# Cleveland Clinic Quarterly

Volume 25 APRIL 1958 No. 2

## PLACENTA PERCRETA

*Report of Two Cases*

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PLACENTA PERCRETA is a rare but catastrophic obstetric complication. Reports of only 10 authenticated cases[1-10] have been found in the English literature; we are presenting two additional cases.

To facilitate understanding of this clinicopathologic entity, we first shall describe pertinent features of normal placentation.

### Aspects of Normal Placentation

The decidua basalis is that portion of the decidua underlying the placenta, and throughout its circumference is continuous with the decidua vera; it is composed of the compact, spongy, and basal layers. The surface of the compact layer is partially replaced by a fibrinous zone that is referred to as Nitabuch's stria and which forms the boundary between fetal and maternal structures of pregnancy (Fig. 1a). Our observations, which indicate that separation of the placenta occurs beneath this plane, are confirmed by the presence of a thin layer of compact decidual tissue on the surfaces of separated placentas (Fig. 1b). The spongy layer contains dilated maternal blood vessels in its stroma as well as occasional isolated trophoblastic cells that represent *unorganized* penetration

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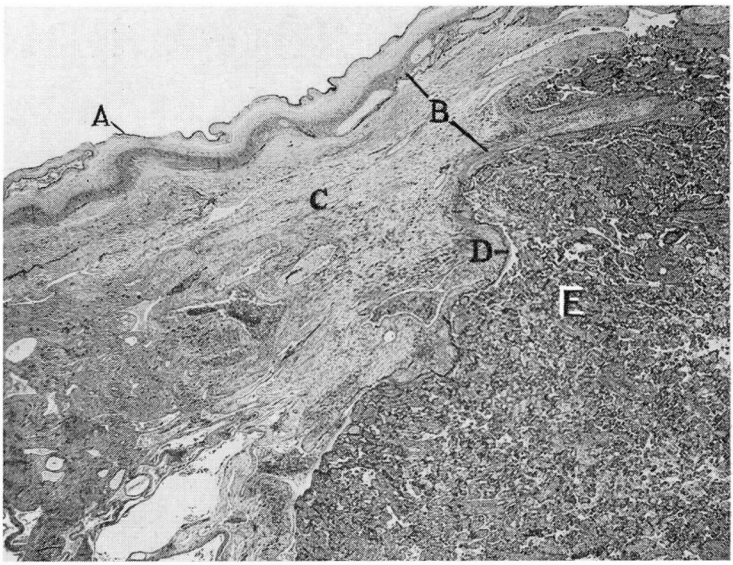
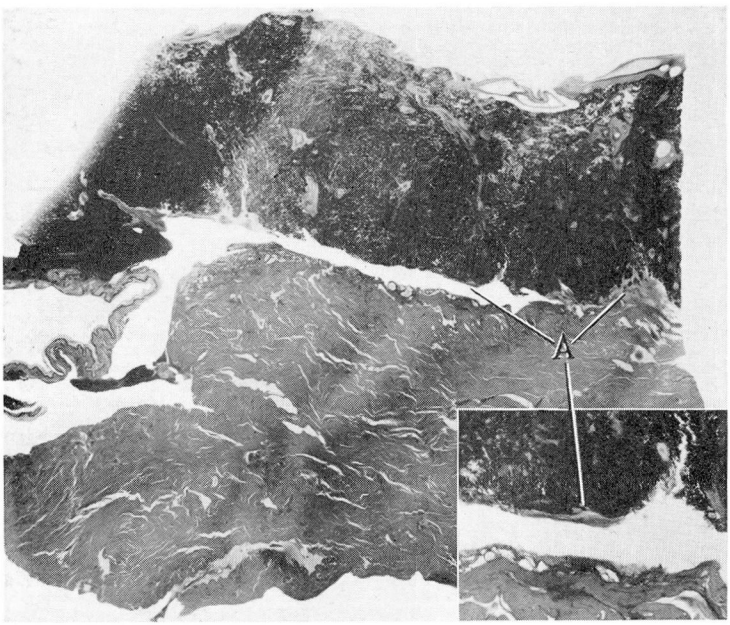



(b)

**Fig. 1.** (a) Normally implanted placenta. (A) amnion, (B) decidua, (C) myometrium, (D) Nitabuch's stria, and (E) chorionic villi. Hemotoxylin-eosin and methylene blue stain; X 10. (b) Celloidin section of normal placenta showing partial separation with (A) segment of Nitabuch's stria attached to placenta. Hemotoxylin-eosin stain; X 1½; insert X 5.

through the compact layer. The basal layer is composed of a single zone of proliferating cells in juxtaposition to the myometrium.

