Perioperative Management of Oral Antithrombotics in Dentistry and Oral Surgery: Part 2

Benjamin J. Statman, DDS

Private Practice in Dental Anesthesiology, SmileMD

Part 1 of "Perioperative Management of Oral Antithrombotics in Dentistry and Oral Surgery" covered the physiological process of hemostasis and the pharmacology of both traditional and novel oral antiplatelets and anticoagulants. Part 2 of this review discusses various factors that are considered when developing a perioperative management plan for patients on oral antithrombotic therapy in consultation with dental professionals and managing physicians. Additionally included are how thrombotic and thromboembolic risks are assessed as well as how patient- and procedure-specific bleeding risks are evaluated. Special attention is given to the bleeding risks associated with procedures encountered when providing sedation and general anesthesia within the office-based dental environment.

Key Words: Antithrombotic; Antiplatelet; Warfarin; Direct-acting oral anticoagulants; Dentistry; Sedation; Anesthesia; Hemostasis; Nasal intubation.

PERIOPERATIVE MANAGEMENT OF PATIENTS TAKING ORAL ANTITHROMBOTICS

Management of oral antithrombotics (OATs) for patients undergoing dental procedures with sedation or general anesthesia requires understanding the factors used to weigh the risks and benefits of continuing or stopping OATs perioperatively and framing treatment modifications to fit with the proposed surgical and anesthetic procedures. Questions that must be addressed include the following: 1) whether OAT agents should be continued, "bridged" (ie, temporarily substituted), or held during the perioperative period; 2) how intraoperative and postoperative bleeding will be managed; and 3) when to resume medications if they have been stopped. These decisions must involve input from a multidisciplinary team consisting of surgeons and operating dentists, sedation or general anesthesia providers, and health care providers managing the patient's OAT regimen.

Received November 28, 2022; accepted for publication January 27, 2023.

Address correspondence to Benjamin J. Statman, DDS, 4486 Baintree Rd, University Heights, OH 44118; benjamin.statman@offorhealth.com.

Anesth Prog 70:37–48 2023 | DOI 10.2344/anpr-70-01-06

Assessment of Thrombotic and Thromboembolic Risk

Thrombotic Risk. The risk of thrombosis in patients has not been evaluated in prospective clinical trials, but observational and retrospective studies have been used to establish a general stratification of thrombotic risk (Table 1). Patients prescribed acetylsalicylic acid (aspirin; ASA) for primary prevention of atherosclerotic complications are at a relatively very low risk for thrombosis. For patients on oral antiplatelet (OAP) agents for secondary prevention of thrombosis, risk is stratified based on the condition (coronary artery disease, peripheral artery disease, cerebrovascular disease), intervention (coronary artery stent or stents, coronary artery bypass graft, angioplasty, medical management), time since intervention, disease course (acute versus stable), stent features (type, generation, diameter, length, bifurcation, in-stent restenosis), and additional comorbidities, such as diabetes mellitus or chronic kidney disease (Table 1).[1,2] In addition to a patient's baseline risk factors, the perioperative environment is also associated with elevation of thrombotic and ischemic risk in part due to surgical trauma, inflammation, sympathetic stimulation, and metabolic dysregulation.[3]

Thromboembolic Risk. The American College of Chest Physicians (ACCP) and the International Society on Thrombosis and Haemostasis (ISTH) have both established a 3-tiered system stratifying patients into high,

Table 1. Periprocedural Thrombotic Risk Stratification[1]

Acute coronary syndrome	*Stable coronary disease*	*Cerebrovascular disease*	*Peripheral artery disease*	*Time since intervention (months)*	*Thrombotic risk*
Medical treatment	PCI + BMS/DES/DEB or CABG*	Ischemic stroke; carotid stent placement	Acute peripheral vascular event + revascularization with DES; chronic occlusions	<3	High
				3–6	Moderate
				>6	Low
PCI + BMS/DES/DEB or CABG	PCI + BMS/DES/DEB or CABG with additional patient or stent risk factors			<6	High
				6–12	Moderate
				>12	Low
PCI + BMS/DES/DEB or CABG with additional patient or stent risk factors, or first-generation DES	PCI + first-generation DES			<12	High
				>12	Moderate

* BMS indicates bare metal stent; CABG, coronary artery bypass graft; DEB, drug-eluting balloon; DES, drug-eluting stent; PCI, percutaneous coronary intervention.

moderate, or low risk for perioperative thromboembolism (Table 2).[4–6] For patients on anticoagulation for atrial fibrillation, risk is based on their CHA_2DS_2VASc score (congestive heart failure, hypertension, age ≥75, diabetes, stroke, vascular disease, age 65–74, sex), history of rheumatic disease, and stroke/transient ischemic attack history. For those anticoagulated due to mechanical valves, risk is based on type of valve, valve position, risk factors for stroke, and history of atrial fibrillation. For patients with a history of venous thromboembolism (VTE), risk stratification is based on time since VTE, number of VTEs, and other prothrombotic conditions.

