Hemostasis in Endoscopic Sinus Surgery

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KEYWORDS
- Sinus • Skull base • Bleeding • Hemostasis • Surgical field • Endoscopic
- Hypotensive anesthesia • Total intravenous anesthesia

KEY LEARNING POINTS
At the end of this article, the reader will:
- Apply key perioperative preventive strategies to minimize bleeding during sinus surgery.
- Critically assess named vessels that may be at risk of injury during sinonasal surgery.
- Understand the rational application of local vasoconstrictors.
- Understand the effects of relevant hemodynamic parameters during general anesthesia on the surgical field.
- Apply a logical approach in the management of intraoperative bleeding.
- Apply a rational approach in the management of suspected orbital compartment syndrome caused by anterior ethmoid artery bleed.

INTRODUCTION

Endoscopic sinus surgery (ESS) is considered to be a moderate bleeding risk surgery. Bleeding is anticipated during sinonasal surgery when treating inflammatory and vascular disorders, and is due in part to the inherently rich blood supply derived from the external and internal carotid arteries in this region. Expected surgical bleeding is encountered from mucosa, bone and vascular tumors, such as juvenile angiofibroma and metastatic renal cell carcinoma.
Adequate hemostasis of the microvascular and macrovascular circulation is needed during endoscopic or open sinus surgery, performed under local or general anesthesia, to accomplish the surgical goals and avoid complications. A thorough risk assessment is required to prevent excessive bleeding. Correct assessment of the source of the bleeding, and a detailed knowledge of surgical vascular anatomy and of hemostatic techniques, is necessary to successfully manage intraoperative bleeding.

### Why is hemostasis important in sinonasal surgery?

- To improve intraoperative surgical field and visualization
  - Avoid injury (vascular, cerebrospinal fluid leak, orbital)
  - Allow completion of the surgical procedure
- Minimize bleeding associated comorbidities
  - Nausea, emesis and aspiration
  - Significant blood loss, hypoxia and blood transfusion
- Prevent the need for nasal packing and related complications
- Prevent postoperative complications and improve healing
  - Hematoma and bleeding
  - Adhesions and scarring

### Risk of mucosal bleeding

- Disorders
  - Chronic rhinosinusitis with nasal polyps, eosinophilic mucus chronic rhinosinusitis (EMCRS), allergic fungal rhinosinusitis (AFRS)
  - Rhinitis medicamentosa
  - Infection, subperiosteal abscess
  - Thyroid eye disease for example, Graves ophthalmopathy
  - Immunopathology; for example, Sarcoidosis, Wegener granulomatosis, Churg-Strauss disease
  - Vascular tumors (juvenile angiofibroma, metastatic renal cell carcinoma)
- Prior surgery, radiotherapy
- Patient
  - Morbid obesity, hypertension
  - Chronic alcohol, liver, kidney disease
  - Smoking
  - Coagulopathies (congenital or acquired)

### Risk of inadvertent vascular injury

- Incorrect diagnosis
  - Internal carotid artery aneurysm, vascular tumor
- Unfavorable vascular and sinonasal anatomy
  - Ethmoidal arteries (anterior, posterior, and sometimes middle)
  - Internal carotid artery
  - Onodi cell
  - Sphenoid sinus septations
- Previous sinonasal surgery
  - Bone dehiscence, scarring, altered or absent anatomic landmarks
- Surgical mistakes
Significant intraoperative bleeding can be minimized by preventive measures preoperatively, intraoperatively, and postoperatively. When bleeding is encountered, either intraoperatively or postoperatively, the surgeon has to be prepared to be able to manage this effectively.

**BLEEDING PREVENTION: PREOPERATIVE**

**Some preventive preoperative hemostasis strategies**

- Assessment and treatment of comorbid patient factors (see Tassler A, Kaye R: Preoperative assessment of risk factors, in this issue)
- Timely cessation of medications that increase the risk of bleeding (see McKean E: Quality control approach to anticoagulants and transfusion, in this issue)
- Minimize sinonasal inflammatory and vascular burden
  - Role of preoperative glucocorticosteroids (GCs)
  - Vascular tumors and preoperative embolization
- Thoroughly examine clinically relevant vascular anatomy on patient’s sinus computed tomography (CT) scans

For a comprehensive review on the assessment and management of patient comorbid risk factors and medications, please see articles elsewhere in this issue (see Tassler A, Kaye R: Preoperative assessment of risk factors, in this issue; and McKean E: Quality control approach to anticoagulants and transfusion, in this issue). It should be noted that herbal supplements, in particular the 4 Gs (ginseng, garlic, ginger, and ginkgo biloba), also affect platelet function and should be stopped 10 days (based on the half-life of platelets) before surgery. Saw palmetto and high doses of vitamin E and omega-3 can increase bleeding risk as well.

Rhinitis medicamentosa should be recognized preoperatively and the offending topical decongestant discontinued as soon as possible. To minimize rebound congestion patients should be started on topical saline and corticosteroids. Chronic topical exposure to sympathomimetics (eg, phenylephrine) or imidazolines (eg, oxymetazoline, xylometazoline) causes dysregulation of vascular tone, thus intraoperative topical vasoconstrictors are ineffective in controlling the microvascular circulation. In such situations, tranexamic acid (TXA) (1 mg during induction), which acts to reduce clot breakdown, can be used.

