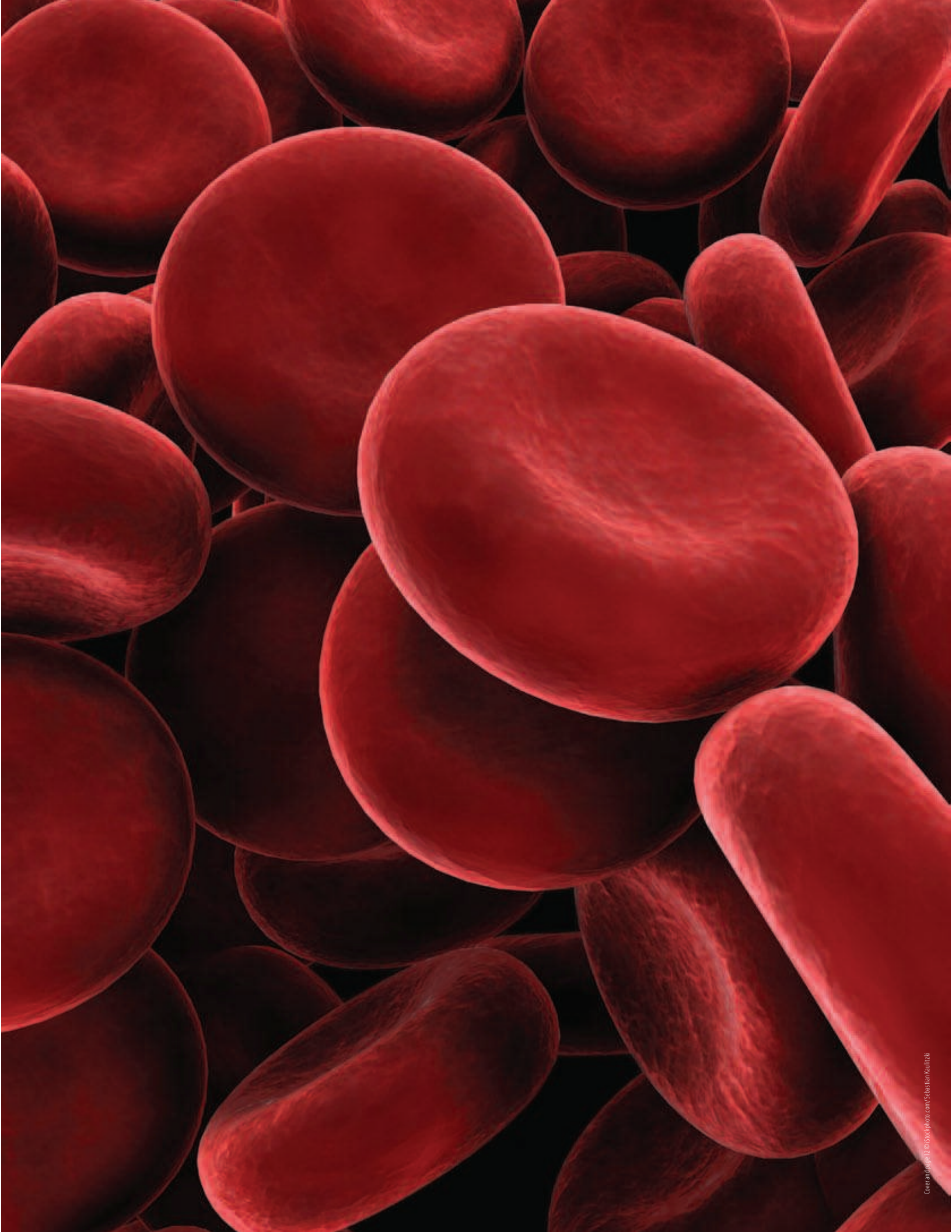

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Blood Clotting Mechanism

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INTRODUCTION

This article is intended as an introduction to the blood clotting mechanism for the student surgical technologist and as a review for those in practice. Effective control of bleeding occurs through a complex process called hemostasis, which will be explained in four basic steps. The basic steps of the blood clotting process are vasoconstriction, platelet activation, thrombus formation, and dissolution of the clot. Basic laboratory tests used to identify blood clotting problems will also be presented.

