Textbook of Oral and Maxillofacial Surgery

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Chapter 13

Hemorrhage and shock

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The ancient Greek words for "blood" and "to burst forth" are the derivations of the modern English word "hemorrhage" and serve as a graphic definition of the subject of this chapter. Included will be the various types of hemorrhage from that expected during surgery, through an annoying oozing during the postoperative phases, to exsanguination. This chapter will discuss the appropriate classifications, etiologies, prevent, and direct treatment of hemorrhages associated with the oral cavity.

Bleeding and Clotting Phenomenon

Hemostasis is the spontaneous or the induced cessation of the flow of blood from ruptures in the integrity of the vascular system. Spontaneous or controlled cessation involves overlapping considerations of intravascular factors, which include the blood platelets, calcium, and coagulation protein; extravascular factors, which include the general metabolism, organ systems, connective tissues, and mucosal and cutaneous tissues; and the vascular factors, which include the general metabolism and the type, size, and location of the blood vessels.

Intravascular factors

Platelets, with their functions of adhesion and aggregation, form a hemostatic plug in small vessels. In large vessels another function of platelets, clot retraction, plays a necessary role in closing an opening or a break in blood vessels. The intravascular clot of platelets does not endlessly propagate itself, and thereby produce serious consequences, because of the conversion of soluble fibrinogen to insoluble fibrin, which absorbs the thrombin produced from prothrombin during blood coagulation. Once hemostasis is established and wound repair begun, the ubiquitous platelets have still another function, the lysis and removal of fibrin.

Blood coagulation. There is an international classification of 13 blood moieties (12 protein and one ionic calcium) that participate in the coagulation of blood (Table 13-1). These coagulation factors are designated by Roman numerals I through XIII, representing the order in which they were identified, not the order in which each plays a role in blood coagulation.

Table 13-1. Coagulation factors

Factor	Descriptive terms and synonyms
I	Fibrinogen
II	Prothrombin
III	Thromboplastin
IV	Calcium
V	Proaccelerin, accelerator, globulin, labile factor, thromboplastin cofactor, Owen's factor
VI	No longer used
VII	Proconvertin, serum prothrombin conversion accelerator (SPCA), stable factor, prothrombin accelerator
VIII	Antihemophilic factor (AHF), antihemophilic globulin, thromboplastinogen
IX	Plasma thromboplastin component (PTC), Christmas factor
X	Stuart-Prower factor
XI	Plasma thromboplastin antecedent (PTA)
XII	Hageman factor, contact factor
XIII	Fibrin-stabilizing factor (FSF), Laki-Lorland factor.

The objective of the first stage of coagulation is concerned with the activation of thromboplastin by the rapid extrinsic tissue system and by the slower-acting intrinsic or extravascular system, which both contribute to a common pathway to produce activated thromboplastin. With regard to the extrinsic system, essentially all tissues contain a complex of materials known as thromboplastin factor (Factor III) that is activated by the enzymatic reaction of the B-globulin, preconvertin (Factor VII), and free calcium (Factor IV). Production of Factor VII by the liver is dependent on vitamin K.

The intrinsic system is initiated by the activation of the Hageman factor (Factor XII) by contacting roughened intima or collagen. (Contact with a glass slide likewise will initiate the blood coagulation and permit in vitro studies.) Following the activation of Factor XII, there is a sequential series of activations in which each step initiates the next. Factor XII activates plasma thromboplastin antecedent (PTA) (Factor XI); Factor XI activates plasma thromboplastin component (PTC) (Factor IX); Factor IX activates the antihemophilic factor (AHF) (Factor VIII). The activation of Factors IX and VIII requires the presence of the ionic calcium ions (Factor IV).

After both the extrinsic and intrinsic systems have begun the activation of thromboplastin, a final pathway common to both systems is activated. Stuart-Prower factor (Factor X), proaccelerin or Owen's factor (Factor V), a platelet factor, and the ionic calcium factor (Factor IV) contribute, in the order given, to the final activation of thromboplastin.

The objective of the second stage is to convert the inactive enzyme prothrombin (Factor II) to thrombin by means of the thromboplastin generated in stage one in the presence of calcium ion. Adequate amounts of Factors V, VII, and X are necessary for this conversion to take place; therefore, a deficiency of any of these factors will result in a prolonged prothrombin time.

The objective of the third stage of the coagulation system is to convert soluble fibrinogen (Factor I) to insoluble fibrin. Fibrinogen, by action of thrombin, is converted to a fibrin monomer that aggregates to form fibrin polymers. Small amounts of the fibrin-stabilizing factor (Factor XIII), which is activated by thrombin and the ever-present calcium ion, produce cross-linking of the fibrin polymers, producing the fibrin clot.

The degree of hypocalcemia that would prolong blood coagulation, less than 2.5 mg per 100 mL, is not compatible with life, therefore lack of calcium is not a cause of aberrant coagulation time.

Extravascular factors

The extravascular factors involved in hemostasis depend on the state of health, the tonicity, and the tautness of mucosa, submucosa and cutaneous, subcutaneous, muscular, and other tissues that surround and support the blood vessels. Disorders that lead to atrophy of subcutaneous tissues (purpura senilis), fragility of skin (Cushing's syndrome), or degeneration of elastic tissues (pseudoxanthoma elasticum) may produce benign purpura and may not be as severe as disorders produced by defective intravascular factors.

Vascular factors

Disorders of the vessels themselves, from capillaries to arteries and veins, may produce a group of vascular purpuras. The etiologies vary from scurvy, caused by a deficit of vitamin C that causes a defect in the intercellular cement of small vessels, to purpuras resulting from infection, chronic renal diseases, and allergies. As a rule, the mechanisms of these purpuras are not well understood.

Laboratory Tests and Analysis of Hemorrhagic Disorders

The studies of the greatest clinical value in analyzing the coagulation factors are prothrombin time, partial thromboplastin time, thromboplastin generation time, and platelet count and clot retraction. To provide an estimate of the integrity of the extravascular and vascular factors, the bleeding time and the tourniquet tests are used.

Prothrombin time (PT) test

In the PT test, plasma that has been rendered incoagulable by an anticoagulant is added to a mixture of equal amounts of active tissue thromboplastin and calcium ion. Thromboplastin activates the prothrombin to form thrombin, which converts fibrinogen (Factor I) into insoluble fibrin. Since fibrin is the end point of prothrombin time, adequate amounts of Factor I must be present to produce a normal finding of 11 to 12.5 seconds with this test. The PT measures only certain factors in stages 2 and 3 of coagulation; stage 1 is bypassed. Deficiencies of Factors I, II, V, VII, and X will result in prolonged PT. Normal PT may be obtained when deficiencies exist in Factors VIII, IX, XI, XII, and XIII and in the phospholipid of platelet factor 3.

Partial Thromboplastin time (PTT) test

The PTT test is a measure of the integrity and activation of the intrinsic system and common pathways of thromboplastin generation by the addition of a lipid form of thromboplastin. The integrity of stage 2 of coagulation and the adequacy of Factor I (fibrinogen) in stage 3 are measured. Deficiencies of Factors VII and VIII and the phospholipid of platelet factor 3 are not detected. There are different ranges of normal for PTT depending on the specific type of PTT test being performed in a given medical laboratory.