Adequate perioperative management of hypertension is imperative to achieve optimum intraoperative conditions. The risk of surgical bleeding is estimated to increase 1.5-fold with aspirin but for most procedures the severity of bleeding is not increased.
In this study, the mean estimated blood loss during ESS in patients on aspirin was slightly higher than in controls, especially when more sinuses were opened. It is generally possible to safely perform ESS in patients on aspirin by using other strategies to manage the surgical field.

Because of the risks associated with a bloody field during ESS and no known reversible agents for aspirin, it is advisable to cease aspirin 10 days earlier. Before stopping aspirin, the risk of doing so needs to be discussed with the patient’s cardiologist or primary care physician because 10% of acute cardiovascular events are preceded by aspirin withdrawal. In at-risk patients, the average time intervals from aspirin cessation to acute stroke and acute coronary syndrome are 14.3 and 8.5 days respectively.

For emergency reversal of the effect of aspirin, donor platelet infusion is effective. In otherwise healthy aspirin-treated individuals, desmopressin (desamino-d-arginine vasopressin [DDAVP]) has been shown to reduce bleeding time, and may be considered intraoperatively in selected situations. However, it is contraindicated in patients at cardiovascular risk and there are no data to support the efficacy of DDAVP in this group.

At present there are insufficient data to make strong recommendations on routine GC use in ESS to improve the surgical field. Despite the limitations of study design and control groups (patient selection, disorder, type of anesthesia, or vasoconstrictor use), there seems to be a trend toward a beneficial effect of preoperative GC in reducing the amount of blood loss during ESS for nasal polyps.

In patients with nasal polyps, a single preoperative dose of 1 mg/kg prednisolone compared with 5 days of therapy may be just as effective in reducing blood loss during ESS. However, a control group without any treatment was not used for comparison. In contrast, a recent randomized controlled study showed no significant hemostatic benefit of preoperative GC. It remains to be seen whether the addition of preoperative GC has a significant benefit in the presence of other hemostatic preventive strategies (eg, adequate patient positioning and general anesthesia conditions). Note also that many anesthetists administer a single dose of dexamethasone during induction for its beneficial effects on perioperative nausea and vomiting, but it may also have an independent positive effect on the surgical field.
Potential vascular hazards can be identified preoperatively by examining high-resolution sinus CT scans systematically. Note that the right and left sides may be asymmetrical. Breaching its boundaries during ESS not only risks serious complications and vascular injury but causes bleeding from surrounding tissues, orbital fat, and dura.

A suggested approach is described, starting off with coronal sections to examine the relationship of the cribriform plate to the frontal recess and ethmoid sinuses, including lateral lamella.

The lamina papyracea can be followed posteriorly into the sphenoid sinus and from the skull base to the level of the roof of the maxillary sinus to look for bone dehiscence in both axial and coronal planes.

Next, determine whether the ethmoid arteries are against the skull base or running within a pedicle suspended below the skull base. This anatomy is best identified on a coronal scan and confirmed with sagittal images. The anterior ethmoid artery (AEA) is more likely to be on a pedicle than the posterior ethmoid arteries. In some patients there is a middle ethmoid artery.

Follow the posterior ethmoid cells and examine their relationship with the sphenoid sinuses. If the posterior ethmoid pneumatization extends beyond the sphenoid sinus (demarcated inferiorly by the choanal roof), it is most likely an Onodi cell, hence closely related to the optic nerve, and in some cases the internal carotid artery (ICA).

The sphenoid sinus should be assessed for the extent of pneumatization, dehiscence, and configuration of the intersinus septum. If the septum is lateralized, it often attaches to bone overlying the ICA. Care needs to be taken when taking down the septum and avoid through-biting instruments and twisting maneuvers closer to its posterior attachment. Here also, the relationship of the pituitary gland and ICA within the sphenoid sinuses can be examined. Determine whether the course of the ICA is predictable or aberrant, and look for vascular malformations, aneurysms, bone dehiscence, and relationship to the posterior ethmoid sinus.
The shape or slope of the skull base can be appreciated on sagittal images. The skull base may be high within the sphenoid sinus, low toward ethmoid sinuses, and high again at the frontal recess and anterior ethmoid sinuses. The extent of low ethmoid skull base can be evaluated in comparison with the adjacent orbital roof height on coronal scans.

In addition, reconfirm that the radiological characteristics are in keeping with the clinical diagnosis. Presence of skull base dehiscence should prompt further assessment with MRI and or CT/MR angiogram to exclude the possibility of disorders such as a vascular tumor (angiofibroma), ICA aneurysm, or meningoencephalocele.

**BLEEDING PREVENTION: INTRAOPERATIVE**

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**Patient Position**

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<td>• Minimum of 10° head elevation to show a benefit in the surgical field</td>
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<td>• Achieve a balance between improved surgical field and cerebral perfusion pressure</td>
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Studies addressing this issue consistently show a reduced blood loss and better surgical field with an elevated head position. A reverse Trendelenburg position can achieve this by reducing venous pressure and mucosal blood flow. The surgical field improves during ESS with a 10° tilt, and the blood flow at the head of the inferior turbinate reduces by 38% with a 20° tilt. Cerebral perfusion and blood flow are preserved with up to 20° to 30° of head elevation. The level of tilt is often underestimated without objective measure. It is advisable to check with a clinometer (available on most handheld mobile devices) while the patient is positioned to appreciate the extent of tilt required to achieve 10° to 20°.