Blood clotting is initiated in one of two ways. The first, referred to as the intrinsic or internal pathway, occurs when a clot forms inside of a blood vessel due to an internal abnormality or an injury to the blood vessel itself.[8] The second, referred to as the extrinsic or external pathway, occurs following an injury, such as a cut, when blood is exposed to the outside environment. No matter how the clotting process is initiated, the clot forms in the same way. This is referred to as the common pathway. Another term used to describe blood clotting is coagulation.

Blood cells called platelets, along with numerous factors—proteins, enzymes, vitamin K, and calcium—found in blood plasma, are involved in the clotting process. Blood clotting factors are referred to by Roman numerals and also have names associated with them.

Injuries leading to extrinsic blood clotting and the related chain of events will be the focus of this article, as this is the type of injury most commonly seen in the surgical environment. An example is provided in the case study below.

FIGURE 1
Layers of the blood vessel wall.

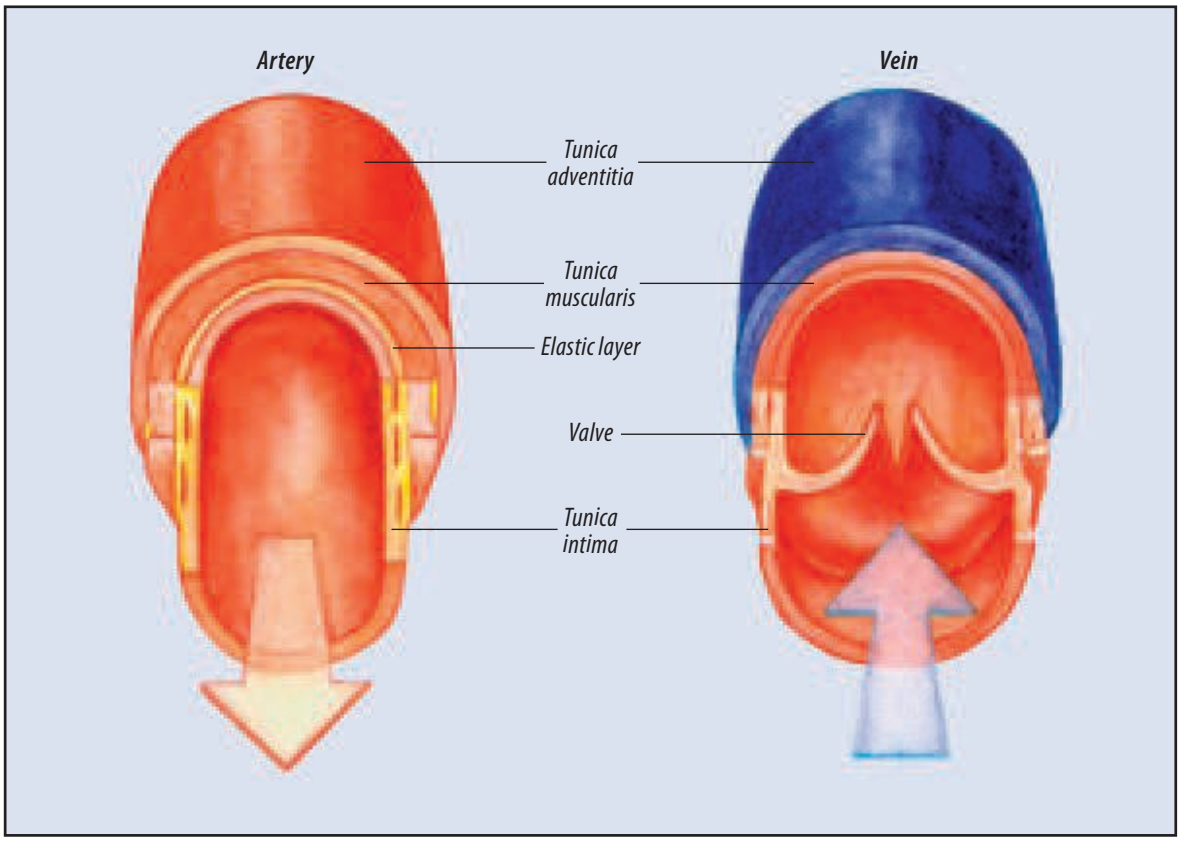


Table 1 Glossary[4,7,8]