Thromboplastin generation time (TGT) test

The TGT test is based on the development of potent thromboplastin activity during the incubation of absorbed normal plasma, normal serum, and normal plasma in the presence of ionic calcium. Rates of development of thromboplastin activity are measured and compared to rates obtained when the patient's plasma, serum, or platelets are substituted for their normal counterparts in additional mixtures. Thus differentiations as to factor deficiencies may be interpreted because normal serum contains Factors VII, IX, X, and XII and lacks Factors I, II, V, VII, and XIII; absorbed plasma includes Factors I, V, VIII, XI, XII, and XIII and platelet factor 3; absorbed normal plasma lacks Factors II, VII, IX, and X.

Platelet count

Platelet count gives a quantitative result not the qualitative aspect of platelet functions in the coagulation mechanisms. Since multiplication factors are used, a difference of only 10 in the counting could result in a difference of 10.000 to 50.000 platelets on the report. Of great value is the description of the platelets and the morphological examination of the blood smear.

Clot retraction

Clot retraction depends on the quantity and quality of platelets necessary to influence the completeness of retraction of the coagulum produced by nonanticoagulated whole blood in a test tube rinsed in normal saline. Multiple variables that do not help to pinpoint the etiology (for example, poor clot retraction as seen in thrombocytopenia, multiple myeloma resulting from viscosity of plasma, and polycythemia caused by an over abundance of erythrocytes) may affect this test.

Bleeding time

The bleeding time test measures the vascular response to hemostasis or the ability of small vessels to contract and retract and achieve a fibrin plug following injury. The test measures the time from injury to cessation of hemorrhage caused by a puncture wound. The test is performed either by an earlobe puncture or by three uniform punctures in the ventral surface of the forearm, respectively, the Duke and the Ivy methods; the time is measured from puncture to cessation of hemorrhage and, in the Duke method, ranges from 1.5 to 4.5 minutes. The test is influenced by the general state of extravascular factors of age and nutrition and by the microphysical factors of the skin and subcutaneous tissues. Prolonged times will be

seen in thrombocytopenic states and pseudhemophilia, that is, von Willebrand's disease, in which there is a deficiency to some degree of Factor VIII plus prolonged bleeding time.

Tourniquet test

The tourniquet test, also termed the Rumpel-Leede test or the capillary fragility test, measures the response of the arteriole-venule junction to internal stresses. Providing the systolic pressure is above 100 mm of mercury, a constant 100 mm of mercury is maintained for 5 minutes. The appearance of multiple petechiae distal to the blood pressure cuff denotes a positive result and may indicate scurvy, thrombocytopenia, and certain purpuras.

Microhematocrit test

The microhematocrit test is a standard procedure in many offices wherein general anesthesia or deep sedation is used with ambulatory outpatients. Obviously, the reason for the test is to obtain an estimate of the quantity of erythrocytes available for transporting oxygen. As Westwood, Tilson, and Margeno-Rowe pointed out, microhematocrit examination is not a substitute for the coagulopathy tests mentioned previously, but it may be indicative of conditions that predispose the patient to hemorrhage. For example, resting on top of the packed erythrocytes following centrifuging is the buffy coat layer that is composed mostly of leukocytes. a large buffy coat may suggest the presence of an infection or a blood dyscrasias such as a leukemia. The plasma layer is above the buffy coat, and a yellow color may indicate various normal metabolic as well as diseased states: a high vegetable diet, liver disease, or diabetes. The preceding are observations and indications that may or may not be present in any given patient, but observation of the hematocrit is part of the total ongoing inspection of the patient that is made by dedicated doctors.

Clinical Abnormalities of Blood Coagulation

The clinical manifestations of abnormalities of blood coagulation may be categorized and summarized according to the following outline:

A. Acquired
    1. Impaired synthesis
        a. Liver disease
        b. Renal insufficiency
    2. Development of circulating anticoagulants
        a. Disseminated intravascular coagulopathy (DIC)
        b. Fibrinolysis
    3. Pharmacotherapeutic agents
        a. Administration of warfarin (Coumadin). Note: Surgery is contraindicated if PT activity is greater than two times the control. The PT test should be performed on the day of surgery.
        b. Heparinized patients
        c. Quinine
        d. Acetylsalicylic acid
        e. Oral contraceptives
        f. Citrate salt agents, as in blood preservatives in blood transfusions

B. Genetically inherited

1. First stage of coagulation deficiencies

a. Factor VIII deficiency (classical hemophilia)

(1) Sex-linked recessive disorder

(2) Female is carrier with no clinical signs or symptoms.

(3) No sons of hemophiliac would have disorders; half of daughters would be carriers.

(4) Unusual bleeding at an early age as a result of trivial trauma

(5) Bleeding into joints is frequent, also skin and gastrointestinal (GI) and genitourinary (GU) tracts.

(6) Treatment

(a) To control hemorrhage, either fresh blood or fresh plasma or cryoprecipitated and glycine-precipitated concentrate is administered.

(b) Preoperatively, a minimum level of 35% of normal concentration of Factor VIII should be maintained by infusions of human Factor VIII concentrates.

(7) Majority of hemophiliacs have had hepatitis; thus precautionary measures should include autoclave sterilizing of instruments.

b. Factor IX deficiency (Christmas disease or hemophilia B)

(1) Sex-linked recessive transmission as with Factor VIII deficiency. The recessive gene may express itself to the degree that symptoms may appear in female heterozygotes.

(2) Hemorrhage may first appear at birth from the umbilical cord or following circumcision.

(3) In later life, bleeding into GI and GU tracts, bone and joint, and skin but less than with Factor VIII deficiency; however, hemorrhage following exodontics and other surgery may be as excessive and persistent as with Factor VIII deficiencies.

(4) Treatment

(a) A concentrate may be used that contains Factors II, VII, IX, and X.

(b) Usually plasma or whole blood is administered and should be used if doubt exists as to the specific deficiency.

c. Factor XI deficiency

(1) Transmitted as an autosomal dominant trait, not sex linked. Therefore, either male or female has a 50% chance of passing the disorder to offspring.

(2) Bleeding is not as frequent nor as severe as with Factor VIII and IX deficiencies.

(3) Rarely, in comparison with Factor VIII deficiency, is there GI and GU tract, bone and joint, and skin hemorrhage.

(4) Following minor surgery such as exodontics, hemorrhage is usually as severe as with Factor VIII and IX deficiencies.

(5) Treatment

(a) Preoperatively, 50% level of Factor XI should be maintained using infusions of Factor XI concentrate. Usually 4.5 mL frozen plasma per kg of body weight will raise level 10%.

(b) Level should be replenished every 2 or 3 days postoperatively even though half-life of Factor XI in blood is 60 hours.

d. Factor XII deficiency

(1) No clinical symptoms of hemorrhagic diathesis; therefore, patients will withstand surgery in a normal manner.

(2) Disorder usually is found by accident when preoperative studies reveal normal PT but extremely prolonged clotting time.

2. *Either* first or second stage disorders

a. Factor II deficiency

(1) Genetic disorder - very rare autosomal trait with mild manifestations

(2) Acquired form

(a) Liver disease

(b) Iatrogenic disturbance of vitamin K metabolism, sterilizing drugs in GI tract, and coumarin class of antiprothrombin drugs

(c) Manifestations are hemorrhage from gingival tissues, nasal passages, GU and GI tracts, and skin - rarely hemarthrosis.