Assessment of Surgical Bleeding Risk

The likelihood as well as the consequences of surgical bleeding should be factored in when determining surgical bleeding risk. The ISTH stratifies procedures as "high, low, or minimal" risk based on the likelihood for major bleeding (≥2%, <2%, ~0%, respectively).[6] For this classification, major bleeding is defined as any bleeding that causes a drop in hemoglobin >2 g/dL, necessitates transfusion of 2+ units of red blood cells, occurs in a critical site (intracranial, intraspinal, airway[7]), requires surgical reintervention and prolonged stay, or any unexpected and prolonged bleeding.[8] Many professional societies have published risk classifications of common procedures; however, most of the data used to substantiate these classifications come from small observational studies or case series.[7] For this reason, many of the classifications are based on expert consensus. Surgical experience should inform and possibly be the primary factor in determining surgical bleeding risk.[9]

Assessment of Patient Bleeding Risk

The risk of bleeding from a dental or oral surgical procedure may be elevated by patient-specific comorbidities. The HAS-BLED score (Table 3) is validated for predicting higher bleeding risk in patients on oral anticoagulant (OAC) agents who are bridged with parenterally administered heparin[10] and is a useful screening tool to determine patients at increased risk of major bleeding. Additional patient history that may be pertinent to bleeding risk should also be gathered. This includes any events of prolonged or excessive bleeding within 3 months, bleeding with a similar procedure, or qualitative and quantitative platelet abnormalities.[5,9]

Table 2. Periprocedural Thromboembolic Risk Stratification[4,6,9]

Risk level	*Mechanical heart valve*	*Atrial fibrillation*	*Venous thromboembolism*
High	Mitral valve prosthesis, caged ball/tilting disc aortic prosthesis, stroke/TIA* <6 mo	$CHA_2DS_2VASc \geq 6$, stroke/TIA <3 mo, rheumatic valvular disease	VTE <3 mo severe thrombophilia (protein C, protein S, or antithrombin deficiency); antiphospholipid antibodies; multiple abnormalities
Moderate	Bileaflet aortic valve prosthesis with either atrial fibrillation, stroke/TIA history, hypertension, diabetes, heart failure, or age ≥75	CHA_2DS_2VASc of 4–6, stroke/TIA >3 mo	VTE 3–12 months; nonsevere thrombophilia (heterozygous Factor V Leiden, prothrombin gene mutation); recurrent VTE; active cancer
Low	Bileaflet aortic valve prosthesis without additional stroke risk factors	CHA_2DS_2VASc of 1–4 with no stroke/TIA history	VTE >12 mo with no other risk factors

* TIA indicates transient ischemic attack; CHA_2DS_2VASc, congestive heart failure, hypertension, age ≥75, diabetes, stroke, vascular disease, age 65–74, sex; VTE, venous thromboembolism.

General Scheme for OAT Management in the Perioperative Period

The general paradigm for managing OATs in the perioperative period should address the following questions: Should OATs be held prior to the procedure, and for how long? If OATs are to be discontinued, does the patient need to be bridged with parenteral agents? When can OATs be resumed postoperatively?

Antiplatelet Agents. Regarding the perioperative temporary discontinuation of antiplatelet agents, many guidelines do not address when the benefit of holding these agents outweighs the risk of thrombosis and instead rely upon clinician judgement. The American College of Cardiology/American Heart Association (ACC/AHA), for example, give guidance in their 2014 guidelines on how to manage discontinuation of dual antiplatelet therapy (DAPT) when the surgical procedure mandates it but do not define what surgical procedures would warrant DAPT discontinuation. The ACC/AHA instead recommend the risks and benefits should be weighed by a consensus of the surgeon and/or the anesthesia provider, the cardiologist, and the patient.[11] However, some general principles that are broadly agreed upon are worth highlighting, and the following recommendations may be considered should the need to interrupt anticoagulation therapy for major surgery be decided by the care team. Firstly, as evidenced in the POISE-2 trial, ASA when used for primary prevention was associated with an increase in major bleeding without any reduction in thrombotic complications in the perioperative period.[12] Therefore, it would be reasonable to hold ASA perioperatively should a mild concern for major perioperative bleeding even exist. However, this scale of hemorrhage, or major bleeding as defined by ISTH or similar, is quite low in a vast majority of dental surgeries compared with major abdominal, orthopedic, and other nondental surgeries. Secondly, the risk for bleeding is least with ASA, followed by the $P2Y_{12}$ antagonists, followed by DAPT. Thirdly, if a patient on a single $P2Y_{12}$ antagonist requires discontinuation perioperatively, recommendations include bridging with ASA until the $P2Y_{12}$ antagonist can be restarted. Finally, any elective procedure that would require DAPT discontinuation should be delayed until completion of the full DAPT course.[13]

