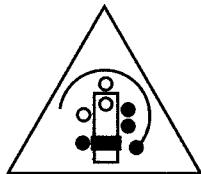


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**OBSTETRIC/GYNECOLOGIC SURGERY**

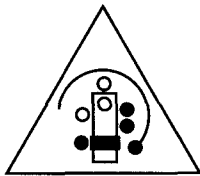


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## INTRODUCTION

### Purpose

The purpose of this module is to provide the learner with information concerning specific procedures in obstetric/gynecologic surgery. The articles that follow originally appeared in *The Surgical Technologist* and have been reprinted in this series. Upon completing this module, the learner will receive 1 continuing education (CE) credit in category 3G and 2 CE credits in category 8G.

### Using the Module

1. Read the information provided, referring to the appropriate figures.
2. Complete the enclosed exam without referring back to the text. The questions are in a multiple-choice format and are taken directly from the corresponding article. Choose your answer based on the information presented in the article itself. Do not choose a response based on your personal experience, but instead select the best answer from the alternatives given.
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5. You will be sent the correct answers to the exam. Compare your answers with the correct answers to evaluate your level of knowledge and determine what areas you need to review.

# Total Abdominal Hysterectomy

ARTICLE BY STEPHANIE ORCUTT, CST

**H**ysterectomy is the surgical removal of the uterus through either the abdominal wall or through the vagina. The elimination of pain, dysfunctional bleeding, and malignancies as well as the achievement of sterility are among the reasons a hysterectomy may be performed. Total abdominal hysterectomy involves the excision of the entire uterus and its attached cervix through an incision in the uterine wall. This procedure is commonly performed on women who have fibroid tumors that have become large enough to press on nearby structures such as the ureters and the bladder. Fibroid tumors actually consist of muscle cells rather than fibrous tissue and are very common, occurring in 20% to 30% of all women. These fibromyomas develop slowly in women between the ages of 25 years and 40 years of age, often becoming large in size and producing pressure symptoms including pain, backache, constipation, and metrorrhagia. When such symptoms occur, the treatment of choice is total abdominal hysterectomy.<sup>1</sup> Ovary removal is usually advised during this procedure in patients approaching menopause, due to the decreasing function of hormone production. However, the ovaries produce hormones that help prevent osteoporosis, so they are not normally removed in women younger than 40 years of age.<sup>2</sup>

## Anatomy of the Uterus

The uterus is a pear-shaped organ that measures approximately

3" x 2" x 1". The uterus consists of three major parts: the fundus, the cavity, and the cervix. The fundus is the uppermost, rounded part of the uterus; the cavity, or body, is the central portion; and the cervix is the canal portion. The cavity is triangular with three openings. The upper two openings receive the fallopian tubes, while the lower opening, called the internal os, leads to the cervical canal opening at the external os situated in the vaginal canal (Figure 1).<sup>3</sup>

The uterus consists of three layers of tissue. Parametrium is the

external loose connective tissue surrounding the uterus. Myometrium is the middle muscular layer that forms the main mass of the uterus. Endometrium is the mucosal layer that lines the cavity of the uterus. Located between the bladder and the rectum in the midpelvis area, the uterus is normally anteflexed, meaning the uterine body lies over the top of the bladder and points forward and slightly upward. Four pairs of major ligaments hold the uterus in place on each side. Broad ligaments are double folds of parietal peritoneum that attach the uterus to