(3) Treatment

(a) Plasma infusions

(b) Concentrate of Factors II, VII, IX, and X plus vitamin K, usually intramuscularly 25 to 50 mg at a rate of less than 5 mg per minute.

b. Factor VII deficiency

(1) Inherited codominantly, and onset of hemorrhage in infancy may be from umbilical cord. GI tract hemorrhages, epistaxis, and bruising are common.

(2) Acquired form is seen with liver disease and administration of coumarin drugs.

(3) Treatment is with concentrates of Factors, II, VII, IX, and X, and a level of Factor VII at 15% to 20% will control spontaneous hemorrhage as well as hemorrhage resulting from surgery. If deficiency is induced by coumarin drugs, treatment is administration of natural vitamin K.

Extravascular Hemorrhage

Extravascular hemorrhage may be classified according to the type of vessels involved and the time of the hemorrhage.

Type of vessels

The character of hemorrhage will depend on the type of vessels severed - arteries, veins, or capillaries. *Arterial hemorrhage* will be distinguished by its pulsating character, vigor of the flow, and bright red coloration of the blood. *Venous hemorrhage* may not have a pulsating quality, the flow will be slightly less rapid, and there will be a darker red coloration. In the head, neck, and maxillofacial tissues, the lack of valves in the veins and the short connections between the jugular system and the terminal branches often will permit awesome surges of venous blood when a major vein is severed. *Capillary bleeding* will be oozing, nonpulsating in character, and an intermediate red, a color between the bright red of arterial blood and the darker red of venous blood.

Capillary bleeding may be quite aggressive in the oral and maxillofacial region as a result of the strong arterial pulse on one side of the capillaries and the open, direct, nonvalved access to the jugular system on the venous side. Classifiable as capillary hemorrhage is the vigorous bleeding encountered during procedures in the vascular shunt area posterior to the mandibular condyle.

Time of hemorrhage

Primary hemorrhage occurs as a normal part of surgery as well as from lacerations incurred in trauma. In most intraoral operations, for example, exodontics and alveoloplasty surgery, the normal bleeding time will provide reasonable hemorrhage control. The application of pressure dressings in the form of gauze packs, immediate dentures, or splints will control the primary hemorrhage; also, the vasoconstrictors in local anesthetic solutions will help to control primary hemorrhage. In some cases in intraoral surgery, clamping and tying or electrocoagulation may be necessary to control bleeding; the latter modalities are, of course, routinely used in extraoral surgical approaches.

Secondary hemorrhage occurs during the postoperative phase. Some doctors use the phrase "intermediate hemorrhage" to describe the unexpected hemorrhage that occurs in the first 24 hours postoperatively and "secondary hemorrhage" for the hemorrhage that occurs after the first 24 hours. Regardless of the time phases, secondary hemorrhage that occurs after the first 24 hours. Regardless of the time phases, secondary hemorrhage following intraoral surgery is most often associated with the presence of foreign bodies in an alveolus. This may be a splinter of bone, a fleck of enamel, or a piece of dental restorative material that cause repeated, delayed organization of a blood coagulum. The result may range from an aggressive oozing hemorrhage of blood that continually fills the oral cavity, to a liver clot, to mere blood-tinged saliva that causes alarm to the uninformed patient.

If the secondary hemorrhage is from a metabolic or blood intravascular coagulopathy, definitive care must include managing the general systemic problem.

Prevention of extravascular hemorrhage from dentoalveolar areas

Following surgery, from simple exodontics to extensive alveoloplasties, the surgical sites are irrigated with normal saline and cleansed with suctioning. It is reasonable for the

doctor to take the suctioning device and inspect all aspects of the surgical site while the oral surgical nurse irrigates with normal saline.

The postoperative pack is a 10 by 10 cm gauze that is dampened with saline and molded over the surgical site. The saline decreases the tendency of the clot to become embedded in the gauze mesh. The molding of gauze is designed so that the tongue is presented only with smooth folded margins, the free edges being toward the cheek. The gauze is tailored so that none of it protrudes from the oral cavity when the lips are closed; for anterior extraction sites it may be necessary to use 5 by 5 cm or 7.5 by 7.5 cm gauzes to ensure that the material will not protrude from the lips and produce unsightly extraoral excretions.

The postoperative instructions are given by the doctor to either the patient or a responsible adult. The instructions are delivered in a deliberate, concerned manner and include impressing on the patient that the gauze must stay in place and that talking, eating, and expectorating should not be done for 2 to 3 hours. An allegory is made to the patient that pressure bandages are frequently taped or wrapped over the site following surgery elsewhere on the body; however, the only way a pressure bandage may be placed in the patient's mouth is by the cooperation of the patient in closing firmly on the gauze for 2 or 3 hours. The time the gauze may be removed is *written* on the instruction sheet.

Each patient is telephoned in the evening to reinforce, among other things, that blood-tinged saliva is normal and should not be confused with hemorrhage.

This regimen, each step of which is critically important, has been highly successful in controlling postoperative problems produced by secondary hemorrhage. Incidentally, when elective surgical procedures are performed only in patients in whom there are no oral and *pharyngeal* infections, there has been a significant reduction in postoperation localized osteitis and other infections. It is logical that deft, delicate surgery performed in an area free of infections and well protected by a vitalized blood coagulum would decrease untoward sequelae.

Extravascular hemorrhage control

Coagulation may be delayed by deficiencies of factors described previously, and this was termed intravascular hemorrhage. However, given a normal coagulation system, the control of extravascular hemorrhage is primarily dependent on a normal vessel contraction, retraction, and fibrin plug. During a surgical procedure, positive control of hemorrhaging vessels is possible with various procedures and agents. It is a sound and a fundamental surgical principle that one leaves a dry field, that is, an area with no macroscopic hemorrhage, at the conclusion of an operation. This prevents hematoma formation that may dissect to involve other areas, such as the airway, or may serve as a bed in which microorganisms can flourish. Extravascular hemorrhage control must be effective, also, to stem aggressive and sometimes life-threatening effusions.

Assuming the patient has a normal coagulation time, the agents and methods for control of extravascular hemorrhage are pressure, direct occlusion with hemostats, coagulation of the hemorrhaging point by the precipitation of proteins, occlusive dressings, production of

an artificial clot, provision of an artificial fibrin network, hastening of the coagulation mechanism to produce vasoconstriction, production of an adherent, protective bonding between the coagulum and the surrounding tissues, and administration of general systemic agents that are calculated to decrease hemorrhage.

Pressure will control most hemorrhages. This may be direct occluding of a pressure point of a major vessel leading to the hemorrhaging site. Pressure points are located between the mandibular gonial angle and the sternocleidomastoid muscle to control the external carotid artery, over the mandibular notch (premasseteric incisure) to control the facial artery, and between the tragus of the ear and the zygomatic process of the temporal bone to control the temporal artery. Pressure may be exerted by pooling of escaped blood that tamponades the ruptured vessels. In a negative sense, the lack of pressure manifested in shock may decrease hemorrhage. Obviously, the best method of control would be directly at the hemorrhage site, and according to surgical circumstances, this may be pressure with intraoral gauzes or splints and extraoral bandages.

Direct occlusion of bleeding vessels is obtainable by the application of mosquito hemostats, so named because of their delicate, fine points. The hemostats may be curved or straight; the curved ones are more versatile and more readily lend themselves to the application of ties.