Table 3. HAS-BLED Criteria*

	Characteristic	*Points*
H	Hypertension (uncontrolled)	1
A	Abnormal renal or hepatic function	1 or 2
S	Stroke	1
B	Bleeding tendency	1
L	Labile INR (if on VKA)	1
E	Elderly (age >65)	1
D	Drugs (ASA/NSAIDs) or excess alcohol	1 or 2

* Total score ≥3 indicates "high risk" for major bleeding. INR indicates International Normalized Ratio; VKA, vitamin K antagonist; ASA, acetylsalicylic acid; NSAIDs, nonsteroidal anti-inflammatory drugs.

There is limited clinical utility for bridging OAP agents with short-acting intravenous (IV) agents, such as IV cangrelor (Kengreal). This bridging therapy would only be considered for an urgent or emergent surgery with a high risk of major bleeding in a patient on ASA and a $P2Y_{12}$ antagonist within 1 month of stent placement.[13]

For most dental procedures performed in the office-based environment, OAP discontinuation is generally not recommended as per ACCP guidelines. However, in instances where more invasive or extensive dental or oral and maxillofacial surgery is planned that ideally requires OAP stoppage, the proposed timelines for when OAPs should be stopped and restarted are presented below (Table 4). The ranges presented allow for some discretion based on the degree of functional coagulation needed to minimize major bleeding.

Table 4. Duration of Withholding Oral Antithrombotics When the Bleeding Risk Warrants Discontinuation[8,13,97–99]

Drug	*Hold prior to procedure (days)*	*Restart postoperatively (hr)*
ASA*	3–7	24–72
Clopidogrel	2–5	24–72
Prasugrel	5–7	24–72
Ticagrelor	2–5	24–72
Vorapaxar	Consult required	Consult required
Dipyridamole	1–2	24
Cilostazol	1–5	24
Warfarin	3–5	12–24
Apixaban	1–2	24–48
Dabigatran	1–4	24–48
Rivaroxaban	1–2	24–48

* ASA indicates acetylsalicylic acid.

Warfarin. The decision to hold warfarin is based on overall bleeding risk, whereas the decision to bridge with parenteral agents is based on thromboembolic risk. Procedures with minimal risk of major bleeding do not warrant discontinuation, whereas those with greater bleeding risk (eg, orthognathic surgery) do.[5,9] Determination of the last warfarin dose is based on the most recent International Normalized Ratio (INR) as well as the target INR. Clinically, if the intent is to reduce the INR of a patient currently in a therapeutic range to a lower value, warfarin is withheld for 5 days prior to the procedure (Table 4),[4] and the INR is confirmed prior to the surgery to verify it is within the expected range.[9] There will be a time during this 5-day period when the INR is subtherapeutic (<2.0 or <2.5) but not fully normalized (>1.0). Patients historically were often bridged with a parenteral anticoagulant during this subtherapeutic window to minimize thromboembolic risk. However, newer data have shown that bridging during this period can triple the risk of excessive bleeding without reducing thromboembolic risk when compared with holding warfarin without bridging.[14,15] Therefore, bridging with heparin is only considered in cases with high risk of thromboembolism in major surgeries other than dental surgery.[5,9]

Warfarin can be resumed postoperatively almost immediately, unless an active, uncontrolled bleed exists or the patient has a high risk of postprocedural bleeding, since its anticoagulant effect will not begin for at least 24 to 72 hours and achieving a therapeutic INR requires 4 to 6 days.[5,16]

Management of Direct-Acting Oral Anticoagulants in the Perioperative Period

Due to the pharmacokinetic profiles of direct-acting oral anticoagulants (DOACs), such as dabigatran, apixaban, and rivaroxaban, there is a shorter preoperative withholding period, a longer interval necessary prior to resuming DOACs postoperatively, and limited utility for laboratory testing (Table 4). For procedures with minimal bleeding risk, like most dental procedures, no interruption of DOACs is usually necessary. However, major surgery with a potential for hemorrhage should be scheduled when the medication level is at a trough or low level, as determined by dosing schedules.[5]

The PAUSE multicenter prospective cohort study established a standardized strategy that has been advocated by many others, which requires no bridging or coagulation testing with low rates of major bleeding or thromboembolism.[8,9,15,17] For procedures where the bleeding risk is low but the surgical team still requires reduced anticoagulation, DOACs are held the day prior to surgery, approximating 3 serum concentration half-lives, and can be restarted the day after surgery according to this strategy. For procedures with a high bleeding risk, DOACs are held for 2 days prior to surgery, approximating 5 half-lives for most DOACs, and can be resumed 2 to 3 days after surgery. The only exception to this directive is for patients taking dabigatran with impaired creatinine clearance values (CrCl <50 mL/min). In this instance, preoperative holding intervals are usually doubled due to dabigatran's significant renal excretion (Table 4).