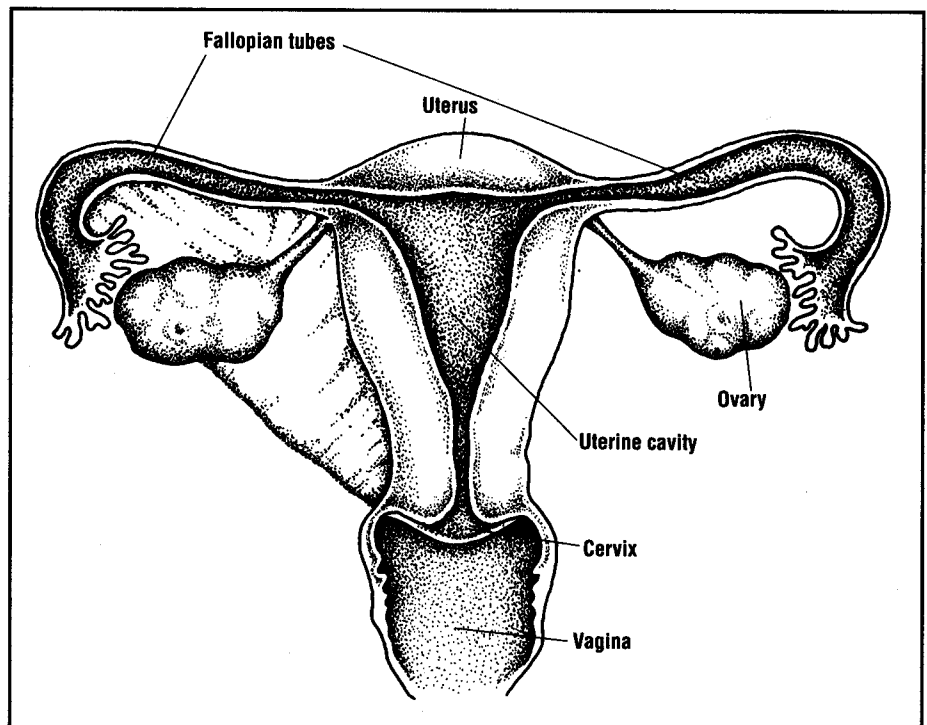


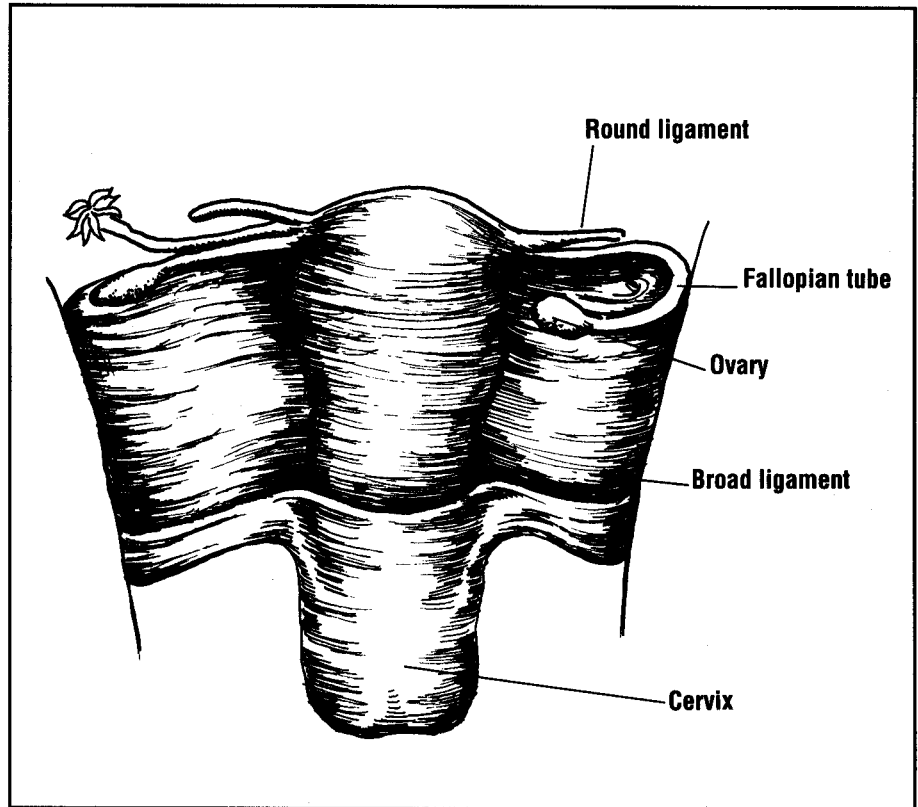
Figure 1. Female reproductive organs.

either side of the pelvic cavity. Consisting of bands of fibrous connective tissue, round ligaments are found between the layers of the broad ligaments (Figure 2). Cardinal, or cervical, ligaments extend below the bottom of the broad ligaments between the pelvic wall, the cervix, and the vagina and are the chief supporting structures of the uterus. These ligaments prevent the uterus from dropping into the vagina. On each side of the rectum are the uterosacral ligaments, which connect the uterus to the sacrum. Blood supply to the uterus is derived from the uterine and ovarian arteries, which are branches of the internal iliac arteries. Autonomic nerves are branches of the superior hypogastric plexus and supply nerves to the uterus. Blood vessels and nerves run through both the broad and the cardinal ligaments to supply the uterus. The three major functions of the uterus include menstruation, pregnancy, and expulsion of a fetus.<sup>3,4</sup>

**Patient Selection and Indications**

Fibroid tumors are noncancerous and are not life-threatening but patients suffer from various symptoms, the most serious being anemia. Depending on the size, shape, location, and number of tumors, the patient may have prolonged and heavy menstrual periods as well as excruciating pain. When these symptoms become severe, an abdominal hysterectomy is indicated. If the patient plans to bear children in the future, an abdominal myomectomy may be performed in which only the tumors themselves are removed from the uterine wall and the uterus remains intact.<sup>5,6</sup>

The patient with fibroid tumors may complain of severe, painful menstruation lasting for several days, as well as fatigue and weakness. Patient assessment begins with a medical history and a physical examination. Fibroid tumors most often occur in women 30 years of age and older, so a patient's age must be considered. A pelvic exam allows the physician to feel any abnormal masses, bumps, or contours of the uterus. A Pap smear should be given to rule out cervical cancer particu-



**Figure 2.** Relationship of the female sexual organs to the anterior abdominal wall.

larly if the patient is obese since this condition makes it difficult for the physician to feel the internal organs. Several tests are given to rule out other conditions including a complete blood count to check for anemia and urinalysis to determine whether the patient has a urinary

**Fibroid tumors...are  
very common,  
occurring in 20% to 30%  
of all women.**

tract infection. Dilation and curettage (D & C) may be performed in a day surgery unit to reach a final diagnosis. The patient is anesthetized, the cervix is dilated, and the uterine lining is scraped with a curette, after which the endometrial tissue is sent to the laboratory for analysis. Small, mushroom-shaped fibroid tumors may be removed with

a D & C procedure, but hysterectomy is recommended in cases where the tumors have become large, rock-like masses embedded in the uterine lining and extending into the myometrium. For women who are plagued by heavy bleeding and pain and who do not plan to bear children, a hysterectomy is the usual choice. If the fibroid tumors are determined to be large, the vaginal approach may cause the patient to hemorrhage. An abdominal hysterectomy is less likely to cause hemorrhaging and may be recommended.<sup>2</sup>

**Patient Preparation**

The patient is admitted to the hospital 1 day prior to surgery. A type and crossmatch for two to four units of blood should be ordered as a precautionary measure. Following the evening meal, the patient is given a soap suds or Fleet's enema to cleanse the lower intestine. Once the enema is given, the patient showers;

a povidone-iodine douche may be ordered to cleanse the vaginal canal. The patient may be given a tranquilizer before midnight to aid in sleep. Nothing should be given to the patient to eat or drink, including oral medications, after midnight. Health care workers should offer the patient emotional support during the preparation. Because the patient will no longer be able to bear children, she may feel that the surgery will affect her ability to function as a woman. It is important that she evaluate her feelings about her femininity prior to surgery.<sup>7</sup>

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**For women who are plagued by heavy bleeding and pain and who do not plan to bear children, a hysterectomy is the usual choice.**

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#### **Preoperative Preparation**

The patient is brought into the operating room and placed on the operating room table in a supine position. A general anesthetic or a spinal anesthetic is then administered. When using a spinal anesthetic, the nerve roots and lower spinal cord are anesthetized and the patient remains awake throughout the procedure. Members of the operating room team must be careful not to discuss the patient and the diagnosis. After the patient is properly positioned and anesthetized, the

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**Health care workers should offer the patient emotional support during the preparation.**

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skin prep begins. The surgeon may order the skin of the upper pubis to be shaved, just prior to the skin scrub. The abdominal prep is done from the nipple line to the pubis. The vaginal prep includes the vagina, vulva, and the upper thighs.

Two separate prep trays are used for this procedure: one for the abdominal prep and the other for the vaginal prep as the vagina is considered an unclean area. Usually two surgical technologists carry out the preps simultaneously. Following the vaginal prep, a Foley catheter is inserted into the bladder to provide urinary drainage throughout the procedure.