The differentiation of Rohr's stria and Nitabuch's stria is not always clear-cut because they are similar histologically although they have different origins. Rohr's stria, of *fetal* origin, is a fibrinous layer that lines the intervillous spaces; in the separated placenta this fibrinous layer is covered by syntrophoblast. Nitabuch's stria is of *maternal* origin and is part of the decidua basalis; it is thought to be structural evidence of decidual antitryptic activity in response to the stimulation of the proteolytic enzyme produced by trophoblastic cells.[11]

## Placenta Percreta

Placenta percreta occurs when chorionic villi penetrate completely through the myometrium, because of an abnormal decidua, manifested by partial or complete absence of Nitabuch's stria, and an abnormal underlying myometrium caused by previous trauma.

Pathologic findings associated with placenta percreta are those so well described by Irving and Hertig[12] in their classic article reviewing the world literature on placenta accreta and include: absence of an intact decidua basalis, penetration of trophoblastic cells into the myometrium, thinning of the uterine wall beneath the placental site, degeneration of the myometrium, and chronic or acute inflammatory reaction. Conditions contributing to these pathologic changes as summarized by McKeog and D'Errico[13] include: cesarean section, dilatation and curettage, manual removal of the placenta, placenta previa, infection, neoplasm, irradiation and caustic drugs, congenital anomalies, and cornual implantation of the placenta. Meyerhardt[10] cites adenomyosis as an etiopathologic factor in placenta accreta.

## Case Reports

**Case 1.** A 36-year-old woman, gravida X, para 0, was first examined at the Cleveland Clinic on July 15, 1957. Her history included nine spontaneous abortions, varying from 6 to 22 weeks of gestation. Dilatation and curettage (D. and C.) was required after eight of the abortions; in addition, after the ninth spontaneous abortion and D. and C., the uterus was packed with gauze because of excessive bleeding.

At the time of examination the uterus was enlarged to the size of a six weeks' pregnancy; the beginning of the last menstrual period was on June 2, 1957. Findings from the complete physical examination otherwise were normal. The hemoglobin content of the blood was 14.4 gm. per 100 ml.; hematocrit value was 41 volumes per cent; leukocyte count was 8,500 per cu. mm.; blood Wassermann test was negative, and results of urinalysis were within the normal range. A diagnosis of pregnancy in a woman who had had habitual abortions was made, and a regimen of bioflavinoids, progesterone, and mineral-vitamin capsules was prescribed.

The pregnancy progressed uneventfully to the fourth month, at which time the patient was admitted to the Cleveland Clinic Hospital (September 26, 1957) because of pain in the abdomen, nausea, vomiting, and diarrhea since the preceding evening. She had fainted twice while erect.

Physical examination revealed a patient writhing with abdominal pain and perspiring

freely. The oral temperature was 95.2 degrees F.; blood pressure was 115/80 mm. of Hg, and pulse rate was 80. The laboratory findings were: hemoglobin content of the blood, 10.4 gm. per 100 ml.; hematocrit value, 32 volumes per cent; leukocyte count, 23,000 per cu. mm., with 94 per cent neutrophils.

Positive findings were limited to the abdomen, which was soft, protuberant, and tympanitic; there were mild, generalized, muscle guarding and rebound tenderness in the right lower quadrant and in the midepigastrium. Peristalsis was active with high-pitched sounds.

The clinical impression was that the patient had severe gastroenteritis. Because of the especial importance of this pregnancy, sedation and antibiotic therapy were immediately instituted. Five hours later the temperature was 97.6 degrees F.; blood pressure was 110/80 mm. of Hg, and pulse rate was 80. The hemoglobin content of the blood was 10.4 gm. per 100 ml.; hematocrit value was 31 volumes per cent; and leukocyte count was 25,000 per cu. mm. with 94 per cent neutrophils.

The patient continued to have nausea and vomiting. She frequently passed liquid stools, cultures of which were negative for *Micrococci*, *Salmonella*, *Shigella*, and parasites; no occult blood was present. Surgical consultation was obtained and the clinical findings were confirmed, but a definite diagnosis could not be made.