CONSIDERATIONS FOR DENTAL SEDATION AND GENERAL ANESTHESIA PROVIDERS

With the increasing number of patients on OATs undergoing dental procedures, providers of sedation and general anesthesia for dentistry who encounter these patients should be comfortable assessing the risks and benefits surrounding perioperative OAT management. They should also expect to participate in multidisciplinary discussions regarding OAT management. Additionally, providers should be cognizant of the bleeding risks associated with common dental procedures and surgeries (eg, multiple dental extractions, extensive soft tissue surgery), as well as those utilized during the provision of sedation or general anesthesia (eg, nasopharyngeal airway [NPA] placement, nasal intubation).

Dental Procedures

Many dental and medical professional societies have published recent guidelines regarding the continuance of OATs for routine dental procedures as bleeding should be minor and well controlled with local hemostatic

measures.[4,6,18–26] As with many broad-based recommendations, limitations and exceptions exist with most of these guidelines. These guidelines have historically been based on studies of ASA, clopidogrel, and vitamin K antagonists (VKAs), and it remains unclear whether the same guidance should be applied to newer OAPs and DOACs.[24,27–29] Secondly, existing guidelines are vague regarding specific dental procedures or are explicitly limited to procedures with bleeding risks similar to simple extractions of fewer than 3 teeth. It should be noted that although the evidence for the safety of continuing VKAs remains robust, evidence is accumulating that supports the notion that continuation of DOACs is at least as safe as the continuation of VKAs for extractions, implant placement, sinus augmentation, and bone grafting procedures.[30–35] Although associated with more bleeding than clopidogrel, the newer $P2Y_{12}$ antagonists prasugrel and ticagrelor similarly appear to be safe to continue even in combination with ASA for simple, uncomplicated dental extractions.[36,37]

Sedation and anesthesia providers are often involved in procedures with bleeding risks greater than simple extractions. For procedures that fall outside of the scope of current guidelines or for procedures or agents with limited data to guide perioperative management of OATs, the directives outlined above can be applied when assessing risk and therapy management. Dental procedures should be stratified into high, low, or minimal bleeding risk much like other surgical procedures to guide decision making for those involved in perioperative OAT management. The procedural bleeding risks presented below may be augmented by patient-specific bleeding risks, the surgeon's procedural experience, and the availability of and familiarity using local hemostatic measures.

Minimal Bleeding Risk Dental Procedures. Procedures with a bleeding risk similar to or lower than a simple extraction would be classified as having a minimal risk of major bleeding for which OATs should be continued. Procedures in this class would include local anesthetic administration, scaling and root planing, direct and indirect restorations (supragingival or subgingival), and most endodontic procedures.[5,38,39]

Low Bleeding Risk Dental Procedures. Procedures with a higher risk of surgical bleeding than simple extractions may fall into the category of low risk (<2% risk of major bleeding). Procedures in this category may include more complex extractions (surgical, >3 teeth, requiring osseous removal or recontouring), procedures that require soft tissue flap elevation, biopsy, or preprosthetic surgery.[5,38,39] There is evidence that procedures in this category can be safely performed in patients on OATs with local hemostatic measures. A retrospective study found that continuation of single antiplatelet therapy and DAPT for alveoloplasty, implant placement, excisional biopsy, and multiple extractions including removal of third molars was safe and presented minimal risk for uncontrolled or excessive bleeding.[40] Also, evidence exists that patients continuing ASA, $P2Y_{12}$ inhibitors, VKAs, or combinations of these medications can undergo periodontal surgery, including flap elevation and gingivectomy, utilizing local hemostatic measures such as oxidized cellulose (Gelfoam), digital pressure, and electrocautery when necessary.[41] However, some references have recommended the consideration of skipping a single DOAC dose due to the slightly increased risk of bleeding associated with some potentially invasive dental procedures.[38] Although the above referenced studies seem to suggest the possibility of performing many procedures without requiring OAP cessation, certain measures may be taken if the surgical team feels the risk for major bleeding is elevated. For example, if a patient is currently taking a $P2Y_{12}$ antagonist, it may be possible to bridge the patient with ASA perioperatively in consultation with the managing health care team.