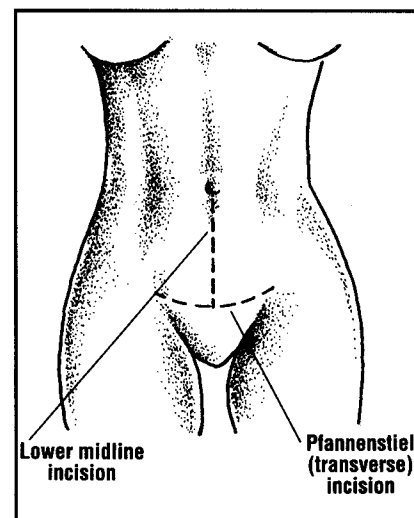
Several special instruments are used during an abdominal hysterectomy. A round, self-retaining retractor capable of retracting the bladder and the sides of the abdomen such as an O'Sullivan-O'Connor is used. Heaney hysterectomy clamps should be available when clamping the uterine ligaments. These clamps are used to grasp and clamp the ligaments before they are divided. Heavy scissors such as curved Mayo scissors can be used to divide the ligament tissue. Long Allis clamps should be available to grasp around the edge of the cervix during its division. The electro-surgical unit is used to stop bleeding vessels when entering the abdomen. All instruments that have contact with the cervix or vagina should be considered contaminated and should be discarded into a separate, designated basin after use.<sup>8</sup>

#### **Surgical Procedure**

A Pfannenstiel or bikini incision, in which the skin is incised just above the pubis, is commonly used for abdominal hysterectomy. Many gynecologic procedures are performed through this incision. However, a low abdominal midline incision may be used in cases where a patient is obese or has large fibroid tumors in order to provide more exposure and to enable the surgeon to feel the other abdominal organs (Figure 3). After incising the skin with a #10 blade on a #3 knife handle, the surgeon deepens the incision through the subcutaneous tissue with a deep knife or cautery pencil. The fascia is nicked with the knife and cut with curved Mayo scissors. The surgeon grasps one edge of the fascial margin and bluntly dissects, using his or her fingers to separate the fascia from the underlying muscle. The muscle lay-

ers and lower fascial margin are bluntly dissected manually. The peritoneum is then nicked with a long knife and the incision is lengthened with Metzenbaum scissors. Using moist lap sponges, the surgeon packs the bowel away from the uterus. Moist packs are placed one on each side of the abdominal incision and a self-retaining O'Sullivan-O'Connor retractor is placed in the wound.

Once the abdomen is opened, the patient may be placed in a slight



**Figure 3.** Common gynecological incisions made through the abdominal wall.

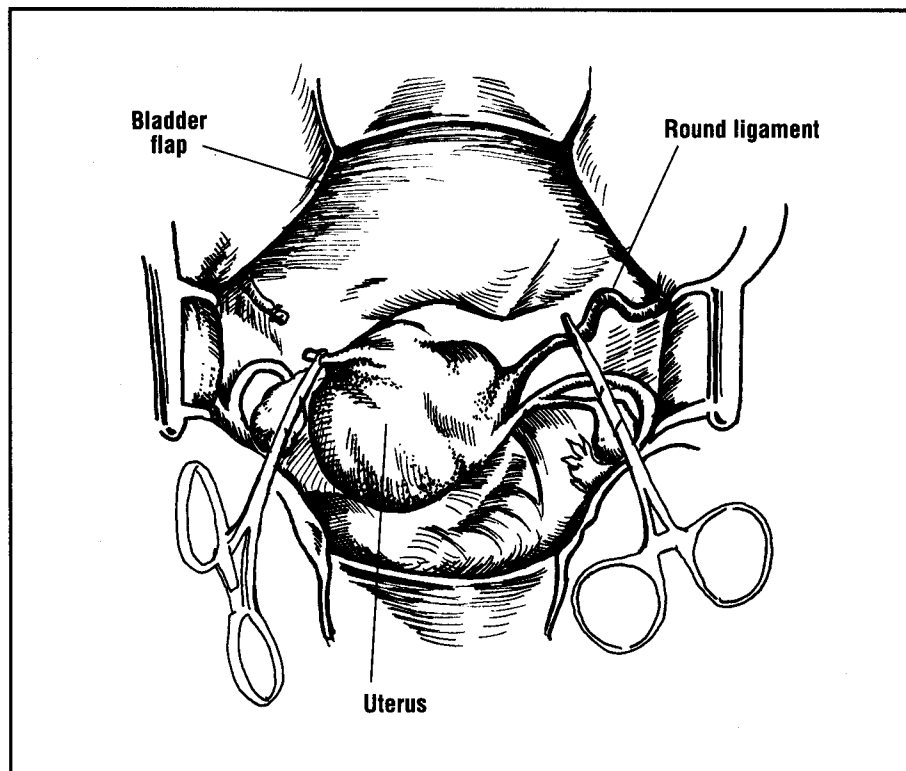
Trendelenburg position. The entire operating table is tilted with the patient's feet higher than the head to provide the surgeon with a better exposure of the surgical area. The surgical technologist must be careful to guard against the Mayo stand touching the patient's feet. Gravity moves the body organs toward the patient's chest and may cause respiratory compromise as the abdominal organs press against the diaphragm. Therefore, the anesthesiologist must carefully monitor the patient to ensure proper ventilation.