**Local Vasoconstrictors**

Local agents are widely used in ESS for mucosal vasoconstriction, reduced bleeding, and decongestion. Most surgeons use a combination of local injection and topical application of vasoconstrictors on pledgets within the nose. Although, for injection, usually varying concentrations of adrenaline mixed with a local anesthetic is most commonly used, the choice of topical vasoconstrictor varies considerably between surgeons worldwide.
Key factors in the efficacy of local vasoconstrictors are to allow time for its action, usually 15 minutes, with peak effect ~30 minutes, and an atraumatic technique. If applied soon after induction, it can work while the ESS setup is performed. In addition to the agent used, the sites of vasoconstrictor application should be considered, bearing in mind the hemostatic goals. It is reasonable to suggest that, where possible, vasoconstrictor application should target the major vascular supply to the sinonasal cavities.

Based on available data, if already using topical vasoconstrictors, additional injection of a vasoconstrictor may not provide further hemostatic benefit during routine ESS. However, injection of a long-acting local anesthetic may be considered for postoperative local pain control if not already diluting topical vasoconstrictors in a local anesthetic solution, such as ropivacaine. The septum and inferior turbinate may be injected if performing a septoplasty and turbinate reduction together with ESS.
Except for cocaine, these vasoconstrictors act directly on endogenous neural mediator adrenergic receptors to regulate the sinonasal vasculature. Cocaine acts indirectly, by preventing the reuptake of noradrenaline, thereby potentiating its vasoconstrictor effects. Cocaine is also a naturally occurring local anesthetic and acts by blocking sodium channels and preventing the propagation of nerve action potentials.

The arterial system is largely innervated by alpha1-adrenoceptors and the venous system by alpha2-adrenoceptors. Stimulation of alpha1 reduces flow into the capillary network, and of postsynaptic alpha2 constricts venous sinusoids and promotes decongestion. In the sinonasal vasculature, postsynaptic alpha2 receptors (mediate vasoconstriction) predominate over presynaptic alpha2 receptors (mediates vasodilation), hence a generalized constriction and decongestion is achieved.

When used as a single agent, adrenaline or cocaine theoretically also reduce mucosal blood flow and congestion, and have recently been suggested to achieve similar surgical field scores and blood loss to each other during ESS.\(^\text{16}\)

### Which topical vasoconstrictors achieve the best surgical field in ESS?

- **Vasoconstrictor options for topical (not injectable) application**
  - Adrenaline: nonselective α1, α2, and β-receptor agonist
  - Oxymetazoline: predominantly α1 receptor agonist
  - Phenylephrine: predominantly α1 receptor agonist
  - Cocaine: potentiates α1, α2, and β activity of endogenous catecholamines
  - Variations of Moffett’s solution (mix of cocaine, adrenaline and sodium bicarbonate solution)

- **Recommend rational use based on knowledge of pharmacologic vascular anatomy and side-effect profile (discussed later)**
  - Single agent
  - Judicious use

- **Amount delivered to mucosa may vary on the surgical pledget (cottonoid, neuropatties or gauze) used. The more absorbent the material, the less available to mucosa**

### What are some relevant vasoactive pharmacokinetic and safety parameters of adrenaline?

- These data are based on the pharmacokinetics of adrenaline administered via the subcutaneous (injected) route in mediating local vasoconstriction
  - Dosage for effective vasoconstriction: 1:50,000 to 1:200,000
  - Onset of action: 5 to 15 minutes
  - Peak effect: 30 minutes
  - Duration: 1 to 4 hours
  - Metabolized in liver, intravenous half-life 2 to 5 minutes, but prolonged when administered subcutaneously

- Adrenaline is deactivated by oxidizing agents, alkalis (including sodium bicarbonate), and halogens

- Beta2 (vasodilatory) effects may predominate on blood vessels after 6 hours of adrenaline stimulation

- For skull base surgery anticipated to extend beyond this time, an alpha1 agonist such as oxymetazoline may be preferred
Adrenaline may be favored rather than cocaine because of the toxic profile of the latter. No significant adverse cardiovascular effects have been described with topical adrenaline of up to 1:1000 in ESS. However, based on the principle of using the lowest concentration to achieve the desired effect, a more dilute formulation of 1:10,000, 1:50,000, 1:100,000, or 1:200,000 may also be effective. However, these studies examined surgical site blood flow and surgical field following adrenaline injection in head and neck and dermatologic surgery.

The reported toxicities for cocaine during sinonasal surgery are confounded by concomitant adrenaline use, thus advocating against mixing adrenaline with cocaine (variations of Moffett solution). Note that sodium bicarbonate, which is helpful for the pharmacologic activity of cocaine, renders adrenaline ineffective. Furthermore, adding adrenaline to cocaine solution makes absorption of cocaine highly unpredictable, without added pharmacologic benefit because cocaine in itself is a potent vasoconstrictor. Ultimately these agents are absorbed, resulting in systemic vasopressor responses, manifested by fluctuating heart rate and blood pressure, which adversely affect the surgical field (discussed later).