High Bleeding Risk Dental Procedures. Orthognathic surgery, excision of large bony or soft tissue pathologies, extensive facial trauma repair via open techniques, and radical and reconstructive surgery for head and neck malignancies are classified by many as high risk for bleeding and would likely require pausing OACs and possibly OAPs (aside from ASA for patients with stents) following evaluation by the managing health care team.[5,42] These procedures are often performed in hospital or ambulatory surgical settings with ample pre- and postsurgical resources, and consultation with multiple specialty services is often necessary regarding hemostatic management.

Local Hemostatic Measures. Local measures to achieve hemostasis in dental procedures include applying pressure with gauze, suturing, electrocautery, oxidized cellulose (Surgicel), resorbable sponges (Gelfoam), cyanoacrylates, fibrin glue, and antifibrinolytics. Tranexamic acid (TXA) and epsilon aminocaproic acid (EACA) are antifibrinolytic lysine analogues that prevent activation of the coagulation cascade degradation factor plasminogen to plasmin. Under typical conditions, plasmin would break down fibrin, fibrinogen, and other clotting factors, but this process is impaired by TXA.[43] The recent POISE-3 study evaluated the use of IV TXA for patients undergoing noncardiac surgeries and found a decrease in bleeding events although noninferiority to placebo in causing adverse cardiac events was unproven.[44] For dental and oral procedures, topical solutions of either TXA or EACA can be irrigated into surgical sites, applied to gauze compressed on the surgical site, or utilized as a

mouth rinse for 2 to 7 days postoperatively. TXA has been shown to decrease incidence of postoperative bleeding for patients on VKAs and reduce delayed bleeding in patients taking novel OACs without increasing thromboembolic risk.[30,43,45–47]

Postoperative Pain Management

Management of pain following dental procedures and surgeries for patients on OATs centers on providing adequate pain control while minimizing excessive bleeding and thrombotic risk. Nonsteroidal anti-inflammatory drugs (NSAIDs), often central to postoperative pain management, function by inhibiting cyclooxygenase (COX). Yet their use is associated with both excessive bleeding and adverse cardiac events.[48,49] While nonselective NSAIDs reversibly block both major isoforms of COX (COX-1 and COX-2) to varying degrees, selective COX-2 inhibitors have minimal interaction with COX-1 leading to diminished bleeding risk with a variable risk of adverse cardiac events.[50]

The combination of OACs and nonselective NSAIDs is associated with a 3- to 6-times greater bleeding risk and a reported elevation in INR as high as 15%.[51,52] Celecoxib, a selective COX-2 inhibitor, has shown no increased risk of major bleeding when combined with warfarin versus warfarin alone. Although celecoxib has similar rates of reported major adverse cardiac events as ibuprofen, additional analysis is needed to understand its safety profile when combined with OACs.[53,54]

Like OACs, combining nonselective NSAIDs with OAPs can increase bleeding risk. Conversely, nonselective NSAIDs inhibit ASA's ability to bind to platelets and thereby increase thrombotic risk.[55,56] The US Food and Drug Administration has mandated a boxed warning on both nonselective and COX-2 selective NSAIDs that cautions their use in patients with cardiovascular disease due to elevated thrombotic risk.[57,58]

Due to the possible deleterious effects of NSAIDs, a multimodal approach seeking to minimize their use may be appropriate to minimize exacerbation of excessive bleeding and thrombotic risk in patients on OATs. Components of this multimodal approach can include administration of long-lasting local anesthetics, liposomal bupivacaine, acetaminophen, opioids, and other pharmacological adjuncts such as gabapentin or magnesium. If NSAIDs will be administered, the choice of agent, duration of use, and timing of dosing must be considered. Use of celecoxib up to 200 mg/day is preferable to ibuprofen or ketorolac when bleeding risk is high.[48] NSAIDs should be used for as short a duration as feasible to minimize both bleeding and thrombotic risk. Additionally, dosing regimens should be designed to minimize drug interactions. For example, ibuprofen and naproxen competitively inhibit ASA's effects on COX-1 and should be given 30 minutes after and 8 hours prior to the ingestion of immediate-release ASA to minimize antiplatelet attenuation.[59]

One final drug interaction that must be considered is in patients taking $P2Y_{12}$ antagonists who will be prescribed opioids postoperatively. The impairment of gut motility induced by opioids can lead to delayed enteric absorption and impaired oral bioavailability of all oral $P2Y_{12}$ inhibitors. Although the current evidence is of low quality, it may be prudent to alter timing and avoid opioid prescriptions in patients on oral $P2Y_{12}$ inhibitors.[60,61]

Anesthesia Procedures

Routes of Medication Administration. The 3 routes for medication administration that may be associated with bleeding are IV, intramuscular (IM), and intraosseous (IO). Both IO and peripheral IV access and even central venous access can be obtained without discontinuation of OATs.[62] Adequate pressure must be applied to access sites during removal to allow for adequate hemostasis, and careful monitoring of catheterization sites for hematoma and extravasation is prudent.

