Surgical Hemostasis

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ABSTRACT: Maintaining appropriate surgical hemostasis is an important component of sound surgical technique and reduces postoperative morbidity and mortality. Intraoperative hemostasis can be achieved by using multiple techniques and hemostatic agents. Factors that must be considered when deciding on an appropriate hemostatic agent include tissue type, characterization of hemorrhage, vessel diameter, and any underlying coagulopathy. The purpose of this article is to review the normal physiologic hemostatic pathways, discuss application of different hemostatic techniques, and provide a detailed description of currently available topical hemostatic agents.

Success in surgical procedures in veterinary patients depends on proper surgical technique. Careful attention to proper surgical technique results in fewer intraoperative complications and minimizes postoperative morbidity and mortality. One of the most common complications encountered during surgery is hemorrhage. Methods to stop hemorrhaging blood vessels include direct digital pressure, ligation with suture, electrosurgery, radiosurgery, laser, and topically applied hemostatic agents. The need to effectively manage hemostasis and improve wound healing after surgery has led to efforts to develop an ideal topical hemostatic agent. Properties of an ideal topical hemostatic agent include biocompatibility, biodegradability, minimal antigenicity, easy application, and no inhibition of wound healing.

OVERVIEW OF HEMOSTASIS

Hemostasis is a complex physiologic process involving multiple cellular interactions, secretion of hormones, and activation of the coagulation cascade. The primary hemostatic pathway involves a complex interplay among vascular endothelium, subendothelial collagen, platelets, and prothrombotic substances that results in formation of a platelet aggregate (Figure 1). The secondary hemostatic pathway involves activation of a series of proenzymes, known as coagulation factors, that results in the formation of a fibrin meshwork (Figure 2).

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fibrin meshwork acts to stabilize the existing platelet aggregate. The goal of the primary and secondary hemostatic pathways is to restore the integrity of the damaged vascular endothelium and minimize blood loss from the intravascular space.

**Primary and Secondary Hemostatic Pathways**

The vascular endothelium is a metabolically active structure that serves to maintain vascular tone, thromboresistance, and selective permeability to cells and proteins. Normal, intact vascular endothelium maintains fluidity of the blood and prevents thrombus formation through secretion and expression of anticoagulant molecules, such as thrombomodulin, heparan sulfate, and prostacyclin. Damage to the endothelium results in exposure of subendothelial type II collagen to the circulating blood and activation of endothelial cells adjacent to the site of injury. Activated endothelial cells secrete a number of prothrombotic substances, such as tissue factor, von Willebrand's factor (vWF), and plasminogen activator inhibitor type 1 that stimulate platelet activation and aggregation. vWF is a glycoprotein found in endothelial cells, subendothelial matrix, megakaryocytes, platelets, and plasma. vWF serves as a signaling factor to mediate platelet adhesion to exposed subendothelial collagen. Platelets bind to vWF via the complex of glycoprotein type Ib–type IX. This results in platelet activation and expression of the transmembrane integrin IIbIIIa, which serves as a receptor for binding of fibrinogen, plasma vWF, and fibronectin. Activated platelets secrete platelet aggregation agonists (thromboxane A2, platelet-activating factor, and ADP) that attract circulating platelets to the site of endothelial damage. Platelets undergo a conformational change in response to aggregation factors and form cellular membrane adhesions through cross-linking with bound fibrinogen and vWF. The end result of this primary hemostatic pathway is the formation of a platelet aggregate that seals the endothelial defect.

The factor VII–tissue factor (extrinsic) pathway is considered to be the most important initiator of the secondary coagulation mechanism. Tissue factor (Hageman factor), a potent stimulator of the extrinsic coagulation cascade, is secreted by the vascular endothelium in response to injury. After adsorption of tissue factor to exposed subendothelial collagen, factor VII is activated, which, in turn, activates factors IX and X in the presence of calcium ions and phospholipids.