The surgeon isolates the uterus by severing it from the uterine ligaments, ovaries, and fallopian tubes. The ligaments are clamped, divided, and ligated in the order in which they appear, beginning with the round ligaments (Figure 4).<sup>4</sup> Ligaments are double clamped with

Heaney hysterectomy clamps, dividing the tissue between the clamps, and the tissue sections are ligated with size 0 or size 1 chromic surgical gut on a heavy tapered Mayo needle. This procedure will be followed for each set of ligaments including the round, broad, cardinal, and uterosacral ligaments. The uterus is mobilized in this manner to the level of the bladder. At this point, the bladder is continuous with the uterus and both are attached by peritoneum. The surgeon separates the bladder and the uterus by dissecting the peritoneal covering away from the bladder with Metzenbaum scissors and long tissue forceps, leaving a bladder flap that will be reattached during closure. Long Allis clamps are placed around the cervix at the level of the internal os. The cervix is divided from the vagina with a long knife or Mayo scissors thereby freeing the uterus from the abdomen. The uterus is passed to the circulator who places it in a basin out of the field; it will be sent as a specimen to the laboratory for analysis following surgery. Throughout the procedure the surgical technologist places all scissors, knives, and any other instruments that have been used on the cervix or the vagina into a separate basin. These instruments are not used again.<sup>1</sup>

**Throughout the procedure the surgical technologist places all...instruments that have been used on the cervix or the vagina into a separate basin.**

Closure of the vagina begins where the cervix was previously attached. Size 0 or size 1 chromic surgical gut sutures on a Mayo taper needle are used for this purpose. As the surgeon sutures, the Allis clamps are removed and the surgical technologist places them in the contaminated instrument basin. The bladder flap is then again peritonealized using a size 2-0 or size 3-0 chromic surgical gut or polyglycolic acid



**Figure 4.** Round ligament grasped, ligated, and cut. (Adapted from Gruendemann.<sup>4</sup>)

suture on a fine swaged-on needle. An absorbable suture must be used to sew the bladder to prevent crystalline stone formation. The O'Sullivan-O'Connor retractor is removed and the peritoneum is closed with a running size 0 chromic surgical gut or polyglycolic acid suture swaged on a taper needle. Muscle tissue is closed with size 4-0 chromic surgical gut sutures. Absorbable or nonabsorbable size 0 or size 2-0 sutures may be used to close the fascial layer. Wound strength lies in the fascial closure. Nonabsorbable sutures may be preferable if the patient is obese. Subcutaneous tissue is closed with a size 3-0 plain surgical gut or polyglycolic acid suture on a taper needle. The skin is approximated with Adson forceps with teeth and is stapled or sutured closed according to the surgeon's preference.<sup>8,9</sup>

**Perioperative Complications**

Many complications may occur during or after an abdominal hysterectomy, the most severe possible complication being injury to one or both

ureters during surgery. The ureters pass close to the ligaments supporting the uterus and must therefore be identified before clamping the ligaments. Identification of the ureters is accomplished by touching them, which causes a peristaltic action. Massive hemorrhaging may occur if the broad ligaments and the incision into the vaginal cuff have not been carefully divided since these areas are highly vascular and the uterus must be properly ligated to prevent profuse bleeding. Careful dissection of the uterus from the bladder is necessary to prevent injury to the bladder wall. Injury to the colon and intestines may occur if these organs are accidentally incised during the opening of the peritoneum. The colon and intestines may become dry during the procedure if the lap sponges are not kept moist or these organs may be damaged by heavy retraction. If the intestines are punctured, peritonitis may occur and death may result.

**Postoperative Care**

The patient's vital signs are moni-

tored in the recovery room until the patient becomes stable and responsive. A fall in blood pressure may indicate abdominal or pelvic bleeding. Pain medication is administered to alleviate incisional discomfort. An antiemetic may be ordered to prevent nausea. The patient should be encouraged to turn, cough, and deep breathe to prevent pneumonia and to aid in the return of circulatory, respiratory, and muscular functions. This sequence should be repeated every 2 hours until the patient is ambulatory. The patient's fluid intake and output should be carefully recorded. If the drainage bag from the Foley catheter is empty and intravenous fluids have been given, damage to the ureters may have occurred. Blood in the urine indicates possible damage to the bladder. The Foley catheter is usually removed 1 to 2 days postoperatively. A liquid diet is ordered until bowel sounds return. The patient can then progress to a diet including solids as tolerated. Intravenous fluids may be stopped once nausea has diminished and the patient is able to tolerate liquids. The patient will usually be encouraged to walk with assistance the same evening as surgery. Dressings should be changed and any abnormal serous drainage recorded. If the vaginal canal contains packing, it should be checked and the packing removed before removal of the Foley catheter. If the patient shows no complications, skin staples are removed within 5 to 7 days and the patient may be discharged. At-home recuperation is approximately 6 weeks long.<sup>8</sup>

### Conclusion

Hysterectomy is a common procedure and one that many surgical technologists will encounter at some point during their time in surgery. As a result, surgical technologists should not only be familiar with the procedure itself but should also understand the emotional impact on the women who undergo such surgery and be prepared to lend support when necessary. Δ

### Acknowledgements

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# Pregnancy-Induced Hypertension as a Precipitating Factor for DIC

ARTICLE BY BOB CARUTHERS, CST, PhD, AND LORI DRIGGS, CST

**E**volution has provided the human being with blood clotting mechanisms intended to prevent blood loss and hemorrhage. Aberrations in the clotting mechanism result in inappropriate or insufficient clotting. Clotting disorders can be divided into two main groups: states of hypercoagulability and bleeding disorders. Disseminated intravascular coagulation (DIC) is one aberration of the normal hemostatic mechanism. In DIC, both inappropriate fibrin formation in small vessels and fibrinolysis are disseminated throughout the vascular bed. DIC is commonly associated with infections, malignancies, shock, trauma, obstetric conditions, blood

## Aberrations in the clotting mechanism result in inappropriate or insufficient clotting.

transfusion reactions, and snake bites. Potentially life threatening, DIC requires rapid treatment of its underlying causes and vigilant patient monitoring.<sup>1,2</sup>

This article (presented in two parts) will focus on the relationship between pregnancy-induced hypertension (PIH) and DIC. In order to explore this relationship, normal blood clotting mechanisms will be reviewed followed by a discussion of the pathogenesis of DIC. PIH will serve as an illustrative example of endothelial trauma leading to DIC; therefore, obstetric conditions

related to PIH will be discussed. Treatment requirements will be briefly reviewed.

### A Fictional Case History

Rebecca A., a 21-year-old primigravida in active labor, with 39 weeks' gestational date by history and fundal height and a history of no prenatal care, was admitted to the high risk labor and delivery unit after recording a blood pressure of 170/116 in the emergency room. The cervix was 80% effaced and dilated 3 to 4 cm. Membranes were intact. A clean-catch urine specimen was checked and a 3+ proteinuria was recorded. The patient's face, hands, and feet were edematous. The patient had no history of drug abuse or other medical problems.