IM administration has been established as a safe method of delivering different vaccines and antibiotics in patients on OATs without hematoma development.[63–66] IM injections with small gauge needles are considered minimal risk for bleeding and do not require discontinuation of OATs.[67] Nevertheless, some recommend consultation with a physician familiar with the patient's bleeding risk for those taking OACs prior to IM administration of medications.[68,69] Techniques that minimize hematoma development with IM injections are the use of a 23-gauge or smaller caliber needle, holding firm pressure for at least 2 minutes over the injection site, and avoiding medications that may inhibit hemostasis such as ketorolac tromethamine.[69]

Laryngoscopy. There have been some case reports of spontaneous epiglottic hematoma development after laryngoscopy in patients on VKAs and induced by trauma in patients not on OACs.[70] These occurrences are quite rare, and laryngoscopy is considered a procedure with minimal bleeding risk that can be performed on patients continuing their OATs.[71,72] To minimize the risk of a major bleed, laryngoscopy should be performed carefully with consideration given to avoiding any tissue trauma.

Nasal Instrumentation. Instrumentation of the nasal passages of a patient on OATs with either an NPA or a nasotracheal tube (NTT) requires providers assess both the risk of major bleeding associated with nasal instrumentation as well as the necessity of nasal instrumentation.

Bleeding risk of nasal instrumentation. The frequency of epistaxis, its sequelae, and the ability to manage significant hemorrhage all factor into the procedural bleeding risk for nasal instrumentation. The rate of bleeding from nasotracheal intubation (NTI) varies based on technique used but ranges in the literature from 18% to 77%.[73–75] If bleeding is severe and if local measures unsuccessfully control bleeding, airway compromise and obstruction may rapidly ensue. For this reason, nasal instrumentation more invasive than flexible nasopharyngoscopy would be considered to have a high risk of major bleeding and would likely require pausing OACs and possibly OAPs (aside from ASA in patients with stents) in consultation with the managing physicians.[71,76–78] If the procedure can be safely completed, albeit with a slight inconvenience either without NTI or NPA placement or alternatively with oral intubation, then the benefits of nasal instrumentation generally do not outweigh the potential risks of discontinuing OATs.

Prevention of nasal bleeding. Even if the patient has discontinued their OATs or in patients who remain on ASA, any strategy that can minimize trauma to the nasal mucosa should be utilized, as hemostasis may not be completely normalized. There are numerous strategies to minimize the risk of bleeding during NTI. Selection of the more patent nasal passage is 1 way to minimize trauma during nasal instrumentation. Of patients reporting equally patent nasal passages, nasal endoscopy found greater than two-thirds of the study population with intranasal pathology significant enough to warrant instrumentation of the contralateral side of the nose to minimize trauma.[79] When nasal endoscopy is not feasible or available, use of the occlusion and spatula tests can be used with reasonable diagnostic accuracy.[80] The occlusion test is performed by having the patient select which nostril has better airflow while occluding the contralateral side. The spatula test is performed by measuring the diameter of condensation on a spatula or a dental mouth mirror held a centimeter from each nostril with the contralateral side occluded.

The pathway that the NTT takes can also impact bleeding risk. Guiding the NTT between the inferior turbinate and the floor of the nose as opposed to between the inferior and middle turbinates reduces mucosal trauma and bleeding. Using a fiberoptic scope to reliably access this pathway is ideal, but if a fiberoptic scope is unavailable, simple lifting of the nasal tip in a cephalad direction increases the likelihood that the NTT will traverse the inferior pathway.[81–83]

Other techniques that have been shown to reduce the risk of epistaxis during NTI include application of a topical vasoconstrictor (eg, cocaine, phenylephrine, oxymetazoline) to the nasal mucosa, lubricating the NTT, thermosoftening the NTT up to 60°C, tunneling the NTT tip into a catheter, NPA, or surgical glove, and obturating the NTT tip with an inflated esophageal stethoscope.[74,84–93] Consideration can also be given to utilizing uncuffed NTTs for certain procedures and patients to reduce the overall diameter and resistance to passage.