The intrinsic pathway of the secondary coagulation mechanism is initiated after exposure of factor XII to negatively charged subendothelial collagen. Factor XII is activated in the presence of prekallikrein, high molecular weight kininogen, factor XI, and factor IX. After activation of factor XII, a cascade of events results in the activation of factors XI, IX, and X. Factor VIII, a nonenzymatic cofactor, circulates within plasma bound to vWF. Activation of factor IX is greatly enhanced in the presence of factor VIII. Phospholipid, an important component of the intrinsic and extrinsic coagulation pathways, acts to sustain the coagulation cascade in a rate-limiting manner. The major source of phospholipid is the platelet membrane. In the presence of calcium ions, coagulation factors II, V, VIII, IX, and X bind to and accumulate at the surface of phospholipids, making them more accessible to the ongoing enzymatic reactions.

Activation of factor X is the final step in the intrinsic and extrinsic coagulation pathways. Factor X is activated in the presence of factor VIIa, factor IXa, phospholipid, and calcium ions. Activated factor X is responsible for the conversion of prothrombin to thrombin.

The final steps in the coagulation cascade involve the formation of a fibrin meshwork that stabilizes the platelet aggregate. Prothrombin is converted to thrombin in the presence of factor Va, factor Xa, phospholipid, and calcium ions. Thrombin, a potent coagulation protease, cleaves fibrinogen into fibrin, which forms covalent cross-links in the presence of factor XIIIa and calcium ions. The fibrin meshwork weaves around the platelet aggregate and attaches it to the vascular wall, thus stabilizing the clot.

**Inhibitory Pathways of the Coagulation System**

Inhibitory pathways of the coagulation system include the antithrombin III–heparin system and the thrombomodulin–protein C–protein S pathway (Figure 3). These pathways exist to prevent excessive thrombus formation. Antithrombin III accounts for approximately 80% of the anticoagulative property of blood. Antithrombin III is a serine protease inhibitor that neutralizes thrombin and inactivates coagulation factors IIa, IXa, Xa, Xla, and XIIa. Heparin, a sulfated glycosaminoglycan, binds to antithrombin III and enhances its neutralization capabilities. Heparin has also been shown to enhance the release of tissue factor pathway inhibitor from endothelial cells and interfere with platelet–vWF complexes.

Protein C is a vitamin K–dependent serine protease zymogen that becomes activated after contact with thrombin bound to thrombomodulin on the surface of intact endothelial cells. Protein S serves as a cofactor and increases the affinity of protein C for phospholipid substrates. The activated thrombomodulin–protein C–protein S complex binds and degrades factors Va and VIIIa through a proteolytic mechanism. Deactivation of these coagulation factors prevents activation of factor X and conversion of prothrombin to thrombin.
Fibrinolytic System

The fibrinolytic system, composed of complex physiologic processes, complements the coagulation pathway in maintaining adequate perfusion to tissues and organs (Figure 4). The role of the fibrinolytic system is removal of fibrin clots from blood vessels once hemostasis is accomplished. Plasmin, an endogenous serine protease, is the key enzyme contributing to fibrinolysis. Plasmin is formed from activation of the inactive zymogen plasminogen, via intrinsic and extrinsic pathways. The intrinsic activation pathway of the fibrinolytic system is initiated after conversion of factor XII to factor XIIa.5,20 Extrinsic activators include tissue plasminogen activator, urinary plasminogen activator, urokinase, and streptokinase.5,18 Activation of plasmin results in the breakdown of fibrin into fibrin degradation products, which are phagocyted by the mononuclear-macrophage system. Inhibitors of the fibrinolytic system include plasminogen activator inhibitors types 1 and 2, \( \alpha \)-aminocaproic acid, \( \alpha \)-antiplasmin, and \( \alpha \)-macroglobulin.5,20

It is important to note that the events involved in the primary and secondary hemostatic pathways, inhibitory pathways, and fibrinolytic system are part of extremely complex sequences of biochemical events whose discussion is beyond the scope of this article. Readers should refer to other sources for a more detailed review.2,3,5–7,11,14–18,20

Figure 1—Primary hemostatic pathway. Gelfoam (Upjohn) acts (a) to stimulate platelet aggregation and serves as a matrix for fibrin deposition. Collagen sponge and microfibrillar collagen serve (b) as a source of type II collagen that activates endothelial cells and stimulates the primary and secondary hemostatic pathways. HemaBlock (Abbott Laboratories), a plant-based polysaccharide powder, acts (c) as a molecular sieve to concentrate platelets at the site of hemorrhage. Note: Bone Wax and Surgicel act independently of the primary and secondary hemostatic pathways and are, therefore, not represented in Figures 1 and 2. PAF = platelet-activating factor; PAI-1 = plasminogen activator inhibitor type 1; \( \alpha \)WF = von Willebrand’s factor.