Routine labs were drawn. The hematocrit was reported as 30.2%, hemoglobin 12.5 g/dL, and a white blood cell count of 9,500. The platelet count was 149,000/mm<sup>3</sup>; prothrombin time, 15 seconds; and activated thromboplastin time, 45 seconds. The patient's blood was typed and screened.<sup>3</sup>

The progress of labor was essentially uncomplicated. Blood pressure remained in the area of 170/110 throughout. The patient delivered vaginally. A second-degree midline episiotomy was performed with 1% lidocaine for local anesthesia. The placenta delivered naturally in 17 minutes. Oxytocin and fundal massage were used to assist the uterus to contract. The episiotomy was repaired with 2-0 and 3-0 chromic sutures. Vaginal bleeding continued during the closure. At completion of the episior-

rhaphy, vaginal bleeding continued. The cervix was rechecked for lacerations but none were found. The uterus was firm. However, the bleeding continued and a vaginal pack was placed. The patient was sent to the recovery room.

Lab tests were repeated. The hematocrit showed a drop to 10.7%. The platelet count was 92,000/mm<sup>3</sup>; prothrombin time, 21 seconds; and activated thromboplastin time, 75 seconds. Fibrinogen levels were depressed while fibrin split products were present. Liver enzymes were elevated.

An initial diagnosis of DIC was made and a treatment regimen initiated.

### Normal Mechanism of Blood Coagulation

Hemostasis is a normal process intended to prevent blood loss. The hemostatic mechanisms are vascular spasm, platelet plug formation, blood coagulation, clot contraction, and fibrinolysis.

The ability of the blood to clot depends on the degree of balance existing between substances supporting coagulation and substances inhibiting coagulation. Substances that support coagulation are referred to as procoagulants; those that inhibit coagulation are known as anticoagulants. Under normal conditions, the balance favors the anticoagulants. When a break occurs in a blood vessel, the procoagulants are activated and normal clot formation begins within 15 to 20 seconds at the site of the break. Prothrombin is converted into thrombin. Thrombin acts enzymatically to convert fibrinogen into fibrin. Platelets,

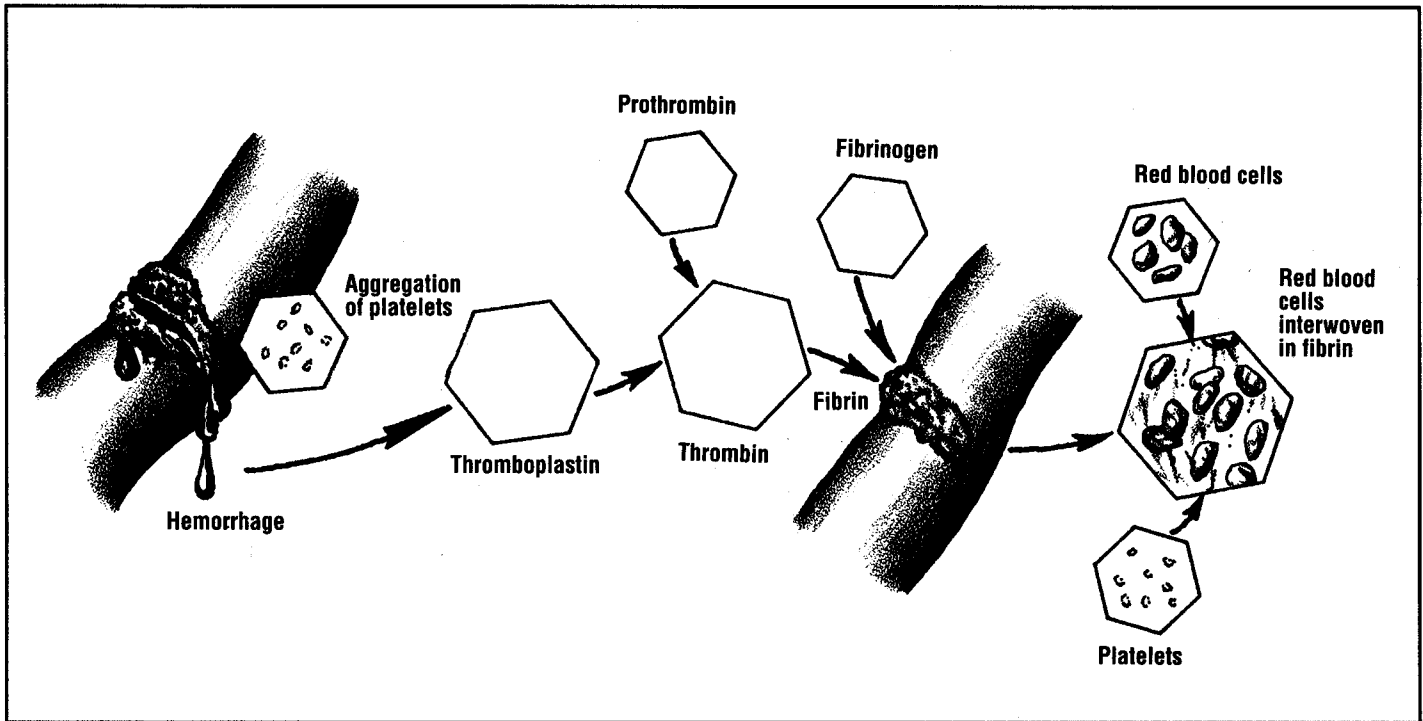


Figure 1. Essential steps in normal blood clotting mechanism. (Adapted from McCance KL et al.<sup>1</sup>)

blood cells, and plasma are caught in the fibrin strands to form a clot (Figure 1).

Two methods exist by which pro-coagulants are activated resulting in the activation of prothrombin. The extrinsic pathway is initiated by trauma to the vessel wall and sur-

**The ability of the blood to clot depends on the degree of balance existing between substances supporting coagulation and substances inhibiting coagulation.**

rounding tissues. The intrinsic pathway is initiated by trauma to the blood itself or blood contact with substances outside the blood vessel endothelium. In both pathways, several different plasma proteins, referred to as blood-clotting factors, are required for normal clotting (Table 1). While generally existing in an inactive state, the activation of these plasma proteins results in the

successive, cascading reactions of the blood clotting process. Except for the first two steps in the intrinsic mechanism, calcium ions are required for all reactions.