Conflicting reports exist regarding placement of an NPA to facilitate NTI by dilating nasal passages. Some authors have found that use of NPAs creates additional trauma,[94] whereas others have found that NPAs do not generate additional trauma and may minimize bleeding when used prior to introduction of the NTT.[95,96]

CONCLUSION

Perioperative management of patients taking antithrombotic agents requires a multidisciplinary approach involving the sedation or anesthesia provider, the surgeon or dentist, and the physician managing the antithrombotic regimen. The risks and benefits of continuing or pausing antithrombotic agents must be weighed, and pharmacologic principles should be used to guide agent disruption. Providers need to be familiar with the bleeding risks of frequently encountered procedures to provide optimal patient care. Guidelines and robust data exist on how to manage the more commonly used OATs for extractions. Additional research elucidating the bleeding risks associated with newer and less frequently used OATs as well as the bleeding risk of NTI and other dental procedures with weak or nonexistent evidence would help guide perioperative management of OATs for providers of sedation and general anesthesia for dentistry.

REFERENCES

1. Vivas D, Roldán I, Ferrandis R, et al. Perioperative and periprocedural management of antithrombotic therapy: consensus document of SEC, SEDAR, SEACV, SECTCV, AEC, SECPRE, SEPD, SEGO, SEHH, SETH, SEMERGEN, SEMFYC, SEMG, SEMICYUC, SEMI, SEMES, SEPAR, SENEC, SEO, SEPA, SERVEI, SECOT and AEU. *Rev Esp Cardiol (Engl Ed)*. 2018;71(7):553–564. doi:10.1016/j.rec.2018.01.029

2. Levine GN, Bates ER, Bittl JA, et al. 2016 ACC/AHA Guideline Focused Update on Duration of Dual Antiplatelet

Therapy in Patients With Coronary Artery Disease: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines: An Update of the 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention, 2011 ACCF/AHA Guideline for Coronary Artery Bypass Graft Surgery, 2012 ACC/AHA/ACP/AATS/PCNA/SCAI/STS Guideline for the Diagnosis and Management of Patients With Stable Ischemic Heart Disease, 2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction, 2014 AHA/ACC Guideline for the Management of Patients With Non-ST-Elevation Acute Coronary Syndromes, and 2014 ACC/AHA Guideline on Perioperative Cardiovascular Evaluation and Management of Patients Undergoing Noncardiac Surgery. *Circulation*. 2016;134(10):e123–155. doi:10.1161/cir.0000000000000404

3. Devereaux PJ, Sessler D, Lalu M. Myocardial injury after noncardiac surgery [La lésion myocardique après une chirurgie non cardiaque]. *Can J Anaesth*. 2022;69(5):561–567. doi:10.1007/s12630-022-02220-5

4. Douketis JD, Spyropoulos AC, Spencer FA, et al. Perioperative management of antithrombotic therapy: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines. *Chest*. 2012;141(2 suppl):e326S–e350S. doi:10.1378/chest.11-2298

5. Doherty JU, Gluckman TJ, Hucker WJ, et al. 2017 ACC Expert Consensus Decision Pathway for Periprocedural Management of Anticoagulation in Patients With Nonvalvular Atrial Fibrillation: A Report of the American College of Cardiology Clinical Expert Consensus Document Task Force. *J Am Coll Cardiol*. 2017;69(7):871–898. doi:10.1016/j.jacc.2016.11.024

6. Spyropoulos AC, Brohi K, Caprini J, et al. Scientific and Standardization Committee Communication: guidance document on the periprocedural management of patients on chronic oral anticoagulant therapy: recommendations for standardized reporting of procedural/surgical bleed risk and patient-specific thromboembolic risk. *J Thromb Haemost*. 2019;17(11):1966–1972. doi:10.1111/jth.14598

7. Tomaselli GF, Mahaffey KW, Cuker A, et al. 2020 ACC Expert Consensus Decision Pathway on Management of Bleeding in Patients on Oral Anticoagulants: A Report of the American College of Cardiology Solution Set Oversight Committee. *J Am Coll Cardiol*. 2020;76(5):594–622. doi:10.1016/j.jacc.2020.04.053

8. Douketis JD, Spyropoulos AC, Duncan J, et al. Perioperative management of patients with atrial fibrillation receiving a direct oral anticoagulant. *JAMA Intern Med*. 2019;179(11):1469–1478. doi:10.1001/jamainternmed.2019.2431

9. Hornor MA, Duane TM, Ehlers AP, et al. American College of Surgeons' Guidelines for the Perioperative Management of Antithrombotic Medication. *J Am Coll Surg*. 2018;227(5):521–536.e1. doi:10.1016/j.jamcollsurg.2018.08.183

10. Omran H, Bauersachs R, Rübenacker S, Goss F, Hammerstingl C. The HAS-BLED score predicts bleedings during bridging of chronic oral anticoagulation. Results from the national multicentre BNK Online bRiDging REgistRy (BORDER). *Thromb Haemost*. 2012;108(1):65–73. doi:10.1160/th11-12-0827