Phenylephrine and oxymetazoline are predominantly alpha1 agonists, with oxymetazoline also exerting partial alpha2 activity. Theoretically, oxymetazoline may provide better vasoconstriction and decongestion, but there is no substantive evidence. It is noteworthy that a systematic review of topical vasoconstrictors in nasal sinus surgery strongly discouraged the use of topical phenylephrine. This advice was based largely on phenylephrine-associated morbidity and mortality, which led the New York State Department of Health to issue guidelines for intraoperative phenylephrine use. In these cases, phenylephrine dosage was excessive; it was frequently not measured and was confounded by a long-acting β-blocker used to treat hypertension resulting in pulmonary edema. Oxymetazoline-associated toxicity is also reported, likely caused by its inadvertent excessive use attributed in part to squeezing the bottle in an inverted position while patient was supine which dispensed a much higher volume. The anesthetist should be made aware of the topical and injectable agents used. The key to avoiding morbidity and mortality due to phenylephrine or oxymetazoline induced hypertension is its diagnosis, because its treatment differs in that, non-specific β-blockers should be avoided. Serious complications with topical application of these two vasoconstrictors have been reported, predominantly in pediatric patients in nonsinus surgeries.

Taking into account the available evidence regarding their efficacy or toxicity profile, there are no solid grounds to advocate the use of one rather than another.

The available evidence lends support to the following suggestions with regard to either oxymetazoline or phenylephrine use:

- Only topical application (not for local injection/infiltration)
- Not to combine its use with other vasoconstrictors, hence single-agent use
- Phenylephrine dosage (assumes 100% bioavailability): Groudine et al.
  - In adults, initial dose should not exceed 0.5 mg
  - In children < 25 kg, should not exceed 20 μg/kg
- Oxymetazoline dosage:
  - No specific guidelines exist, use manufacturer’s instructions
- Exercise caution by using:
  - A measured amount
  - The least amount required to achieve hemostasis
General Anesthesia

It is generally accepted that hemodynamic parameters during general anesthesia have a significant effect on the surgical field during ESS. There are several studies in the area, with inherent confounding factors that preclude a meta-analysis and firm recommendations regarding specific anesthetic agents and techniques for obtaining the best surgical field. Hence, it is important to define the key questions relevant to sinonasal hemostasis. First, some current questions are addressed here.

Does Hypotensive Anesthesia Achieve the Best Surgical Field in Endoscopic Sinus Surgery?

Hypotensive anesthesia, also known as controlled hypotension, is defined as a reduction of the systolic blood pressure to 80 to 90 mm Hg, a reduction of mean arterial pressure (MAP) to 50 to 65 mm Hg, or a 30% reduction of baseline MAP. Hypotensive anesthesia is hypothesized to reduce organ blood flow and consequently improve surgical field and blood loss. To answer this question, and to rationally interpret the available data for ESS, an understanding of the fundamental physiologic parameters that affect end-organ blood flow is helpful.

Cardiac output = HR × Stroke volume

In addition, the volume of blood delivered to the sinonasal surgical field is influenced by locoregional vascular autoregulation, effect of topical vasoconstrictors, inflammatory disorders, and extent of upper airway resistance.

In most published ESS studies, a common factor associated with a good surgical field is hypotension without reflex tachycardia. This physiologic response can be appreciated by the following:

MAP = Heart rate × Stroke volume × Systemic vascular resistance

If vasodilators are used to reduce the MAP by primarily decreasing systemic vascular resistance (SVR), the surgical field does not improve significantly, owing to reflex tachycardia that maintains cardiac output. However, if HR and/or cardiac contractility (affects stroke volume) are reduced by centrally acting presynaptic alpha2 agonists (clonidine, dexmedetomidine), β-blockers (eg, esmolol), or magnesium sulfate, cardiac output is reduced and consequently results in good surgical field scores. This result is also evident from studies showing independent correlation between HR and surgical field.

Is Total Intravenous Anesthesia Necessary to Achieve the Best Surgical Field?

The term total intravenous anesthesia (TIVA) refers to maintenance anesthesia delivered solely intravenously, without inhalational agents such as sevoflurane. TIVA, using propofol alone or in combination with an opioid such as remifentanil, is generally advocated to achieve the desired conditions of hypotension without reflex tachycardia. Compared with inhalational maintenance anesthesia, TIVA causes less variability in MAP and HR, and thus reduces the need for additional antihypertensive or β-blocker medication during surgery.

However, these stable hemodynamic parameters can also be achieved with a combination of inhalational anesthesia and remifentanil infusion. Remifentanil is a short-acting synthetic opioid that acts specifically as a mu-receptor agonist. Among other physiologic effects, it independently reduces HR and MAP. It therefore follows that, if ideal physiologic parameters were obtained with means other than propofol TIVA, the impact on surgical field would not be significantly different. Thus if a
remifentanil infusion with inhalational sevoflurane maintenance anesthesia achieves a stable MAP and HR, then this would be ideal from both anesthetic and surgical viewpoints, because one of the challenges with TIVA (using propofol) is of monitoring adequate depth of anesthesia and its longer duration of action (see Cordoba Amorocho M, Fat I: Anesthetic techniques in endoscopic sinus and skull base surgery, in this issue).

It cannot be emphasized enough that good communication with the anesthetist throughout surgery is invaluable for achieving hemodynamic conditions that are ideal for ESS. Ultimately it rests with the anesthetist to use an effective technique that achieves a balance between the physiologic parameters ideal for the surgical field and the level of hypotension and bradycardia that can be safely tolerated by an individual patient.

