and the uterus and vagina packed. Soon bleeding occurred through this packing. The patient's condition was fair, as 1,000 cc. of blood had been administered.

Under ether anesthesia supravaginal hysterectomy was now performed while transfusion continued. The uterus showed many areas of subserosal hemorrhage and the broad ligaments were infiltrated with blood. The patient's postoperative condition remained fair. Two hours later, however, vaginal bleeding recurred and the patient died in profound shock. Pathologic report on the uterus showed extensive subserosal and myometrial hemorrhage.

CASE XI. A young primipara in her eighth month of pregnancy complained of mild irregular abdominal pains associated with bloody show. Her blood pressure was 150/100. The uterus was very large, tense and tender, and no fetal heart sounds were heard. Urinalysis showed heavy proteinuria and the hemoglobin was 37 per cent. The diagnosis was preeclampsia complicated by polyhydramnios and secondary anemia. Twelve hours later several small blood clots were passed. Since uterine tenderness was still present, partial separation of the placenta was suspected and the membranes were ruptured with escape of about 1,500 cc. of clear fluid. She then received 1,000 cc. of blood. Five hours afterward regular strong uterine contractions began and continued for six hours, at which time caput appeared. Low forceps delivery of a stillborn fetus under saddle block anesthesia was followed by profuse vaginal bleeding, so the placenta was delivered manually together with several old clots. Ergotrate and pituitrin failed to control hemorrhage, so the uterus and vagina were tightly packed. The patient was now in profound shock and in no condition for hysterectomy. With another blood transfusion the patient gradually recovered from shock. Four hours after delivery she had received 2,500 cc. of blood and 2,000 cc. of plasma, it having been estimated that she had lost at

least 3,000 cc. of blood. It was then seen that she was bleeding actively through the pack. Hysterectomy was decided upon and a supravaginal hysterectomy performed under local anesthesia. The uterus was found to have been well packed but was flabby and showed numerous areas of subserosal hemorrhage. Death occurred two hours following the operation. The pathologist reported subserosal and myometrial hemorrhage with edema involving chiefly the outer third of the muscular layer.

Questions. (1) How may the diagnosis of Couvelaire uterus be made? (2) What is the treatment? (3) When is hysterectomy indicated? (4) What type of anesthesia is best? (5) Of what value are the uterine and vaginal packs?

Answers. The diagnosis of Couvelaire uterus, or "apoplexie utero-placentaire" as it was designated by that French obstetrician in 1911, is certainly not difficult once the abdomen has been opened. The uterus has a characteristic deeply purpled mottling due to subserosal hemorrhages involving the fundus and occasionally the broad ligaments. Areas of hemorrhage are more numerous over the placental site.

Erroneously the impression exists that the "Couvelaire uterus" demands hysterectomy. In Couvelaire's original papers he stated that uterine contraction may be prompt and vigorous with no postpartum hemorrhage despite the presence of uteroplacental apoplexy. Little has been contributed to our knowledge of the pathology of this complication since his description. Extensive hemorrhage involves chiefly the serosal and outer third of the uterine muscle, occasionally the middle third and rarely all layers. The degree of muscular dissociation resulting from hemorrhagic infiltration cannot be determined by the gross appearance of the uterus. Hysterectomy is indicated only when persistent bleeding results from uterine atony. *No uterus should be removed because it has a Couvelaire appearance.* Hysterectomy not only adds to the risk of section but also may tip the scales adversely for a woman who is already gravely ill. Cesarean section may be successfully completed under local anesthesia; but if hysterectomy is added, a general anesthesia with its additional risks will become necessary. Lower segment cesarean section with horizontal elliptic incision will avoid the hemorrhagic and friable muscle of the upper segment. If atony and hemorrhage should follow, little time will be lost in ligating the uterine vessels which will easily be found at the lateral edges of the uterine incision. If the resulting ischemia produces firm uterine contraction, the uterus may be safely left; if not, little time has been lost and hysterectomy may be completed.

Local should be the anesthesia of choice whether delivery be by the abdominal or vaginal route. When cesarean section is indicated and oliguria has persisted in spite of successful shock therapy, continuous spinal anesthesia should be used in an attempt to relieve renal spasm providing a qualified physician anesthetist is available.

As stated in our clinic on postpartum hemorrhage, we do not use vaginal or uterine packs. That they conceal rather than prevent hemorrhage is likely. The false sense of security they lend leads to further loss of both blood and time. Packing may conceal bleeding until it is too late to use better methods.