The ties may be nonabsorbable black silk, 3-0 or 4-0, usually placed on named vessels, that is, vessels large enough to be noted by an anatomical name. Smaller vessels are tied with 3-0 or 4-0 plain gut or polyglycolic acid sutures. These absorbable sutures will persist for varying lengths of time. Plain gut will persist the shortest time (measured in weeks), the polyglycolic acid the longest (measured in months). The usual technique involves placing two hemostats on a vessel, points curved to the area to be divided; the vessel is cut, and with the points directed upwards, the ties are placed. Stick ties are those in which sutures are placed in the soft tissues lateral to the free end of the vessel that has been clamped; the knot is drawn tight to occlude the vessel by compression from the surrounding tissues when the hemostat is removed.

Coagulation of the hemorrhaging point may be produced by a variety of modalities that precipitate proteins. Electrocoagulation and cryotherapy are controlled, destructive techniques that produce occlusion of vessels. Styptic and astringent agents have been employed for hundreds of years; among the most popular have been ferric subsulfate, in the form of Monsel's solution, and tannic acid, a home remedy in the form of tea bags. In general, the chemical agents have been replaced by the use of thrombin and absorbable gelatin sponge for the control of intraoral secondary hemorrhages.

Bone wax (beeswax and salicylic acid) may be used in small amounts to occlude bony canals that bear hemorrhaging vessels. These vary from walls of dental alveoli to foramina through which the neurovascular bundles have been avulsed.

An artificial clot may be produced by oxidized cellulose (Novacell, Oxycel) and regenerated oxidized cellulose (Surgicel). Oxidized cellulose attracts erythrocytes and thus produces an artificial clot. The material should be used dry, and, in the absence of infection and in areas where bone repair is not necessary, it provides rapid control of capillary and

venous oozing hemorrhages. The material may be placed over, not in, an alveolus and maintained with a pressure gauze to help control primary hemorrhaging. Remnants of the gauze may be removed by flushing with normal saline or an alkaline solution.

An artificial network that will disrupt platelets is created by application of an absorbable gelatin sponge (Gelfoam). The combined action of the platelets and the gelatin will control capillary hemorrhage and may be enhanced by the addition of thrombin.

Thrombin, used as a *topical agent*, directly clots fibrinogen to produce rapid hemostasis. Gelatin sponges moistened with thrombin provide very effective coagulation of hemorrhages from small veins and capillaries. In dental alveoli, particularly in mandibular posterior regions, gelatin sponges may absorb oral microorganisms and cause alveolar osteitis, a painful condition that will delay repair.

The vasoconstriction of bleeding beds may be produced by the use of epinephrine, either topically or in combination with local anesthetic solutions. The topical solution, 1:1.000, may produce blood pressure responses nearly equal to those resulting from an intramuscular injection. Minimal concentration should be used to avoid untoward reactions. In most instances, another modality, such as thrombin or regenerated oxidized cellulose, should be used in oral surgery to control capillary and venule hemorrhages. In some cases, the injection of 1.8 mL of local anesthetic solution containing 1:100.000 epinephrine will suffice to control annoying primary hemorrhages.

Cyanoacrylate adhesive monomers are under animal laboratory investigations for use as hemostatic agents following surgery in the oral cavity and these agents have had some controlled clinical applications. Polymerization of the monomer occurs extremely rapidly in the oral environment and bonds to oral tissues as well as to the coagulum. This produces a protective, hemostatic dressing for surgical sites. Controlled clinical investigations will probably produce approval and acceptance of this modality of hemostasis in oral surgery.

Agents given systemically are reported from time to time for use in otherwise normal individuals to control postoperative hemorrhage. Aside from the fact that these agents have effectiveness that ranges from none to varying, the manipulation of an entire biological system does not seem warranted to control the normal physiologic response of bleeding and coagulation.

Oral and Maxillofacial Arteries

The external carotid arteries are the major arterial supply to the oral and maxillofacial tissues. Pressure on these arteries should stem the flow from hemorrhaging peripheral arteries. Anastomosing arterial networks from the opposite side as well as the rich venous bed may make pressure to the external carotid arteries only partially effective in controlling hemorrhage. Following consultations and transfers, if appropriate, the external carotid artery is controlled as follows. The patient is placed in a supine position with the head rotated and the neck slightly extended towards the contralateral side. An incision is made through the skin and superficial fascia overlying the anterior slope of the sternocleidomastoid muscle and is centered over the hyoid bone. Variations in the direction of the incision through the skin are seen; however, the hyoid bone landmark is about the level of the common carotid artery

bifurcation and allows access to its branches. Blunt dissection will expose the carotid triangle and its contents, the blue jugular vein, yellow vagus nerve, and the red common carotid and external and internal carotid arteries. An umbilical tape is secured around the external carotid artery to assess the effect of occluding the artery. If indicated, permanent ties are placed and the artery is sectioned.

Temporal arterial hemorrhage is readily accessible to identification of bleeding points, hemostatic control, and ties. A pressure point exists over the root of the zygomatic process of the temporal bone that will give hemostatic control to control arterial hemorrhages.

In the event of a scalping injury of the cranium, the freely anastomosing subcutaneous arteries may produce a profound, aggressive hemorrhage. Treatment should include direct ligation or electrosurgical coagulation and pressure bandages.

The maxillary artery is a direct continuation of the external carotid artery. Injuries to this artery result in massive and, possibly, life-threatening hemorrhages. Control may be difficult if the injury to the artery is in a fairly deep wound, such as on the medial side of the condylar neck when approached by a preauricular incision. Tight packing of the wound and pressure and ties of the external carotid artery are indicated. In hemorrhages from the maxillary artery, blood replacement and prevention of shock become of paramount importance.

The masseteric artery, traversing the sigmoid notch form the maxillary artery to the masseteric muscle, will produce a copious hemorrhage if torn by either retractor or by cutting during procedures performed on either the medial side of the mandible, such as a midsagittal split of the ramus, or on the lateral side of the ramus, such as oblique osteotomies when approached either intraorally or extraorally. Because of the depths of and limited access to the surgical sites, pressure from gauzes soaked in hot saline maintained for 10-minute increments has been effective.

The facial artery has a pressure point at the premasseteric incisure, the mandibular notch, or the antigonial notch as it is variously termed. Digital pressure should stem distal hemorrhage flows. However, other arteries that contribute to the nourishment of the facial tissues - infraorbital, mental, transverse facial, and buccal - and anastomosing flows from the opposite side may make the application of pressure futile. In any case the accessibility of the facial tissues to inspection and identification of hemorrhaging points makes the usual treatment of choice the application of hemostats and subsequent ties and coagulation methods. Though dramatic, facial lacerations and hemorrhages are not in themselves usually life threatening. Blood from a through-and-through buccal laceration may pool in the airway and produce a truly emergent condition of airway embarrassment.

In the event of lingual hemorrhage that cannot be directly controlled, one may need to resort to approaching the carotid triangle to ligate either the external carotid or the lingual artery. The lingual artery is approachable, according to textbooks, through Lesser's triangle. Many oral surgeons, head and neck general surgeons, and emergency room general surgeons queried stated that none had ever exposed the lingual artery by way of this triangle. However, the approach is described as a curvilinear incision from the gonial angle to the mental region of the mandible, extending inferolaterally to overlay the hyoid bone. Exposure of the anterior

and posterior bellies of the digastric muscle and the hypoglossal nerve completes the triangle. Within the triangle the vertical fibers of the hypoglossus muscles are separated, revealing, at least in cadaver dissections, the lingual artery, which may then be clamped.