Tranexamic Acid

**What is the current evidence for the hemostatic effect of TXA in ESS?**

- TXA prevents clot breakdown (antifibrinolytic)
- Systemic use preoperatively\(^\text{32,33}\)
  - Significantly improved visualization, reduced bleeding
- Some concern regarding systemic TXA use and risk of deep vein thrombosis
- Topical TXA (5%) in sinus surgery compared with no TXA\(^\text{34}\)
  - Significantly improved surgical field (in the first 30 minutes)
  - Overall less bleeding in the TXA group

TXA has been used during surgery to reduce blood loss and is the standard treatment used to reduce the rate of perioperative transfusion in cardiac surgery.\(^\text{35}\) Its efficacy has also been suggested in orthopedic and liver surgery.\(^\text{36,37}\) In ESS, a single systemic preoperative dose has shown significant benefit in surgical field and reduced blood loss. Because of some concern with the risk of deep vein thrombosis, topical TXA may be used with the desired hemostatic effect. TXA use in ESS may be considered, especially in diseases with high risk of bleeding (eg, nasal polyps) and where topical vasoconstrictors may not be effective (eg, rhinitis medicamentosa).

Surgical Technique

**What are some surgical techniques that minimize bleeding during ESS?**

- Prevent unwanted mucosal injury
  - Precise and strategic placement of endoscope and instruments
  - Use suction with atraumatic tip
  - Minimize direct suction of normal tissues
- Avoid unnecessary mucosal stripping
  - Use sharp, through-biting or cutting powered instruments
- Methodical surgical approach
  - Work quickly to debulk inflammatory tissue; for example, nasal polyps
  - Where possible work from inferior to superior to avoid blood tracking down the endoscope
- Avoid potential vascular injury
  - Recent patient CT scans should be available in the operating theatre
    - No scans, no surgery
Identifying the source of bleeding is key to obtaining adequate hemostasis. Bleeding from inadvertent vascular injury is often evident immediately because of the onset of sudden and brisk bleeding. Attempts should be made to obtain adequate visualization and to implement vascular control (see Lin G, Bleier B: Surgical management of severe epistaxis, in this issue; Alobaid A, Dehdashti AR: Hemostasis in skull base surgery, in this issue; Gardner PA, Snyderman CH, Fernandez-Miranda JC, et al: Management of major vascular injury during endoscopic endonasal skull base surgery, in this issue; and Valentine R, Padhye V, Wormald P-J: Simulation training for vascular emergencies in endoscopic sinus and skull base surgery, in this issue for more detail on visualization and control of sphenopalatine and internal carotid arteries). With persistent generalized ooze obscuring the visual field, saline irrigation can help identify specific bleeding sites for targeted control.

<table>
<thead>
<tr>
<th>MANAGEMENT OF INTRAOPERATIVE BLEEDING</th>
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<td>Identify the source of bleeding</td>
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<td>- Bone</td>
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<td>- Named vessel</td>
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<th>What are some strategies to control mucosal bleeding in ESS?</th>
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<td>- Communicate with the anesthetist</td>
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<td>- Check hemodynamic parameters</td>
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<td>- Topical vasoconstrictors</td>
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<td>- Cautery</td>
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Bleeding from diseased mucosa generally settles as this is removed, and working quickly to achieve this is helpful. During this time, maintaining good visualization is essential. Communicate with the anesthetist to achieve optimum hemodynamic parameters (MAP and HR). Bleeding from mucosal edges can be managed by topical vasoconstrictors and gentle pressure with pledgets. With nasal polyps, initial application of topical vasoconstrictors may not reach all areas, and reapplication is helpful as the polyps are debulked, exposing fresh mucosa. In addition, cauterization of specific bleeding points with monopolar suction diathermy or bipolar forceps is often sufficient. If using monopolar cautery, care should be taken to limit its use posteriorly to the sphenoid rostrum, and at the level areas below the level of the orbital floor (regions of the root of inferior turbinate, middle turbinate, SPA) to avoid inadvertent skull base or orbital injury.
With continued ooze, TXA can be used either topically (5% TXA) or systemically (1 g of TXA). The beneficial effect of TXA relies on intact patient platelet function and coagulation pathway. Topical hemostatic agents can also be used (for a thorough discussion, see Barham HP, Sacks R, Harvey RJ: Hemostatic materials and devices, in this issue). In unrecognized mild hemophilia A, von Willebrand disease, and congenital or acquired platelet dysfunction such as is caused by aspirin or uremia, DDAVP is helpful. DDAVP has an immediate effect, with 2-fold to 6-fold increase in plasma concentration of coagulation factor VIII, von Willebrand factor, and tissue plasminogen activator, and in platelet adhesiveness.

Another strategy to consider with persistent mucosal bleeding is to reduce blood flow by targeting the major blood supply, by either injecting vasoconstrictor or cauterizing the sphenopalatine artery and its branches, including the posterior septal artery.

### What are some options for continued mucosal bleeding?

- Hot saline irrigation
- TXA (if not already given)
- Topical hemostatic agents
- DDAVP
- Vasoconstrictor injection or cauterization of major feeding vessels
  - Posterior septal artery
  - Sphenopalatine artery

Does hot saline irrigation improve the surgical field in ESS?