Generally, clotting is initiated by both pathways simultaneously following rupture of a blood vessel. Due to the chemical process involved, the activation of the extrinsic pathway secondarily activates the intrinsic pathway. It is possible for the intrinsic pathway to be activated alone in cases of antigen-antibody reactions or drug reactions. An important difference between the two pathways is the speed with which clotting occurs. The extrinsic pathway can produce clotting within 15 seconds, whereas the intrinsic pathway requires 1 to 6 minutes to produce clotting.

Trauma to the vascular wall and surrounding tissue initiates the extrinsic pathway. Several factors, referred to as tissue thromboplastin, are released by traumatized tissue. The lipoprotein complex of tissue thromboplastin interacts with serum prothrombin conversion accelerator (factor VII). In the presence of tissue phospholipids and calcium ions,

this new complex activates the Stuart factor (factor X). Factor X interacts with tissue phospholipids and proaccelerin (factor V) to form prothrombin activator, a substance that splits prothrombin into thrombin.

**Platelets, blood clots, and plasma are caught in the fibrin strands to form a clot.**

Thrombin provides positive feedback, acting on factor V, thereby accelerating the process of splitting prothrombin into thrombin.

Trauma to the blood or contact between the blood and collagen in a traumatized vascular wall initiates the intrinsic mechanism for clot formation. Trauma or contact with collagen affects the Hageman factor (factor XII) and the platelets. Contact with collagen causes factor XII to become an activated proteolytic enzyme and damage to the platelets. The damaged platelets release platelet factor 3, a lipoprotein in the platelet phospholipid. Activated

**Table 1. Clotting Factors and Synonyms in Coagulation Terminology**

Clotting Factor	Synonym
fibrinogen	factor I
prothrombin	factor II
tissue thromboplastin	factor III
calcium	factor IV
proaccelerin	factor V
serum prothrombin conversion accelerator	factor VII
antihemophilic factor	factor VIII
plasma thromboplastin component	factor IX
Stuart factor	factor X
plasma thromboplastin antecedent	factor XI
Hageman factor	factor XII
fibrin-stabilizing factor	factor XIII
prekallikrein	Fletcher factor
kininogen	HMWK
platelets	.....

factor XII in the presence of high-molecular-weight kininogen enzymatically activates plasma thromboplastin antecedent (factor XI), which in turn activates plasma thromboplastin component (factor IX). Activated factor IX in the presence of antihemophilic factor (factor VIII), platelet phospholipids, and platelet factor 3 activate factor X. As previously discussed regarding the extrinsic mechanism, factor X interacts with tissue phospholipids and proaccelerin (factor V) to form prothrombin activator. This substance splits prothrombin into thrombin. Thrombin provides positive feedback, acting on factor V, and thereby accelerates the process of splitting prothrombin into thrombin.

Formation of prothrombin activator is the end product of both the extrinsic and intrinsic pathways. The formation of prothrombin activator is the rate-limiting factor in blood coagulation.

Thrombin acts on fibrinogen causing polymerization of molecules into fibrin strands within 15 seconds. In this part of the process, thrombin acts on fibrinogen, a protein produced by the liver, to produce a fibrin monomer. The fibrin monomer polymerizes with other fibrin monomers. Fibrin-stabilizing factor, activated by thrombin, forms covalent bonds between the fibrin

strands and strengthens the framework of the forming clot. Blood cells, platelets, and plasma are trapped in the meshwork formed by these fibrin strands, which adhere to the damaged surfaces of blood vessels. Further blood loss is prevented by the blood clot created.

Platelets in the clot actually bind the fibrous strands together and continue to release procoagulant substances. One of the substances acts to cause a growing number of cross-linked bonds between fibrin strands. Substances released by the platelets also directly cause the clot to contract. As the clot begins to retract a few minutes after its formation, serum is expressed. Contraction of the clot pulls the edges of the blood vessel together, assisting with hemostasis.

The continued action of thrombin promotes more clotting, which in turn produces more thrombin. Unless actively inhibited, the blood clot will continue to increase in size and density. Two anticoagulants, the fibrin strands and antithrombin-heparin cofactor, stop the thrombin-driven clotting cycle.

Approximately 85% to 90% of all thrombin formed from prothrombin becomes absorbed into the fibrin strands. The remainder combines with antithrombin-heparin cofactor and is inactivated within 20

minutes.

The lysis of blood clots is achieved over a period of several days. Extraneous blood clots are removed and small vessels may be opened by the mechanism of fibrinolysis. Blood clots are broken down by fibrinolysin, a proteolytic enzyme. Profibrinolysin, as well as other plasma proteins, is held in the blood clot. Injured tissue and the vascular endothelium release tissue fibrinolysin activator, which converts profibrinolysin to fibrinolysin in approximately 1 day. The proteolytic activity of fibrinolysin breaks down the clot.<sup>4A</sup>

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*Lori Driggs, CST, a graduate of Austin Community College, assisted Dr Caruthers with the research for this article.*

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# Pregnancy-Induced Hypertension as a Precipitating Factor for DIC

ARTICLE BY BOB CARUTHERS, CST, PhD, AND LORI DRIGGS, CST

**I**n the second part of this article, disseminated intravascular coagulation (DIC) is discussed in relation to pregnancy induced hypertension (PIH).

## Disseminated Intravascular Coagulation

DIC is a complex syndrome representing an aberration of normal hemostasis characterized by indiscriminate clot formation followed by active bleeding. Indiscriminate clot formation consumes fibrinogen and other procoagulants while stimulating the fibrinolytic system. The syndrome is paradoxical in nature; extensive clotting results in systemic bleeding.