Shortly thereafter, while receiving nursing care, the patient was turned on her left side and suddenly went into shock, exhibiting pallor, apprehension, and air hunger. The blood pressure dropped to 85/50 mm. of Hg; the pulse rate rose to 160; the hemoglobin content of the blood was 8 gm. per 100 ml.; the hematocrit value was 24 volumes per cent; and the leukocyte count was 18,500 per cu. mm. Fluids and blood were administered intravenously. A diagnosis of massive intraabdominal hemorrhage was made and an immediate laparotomy was performed under general anesthesia. The abdominal cavity contained approximately 3,000 ml. of both liquid and clotted blood. The uterus was enlarged to the size of that of a four-month pregnancy, and high on the posterior wall there were two perforations measuring approximately 3 cm. in diameter through which placental tissue protruded. Because of the patient's precarious condition, a rapid supracervical hysterectomy was performed. During this procedure 5 units of whole blood was given.

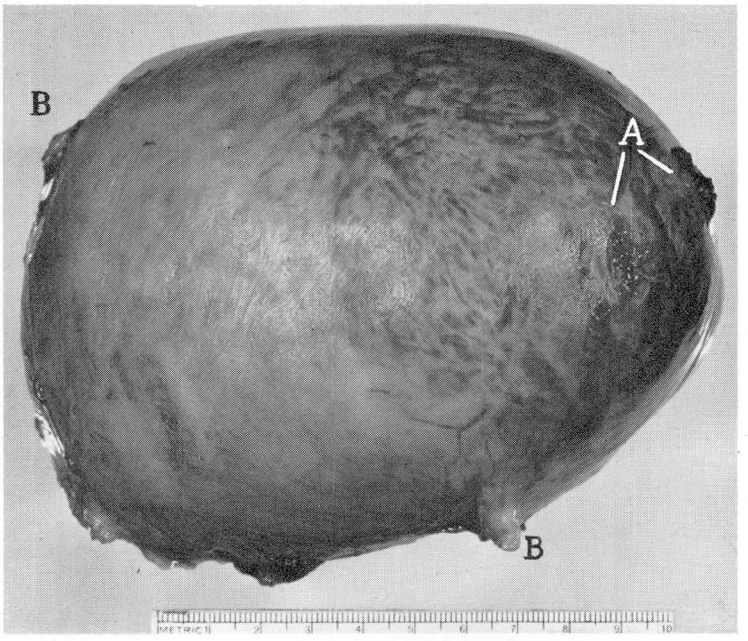
The patient had an uneventful recovery except for superficial thrombophlebitis, and she was discharged on October 9, 1957.

*Pathologic findings.* The gross specimen consisted of a supracervically amputated uterus with a soft globular protrusion, 5 cm. in diameter, on the posterior wall in the region of the right cornu. In the center of this protrusion two soft red masses of friable tissue measuring 2 cm. by 2 cm. issued from perforations of the myometrium (Fig. 2). Within the uterus, the amniotic cavity contained 150 ml. of fluid and a fetus measuring 10.5 cm. in length and weighing 40 gm. The placenta was implanted in the right cornu of the uterus, where the entire thickness of the myometrium had been replaced by placental tissue (Fig. 3). Elsewhere grossly the myometrium was normal.

In stained sections of the placental uterine junction, chorionic villi and trophoblastic cells and masses of Langerhans cells extended directly into the myometrium; the decidua basalis was absent. Scattered individual trophoblasts were in the adjoining muscle. In sections taken at the site of perforation the myometrium was completely replaced by chorionic villi (Figs. 4 and 5). The diagnosis was placenta percreta.