To repeat, positive control of the lingual artery may be achieved at its origin from the carotid artery. The exposure of the external carotid is as described above; the lingual artery is the second anterior branch above the bifurcation of the external and internal carotid arteries. Even though clamped, the peripheral hemorrhage may go unabated as a result of the rich venous blood supply and the anastomosing arteries from the opposite side. The best control of lingual hemorrhage, though difficult, is directly at the injured site, with due regard, of course, for the challenges to the airway of accumulated blood and from expanding hematomas.

The posterior superior alveolar artery may be bound by periosteum to the posterolateral wall of the maxillary tuberosity, and occasionally it may produce an osseous depression. In these instances, the artery cannot move away from a local anesthesia injection needle and, if lacerated, will produce a rapidly expanding hematoma. Treatment consists of ice bag pressure packs and reassurances to the patient. As a general rule, the planned surgical or other intraoral procedure should be deferred.

Oral and Maxillofacial Veins

The abundant veins of the head and neck, devoid of valves, provide a generous vascular bed for movement of the massive quantities of blood required by the brain and the oral and maxillofacial tissues. Although the arteries are often implicated in the untoward sequelae in facial and related hemorrhages, it is the veins that often are the more dangerous structures in terms of risk and more difficult in terms of control. Whereas one may ligate the external carotid artery to help control an arterial hemorrhage, a laceration of, say, the retromandibular vein may not be approachable for ligation *above* the injured site. In a like manner, massive basilar skull fractures may open venous sinuses and channels of hemorrhage that will rapidly lead to hypovolemic shock. Furthermore, the lack of valves may help provide direct communication for the passage of infections in a retrograde direction to the cavernous sinuses or other intracranial areas.

The pterygoid plexus of veins, with its rather far-reaching extensions, envelops the origins and the spaces between the medial pterygoid muscle and the anterior heads of the lateral pterygoid muscle. Local anesthetic injections to the inferior alveolar foramen may pass through and lacerate the delicate venule channels and permit a pooling of blood in the inverted pyramidal-shaped pterygomandibular space at the site of the foramen. Aspiration will pick up this pooled blood and give the impression of an intravascular placement of the needle tip.

Local anesthetic injections for the posterior superior alveolar nerve are frequently accompanied by a rapidly developing hematoma. As mentioned previously, this hematoma is probably associated with laceration of the posterior superior alveolar artery and not, as sometimes described, the pterygoid plexus of veins. Pressure gradients within the veins, especially the small veins, probably are not sufficient to produce an expending hematoma in the plexus area.

The sublingual, or the ranine, vein may be a direct contributor to the jugular vein or it may contribute first to the lingual vein. In either case, opening of this vein provides a direct, explosive outlet for the jugular veins. Suctioning, clamping, and pressure dressings may control this effusion that could follow frenoplasty for ankyloglossia or a laceration. A lingual artery hemorrhage possibly may be controlled with ligation of the external carotid artery, but one is not able to approach in a reasonable manner the jugular vein superior to the injured site.

The sizeable posterior facial vein, the retromandibular vein, is especially vulnerable to closed and semiclosed operations that pass cutting and stripping instruments posterior to the ramus of the mandible. Also, it is conceivable that hemorrhages following a midsagittal split of the ramus may be produced by sharp bony margins that could lacerate this vein. The first treatment would be directed toward packing the area with gauze dampened with hot saline and applying pressure.

Other Maxillofacial Vessels

The inferior alveolar neurovascular bundle contains the named vessels most likely to be damaged by oral surgical procedures. The inferior alveolar canal is an actual bony canal with perforations, in dentate individuals, for the passage of dental neurovascular structures. When a tooth is removed, these structures are severed and the bony canal, in time, closes over the perforations. Thus in an edentulous individual the canal becomes a smooth bony tube that traverses the mandible from the inferior alveolar foramen to the mental foramen. In some cases, the bony canal is not distinct in either the edentulous or dentulous individual. In these instances the clinical impression that one gains when performing body of the mandible ostectomy procedures is that there are multiple filaments and strands of neurovascular bundles passing through the body of the mandible.

The mandibular third molar area is where the inferior alveolar vessels are most likely to be damaged during surgery. The location of the inferior alveolar canal may be ascertained by shifting intraoral radiographs; in this case the shifts should be vertical rather than horizontal, which is usual. In general the neurovascular bundle will be located buccal to the apical area of the third molars. Obviously, if the inferior alveolar canal is in juxtaposition to the area of proposed surgery, the patient should be informed and consent obtained because of this special surgical hazard.

If encountered during surgery, the hemorrhage from the inferior alveolar vessels may be quite brisk. Though dismaying to the doctor and the team, this hemorrhage will not be life threatening if adequately treated. Direct treatment includes controlling the hemorrhage by clearing the excess blood by aspirating and then packing the site with oxidized cellulose and injecting the area and reinjecting the inferior alveolar foramen area with vasoconstrictors in local anesthetic solutions.

Following these procedures, a saline-dampened sponge should be placed over the area and held in place for at least 10 minutes. If on reinspection the hemorrhage should recur, one may repeat the entire procedure. Then, if on reinspection the hemorrhage should again recur, one may wish to pack a gelatin sponge containing thrombin over the oxidized cellulose, suture the overlying mucoperiosteum, place a 3-hour intraoral pack, and either give the patient

another appointment for continuation of the surgical procedure in 7 days or refer the patient to an oral surgeon. If on a subsequent appointment the hemorrhage should again be present or if there have been intermittent recurring bouts of hemorrhage, then one may be faced with a scalped vessel, a vessel that has been partially severed, and the remaining attachments of the vessel prevent its retraction and contraction. In this case, the area should be cut with a curet to ensure severance of the vessel and then treated as a new surgical site.

The buccal vessels may be encountered in reflection of flaps from the area of the mandibular second molars to the retromolar trigone. Although the sudden velocity of blood is somewhat surprising, the problem is quickly remedied by hemostats followed by ties, electrocautery, cryotherapy, or injection of vasoconstrictors in local anesthetic solutions.

The mental vessels emerging from the mental foramen are predictably located 10 to 12 mm above the base of the mandible. Thus in individuals with either normal or elongated alveolar processes, there is only a slight possibility of encountering these vessels during usual intraoral surgery. In the event that the neurovascular bundle is likely to be encountered, great care should be taken to prevent its severance because of the possibility of a paresthesia or anesthesia in the inferior lip. The hemorrhage problem is managed handily by pressure dressings to avoid clamping filaments of sensory nerves to the lips, by hemostat controls, or, if the vessel has been severed at the foramen, by packing gelatin sponges or oxidized cellulose into the foramen.