- What temperature to use? Animal study of sinus mucosal histology after hot water exposure
  - Greater than 52°C, cell necrosis
  - From 48°C to 50°C, moderate mucosal change, no necrosis
  - At 46°C, slight mucosal change, no necrosis
  - From 40°C to 44°C, no significant mucosal change
- In ESS, hot saline irrigation at 49°C
  - Improvement in surgical field and blood loss for surgeries of greater than 2 hours’ duration

Hot water irrigation has been used to successfully treat posterior epistaxis and reduce intraoperative bleeding with adenoidectomy. It has been widely used in neurosurgery, skull base surgery, and more recently in sinus surgery. The precise mechanism of hemostasis is not known. Animal studies suggest that the physical characteristics of blood vessels exposed to hot water include vasodilation, followed by interstitial mucosal edema and compression of the blood vessel lumen, and this may reduce mucosal blood flow into the surgical field. The procoagulative activity of hot water, or saline, is also proposed but requires confirmation.

A comparison of the effectiveness of hot (49°C) versus room-temperature (18°C) saline irrigation in patients with polyp and nonpolyp chronic rhinosinusitis undergoing ESS showed that the most benefit with respect to visual field and blood loss occurred after 2 hours of surgery. It is also suggested that, for hemostasis, hot saline irrigation is as effective as topical TXA in ESS. Further studies are required in this area, taking into account the different conditions of sinus disease, anesthesia, and perioperative management.
In addition to the potential independent hemostatic effects, saline irrigation is invaluable in improving the surgical field by clearing the visual field and identifying precise bleeding points for targeted vascular control.

**What are Some Strategies to Control Bone Bleeding?**

Bleeding from bone is typically encountered when mucosa is stripped or while drilling. If bone bleeding continues despite topical application of vasoconstrictors, this is expected given the microvascular bone anatomy. The intraosseous vessels do not respond well to vasoconstriction and generally rely on clot formation or physical occlusion for hemostasis. The clotting process can be facilitated by topical application of a hemostatic material or TXA, and through physical occlusion by diamond burr or Surgifoam application followed by gentle pressure with a pledget.

**What are Some Strategies to Control Named Vessel Bleeding?**

Common named vessels and their potential sites for injury are summarised in Table 1. During middle or inferior turbinate surgery, exposure of the feeding vessels (branches of the sphenopalatine artery) is common, and can be controlled effectively with focal cautery. Similarly, while performing a sphenoidotomy, if encountered, the posterior septal artery can be cauterized at the sphenoid rostrum. This vessel is found at variable distances below the natural sphenoid ostium during inferior extension of the sphenoidotomy. In conditions requiring resection of the palatine bone posteriorly toward the posterior wall of maxilla (e.g., nasal polyps, inverting papilloma [endoscopic medial maxillectomy and variations thereof]) the descending palatine artery may be encountered, and, further posteriorly, the

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<td>Vidian and posterior pharyngeal artery (see Lin G, Bleier B: Surgical management of severe epistaxis, in this issue; and Snyderman C, Pant H: Endoscopic management of vascular sinonasal tumors including angiofibroma, in this issue)</td>
<td>Vidian neurectomy</td>
</tr>
<tr>
<td>Greater palatine artery</td>
<td>Extended antrostomy, Medial maxillectomy</td>
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<tr>
<td>Branch to the Little area at incisive foramen (Stensen canal)</td>
<td>Septoplasty, septal flap surgery</td>
</tr>
<tr>
<td>Anterior and posterior ethmoid artery (see Lin G, Bleier B: Surgical management of severe epistaxis, in this issue; and Shaftel SS, Chang S-H, Moe KS: Hemostasis in orbital surgery, in this issue)</td>
<td>Ethmoid sinus surgery</td>
</tr>
</tbody>
</table>
sphenopalatine artery. These vessels too can be effectively controlled with monopolar or bipolar cautery. Occasionally excessive bleeding occurs from the greater palatine artery branch coursing through the nasal septum floor, approximately 1 cm posterior to the nasal spine. Topical vasoconstrictors are generally not sufficient, hence requiring cauterization.

The incidence of AEA and ICA injury (reported between 0.1%–0.3%) during routine ESS is low. The ICA is most at risk during sphenoid sinus surgery or posterior ethmoid surgery, the latter most likely to be an unrecognized Onodi cell. The AEA is at risk during anterior ethmoid and frontal recess surgery. Surgeons should be prepared to proficiently and adequately manage these to minimize or prevent serious patient morbidity and mortality (see Lin G, Bleier B: Surgical management of severe epistaxis, in this issue; Shaftel SS, Chang S-H, Moe KS: Hemostasis in orbital surgery, in this issue; and Gardner PA, Snyderman CH, Fernandez-Miranda JC, et al: Management of major vascular injury during endoscopic endonasal skull base surgery, in this issue).

**MANAGEMENT OF POSTOPERATIVE BLEEDING COMPLICATIONS**

Only approximately 15% to 25% of postoperative hemorrhage occurs within 24 hours of ESS. A significant bleed can occur up to 6 weeks after ESS and the most common time frame is between 1 and 2 weeks after ESS. Patients should be advised to attend the nearest emergency department for acute management. A thorough assessment for site of bleeding should include a nasal endoscopy to evaluate the source of bleeding, which is most commonly from branches of the sphenopalatine artery (posterior septal artery, and artery to middle and inferior turbinates).