Artery	Large blood vessel that carries blood away from the heart
Arteriole	Small artery that delivers blood rich in oxygen and nutrients to a capillary
Capillary	Tiny blood vessel that allows oxygen and nutrients to be delivered to the tissues and carries waste products away
Endothelium	Smooth flat cells that line the heart, as well as blood and lymphatic vessels
Lumen	The inside of a tubular structure (eg, the inside of a blood vessel or intestinal tract)
Tunic	Coat or layer of a structure
Tunica Adventitia (Externa)	Outer layer of a blood vessel consisting of connective tissue
Tunica Intima (Interna)	Smooth inner layer of a blood vessel consisting of endothelium
Tunica Muscularis (Media)	Middle layer of a blood vessel consisting of muscle tissue
Vein	Large blood vessel that carries blood toward the heart
Venule	Small vein that carries blood and waste products away from the capillary to a vein

CASE STUDY

George, an eighteen-month-old male, has fallen while playing on the patio at his home. As he fell, he hit his chin on a terra cotta flower pot and cut himself. His mother rushed to his aid. First, she cleaned the wound with a soft moist cloth; she then applied pressure and ice to help control the bleeding and held George in a sitting position on her lap to calm him. It was determined that George needed stitches in his chin, and he was taken to the urgent care center for further treatment. By the time of his arrival at the urgent care center, the bleeding from his cut had stopped.

ANATOMY REVIEW

To facilitate understanding of the process of vasoconstriction, blood vessel wall anatomy will be reviewed. Please refer to Table 1 for a brief glossary of terms related to blood vessel wall anatomy.

Walls of blood vessels, specifically veins and arteries, consist of three main layers called tunics (Figure 1). The outer tunic is called the tunica externa or tunica adventitia and consists of the connective tissue that maintains the cylindrical shape of the blood vessel. The middle tunic is called the tunica media or tunica muscularis and consists of smooth muscle tissue. The inner tunic is called the tunica interna or tunic intima and consists of smooth epithelial tissue that allows the blood to flow freely within the vessel. Additionally, a layer of elastic tissue is present to allow for expansion (vasodilation) and contraction (vasoconstriction) of the vessel.

The muscular layer is thicker in arteries than it is in veins, and veins have additional structures called valves, which keep the blood from flowing backward. The exception to the three-layer structure of blood vessel walls are the capillaries that consist of only a single layer of cells. This layer allows the passage of fluids, cells, and molecules to and from the tissue cells.[7]

VASOCONSTRICTION

Vasoconstriction is the term used to describe tightening of the muscle in the blood vessel wall. When an injury occurs, nerves in the tissue surrounding the blood vessel stimulate the muscle of the blood vessel wall to constrict, narrowing the lumen of the vessel, thereby temporarily restricting the flow of blood from the wound. This initial vasoconstriction lasts only about a minute, but then a secondary mechanism takes over that sustains vasoconstriction at the site of the wound. The secondary mechanism involves release of chemicals, such as serotonin and epinephrine, from blood cells called platelets that have started to gather at the site of the injury. Platelets continue to stimulate the nerves, which causes contraction of the muscle of the vessel for several more minutes.[4]

First aid measures such as those described in the case study—application of ice and pressure to the wounded area, as well as elevating the wounded area—all assist in the vasoconstriction process.

Table 2 Blood Clotting Factors[5,6]

Roman Numeral	Name
I	Fibrinogen
Ia *(a = activated)*	Fibrin
II	Prothrombin
IIa	Thrombin
III	Thromboplastin (also called tissue factor)
IV	Calcium
V	Proaccelerin (also called labile factor and accelerator globulin (AcG))
VI	Accelerin
VII	Proconvertin (also called serum prothrombin conversion accelerator (SPCA), cothromboplastin, autoprothrombin I, prothrombokinase and stable factor)
VIII	Antihemophilic factor A (also called antihemophilic globulin (AHG))
IX	Antihemophilic factor B (also called Christmas factor, plasma thromboplastin component (PTC), and autoprothrombin II)
X	Stuart-Prower factor (also called autoprothrombin C, Prower factor and thrombokinase)
XI	Antihemophilic factor C (also called plasma thromboplastin antecedent (PTA))
XII	Hageman factor (also called glass, contact or activation factor)
XIII	Fibrinase (also called fibrin stabilizing factor (FSF) and Laki-Lorand factor (LLF). The inactive form is also called protransglutaminase, and the active form as transglutaminase.)