**GENERAL PRINCIPLES AND TECHNIQUES OF HEMOSTASIS**

Hemorrhage occurs after disruption of the vascular wall. In surgical patients, there is potential for hemorrhage anytime an incision is made into the skin, subcutaneous tissue, muscle, and organ parenchyma. A significant hemorrhage may be encountered during various orthopedic and neurosurgical procedures. Regardless of the procedure, careful attention must be paid to minimize the volume of blood loss. Intraoperative hemorrhage with subsequent loss of intravascular volume can have detrimental effects on tissue perfusion, cardiac output, tolerance to anesthetic agents, coagulation, and postoperative healing. A number of techniques are used to achieve intraoperative hemostasis. Success of hemostatic methods depends on proper application and adherence to sound surgical principles.

Important considerations when a hemostatic technique is chosen are tissue type, character of the hemorrhage, and size of the bleeding vessel. The goal of this section is to briefly discuss these considerations as they apply to different hemostatic techniques.

**Direct Pressure and Ligation**

Direct digital pressure or pressure applied via saline-soaked gauze is a rapid, first-line method for minimizing blood loss. Pressure on the disrupted vessel reduces turbulent blood flow and facilitates formation of a platelet aggregate. This technique can be applied to all types of tissue and sizes of blood vessels. Gentle pressure to the area minimizes damage to surrounding soft tissues. A wiping motion should be avoided because it can disrupt the platelet aggregate and fibrin clot. In general, direct pressure is effective for reducing capillary oozing and achieving temporary hemostasis of large vessels that require an adjunctive hemostatic technique.

Hemorrhage from large (>2 mm in diameter) blood vessels often requires ligation with suture to achieve hemostasis. Before suture placement, temporary hemostasis can be achieved through application of hemostatic forceps directly on the vessel. During application of hemostatic forceps, care should be taken to incor-
porate as little surrounding tissue as possible to maximize the holding power of the ligature. Monofilament or braided, absorbable suture can be used. Appropriate suture size is based on the diameter of the vessel to be ligated.

### Electrosurgery

A common method of surgical hemostasis is electrocoagulation using monopolar electrosurgery. Electrosurgery was introduced nearly 200 years ago by surgeons in England. Electrosurgery units produce a controlled, high-frequency electric current, ranging from 1.5 to 7.5 MHz, that is applied to tissue via a handpiece. After application of the tip of the handpiece to the blood vessel, electrical energy is absorbed and converted to thermal energy to produce heat. The generated heat seals the bleeding vessel. Unabsorbed electric current passes through the patient’s body to a ground plate. Electrosurgery is most effective for arteries up to 1 mm in diameter and veins up to 2 mm in diameter. The area to be coagulated must be free of blood and other fluid because the heat generated by the electrosurgery tip does not conduct current through fluid. Hemostasis can be achieved through direct contact of the electrosurgery tip to the blood vessel (obliterative coagulation) or by touching the tip to an instrument occluding the vessel (coaptive coagulation).

Electrosurgery can be used in a wide range of tissue types; however, it should be used cautiously in vessels of organ parenchyma and tissues of the central nervous system as well as during ophthalmic procedures. Placement of a grounding plate with maximal contact with the patient is necessary to minimize absorption of current by surrounding soft tissues. Electrosurgery can be used in a wide range of tissue types; however, it should be used cautiously in vessels of organ parenchyma and tissues of the central nervous system as well as during ophthalmic procedures. Placement of a grounding plate with maximal contact with the patient is necessary to minimize absorption of current by surrounding soft tissues.