As noted in Part I of this article, the extrinsic and intrinsic pathways of blood clotting differ primarily in initial stimulation. Once factor X interacts with tissue phospholipids and proaccelerin (factor V) to form prothrombin activator, the physiological response is the same. DIC, therefore, can be triggered by either the extrinsic or intrinsic pathway or both. Activation of these pathways results in system-wide intravascular coagulation as microthrombi are produced. Among the effects of disseminated intravascular coagulation are (1) the consumption of clotting factors and utilization of platelets and (2) the activation of the fibrinolytic system. Consumption of clotting factors and platelet utilization result in thrombocytopenia, or the decrease in the number of platelets, and deficiencies of coagulation factors. Both conditions contribute to the failure of normal

hemostasis. The activation of the fibrinolytic system results in the digestion of fibrin clots with the release of fibrin degradation products, which act as anticoagulants. Circulating fibrin degradation products inhibit platelet function and coagulation and contribute to the failure of normal hemostasis. Four factors result in hemostatic failure: deficiencies of coagulation factors, thrombocytopenia, inhibited platelet function, and inhibited coagulation.<sup>1,2</sup>

Numerous precipitating mechanisms have been associated with DIC. For the purpose of this article, obstetric conditions, particularly hypertension, will be considered. It is important to note that each precipitating mechanism has a tendency to activate either the extrinsic or

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**The syndrome is paradoxical in nature; extensive clotting results in systemic bleeding.**

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the intrinsic pathway to blood clotting. Although the precipitating mechanism does not affect the direct pathophysiological process of DIC beyond the initial activation of the coagulation pathways, it is important to the therapeutic approach discussed later.

DIC affects the production of microthrombi resulting in a failure of normal hemostasis. The clinical picture associated with DIC reflects these outcomes. Thrombosis in the microcirculatory system affects var-

ious organs. Hematuria and kidney failure may result from damage to the renal cortex. Shortness of breath, chest pain, and decreased blood oxygenation may result from pulmonary infarctions. Liver damage may result in decreased production of proteins and toxin clearance. Adrenal damage may produce lowered production of steroids and epinephrine. Brain infarctions may produce confusion, delirium, coma, and multifocal neurologic deficits. The gastrointestinal tract is prone to ulceration and bleeding. Localized ecchymoses and necrosis may appear on the skin. The hemorrhagic component may produce persistent oozing from venipuncture sites; bleeding around catheters, drains, and tubes; and hematuria, melena, or hemoptysis.<sup>2,3</sup>

Laboratory findings reflect the effects of the disseminated intravascular clotting and the fibrinolysis (Table 1). The platelet count is lowered, fibrin split products are present, and fibrinogen is depressed. Prothrombin time, partial thromboplastin time, activated partial prothrombin time, and thrombin times are prolonged. Clotting time is normal, and euglobulin lysis time is shortened. Ethanol or gelatin tests demonstrate the presence of fibrin monomers.

## Hypertensive Disorders of Pregnancy

Brinkman<sup>4</sup> cites the occurrence of hypertensive disorders in pregnancy to fall between 25% and 36%, a range accounted for by variances in definition and ethnic factors. Hypertensive disorders in pregnancy lead to increased mater-

nal and perinatal morbidity and mortality. The American College of Obstetricians and Gynecologists recommends the following classification system for the hypertensive disorders of pregnancy: (1) preeclampsia and eclampsia, (2) chronic hypertension with superimposed preeclampsia, and (4) transient hypertension.<sup>4</sup> In general, the following requirements should be met in order to clinically diagnose hypertension in pregnancy: (1) an absolute blood pressure of 140/90 mm Hg or higher, (2) an increase of 30 mm Hg in the baseline systolic pressure or 15 mm Hg above the baseline diastolic pressure, (3) readings taken with the patient at rest, (4) readings taken at least 6 hours apart, and (5) readings taken with the patient sitting and with notation of both fourth and fifth Korotkoff's sounds.

Preeclampsia is commonly referred to as PIH. PIH most commonly occurs in young women in the last trimester of a first pregnancy. Earlier onset suggests hydatidiform mole, a grapelike neoplastic mass of enlarged chorionic villi, or choriocarcinoma, an epithelial malignancy of fetal origin developing from the outermost embryonic membrane. Diagnosis is made in the presence of pathologic edema, hypertension, and proteinuria. Preeclampsia may be further categorized as mild or severe. Severe preeclampsia is diagnosed in the presence of blood pressure readings greater than 160/110, proteinuria greater than 5 gm in 24 hours, 3+ to 4+ dipstick readings, and edema of the hands and/or face. The diagnosis of severe preeclampsia is made if any of the following conditions are superimposed on mild eclampsia: oliguria, signs of increased intracranial pressure, pulmonary edema or cyanosis, right upper quadrant or epigastric pain, significantly altered liver function, or significantly lowered platelet count. The diagnosis of eclampsia is made if grand mal seizures are present with the symptoms of either mild or severe preeclampsia.

If the hypertensive disorder predates the pregnancy or is discovered

**Table 1.** Comparison of Test Values for Normal Pregnant Patient and Patient With PIH Resulting in DIC

Lab Test	Test Measures	Normal Pregnant Values	DIC Values
platelets	platelets	normal	lowered
fibrin split products	plasmin action on fibrin	absent	present
fibrinogen	fibrinogen	300-500 mg/100ml	depressed
prothrombin time	extrinsic & common pathways	shortened <11-13 sec.	prolonged
partial thromboplastin time	intrinsic & common pathways	shortened <40-60 sec.	prolonged
activated partial prothrombin time	intrinsic & common pathways	shortened <25-45 sec.	prolonged
clotting time	intrinsic & common pathways	normal 6-12 min.	normal
thrombin time	fibrin conversion rate	shortened 12-18 sec.	prolonged
euglobulin lysis time	fibrinolysis rate	normal	shortened
ethanol test	fibrin degradation products	absent	monomer present

before the 20th week of pregnancy in the absence of a hydatidiform mole or choriocarcinoma, the diagnosis is chronic hypertension. The clinical picture may be confused if the stress of pregnancy exacerbates a prior and undiagnosed condition of chronic hypertension or if PIH is superimposed on chronic hypertension. The hypertensive disorder may be characterized as transient if it occurs in the second half of pregnancy, during labor, or within 48 hours of delivery without attendant proteinuria or renal disease.<sup>4</sup>

Weinstein<sup>5</sup> named a multisystem and potentially life-threatening disorder resulting from PIH the HELLP syndrome, for hemolysis, elevated liver enzymes, and low platelet count.<sup>6,7</sup> Weinstein added the HELLP syndrome to the criteria for

severe preeclampsia and argued for aggressive treatment of this disorder. Unlike pure preeclampsia, HELLP is likely to appear in women who are multiparous, more than 25 years of age, and at less than 36 weeks' gestational age. Approximately one in five presentations occurs in the absence of hypertension.