<table>
<thead>
<tr>
<th>What steps can be taken at the end of surgery to minimize the risk of postoperative bleeding complications?</th>
</tr>
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</table>
| • Expose persistent bleeding areas:  
  ○ Use saline irrigation to wash away blood clots  
  ○ Request a Valsalva maneuver by the anesthetist  
| • If operating in the vicinity, ensure that posterior septal artery (sphenoid rostrum), sphenopalatine artery, and vessels at the root of the middle and inferior turbinates are adequately controlled  
| • If performing a septoplasty, a drainage-hole on 1 mucosal surface suturing the mucosal flaps or temporary septal splints may help reduce the risk of septal hematoma  
| • Routine generalized nonabsorbable nasal packing is not necessary  
| • Focal or targeted application of a topical hemostatic agent may be considered  
| • In select situations, generalized application of a topical antihemostatic agent may be required |
Frequent eye monitoring is imperative. Decision for a canthotomy, cantholysis and return to the operating room should be based on clinical assessment and not delayed on the premise of getting further imaging or ophthalmology consultation. Best results for potential orbital compartment syndrome are obtained with treatment within 1 hour.

What are some steps and considerations in the management of post-ESS orbital hematoma (see Shaftel SS, Chang S-H, Moe KS: Hemostasis in orbital surgery, in this issue)?

AEA bleed into the orbit is a serious complication of sinus surgery. Some immediate management principles are highlighted in the following scenario (developed together with Shaftel SS, Chang S-H, and Moe KS).

Scenario

A patient is in recovery after bilateral ESS. Left proptosis and epistaxis are noted. Clinical suspicion is of left AEA injury and retraction into orbit.

Question 1: What are some first steps in the management?
Answer: Remove any intranasal packing.
Check the patient’s visual acuity and pupillary responses. Palpate the globe and orbit. If possible, measure intraocular pressure (IOP).
If readily available, immediately start medical management for increased IOP.
Question 2: When should canthotomy and cantholysis be performed? What are the pathophysiologic goals of canthotomy and cantholysis?
Answer: Canthotomy and cantholysis should always be performed without delay for imaging or consultation in the setting of suspected orbital compartment syndrome. The goal is to reduce the IOP by reducing the compression of the eye by a tight eyelid.
Question 3: Is there enough time to get an ophthalmology colleague to measure IOP?
Answer: Unless IOP can be measured without delay it is not advisable to delay treatment of potential orbital compartment syndrome. Irreversible damage can result in as little as 1 hour and a properly performed canthotomy and cantholysis usually heals well by secondary intention.
Question 4: It was possible to measure IOP within few minutes. Despite some proptosis, the IOP does not show an increase. Should cantholysis be performed?
Answer: There is no benefit of performing cantholysis in the absence of an increased IOP.
The epistaxis has settled. Repeat test shows that the IOP is now elevated. A canthotomy and inferior cantholysis is performed.
Question 5: Is there a risk of rebleeding of the AEA if the tamponade effect is reduced by canthotomy and cantholysis?
Answer: There is a theoretic risk of rebleeding, but the benefit of restoring ocular perfusion significantly outweighs this.
Question 6: Despite inferior cantholysis, the IOP continues to increase. How much time is available, and what should be done next?
Answer: The principal goal is to save vision. If IOP continues to increase, an upper cantholysis can be performed.
If still not adequately reduced, the patient should be returned to the operating theatre emergently. Attempts can then be made to perform orbital wall decompression and identification of active hemorrhages.
SUMMARY

The endoscopic approach to sinonasal surgery relies on hemostasis for visualization so that surgical goals can be accomplished without increasing the risk of serious complications or compromising patient outcomes. This article systematically addresses many factors that influence intraoperative and postoperative bleeding, and strategies of bleeding control. These are summarized and a suggested approach to the prevention and management of intraoperative bleeding is presented in Table 2 and common pitfalls are addressed in Table 3. Despite best efforts, if bleeding compromises endoscopic visualization, it is advisable to stop pursuing surgical goals, focus on bleeding control and return at a later date. Proper surgical planning and prevention of bleeding where possible afford the best results. Although major vascular injury such as AEA or ICA bleed is uncommon during ESS, the surgical team and institution should have protocols in place and be prepared to execute proficient management to achieve best patient outcomes.

Table 2
Summary and suggested approach to the prevention and management of intraoperative bleeding