Bipolar electrosurgery is recommended for ophthalmic and neurosurgical procedures. Bipolar electrosurgery units consist of a foot switch–activated tissue forceps handpiece. Tips of the handpiece are directly applied to a bleeding vessel, with a slight gap left to allow flow of current. Coagulation is achieved as current travels between the tips of the handpiece. Advantages of bipolar electrosurgery over monopolar electrosurgery include a smaller current requirement, reduced risk of injury to surrounding tissues, and successful coagulation in a wet surgical field.

### Radiosurgery

Radiosurgery is used in a similar fashion as electrosurgery for soft tissue incision and coagulation. Current of various waveforms is produced by the radiosurgery unit and is applied to tissue via a handpiece. Energy is converted to heat through tissue resistance. The main difference between radiosurgery and electrosurgery is the source of generated heat. With radiosurgery, heat is generated within the tissues while the tip of the handpiece remains cold. Thermal energy is not transmitted to adjacent tissue; therefore, no ground plate is necessary.

### Lasers

The use of carbon dioxide and diode lasers has become increasingly popular among veterinary surgeons. Lasers are currently used in the clinical setting to perform incisions, excisions, soft tissue ablation, and elective surgical procedures. A reported advantage of laser surgery is establishment of effective hemostasis while tissue is being incised. Carbon dioxide lasers are most effective at achieving hemostasis of vessels with a diameter of 0.6 mm or smaller. For larger vessels, the laser
can be defocused and moved in a sweeping motion over the vessel to stimulate coagulation. Other advantages of using the laser include the absence of heat conduction through the patient's body, which minimizes the risk of thermoelectric burns, and a more precise focal point, which reduces damage to surrounding soft tissue. Risks include production of airborne contaminants; reflection of the laser beam, with exposure of personnel to the laser; and possible fire hazards. Risks and complications can be minimized with training and education of personnel to ensure proper application techniques.

**TOPICAL HEMOSTATIC AGENTS**

Topically applied hemostatic agents have been used in human and veterinary surgery for many years. Materials that can achieve intraoperative hemostasis include bone wax, gelatin sponges, oxidized regenerated cellulose, collagen sponges, microfibrillar collagen, topical thrombin, fibrin sealants, and, recently, microporous polysaccharide powder. These topical agents have a wide range of clinical applications and are highly effective in obtaining rapid, sustained hemostasis.

Properties of an ideal hemostatic agent include biocompatibility, biodegradability, minimal antigenicity, easy application, and no inhibition of wound healing. Clinicians choosing a topical hemostatic agent should consider the type of tissue, surface area, character of bleeding, patient's status, cost, and availability. Regardless of the product that is used, the primary function of topical hemostatic agents is to facilitate rapid hemostasis through stimulation of platelet activation and aggregation, activation of the secondary hemostatic pathway, and formation of a stable fibrin clot.

**Bone Wax**

Bone Wax (Ethicon), a mixture of semisynthetic beeswax and isopropyl palmitate, is used to control bleeding from the surface of bone. Hemostasis is achieved through mechanical tamponade of bleeding vessels and capillaries, with no direct effect on the coagulation cascade. Bone Wax comes packaged in sterile 2.5-g foil envelopes. Warming of the wax by digital manipulation or immersion of the unopened foil envelope in warm sterile solution is recommended before application. Bone Wax has been reported to be moderately antigenic and nonabsorbable and can delay bone healing through inhibition of osteogenesis.

When Bone Wax was applied to cancellous bone procurement sites, it produced an intense foreign-body reaction and inhibited new bone formation. On the basis of these findings, Bone Wax should be used cautiously at fracture fixation or osteotomy sites where osteogenesis is desired for healing.