**Case 2.** *A 36-year-old woman, the wife of a physician, gravida VI, para III was ad-

**A report of this case in regard to the use of the antigravity suit is to be published.*




(a)

(b)

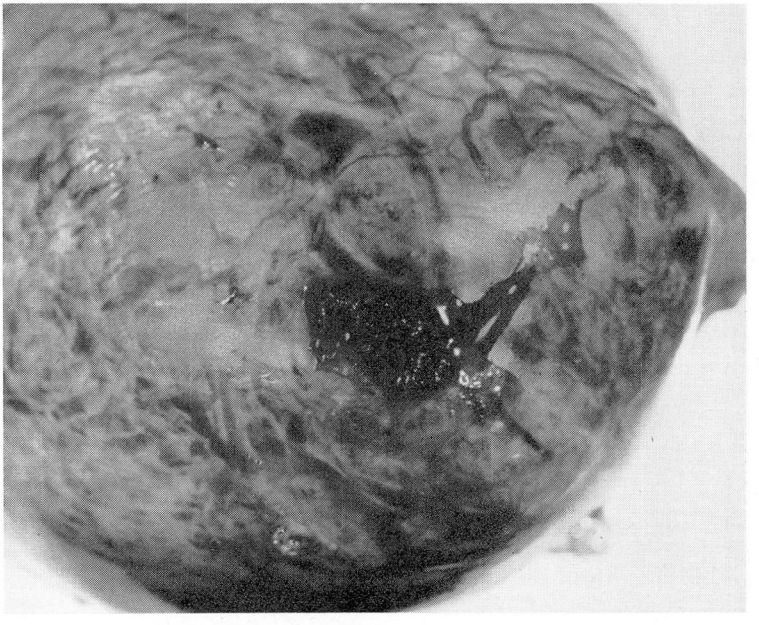


**Fig. 2.** (a) Photograph of the uterus showing bulging of the right cornu with two areas of perforation (A); round ligaments (B). (b) Enlarged photograph of area of perforation.

mitted to the hospital* on the afternoon of April 3, 1955, because of irregular uterine contractions and bleeding. Her history included two previous vaginal deliveries; the second delivery had necessitated extensive cervical and perineal repair. The third child had been delivered by an elective transverse laparotrachelotomy in order to preserve the cervix and the perineum. Since then the patient had had two spontaneous early abortions, each of which required dilatation and curettage and was followed by excessive bleeding that was controlled by intrauterine packing and transfusions. During the first five months of the sixth pregnancy there had been almost continuous spotting without cramps. The membranes spontaneously ruptured at the twenty-fourth week of pregnancy and 48 hours later she had slight spotting with irregular uterine contractions.

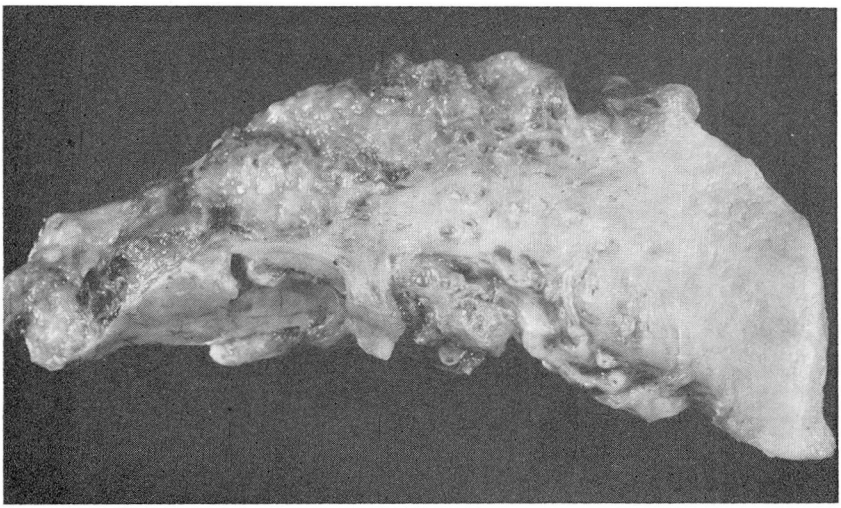


**Fig. 3.** Gross section of the uterine wall showing progressive placental invasion of myometrium with perforation at the extreme left.