<p>| I. Prevention |
| Make the correct diagnosis |
| Assess preoperative sinus imaging |
| Assess and manage: |
| Patient comorbid factors |
| Medications (off antiplatelet medication where possible) |
| No smoking 4 weeks before and after surgery |
| Preoperative embolization for vascular tumors |
| Intraoperative |
| Patient position: reverse Trendelenburg 10° to 20° |
| Maintain body temperature |
| Local topical vasoconstriction: single-agent use |
| Suggestion: 1:10,000 adrenaline in 0.75% ropivacaine (1 mL of 1:1000 adrenaline mixed with 9 mL of 0.75% ropivacaine, total 10 mL) or oxymetazoline; refer to dosage on the preparation |
| Maintain favorable intraoperative hemodynamics (HR and blood pressure) |
| Talk to anesthetist |
| TXA: in patients at high risk of mucosal bleeding |
| Nasal polyps, EMCRS, AFRS |
| Meticulous surgical technique |
| II. Management of intraoperative bleeding |
| Assess site of bleeding: mucosa, bone, named vessel |
| Mucosal bleeding |
| Communicate with the anesthetist, check hemodynamics (HR, blood pressure) |
| Hot saline irrigation (40°–49°C) to clear clots and identify bleeding site, also independent hemostatic effect |
| Reapply topical vasoconstrictor, inform anesthetist |
| Cautery to specific bleeding sites |
| Topical hemostatic agents |
| TXA (if not given already) |
| DDAVP |
| Cautery of posterior septal artery or sphenopalatine artery |
| To prevent postoperative bleeding |
| At the end of surgery, Valsalva maneuver to expose and control bleeding sites |
| Other measures may be used, such as absorbable nasal packing, Silastic splints; elevation of head of bed, and topical oxymetazoline spray |</p>
<table>
<thead>
<tr>
<th>Problem</th>
<th>Likely Explanations and Suggestions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhinitis medicamentosa</td>
<td>Topical vasoconstrictors may not work effectively. TXA can be of benefit intraoperatively.</td>
</tr>
<tr>
<td>Use of natural or herbal therapies</td>
<td>Many have antplatelet activity and should be stopped 10 d before surgery. Consider DDAVP intraoperatively.</td>
</tr>
<tr>
<td>Ineffective reverse Trendelenburg position</td>
<td>It might not be a 10°–20° tilt. Confirm with a clinometer.</td>
</tr>
</tbody>
</table>
| Ineffective topical vasoconstrictor effect | 1. Strategic and atraumatic placement of vasoconstrictors at sites of vascular supply.  
2. Allow time to work: peak vasoconstrictor effect noted ~ 30 min from application.  
3. If using adrenaline, do not dilute in alkaline solutions; it reduces its efficacy. |
| Using primary vasodilators to reduce MAP | This reduces SVR and causes reflex tachycardia, which increases rate of blood flow to sinuses and no significant change to surgical field. |
| Blood frequently tracking along the endoscope | Check the mucosa in the area where the endoscope is placed in the nose; this may be traumatized and requiring bleeding control. |
| Bone bleeding not settling with topical vasoconstrictors | Bone vessel hemostasis usually relies on clotting mechanisms. Suggest topical Gelfoam, Surgifoam, or TXA application to facilitate control. |
| Use of room-temperature or lukewarm saline irrigation | Hot irrigation rather than cold is shown to have hemostatic properties. |
| Completely covering eyes with tape or drapes during ESS | This precludes or hinders intraoperative:  
• Palpation of the ipsilateral globe intermittently to check for lateral lamina bulge or dehiscence.  
• Early detection of potential intraorbital hemorrhage by restricting proptosis, and causing early increase in IOP.  
Suggest keeping eyes within the surgical field protected with copious amounts of lubrication ± consider taping the lateral canthus. |
| Unrecognized Onodi cell | Surgeons may not appreciate that they are working in an area more superior and even lateral to the sphenoid sinus boundaries, hence placing the optic nerve and ICA at risk. This needs to be identified on CT scans pre-operatively. |
| Anterior ethmoid artery on a pedicle below skull base | If not recognised pre-operatively, puts this vessel at risk during ethmoidectomy and frontal recess surgery. If injured, it also risks an intraorbital bleed. |
| Overzealous superior and inferior extension of sphenoidotomy | Superior:  
• This risks injury to posterior ethmoid artery, and may cause a cerebrospinal fluid leak. Note that the dura dips to a varying extent at the junction of the roof of sphenoid (planum) and ethmoid bones, and a through-biter or rongeur can bite through this fold of dura against the skull base.  
• Suggestion: use a straight curette to gauge the level of skull base immediately behind the sphenoid opening.  
Inferior:  
• Injury to the posterior septal artery at the inferior limit of sphenoidotomy; this can also bleed postoperatively. |
### Post-Test Questions (Correct answers are in italics)

1. Risk factors for increased mucosal bleeding during nasal/sinus surgery include all of the following except:
   - a. Rhinitis medicamentosa
   - b. Graves disease
   - c. Morbid obesity
   - d. *Self-medication with St John’s wort*

2. What is the optimal positioning of the patient to decrease venous bleeding?
   - a. Trendelenburg position
   - b. *Between 10° and 20° of head elevation*
   - c. Maximal extension of the neck
   - d. Head elevation to 45°

3. What is the best strategy for the use of topical vasoconstrictors?
   - a. *Use a single agent*
   - b. Combine adrenaline and cocaine for maximum efficacy
   - c. Use an agent with selective alpha2 agonist activity
   - d. Combine adrenaline with sodium bicarbonate to potentiate its effects

4. Which of the following procedures are at risk for bleeding from the sphenopalatine artery?
   - a. Nasal septoplasty
   - b. *Sphenoidotomy*
   - c. Vidian neurectomy
   - d. *Medial maxillectomy*

5. Which of the following are effective in decreasing bleeding during ESS:
   - a. TIVA
   - b. Irrigation with saline greater than 52°C
   - c. *Topical administration of TXA*
   - d. Intravenous administration of adrenaline

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### SUPPLEMENTARY DATA

Supplementary PDF slides related to this article can be found online at [http://www.oto.theclinics.com/](http://www.oto.theclinics.com/).

### REFERENCES