**Gelfoam**

Gelfoam (Upjohn), a purified gelatin sponge, is applied topically to achieve hemostasis of oozing capillaries. After contact with blood, gelatin particles swell to provide a tamponade effect on the bleeding capillaries. Platelet aggregation is promoted, and the uniform porosity of the gelatin particles allows them to act as a matrix for fibrin strand formation. Gelfoam comes packaged in strips of multiple sizes that are easily cut and handled with surgical instruments. It is readily absorbed within 4 to 6 weeks, has minimal antigenicity, does not inhibit wound healing, and easily follows the contour of tissue surfaces. Gelfoam is effective in controlling bleeding from the spleen, liver, periosteal surface, and capillaries of the spinal cord. In a study of experimental splenic lacerations in rabbits, use of Gelfoam as the sole method of hemostasis reduced mortality by 78% when compared with a control group. When Gelfoam was applied to cancellous bone procurement sites on the iliac crest in dogs, effective hemostasis and histopatho-
logic evidence of bone regeneration were observed. Another study found a 4-month delay in cancellous bone regeneration when bone defects in the proximal humeral metaphysis of dogs were packed with Gelfoam.

Gelfoam is not recommended in the presence of infection because of its potential to serve as a nidus for abscess formation. Gelfoam should be used cautiously during spinal surgery because it was shown to cause formation of dural adhesions and dural fibrosis. Disadvantages of Gelfoam include difficulty in handling and decreased adherence when saturated, disruption of the platelet aggregate when removed, and longer time to effective hemostasis when compared with other topical hemostatic agents.

Despite these disadvantages, Gelfoam is an effective topical hemostatic agent with a wide range of applications.

**Surgicel**

Surgicel (Johnson & Johnson), an oxidized regenerated cellulose mesh, achieves hemostasis through formation of an artificial clot of cellulose acid independent of the coagulation pathway. The hemostatic effect of oxidized regenerated cellulose has been postulated to be due to the acidic pH (3.5 to 4.5) and the affinity of polyanhydroglucuronic acid for hemoglobin, which forms a hydrated aggregate that “plugs” traumatized vessels. Surgicel is available as sterilized mesh in sizes of $5.1 \times 35.6$ cm, $10 \times 20.3$ cm, $5.1 \times 7.6$ cm, and $1.3 \times 5.1$ cm. The mesh is flexible, is easily manipulated, and conforms well to irregular surfaces. These properties make Surgicel most effective at achieving hemostasis when applied to denuded surfaces of organ parenchyma, and Surgicel was effective at achieving hemostasis in cases of experimental liver trauma in rats. Platelet aggregation, platelet activation, and overall clotting time are much slower with Surgicel compared with the properties of other topical hemostatic agents. The main advantages of Surgicel are that it is readily available, is easily applied and manipulated, and induces minimal inflammation and fibrosis.

**Collagen Sponges**

Collagen sponges (Actifoam [MedChem Products], Helistat [Integra Life Sciences], Instat [Johnson & Johnson], and microfibrillar collagen (Avitene [MedChem Products]) are topical hemostatic agents that are derived from bovine deep digital flexor tendon containing an abundance of type II collagen. Type II collagen is the predominant form of collagen in the subendothelium and is responsible for the attraction of platelets to the site of endothelial injury during normal physiologic hemostasis. The dependence of collagen-derived topical hemostatic agents on a functional coagulation pathway has led to speculation that they would produce effective hemostasis in patients with an underlying coagulopathy. Early in vivo studies found that bleeding times were prolonged after application of microfibrillar collagen to arterial hemorrhage in thrombocytopenic patients; however, effective hemostasis was still accomplished in greater than 70% of these patients. In vitro studies showed that collagen-based hemostatic agents induced more rapid platelet deposition and aggregation when compared with gelatin sponges and oxidized regenerated cellulose. When collagen-based agents were applied to traumatic spleen and liver injuries, hemostasis was rapidly achieved, resulting in less intraoperative blood loss and a reduced need for postoperative blood transfusions.

Collagen sponges are pliable, are easily manipulated with surgical instruments, and conform to irregular surfaces.
surfaces. These sponges are most effective when applied to slowly bleeding organ parenchyma with a large surface area. Microfibrillar collagen is a pliable, absorbent, flour-like material that is applied via syringe. When exposed to blood, microfibrillar collagen acquires a paste-like consistency and easily conforms to the tissue surface. After establishment of hemostasis, excess collagen material is easily removed by irrigation with sterile solution. Collagen-based hemostatic agents should not be used on skin edges because delayed wound healing may occur. Other reported adverse effects include abscess formation, hematoma, wound dehiscence, foreign-body reaction, and hypersensitivity reaction. Collagen-based hemostatic agents are superior to Gelfoam and Surgicel for achieving rapid, sustained hemostasis; however, the low availability and the expense of collagen-based agents limit their use in veterinary medicine.