PLATELET ACTIVATION/AGGREGATION

Following damage, such as an accidental cut or a surgical incision, blood is exposed to collagen from the nearby tissues. At the time of injury, two things happen almost instantly and simultaneously. First, two substances—fibrinogen, found in collagen, and prothrombin, found on the surface of the platelets—are activated. (See Table 2) The platelets, or thrombocytes, begin to adhere to the exposed collagen. This initial step in blood clotting, or coagulation, is called platelet activation or platelet aggregation.

As the platelets begin to assemble at the site of the injury, several factors are released from the platelets. One of them, the von Willebrand factor (vWF), binds the platelets to the collagen fibers. Several substances are also released from the platelets, including adenosine diphosphate (ADP), which attracts more platelets until a seal is formed over the opening in the blood vessel wall.

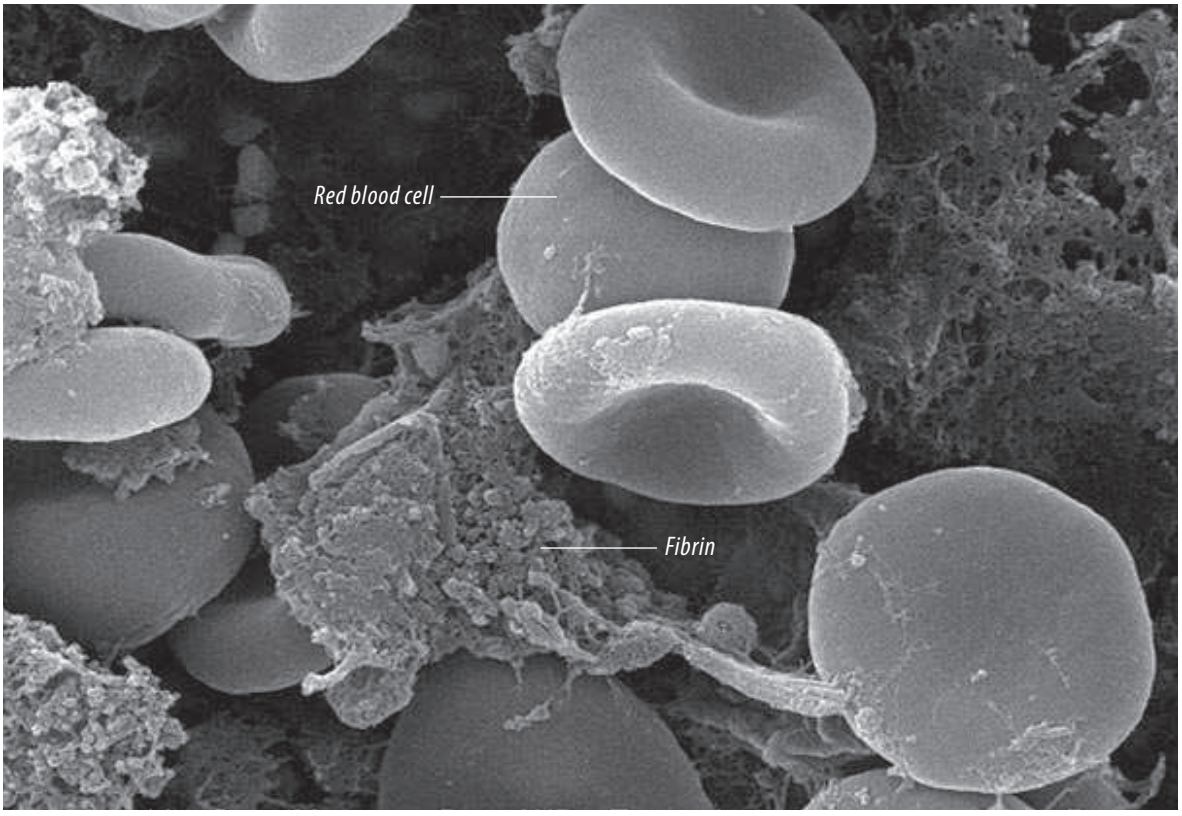
The clump of platelets is known as the platelet plug. The platelet plug may be sufficient to seal a small vessel. However, in larger vessels, the platelet plug is temporary and will be replaced by a thrombus.[6] The platelet plug does not contain red blood cells; therefore, it is sometimes referred to as the white thrombus.[1]