Hemorrhage may occur from the mylohyoid vessels in the mandibular first molar area back to the third molar area. The bleeding may follow flap elevation or fracture of the medial alveolar bone, especially in the second and third molar regions. Control by pressure or by hemostats is usually effective and must be maintained for at least 10 minutes. Since the vessels are bound to the bony canal, ties are nearly impossible to place, and electrosurgical cauterization may be necessary. If a destructive modality such as electrosurgery or cryotherapy is employed, it should be precise to avoid damage to the lingual nerve, which is located just medial to this area. Secondary hemorrhage of the mylohyoid vessels could produce a dissecting hematoma in the sublingual and submandibular spaces. Posterior extensions of hemorrhage in the lateral pharyngeal space may lead to a series of worsening problems. Therefore, accurate treatment of the primary hemorrhage is of fundamental importance.

The palatine vessels, occupying the canal made by opposing grooves in the maxillary and palatine bones, may produce troublesome hemorrhage during orthognathic surgery wherein osteotomies are performed to move the middle facial skeletal segments. Attention given to using sharp chisels, placing lines of cleavage, and being prepared with adequate light, suctioning, and clamping instruments will make hemorrhage from the descending pallatine arteries and veins an insignificant incident in the course of an orthognathic surgical procedure.

Hemorrhage may be encountered from the anterior or the greater palatine artery during the elevation of palatal mucoperiosteal flaps, and control may be difficult to obtain because of poor visibility. When compared to the visibility available with buccal and labial flaps, the lines of sight into the palatal vault, either direct or indirect through a mirror, are more difficult for the operating team, especially if uncontrolled blood is flooding the region, the rotated tissue is thick and dense, and the flap is rotated toward the doctor, not away as in the

case with flaps lateral to the dentoalveolar processes. Control of the anterior or greater palatine artery hemorrhage may be directed toward clamping, followed by either tying or coagulation by electrical techniques. For the clamping and subsequent procedures to be accomplished, the flap may need to be further elevated anteriorly and toward the midline to clearly uncover the offending vessels. If a definite bleeder cannot be isolated, then the doctor may resort to the use of pressure and deep sutures or stick ties.

The nasal palatine vessels seldom contribute to troublesome hemorrhage. The passage of arterial blood may be from either the palatal towards the nasal or vice versa or both. The low pressure gradients in these vessels, which are near the end of the maxillary artery, minimize complicating hemorrhage. If these vessels are severed in the raising of a palatal flap, a piece of gelatin sponge may be placed in the canal to minimize postoperative hematoma formation.

Hemorrhagic Lesions

There are two entities that constitute absolute contraindications to exodontics: arteriovenous or sinusoidal aneurysms and central hemangiomas. Removal of teeth in situations in which the root structures are involved with one of these lesions may produce death by one of several modalities. The patient may exsanguinate, develop shock, or aspirate the high velocity and volume of blood.

Identification of these lesions may be fairly simple. There may be a history of hemorrhage from the gingival cuffs around the teeth, loosening of teeth accompanied by hemorrhage, mobility of teeth, a palpable thrill over the lesion, or a bruit noted by stethoscopic auscultation, or endless blood, sometimes under pressure, may be aspirated from the area. Roots of teeth may be eroded, as noted on radiological examination, overlying tissues may have color and contour changes, and pain or paresthesia may be present. On the other hand, any or all of these findings may be absent.

Radiological interpretation is not reliable. There appear to be no consistent findings of radiodensities, margins, locations, trabeculations, or presence of calcific bodies. Obviously, if there are otherwise clinically healthy teeth in an area of radiolucency, then further investigation is indicated to diagnose the radiolucent lesion. Nonrestorable, diseased teeth may be situated adjacent to an area that is a potentially devastating central hemangioma or aneurysm. Therefore, one should consider aspirating central radiolucent lesions prior to performing invasive surgical procedures including simple exodontics.

In the event a tooth is removed and an otherwise undiagnosed central vascular lesion is exposed, the tooth may be replaced immediately in the alveolus as a stopper. The patient should be transferred to an inpatient facility without delay for evaluation and definitive care. When performing a biopsy of a central lesion of the mandible, one should consider aspiration before opening widely into an unknown area. If the bone is too thick to permit passage of a needle for aspiration, it may be thinned with a round bur and thus permit perforation with the aspirating needles.

Management of central aneurysms and hemangiomas includes surgical excision, irradiation, curettage, and embolization. If resection is performed, Moose pointed out that the

section removed may be replaced as an autogenous bone graft. Angiographic techniques have led to embolization capabilities that may precisely occlude the major feeding vessels to a hemorrhagic lesion.

Occasionally lakes of blood with large feeders are encountered in central areas in the maxillofacial skeleton. These may be managed by occlusive packing of a strip of 1-cm-wide gauze, perhaps following moistening with thrombin. These packs may be left in place for a week; on removal there usually is a bed of granulation tissue without the vascular challenge.

Hemorrhages from soft tissue hemangiomas may be anticipated in most instances. Hemangiomas have camouflaged themselves as ranulas and mucoceles, and therefore, bluish- and reddish-colored soft tissue lesions should be aspirated, as with the central lesions. However, palpation, resultant blanching, and then rapid refilling often constitute differential physical examination points for diagnosing isolated peripheral hemangiomas.

Treatment of soft tissue hemangiomas and aneurysms consists of cryotherapy, introduction of fibrosing solutions such as sodium morrhuate, and resection or combinations of these modalities. Multiple treatments with cryotherapy or by fibrosing with sodium morrhuate are usually necessary before the lesion is reduced enough in size to permit its excision.

Operations on bone underlying soft tissue hemangiomas may not pose a problem, although, obviously, precautions should be considered to include typing and cross-matching the patient's blood and having several units on standby.

Lymphangiomas usually do not constitute a hemorrhagic threat. These compressible, asymptomatic (except for their presence) lesions may be reduced in size or excised by surgical resection or by cryotherapy.

Clinical Management of Typical Secondary Hemorrhage

The treatment in a clinic of an extravascular secondary hemorrhage originating from an intraoral source would begin by separating the patient from the usual retinue of relatives and friends and calming the patient. The oral cavity is cleansed with suctioning and mouth rinses, the hemorrhaging source identified, regional anesthesia obtained, and treatment performed according to the etiological and anatomical factors.

Preoperative considerations in management of dentoalveolar secondary hemorrhage include patient cooperation and an adequate light. Patient cooperation may be enhanced with the use of a sedative or a minor tranquilizer. A headlight or fiberoptic-illuminated instruments should be considered. The area is cleansed by suctioning with a large tonsillar suction tip. The standard preanesthetic injection preparation of drying and painting with an antiseptic solution is followed by injection of regional anesthesia solution without vasoconstrictors, for example, 3% mepivacaine. While the local anesthetic is taking effect, a 10 by 10 cm gauze is placed int he oral cavity over the hemorrhaging site, and the patient is asked to close firmly on the gauze without talking or expectorating. Following the onset of local anesthesia, a radiograph is made of the area. Assuming the area is free of foreign bodies based on clinical and radiographic examinations, the mouth is irrigated with normal saline and the hemorrhaging

site cleared and cleansed with a small tip suction device. Inspection and palpation are used to determine if the alveolar bony walls are intact, including bone in the bifurcation and trifurcation areas. In the event there is a fracture of the dentoalveolar process, the bone should be removed, even if a soft tissue flap needs to be raised, and the site treated as though it were a new surgical site.

Although the hemorrhage may be diminished by the local anesthetic that does not contain a vasoconstrictor and by the pressure gauze, one may usually detect where the primary bleeding site was, either local mucoperiosteal gingival tissues, alveolar bony wall, or apical vessels.