**Recent Developments**

More recent developments in topical hemostatic agents have focused on the use of thrombin mixed with a gelatin matrix (FloSeal [Fusion Medical Technologies] and Proceed [Fusion Medical Technologies]). The gelatin matrix, derived from bovine corium, is supplied as small granules hydrated within a syringe. “Thrombin (Thrombin-JMI [Topical Thrombin, Parke, Davis & Co.]) is commercially available in the United States and is supplied as a sterile, freeze-dried powder that is reconstituted with a sterile diluent before application. After reconstitution, the thrombin is added to the gelatin matrix and delivered to the site of hemorrhage via syringe. When in contact with blood or other fluids, the gelatin particles swell to produce a tamponade effect and restrict blood flow, with maximum swelling achieved in 10 minutes. The thrombin interacts with blood to convert fibrinogen to fibrin, which results in a fibrin clot that incorporates into the gelatin matrix to form a seal (Figure 2). FloSeal and Proceed are easily applied, and excess material can be removed through irrigation without disruption of the hemostatic seal. Resorption of the material occurs in 6 to 8 weeks.

FloSeal is superior for achieving rapid hemostasis compared with Gelfoam soaked in thrombin. In a study of 93 humans undergoing various cardiac procedures, FloSeal stopped bleeding in 94% of patients within 10 minutes compared with 60% in the group treated with a Gelfoam–thrombin combination. In the same study, FloSeal achieved effective hemostasis in 3 minutes in 72% of patients. Less postoperative morbidity and faster recovery times were also noted in the patients treated with FloSeal. A reported complication with FloSeal was development of antibodies to the bovine-derived thrombin with associated bleeding diathesis. This was an isolated case report; however, development of antibodies to topical thrombin has been reported in 15% to 23% of patients.

To our knowledge, there have been no reports on the use of FloSeal in veterinary medicine. FloSeal may have the advantage of achieving rapid hemostasis after accidental rupture of a large vessel that may be encountered during procedures, such as patent ductus arteriosus ligation, application of an amniotic constrictor for porto-systemic shunt correction, adrenalectomy, or removal of a tumor that has invaded a major blood vessel. Proposed advantages of FloSeal over Gelfoam or Surgicel are more precise application, easier manipulation, and more rapid hemostasis. The intended area of application does not need to be free of blood or other fluids, which makes FloSeal beneficial in areas of rapid hemorrhage. Because the ability of this product to achieve hemostasis relies on the natural conversion of fibrinogen to fibrin, it may be ineffective in patients with an underlying coagulopathy. FloSeal and Proceed are much more expensive than are other commercially available topical hemostatic agents, which limits their use in veterinary medicine.

HemaBlock (Abbott Laboratories) is a new topical microporous polysaccharide powder that is synthesized from a plant-based polysaccharide source. When applied to a bleeding surface, the particles act as a hydrophilic molecular sieve that accelerates natural hemostasis through concentration of platelets, red blood cells, thrombin, and fibrinogen at the site of hemorrhage (Figure 1). Effective hemostasis has been reported in less than 30 seconds after application. HemaBlock is indicated for management of bleeding associated with lacerations, abrasions, and dental extractions, and after surgical debridement of tissue. HemaBlock has also achieved temporary hemostasis during general surgical procedures. HemaBlock comes packaged in boxes of 10 0.5-g applicators within individually sterilized foil packages. Once used, the applicators cannot be resterilized. The polysaccharide powder is liberally applied to the bleeding surface. Light pressure with a gauze sponge for 1 to 2 minutes ensures contact of the powder with blood and tissue. If adequate hemostasis is achieved, excess powder is removed via gentle irrigation of the area with sterile solution. HemaBlock powder should not be injected directly into blood vessels because this may result in emboli formation.