THROMBUS FORMATION

A more stable clot, called a thrombus, must form to make a longer lasting seal on larger injured blood vessels. The thrombus must be able to maintain a seal on the damaged blood vessel wall, especially that of an artery where the blood pressure is higher than in a vein, even after the blood vessel walls are no longer constricted. Fortunately, the clotting process is self-limiting. Substances called antithrombins prevent the clot from continuing to enlarge; otherwise the established clot would block the flow of blood within the vessel.[6]

Activation of prothrombin in the platelet aggregation phase sets off a series of additional events, starting with activation of factor X, that leads to formation of the thrombus. Activated factor X (Stuart-Prower) leads to activation of factor II (prothrombin). Prothrombin in its active form is referred to as factor IIa (thrombin). Several substances, including thromboplastin

FIGURE 2
Red thrombus.



(factor III), calcium, and vitamin K, play a role in activation of prothrombin to thrombin.[1]

Thrombin, in turn, helps to convert fibrinogen (factor I) to fibrin (factor Ia). Fibrin is an insoluble protein that, when it reacts with other substances, polymerizes to form strands that web together to form the basis of the thrombus. The web of fibrin is referred to as fibrin mesh. Factor XIII (fibrinase) is activated to serve as additional stabilization of the fibrin mesh. When viewing the fibrin mesh that has been stabilized with fibrinase under a microscope, it is said to have the appearance of cross stitching on loosely woven fabric.[3]

Within a few hours of the injury, as part of the inflammatory process that normally occurs following damage to tissue, leukocytes (white blood cells) gather at the site of the injury to help reduce the risk of infection. Local inflammation is identified by the presence of pain (dolor), redness (rubor), heat (calor), swelling (tumor), and loss of function (functio laesa). Systemic inflammation is identified by the presence of fever.[7]

The thrombus may contain red blood cells; therefore it is sometimes referred to as the red thrombus (Figure 2).[1]

DISSOLUTION OF THE CLOT

Dissolution of the clot, also called fibrinolysis, occurs when a negative feedback system signals that sufficient healing of the blood vessel wall has occurred, and a substance called plasminogen is activated. Plasminogen in its active form is called plasmin. Plasmin signals the clotting mechanism to inactivate the procoagulant (clotting) process and begins the anticoagulant process, with the use of naturally occurring heparin. This is achieved by digesting the fibrin, which is the main component of the thrombus.[6] The body then returns to its state of balance, called homeostasis.

IDENTIFICATION OF BLOOD CLOTTING PROBLEMS

Due to the complexity of the blood clotting mechanism, numerous problems can occur that will impact the clotting ability of the injured patient or the patient scheduled for surgery. Blood clotting disorders will not be discussed in this article; however, knowledge of the status of the patient's blood clotting mechanism prior to and during surgical intervention is valuable to the surgical team members in planning the care of the patient.

Several laboratory tests are available that will help determine if the patient has normal or abnormal clotting ability. If an abnormality is detected, intervention—such as providing blood products, procoagulants, or anticoagulants—may be indicated.

BLOOD CLOTTING TESTS

The following is a list of some of the more common blood clotting tests, as well as brief descriptions of each.