The hemolysis of HELLP is of the type called microangiopathic hemolytic anemia. Of the microscopic signs of this activity, the presence of fragmented red blood cells is most illustrative for the present purpose. Fragmentation occurs when red blood cells are forced through the fibrin-roughened lumens of small blood vessels. Fibrin deposits are secondary to intimal damage in the vessels from the

effects of hypertension. Liver damage results in elevated serum glutamic-oxalocetic transaminase (SGOT) and lactic dehydrogenase (LDH) levels. Platelet aggregation occurs at the site of the intimal damage within the vascular system thereby reducing the platelet count. HELLP criteria require a platelet count of less than 100,000 mm<sup>3</sup>. The HELLP syndrome, worthy of exclusive consideration itself, provides a clinical picture showing some of the same causative conditions as those found in DIC precipitated by PIH.

Although the exact etiology of PIH is still debated, the most likely hypothesis relates preeclampsia with uteroplacental ischemia.<sup>4</sup> The underlying pathophysiology of PIH is accepted as systemic vasospasm. Increased systemic vascular resistance results in an elevated blood pressure. The vasospasm appears to be related to angiotensin II, a strong pressor. Women who develop PIH

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### **Increased pressure in the vascular system may cause severe organ damage.**

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have an excessive response to angiotensin II. Arteriolar vasoconstriction occurs in all vascular beds. The resultant segmental spasm and dilatation damages the endothelium of the vessels. Platelet aggregation occurs at the sites of this damage.

Several maternal consequences result from PIH. Angiotensin sensitivity increases. Weight gain and edema occur as a result of the third spacing of fluid. Increased resistance causes elevated blood pressure. Blood flow to the heart is decreased. The hematocrit is increased. Increased fluid volume results in pulmonary edema and congestive heart failure. Afferent arteriolar constriction results in increased glomerular permeability to proteins, resulting in proteinuria. In some women, thrombocytopenia may result in prolonged bleeding times. Increased pressure in the vascular system may cause severe organ damage thus affecting renal,

liver, placental, and central nervous system function. Long-term effects are minimal or absent in women with uncomplicated preeclampsia or eclampsia. Complications may cause numerous long-term effects including renal failure and cerebrovascular accidents. Intrauterine growth retardation and fetal distress may have long-term effects on the child and prenatal morbidity and mortality are clearly affected. Maternal mortality is essentially nonexistent with preeclampsia but approximately 10% mortality has been reported in cases of eclampsia and HELLP.

Treatment of PIH is determined by its severity and response to treatment. Preeclampsia may be treated with short-term rest and a no-salt diet, short-term rest only, long-term bed rest, and/or induction of labor if gestational age is greater than 36 weeks. Antihypertensive and anti-convulsive drug therapy may be required. Careful management of fluid balance is necessary if intravenous therapy is received. HELLP is treated with delivery when the gestational age is greater than 34 weeks or sufficient lung maturation is demonstrated. Deteriorating maternal and fetal conditions are treated with delivery by cesarean section.<sup>4</sup>

### **PIH as Precipitating Factor to DIC**

Several pregnancy-related conditions may precipitate DIC including abruptio placentae, fetal death in utero, endotoxemia, amniotic fluid embolism, molar pregnancy, and saline-induced abortion. Tissue trauma associated with childbirth or surgical intervention may also precipitate DIC. In each case, thromboplastic substances are released into the circulatory system activating the extrinsic pathway of blood clotting. Intravascular coagulation occurs with attendant activation of the fibrinolytic system and consumption of clotting factors. Thus, the DIC mechanism is activated.

Under normal conditions, the endothelial surface of the vascular system is efficient at inhibiting coagulation. The ability to inhibit intravascular clotting is dependent

upon three factors. First, the endothelium provides a naturally smooth surface over which the blood flows. The smoothness of this surface prevents damage to the circulating blood cells, thereby inhibiting contact activation of the intrinsic blood clotting system. Second, a mucopolysaccharide is absorbed into the inner surface of the endothelium. Called glycocalyx, this layer repels clotting factors and platelets, inhibiting the activation of the clotting process. Third, a protein substance called thrombomodulin is bound to the endothelium. Thrombomodulin also binds thrombin, removing it from any potential clotting reaction, thereby activating other anticoagulants. PIH is produced by systemic vasoconstriction, an overresponse to the presence of angiotensin II. The alternating pattern of vasospasm and dilatation damages the endothelial lining of the blood vessels. Damage to the

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### **Intravascular coagulation occurs with attendant activation of the fibrinolytic system and consumption of clotting factors.**

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vascular bed may be severe enough to cause the release of thromboplastic substances into the circulatory system and activate the extrinsic pathway of blood clotting. In cases where damage is severe, PIH precipitates DIC through a traumalike event. Microscopic damage to the endothelium causes a loss of both the natural smoothness of the vessel and the glycocalyx-thrombomodulin layer. Both platelets and factor XII are activated in this instance, initiating the intrinsic pathway of blood clotting. PIH, then, is capable of exciting both the extrinsic and intrinsic pathways that, once activated, produce microthrombi. Activation of the fibrinolytic system, consumption of clotting factors, and utilization of platelets follow. Hemostatic failure is the end result.

### Treatment of DIC

Conceptually, the treatment plan for DIC is simple. First, treat the precipitating conditions. For example, in cases of abruptio placentae, fetal death in utero, amniotic fluid embolism, and saline-induced abortion, removal of the fetus and placenta eliminates the causative agent, the thromboplastic substances. Generally, attending to the precipitating condition is the only treatment needed. The liver usually replaces plasma factors within 48 hours. Platelet counts should return to normal within 7 days. Second, treat for severity of bleeding if necessary. This treatment may be necessary to restore circulating blood volume, return red blood cell counts to a sufficient level, or to correct procoagulant deficiencies. Whole blood and/or fresh frozen plasma may achieve these ends.

### Summary

PIH can damage the endothelium of the vascular system extensively enough to activate both the intrinsic and extrinsic blood clotting pathways. This systemic activation results in the production of microthrombi, which consume clotting factors and activate the fibrinolytic system. The end result is a paradoxical loss of hemostasis, known as DIC. Mortality and morbidity rates for both mother and fetus increase because of this syndrome. Primary treatment is aimed at correcting the precipitating problem. Δ

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