Experience with use of this product at the Veterinary Medical Teaching Hospital at the University of Missouri–Columbia has yielded promising results. Rapid hemostasis (<30 seconds) was observed when HemaBlock was applied to bleeding surfaces of the oral
cavity after excision of gingival and cutaneous tumors. Also, HemaBlock has been advantageous in controlling oozing hemorrhage associated with excision of masses from intraabdominal organs. HemaBlock is approximately twice as expensive as other readily available topical hemostatic agents, and scientific reports addressing the effectiveness and potential applications of HemaBlock in veterinary medicine are currently not available. On the basis of minimal clinical experience, it is our opinion that HemaBlock has the potential to become a widely used adjunctive topical hemostatic agent. Additional clinical experience with this product and scientific testing are necessary to determine the full spectrum of applications.

CONCLUSION

Many techniques have been developed to establish intraoperative hemostasis with the goal of minimizing blood loss. Achieving adequate hemostasis is an important aspect of proper surgical technique, and adhering to sound surgical principles will ensure optimal outcomes for patients. Development of topical hemostatic agents has greatly improved a surgeon’s ability to achieve and maintain hemostasis, reduce patient blood loss, and decrease injury to the surrounding tissue that may occur with conventional hemostatic techniques. An ideal hemostatic agent has yet to be developed; however, with increased understanding and continued research on the coagulation mechanism, a number of new, commercially available products will begin to meet the criteria for the ideal agent.

REFERENCES

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1. ______ is not a function of the vascular endothelium.
   a. Maintenance of vascular tone
   b. Prevention of thrombus formation
   c. Secretion of vWF
   d. Secretion of thromboxane A₂

2. Which statement regarding the primary hemostatic pathway is true?
   a. Exposure of subendothelial type I collagen directly stimulates platelet aggregation.
   b. vWF is secreted only by activated platelets.
   c. Activated platelets undergo a conformational change in response to platelet aggregation agonists.
   d. The end result of the primary hemostatic pathway is formation of a stable fibrin meshwork.

3. Which statement regarding the secondary hemostatic pathway is true?
   a. The intrinsic pathway is the most important initiator of the secondary hemostatic pathway.
   b. Tissue factor is secreted by the vascular endothelium and stimulates the extrinsic system.
   c. The major source of phospholipid for potentiation of the intrinsic and extrinsic system is the vascular endothelium.
   d. Thrombin is the final product of the secondary hemostatic pathway.

4. The anticoagulative activity of antithrombin III is enhanced when bound to
   a. protein C.
   b. protein S.
   c. plasminogen.
   d. heparin.

5. Which agent is not an activator of the fibrinolytic system?
   a. α₂-antiplasmin
   b. tissue plasminogen activator
   c. streptokinase
   d. factor XIIa

6. Electrosurgery is most effective at achieving hemostasis when applied to
   a. veins up to 1 mm in diameter.
   b. arteries up to 1 mm in diameter.
   c. veins up to 20 mm in diameter.
   d. arteries up to 1.2 mm in diameter.

7. Which statement about electrosurgery is false?
   a. Obliterative coagulation is achieved through direct contact of the electrosurgery tip to the bleeding vessel.
   b. Bipolar electrosurgery requires less current than monopolar electrosurgery.
   c. Touching the tip of the electrosurgery handpiece to an instrument occluding a bleeding vessel is an example of coaptive coagulation.
   d. A ground plate is not necessary for monopolar electrosurgery.

8. Which topical hemostatic agent inhibits osteogenesis?
   a. gelatin sponge
   b. collagen sponge
   c. Bone Wax
   d. oxidized regenerated cellulose

9. Severe thrombocytopenia may reduce the efficacy of all topical hemostatic agents except
   a. oxidized regenerated cellulose.
   b. the gelatin sponge.
   c. Bone Wax.
   d. a and c

10. Properties of an ideal topical hemostatic agent include
    a. biocompatibility and biodegradability.
    b. minimal antigenicity.
    c. easy application.
    d. all of the above