On the evening of admission there was moderate vaginal hemorrhage followed by regular progressive uterine contractions and a decrease in the amount of bleeding. The blood pressure was 134/72 mm. of Hg. No fetal heart tones were audible. Labor progressed slowly and at 3:45 a.m. on April 4, under spinal anesthesia, a stillborn fetus was delivered, and immediately thereafter severe bleeding occurred. Loss of blood was estimated to be 1,500 ml. Prompt manual exploration of the uterine cavity revealed the upper half of the placenta to be completely detached from the anterior uterine wall, but the lower half of the placenta, which extended downward to the external cervical os, was tightly adherent to the uterine wall. The separated portion of the placenta was removed manually, and gentle attempts were made to separate the lower section. It was apparent that this was impossible, and a diagnosis of placenta accreta was made. Since the patient was showing signs of shock, the uterus was tightly filled with 10 yards of intrauterine

**This patient was delivered at Saint Luke's Hospital in Cleveland, Ohio. We wish to acknowledge the skill and assistance of Dr. Donald M. Glover, Chief of Surgery, Dr. B. B. Sankey, Chief of Anesthesiology, and their staffs, especially the blood bank personnel.*

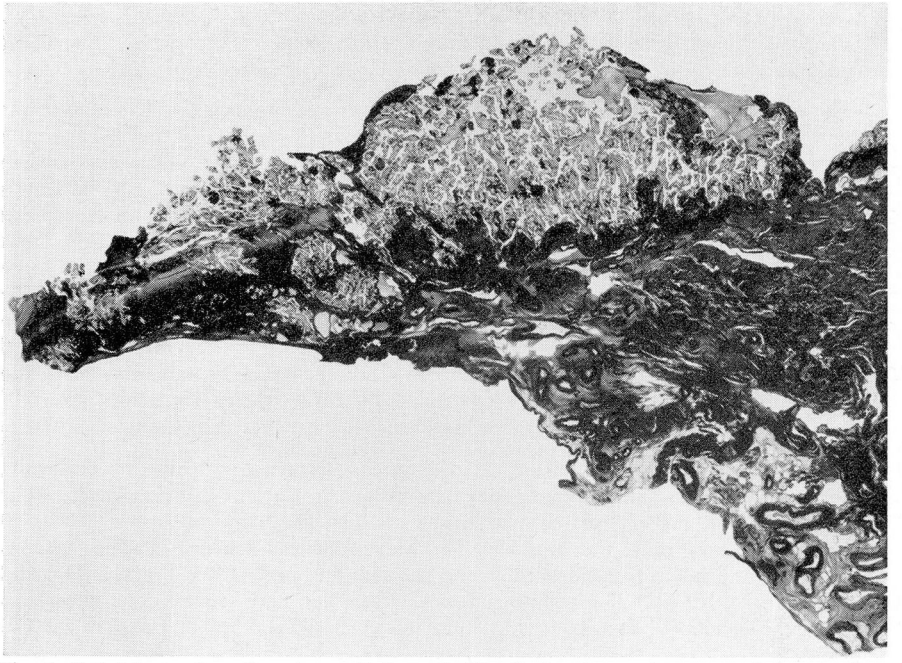


**Fig. 4.** Celloidin section of uterine wall shown in Fig. 3, showing replacement of myometrium by chorionic villi with site of perforation at extreme left. Hemotoxylin-eosin and methylene blue stain; X 1.5.

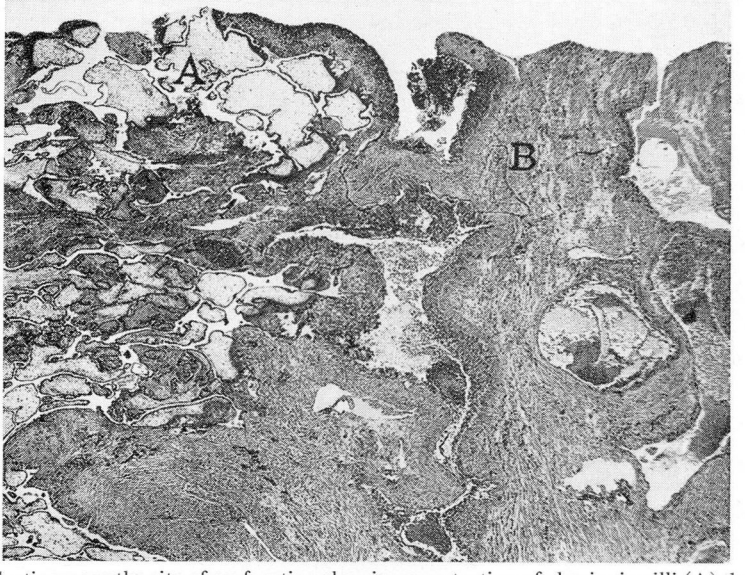



**Fig. 5.** Section near the site of perforation showing penetration of chorionic villi (A) deep into the myometrium (B); decidua basalis is absent. Hematoxylin-eosin and methylene blue stain; X 14.

packing, and she was given fluids and one unit of whole blood intravenously. The patient rallied quickly from the state of shock, and an emergency hysterectomy was begun.