Gingival and mucoperiosteal hemorrhaging sites may be treated by local anesthetic injections with vasoconstrictors, for example, lidocaine with 1:100.000 epinephrine and the placement of sutures under tension to provide long-term pressure over the bleeding site. In some instances, mosquito hemostats will be necessary to control the hemorrhage. The hemostat can serve its usual role in tying bleeders, it may be used to conduct an electrocoagulation current, or it may be left in place for an extended period of time, for example, 30 minutes, and then removed to ascertain if the crushing of the offending vessel by the hemostat was effective in occluding the vessel.

Alveolar bone surrounding the tooth socket has passages to accommodate the vessels that bring nutrients to the periodontal ligament as well as an opening in the apex for the passage of the dentoalveolar neurovascular bundle. The vessels in these osseous canals may or may not have sufficient lateral hydrodynamic pressures, as do vessels in soft tissues, to ensure their contractile state, and recurrent hemorrhages may occur as the vessels relax. Nutrient canals are in all areas of the alveolar process and may contain vessels that have the capability of producing troublesome primary or secondary hemorrhages in the same manner as the vessels that pass through lamina dura to the alveoli. Burnishing the bleeding bone or crushing the areas with hemostats are time-tested methods of controlling alveolar process hemorrhages. Bone wax may be pressed into the osseous bleeding sites; in situations in which the hemorrhage from the alveolar process bony areas is unusual aggressive, the socket may be packed with gelatin sponge that was moistened with thrombin. In every case, the patient will be required to hold a 10 x 10 cm gauze in the mouth for 2 hours postoperatively.

To summarize the management of the usual secondary hemorrhage from a dentoalveolar surgical site, the following steps should be considered:

1. Instruct the patient to place a large gauze sponge, linen cloth, or handkerchief (never absorbent cotton) over the hemorrhaging site, close firmly, and the come to the responsible doctor's clinic for evaluation and indicated treatment.

2. With only the doctor and nursing assistant in attendance, prepare and drape the patient as for oral surgery.

3. Cleanse the area with a tonsillar suction device and 10 x 10 cm gauzes.

4. Obtain an effective light; consider a head light or fiberoptic-illuminated retractors.

5. Prepare the oral cavity and administer a local anesthetic without a vasoconstrictor.

6. Obtain a radiograph of the area.

7. If indicated, administer a simple sedative (50 mg secobarbital (Seconal), intravenously) or a minor tranquilizer (10 mg diazepam (Valium) intravenously). (If the doctor has had training, experience, and current competence in general anesthesia or deep sedation modalities, additional intravenous or inhalation agents could be administered as indicated.)

8. If the general physical status indicates dehydration and a fasting state, begin an intravenous infusion of 5% dextrose in water.

9. Ascertain the source of the hemorrhage.

a. If present, the foreign body or fractured bone should be removed.

b. If from soft tissue, local anesthesia with vasoconstrictor injection, clamping, clamping and tying, electrocoagulation, or cryotherapy may be used; sutures under tension may be placed over the offending tissues.

c. If in bone, crushing of bone and small amounts of bone wax may be indicated.

d. If generalized from the alveolus, packing the socket with gelatin sponge gauze moistened with thrombin may be indicated.

10. Suture the mucoperiosteum, and then place a pressure gauze in the mouth with instructions to close on it for 2 hours.

Shock

In the past, shock was defined and identified almost solely with hypotension. While a decrease in arterial blood pressure usually accompanies the shocklike state, current thinking defines shock at the cellular level. A suitable definition would then be "inadequate blood flow to vital organs or failure of the cells of vital organs to utilize oxygen". Thus, strictly speaking, one can produce show with normal or increased blood pressure but with poor compensation with decreased flow to vital organs or with a generalized metabolic state whereby cell damage occurs and oxygen cannot be appropriately utilized.

Traditionally shock has been classified as hypovolemic, cardiogenic, septic, and neurogenic. Decreased volume and pump failure are still valid causes of shock but the latter two classifications can perhaps be better defined as peripheral pooling with decreased venous return and decreased cellular uptake resulting from such states as sepsis. Regardless of its pathogenesis, shock produces a cycle of events that, if not interrupted, will ultimately lead to uncompensated declining homeostasis and death.

Thus in this context the common denominator is either poor oxygen delivery or poor oxygen utilization at the cellular level. The net result is a shift from aerobic to anaerobic metabolism by the cells with the resultant production of lactic acid from pyruvate. This

assumes significant clinical importance as studies have shown that there is a direct relationship between mortality as a result of irreversible shock and serum lactate levels. The accumulation of the latter will be reflected in altered blood gases, usually indicating a pattern of primary metabolic acidosis with a compensatory respiratory alkalosis.

The above cycle of events not only leads to increased serum lactate levels but to a wide variety of cellular events that, if not treated early, lead to irreversible cellular changes. Poor energy utilization with attending membrane instability is the best known phenomenon. Electrolyte balance is dependent on cellular work and active transport. In shock, passive equilibration tends to occur with ensuing passing of sodium and water into cells and potassium into the serum. The attending electrolyte and water shifts further aggravate any pre-existing volume losses. Thus if vascular volume is depleted by blood loss or dehydration, fluid from the extracellular space shifts to the vascular compartment to compensate for this reduced volume; the actual depletion of the extracellular space is greater than the volume loss because of coincident passive movement of water intracellularly produced by the previously mentioned cellular shifts.

The shifts of fluid previously mentioned are the rationale for the use of crystaloid solutions in the primary therapy of shock, prior to blood replacement. Often a proportionately larger amount of fluid must be used to restore homeostasis than the amount lost. This is partially caused by intracellular shifts of fluid and partially caused by the fact that vascular volume must be replaced to maintain cardiac output but crystaloids rapidly equilibrate between the vascular and interstitial space. Since the vascular space is about 5% and the total extracellular space about 20% of the body weight, a four-fold increase over blood loss is often necessary to restore vascular volume if crystaloid is used. It is obvious that this is a temporizing measure, and lost blood should be replaced with whole blood or component blood therapy. This measure does assume great importance, however, when the volume deficit is caused by dehydration or loss of body fluid other than blood. Dextrose and water alone has no place in the treatment of shock since this fluid will equilibrate with the intracellular space and little will remain in the vascular space to maintain cardiac output.

Assessment

The recognition of shock and assessment of its severity depend largely on awareness of the compensatory mechanisms that act to maintain homeostasis. These can be noted as clinical signs and symptoms. Uncompensated shocklike states lead to death. The compensatory mechanisms with different types of shock are similar, but the oral surgeon will most often have to deal with hypovolemic shock. The following events occur in compensation for shock and are usually easily recognized:

1. Decreased cardiac output - A decrease in cardiac output is based first on decreased venous return in hypovolemic shock. If the condition persists, decreased cardiac nutrition occurs and primary pump failure may result.

2. Cool clammy extremities - Vasoconstriction to skin and other nonvital organs occurs with resultant shunting of blood to brain, heart, and kidneys. There is a preferential shifting of fluids from the skin first, the gastrointestinal system next, and the renal, brain, and heart

systems last. Thus cool clammy skin and adequate urine flow indicate early compensated shock.