- **Bleeding time**—Measures the time elapsed from infliction of a small cut until active bleeding has stopped. Normal bleeding time is three to eight minutes.
- **Coagulation factor assay**—Measures specific proteins (clotting factors, as well as anticlotting factors) in a volume of blood for normalcy, deficiency or absence.
- **Complete blood count**—A group of several tests performed on a volume of blood to detect the number and size of certain cells or cell fragments, including red blood cells, white blood cells (including the various types of white cells), and platelets present in the blood.
- **Fibrinogen test**—Measures the levels of fibrinogen (Factor I) in the blood.
- **Miscellaneous**—Various tests may be performed as needed to detect vitamin deficiencies, liver malfunction, or leukemia that may affect the blood clotting mechanism.
- **Partial thromboplastin time (PTT)**—Measures the amount of time needed (in seconds) that it takes for clotting to occur when reagents are added to plasma in vitro. Normal partial thromboplastin time is 30-45 seconds.
- **Platelet aggregation test**—Measures the amount of time needed for platelets to aggregate (adhere to) and seal off the lumen of a vacuum tube that had been coated with collagen and either epinephrine (EPI) or adenosine diphosphate (ADP). As being drawn through

the tube, the coating activates the platelets promoting platelet aggregation. The term closure time (CT) is used to refer to the amount of time it takes for a clot to form inside the glass tube and prevent further blood flow.

- **Platelet count**—Detects the number of platelets present in the blood. It is typically included in the CBC, but may be ordered independently. The normal platelet count is between 150,000 to 450,000 platelets per ML.
- **Prothrombin time (PT)**—Measures the amount of time needed for blood to clot in vitro. Normal prothrombin time is 10–15 seconds.[7]

CONCLUSION

Many factors influence hemostasis, and the clotting process is a very complex series of chemical interactions that are not all listed in this brief overview. Keep in mind the four basic steps of the blood clotting process (vasoconstriction, platelet activation, thrombus formation, and dissolution of the clot) as you think back to the case study, and try to answer the following questions:

1. **Why did George's mother apply ice and pressure to the wound?**
   George's mother applied ice and pressure to the wound to assist his body in achieving and maintaining vasoconstriction.

2. **Why was George kept in a sitting position following his injury?**
   George was kept in a sitting position while his mother was comforting him to elevate the injured area allowing gravity to direct blood away from the wound.

3. **Why do you think that the bleeding had stopped by the time George arrived at the urgent care center for stitches?**
   George's bleeding had stopped by the time he arrived at the urgent care center for stitches, because all the steps involved in his blood clotting mechanism worked correctly.

ABOUT THE AUTHOR

Teri Junge, CST, CFA, is surgical technology program director at San Joaquin Valley College in Fresno, California. She is also the editorial reviewer for this journal.

Editor's note: To review the main constituents of the blood, please refer to "Blood Components" by this author in the July, 2003 issue of this journal. For more information about fibrin's extraordinary elasticity and strength, please visit ***http://www.wfu.edu/wfunews/2006/2006.08.03.f.html*** *and* ***http://www.madgadget.com/archives/2006/08/fibrin_superher_1.html.***

References

1. Atkinson L, Fortunato N. *Berry & Kohn's Operating Room Technique,* 10th ed. USA: Mosby; 2003.
2. Bhonoah Y. Blood Coagulation. Imperial College of Science, Technology and Medicine: Department of Chemistry. Available at: ***http://www.chemsoc.org/exemplarchem/entries/2003/imperial_Bhono/bloodcoagulation.html.*** Accessed June 19, 2006.
3. Carlson C. How Blood Clots. Available at: ***http://www.med.uiuc.edu/hematology/PtClotInfo.htm.*** Accessed June 1, 2006.
4. Cohen B. *Memmler's The Human Body in Health and Disease*, 10th ed. USA: Lippincott Williams & Wilkins; 2005.
5. Corrigan D. *The Blood*. Englewood, CO: Association of Surgical Technologists; 1994.
6. King M. Blood Coagulation. Available at: ***http://web.indstate.edu/thcme/mwking/blood-coagulation.html.*** Accessed June 3, 2006.
7. Price P, Frey K, Junge T, eds. *Surgical Technology for the Surgical Technologist: A Positive Care Approach,* 2nd ed. USA: Thomson Delmar Learning; 2004.
8. Stedman J. *Stedman's Medical Dictionary for the Health Professions and Nursing Illustrated*, 5th ed. USA: Lippincott Williams & Wilkins; 2005.
9. Tiscali Encyclopedia. Available at: ***http://www.tiscali.co.uk.*** Accessed June 3, 2006.