The abdominal cavity was entered through a lower midline incision. The fundus was found to be well contracted; however, on the dilated anterior surface of the lower uterine segment there was a roughly circular area, about 20 cm. in diameter, which appeared to be similar to the fetal surface of the placenta with large, dilated, interlacing blood vessels coursing beneath a thin membrane. The intrauterine pack was removed, and the patient again went into a state of shock. The bladder reflection could not be mobilized from what was presumed to be the anterior uterine wall. The fascial plane containing the large dilated veins was penetrated, disclosing placental tissue immediately beneath it. The placenta had completely invaded and replaced the myometrium of the lower uterine segment. The uterine arteries were ligated and a supracervical hysterectomy was carried out. There was troublesome bleeding from the placenta, which remained beneath the bladder. The bleeding was partially controlled by figure-of-eight ligatures, Oxycel* gauze, and the pressure of the advanced bladder reflection over this oozing stump.

Because of the patient's condition rapid closure was carried out. Since the delivery, four hours previously, and the emergency laparotomy the patient had received 11 units of blood and 3,000 ml. of 5 per cent dextrose in water. The blood pressure was 110/50 mm. of Hg, and the pulse rate was 130. Ten hours after the delivery, despite the continued use of multiple transfusions, the blood pressure dropped to 70/60 mm. of Hg, and the pulse rate rose to 140. The abdomen became distended and tympanitic, and respiratory distress developed. It was obvious that massive intraabdominal hemorrhage had occurred.

Eleven hours after delivery the patient was moved to the operating room and under cyclopropane anesthesia a vaginal pack was inserted and the abdomen was reopened. An estimated 3,500 ml. of clotted and liquid blood was removed from the peritoneal cavity, 1,000 ml. of which was filtered and retransfused. At the time, the patient was receiving transfusions of blood or fluids into four veins simultaneously. Bleeding was chiefly from a spongelike mass of placental tissue beneath the bladder. There was bleeding from both broad ligaments and from the stumps of the round ligaments and tubes; these were re-ligated. More figure-of-eight sutures were placed in the remaining placental tissue, and for a while all bleeding seemed to be controlled. However, within a few minutes, as the patient was receiving approximately her thirtieth transfusion, oozing developed from many sources and blood was found in the urine. The right hypogastric artery was ligated without effect and temporary ligatures of the common iliacs had no effect. The bleeding was from the peritoneal surfaces as much as from the area about the retained placenta. In addition to citrated blood, the patient received 1,000 ml. of whole blood by silicone syringes, 3 units of fibrinogen, 1 unit of serum albumin, Cortef** (intravenous), and 20 ml. of calcium gluconate. Blood was simultaneously administered intraarterially and intravenously several times during the long operation to keep systolic pressure above the level of shock.

After six hours of anesthesia and attempts to control bleeding, the prognosis appeared hopeless. Therefore, since massive packs with pressure had been the only means capable of somewhat controlling the bleeding, nine large abdominal tapes (each 8 in. by 36 in.) were placed in the abdomen, the incision was partially closed, and the patient was returned to her bed. Sand bags were placed on the abdomen; the legs were wrapped with elastic bandages; and the foot of the bed was elevated on shock blocks. Within 18½ hours

*Parke, Davis & Company, Detroit, Michigan.*
**The Upjohn Company, Kalamazoo, Michigan.*

of delivery, the patient had received 55 units of blood (including two units recovered from the peritoneal cavity, and two units of fresh blood), and approximately 10,000 ml. of other fluids. While two additional units of blood were being transfused, the blood pressure was 86/62 mm. of Hg and the pulse rate was 144. At this juncture the antigravity ("G") suit was applied and inflated to 20 mm. of Hg. Within five minutes the blood pressure rose to 104/72 mm. of Hg.

The rate of transfusion was slowed and the blood pressure gradually rose to 114/80 mm. of Hg, while the pulse rate increased to 160. Within 24 hours of delivery the patient had received 58 pints of blood,* the blood pressure had become stabilized, and the pulse rate had begun to decrease. The 24-hour output of urine was 415 ml.