3. Tachycardia and tachypnea - Both represent a compensatory increase in oxygen delivery to combat hypoxia. Both are early symptoms of shock. Tachypnea may be aggravated by the metabolic acidosis with a compensatory exercise of carbon dioxide. However, these symptoms may also be present with anxiety.

4. Arterial blood pressure - Blood pressure may be maintained in the supine position in early shock, but decompression occurs on standing. Postural hypotension is often the most sensitive sign of early hypovolemic shock and should be a routine portion of the early examination.

5. Central venous pressure - In hypovolemic shock the central venous pressure will be decreased because of poor venous return and a depleted vascular volume. If shock progresses to pump failure, the central venous pressure will rise. Thus an isolated reading is of less value than serial determinations to assess the status of the patient. A clinical estimate may be made of central venous pressure on the basis of evaluation of neck veins in the supine and sitting state.

6. Arterial blood gases - Modern therapy of shock dictates the use of arterial blood gas determinations. Serial gas studies may be necessary to monitor the effects of treatment. Basically the degree of hypoxia and the acid-base status of the patient are important. The oxygen partial pressure (pO_2) should not be allowed to drop beyond 60 if possible. Fair reserve exists with pO_2 levels of 60 to 90, but as a result of the shape of the hemoglobin dissociation curve, small changes in pO_2 after 60 lead to large changes in oxygen saturation and decompensation may occur with minor changes in clinical conditions. In addition, because of lactic acidosis, the pH may be low and the bicarbonate radical (HCO_3) and carbon dioxide partial pressure (pCO_2) decreased, representing compensated metabolic acidosis.

7. Renal function - In early shock, renal blood flow is maintained at the expense of skin and the gastrointestinal system. As further shock occurs renal blood flow decreases with a concomitant fall in glomerular filtration rate and urine output. Thus decreased urinary output (less than 20 mL per hour) indicates fairly advanced shock. In addition, if decreased renal perfusion is allowed to continue over a long enough period of time, ischemia and tubular necrosis may ensure. Therefore, at this stage, therapy should be prompt and vigorous.

8. Hematocrit - Changes in hematocrit will vary according to the cause of shock and the stage. In early blood loss no change occurs and a drop in hematocrit does not become manifest until transcapillary refill occurs from the interstitial space. This may go on for 12 to 24 hours after blood loss. On the other hand, if volume depletion is associated with dehydration, then the hematocrit will be proportionally high because there has been no loss of red cell mass. Serial hematocrit determinations are most useful in assessing the progress of shock and monitoring for continuing blood loss.

Principles of treatment

1. Assure oxygen exchange - Good oxygen exchange is mandatory, and ways to assure it may range from relief of obstruction to oxygen therapy to assisted ventilation by way of an endotracheal tube. A tracheotomy under emergency conditions is rarely indicated and, if necessary, should almost always be done under controlled conditions after endotracheal intubation.

2. Ensure homeostasis - Any ongoing bleeding should be controlled until definitive therapy can be rendered. This may be accomplished by pressure, tourniquets, or direct ligation of severed vessels.

3. Maintain homeostasis - One and sometimes two large indwelling catheters should be placed, one preferably central. Initial therapy should be directed toward replenishing the vascular volume and the depleted extracellular space. One to two liters of Ringer's lactate or equivalent solution may be helpful after the patient is typed and crossmatched for blood therapy. If the cause of shock is blood loss, further therapy should be either fresh whole blood or component blood therapy. The latter is preferable if five or more units are needed since citrate intoxication, clotting factor depletion, and platelet depletion are minimized. There are numerous regimens, but an accepted one is packed red cells and plasmanate supplemented by three units of fresh frozen plasma with every ten units and six platelet packs with every 20 units. The above regimen minimizes bleeding, transfusion reactions, and electrolyte imbalance. Volume should be restored on a one-to-one basis with appropriate attention paid to monitoring all systems. As mentioned previously there is no place for glucose and water and little indication for artificial plasma expanders such as dextran in modern hospital practice.

4. Position - The preferred patient position is with the legs elevated and the body supine. With the head down or body tilted, impingement of the abdomen contents on the diaphragm may occur and cause poor respiratory function. In addition, the patient should be kept warm and dry, but the use of excess cover should be discouraged.

5. Relief of symptoms - Early wound care and relief of pain and anxiety is important. Wounds should be dressed and sutured and fractures set as soon as possible. Assurance and appropriate use of pain medication is also desirable to prevent neurogenic shock. Only enough medication should be used to control pain without depressing respiration. It is probably wiser to use small doses of intravenous narcotic (morphine) than unpredictable larger doses of intramuscular medication.

6. Monitor - Several parameters should be noted at appropriate intervals to assess the state of the patient.

a. Vital signs - Blood pressure should remain stable and orthostatic changes disappear during treatment. Both tachypnea and tachycardia should diminish as well. Worsening of these conditions indicate ongoing or inadequately treated shock.

b. Renal flow - Urine flow should be greater than 20 mL per hour and preferably greater than 30 mL per hour. If renal flow is adequate and the urinary specific gravity greater than 1.020, then the patient is in early shock and still has some reserve available. Decreasing

urinary output may mean either inadequate volume replacement or early renal failure. This distinction is important and must be made on the basis of such parameters as urine sodium and specific gravity and other clinical and historical clues.

c. Arterial blood gases - The goal is maintenance of sufficient pO_2 (greater than 60) and normal or reasonable acid-base balance. Fluid replacement usually corrects the metabolic acidosis and rarely is bicarbonate therapy necessary.

d. Central venous pressure - A low central venous pressure may indicate decreased venous return and vascular depletion while elevated central venous pressure may indicate fluid overload or cardiac failure. Thus the response to therapy shown by serial measurements is of more value than an isolated value.

e. Hematocrit - As mentioned previously, in case of blood loss, changes in hematocrit are delayed until transcapillary refill occurs. Thus early hematocrit readings may be normal and not reflect volume depletion. Serial hematocrit measurements are valuable in estimating total original blood loss or monitoring ongoing loss. It is not necessary to restore the hematocrit to normal levels, but values should be kept above 30 to assure adequate oxygen exchange at the tissue level.

7. Drugs - The use of drugs in shock resulting from hypovolemia has a very limited role. Pain should be relieved by appropriate medication, antibiotics prescribed for contaminated wounds, and tetanus prophylaxis given. If signs of cardiogenic shock are present, the use of digitalis may be considered.

Controversy still exists regarding the role of steroids and vasopressors. While not conclusive, current thinking regarding steroids is that they have limited value in the absence of adrenal insufficiency or septic shock. If the former is present, prompt response to relatively small doses of intravenous steroids usually occurs. With the latter, massive doses are indicated and are considered adjunctive. Vasopressors are also of limited value in early compensated shock, since usually the body is already in maximum sympathetic tone. They may be useful if volume is restored and decompensation of peripheral vasculature or cardiac output occurs.

It is thus clear that the successful management of shock lies in recognizing and treating the state during the compensatory stage and prior to irreversible cellular dysfunction. This requires meticulous attention to detail in diagnosis and assessment and intelligent therapeutic intervention.

Conclusion

In medicine, gathering and organization of information is possible by taking the history and performing a physical examination. If the history is taken and physical examination performed, nearly every one of the conditions described in this chapter, many of which are awesome in their manifestations and consequences, will be prevented. The history and the physical examination, augmented by selected radiographic and laboratory studies, are basic.