Thirty-six and one-half hours after delivery, and after 18 hours in the G-suit (which was deflated and removed after a total of 24 hours), the blood pressure was 130/78 mm. of Hg, and the pulse rate was 130. Seventy-two hours after operation, the abdomen was reopened, a quantity of clot was evacuated, and the abdominal and vaginal packs were removed. There was no further bleeding, and the vital signs remained stable. Subsequently there were ileus, atelectasis, and fever. In the fifth week, a left subphrenic abscess had to be drained abdominally. The patient was discharged on the fifty-fourth hospital day. From then on convalescence was uneventful except for a mild episode of hepatitis that developed on the one hundred and twenty-ninth day.

## Discussion

It is impossible to make an antepartum diagnosis of placenta percreta that does *not* penetrate into the abdominal cavity (Case 2). When symptoms of intraabdominal hemorrhage occur during the second or third trimester of pregnancy (Case 1) placenta percreta as well as spontaneous rupture of the uterus should be considered in the differential diagnosis. History of repeated intrauterine insult, absence of uterine contractions, prior to onset of symptoms, and gradually increasing symptoms of intraabdominal hemorrhage suggest placenta percreta rather than spontaneous rupture of the uterus. Upon diagnosis of intraabdominal hemorrhage, treatment for shock and a laparotomy become mandatory. When placenta percreta is encountered, hysterectomy, preferably total, usually is indicated. Careful examination of the excised uterus may be necessary to distinguish between placenta percreta and spontaneous rupture of the uterus with herniation of the placenta.

Severe loss of blood may require massive replacement transfusions. Uncitrated fresh whole blood, calcium gluconate and fibrinogen may be administered to aid in maintaining the blood-clotting mechanism. In spite of these heroic measures bleeding from serous surfaces may at times demand measures of tamponade, such as intraabdominal packs and use of the antigravity suit. This seems to be a means of "buying time" until the patient's own clotting mechanisms and normal capillary permeability can function.

We believe that placenta percreta results from deficiencies of the endometrium and myometrium, caused by previous (and often repeated) insults, such as cesarean section, dilatation and curettage, and manual removal of placenta, which permit invasion of the entire thickness of the uterine wall by

**This 29,000 ml. of blood represents, to our knowledge, the largest amount of blood transfused in a patient during a 24-hour period.*

trophoblastic elements. Placenta percreta, by definition, has perforation as a characteristic feature, and perforation can occur into the abdominal cavity (Case 1), beneath the bladder reflection (Case 2), or between the sheaths of the broad ligaments.

The basic process of invasion of the myometrium by trophoblastic cells may be compared to the condition present in tubal pregnancy, leading to invasion and perforation of the tubal wall, wherein the mucosa and muscularis of the tube are structurally not suitable for placental implantation. In either condition it is the character of the tissue underlying the area of nidation which determines the extent of the penetration of the chorionic villi, and not, as some have suggested,[14] overaggressiveness of the trophoblast.

When deficiency of the endometrium alone exists, placenta accreta may develop; when the deficiency extends into the superficial layers of the myometrium, placenta increta is likely to develop (Figs. 2 and 3); when defects extend deeply into the myometrium, placenta percreta becomes a distinct probability.

## Conclusions

1. Placenta percreta is a serious obstetric complication. It results when trophoblastic cells and chorionic villi traverse the uterine wall, culminating in perforation of the uterus. Causative are various pathologic changes that are due to previous, repeated, severe intrauterine insults leading to the formation of an abnormal endometrium and an abnormal underlying myometrium. A manifestation of this deficiency may be partial or total absence of Nitabuch's stria when implantation takes place. If Nitabuch's stria is absent, an *organized* invasion by the trophoblast of the underlying uterine wall may occur.

2. Placenta percreta may be suspected clinically in the second or third trimester of a pregnant woman who, in addition to a typical history of repeated intrauterine manipulation, exhibits signs of gradually increasing intraabdominal bleeding. Placenta percreta may be difficult to differentiate from spontaneous rupture of the uterus.

3. Treatment for shock and a hysterectomy in most instances are the only therapy; together they constitute a lifesaving procedure. In administering repeated transfusions, precautions to prevent incoagulability of the blood and abnormality of capillary permeability must be considered.

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