11. Fleisher LA, Fleischmann KE, Auerbach AD, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014;130(24):e278–333. doi:10.1161/cir.0000000000000106

12. Devereaux PJ, Mrkobrada M, Sessler DI, et al. Aspirin in patients undergoing noncardiac surgery. *N Engl J Med*. 2014;370(16):1494–1503. doi:10.1056/NEJMoa1401105

13. Godier A, Garrigue D, Lasne D, et al. Management of antiplatelet therapy for non elective invasive procedures of bleeding complications: proposals from the French working group on perioperative haemostasis (GIHP), in collaboration with the French Society of Anaesthesia and Intensive Care Medicine (SFAR). *Anaesth Crit Care Pain Med*. 2019;38(3):289–302. doi:10.1016/j.accpm.2018.10.004

14. Douketis JD, Spyropoulos AC, Kaatz S, et al. Perioperative bridging anticoagulation in patients with atrial fibrillation. *N Engl J Med*. 2015;373(9):823–833. doi:10.1056/NEJMoa1501035

15. Spyropoulos AC, Al-Badri A, Sherwood MW, Douketis JD. Periprocedural management of patients receiving a vitamin K antagonist or a direct oral anticoagulant requiring an elective procedure or surgery. *J Thromb Haemost*. 2016;14(5):875–885. doi:10.1111/jth.13305

16. Kuruvilla M, Gurk-Turner C. A review of warfarin dosing and monitoring. *Proc (Bayl Univ Med Cent)*. 2001;14(3):305–306. doi:10.1080/08998280.2001.11927781

17. Tafur A, Douketis J. Perioperative management of anticoagulant and antiplatelet therapy. *Heart*. 2018;104(17):1461–1467. doi:10.1136/heartjnl-2016-310581

18. Vlisides PE, Moore LE, Whalin MK, et al. Perioperative care of patients at high risk for stroke during or after noncardiac, non-neurological surgery: 2020 Guidelines From the Society for Neuroscience in Anesthesiology and Critical Care. *J Neurosurg Anesthesiol*. 2020;32(3):210–226. doi:10.1097/ana.0000000000000686

19. AAOM Clinical Practice Statement: Subject: Management of Patients on Warfarin Therapy. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2016;122(6):702–704. doi:10.1016/j.oooo.2016.06.015

20. Hirsh J, Fuster V, Ansell J, Halperin JL. American Heart Association/American College of Cardiology Foundation guide to warfarin therapy. *J Am Coll Cardiol*. 2003;41(9):1633–1652. doi:10.1016/s0735-1097(03)00416-9

21. Armstrong MJ, Gronseth G, Anderson DC, et al. Summary of evidence-based guideline: periprocedural management of antithrombotic medications in patients with ischemic cerebrovascular disease: report of the Guideline Development Subcommittee of the American Academy of Neurology. *Neurology*. 2013;80(22):2065–2069. doi:10.1212/WNL.0b013e318294b32d

22. Wahl MJ. The mythology of anticoagulation therapy interruption for dental surgery. *J Am Dent Assoc*. 2018;149(1):e1–e10. doi:10.1016/j.adaj.2017.09.054

23. Oral anticoagulant and antiplatelet medications and dental procedures. American Dental Association. Updated September 14, 2020. Accessed March 21, 2022. https://www.

ada.org/resources/research/science-and-research-institute/oral-health-topics/oral-anticoagulant-and-antiplatelet-medications-and-dental-procedures

24. Lewandowski B, Myszka A, Migut M, Czenczek-Lewandowska E, Brodowski R. Analysing the effectiveness of topical bleeding care following tooth extraction in patients receiving dual antiplatelet therapy-retrospective observational study. *BMC Oral Health*. 2021;21(1):31. doi:10.1186/s12903-021-01391-9

25. Wahl MJ. Dental surgery and antiplatelet agents: bleed or die. *Am J Med*. 2014;127(4):260–267. doi:10.1016/j.amjmed.2013.11.013

26. Grines CL, Bonow RO, Casey DE Jr, et al. Prevention of premature discontinuation of dual antiplatelet therapy in patients with coronary artery stents: a science advisory from the American Heart Association, American College of Cardiology, Society for Cardiovascular Angiography and Interventions, American College of Surgeons, and American Dental Association, with representation from the American College of Physicians. *J Am Dent Assoc*. 2007;138(5):652–655. doi:10.14219/jada.archive.2007.0237

27. Miller CS. A perspective on "The mythology of anticoagulation interruption for dental surgery". *J Am Dent Assoc*. 2018;149(1):3–6. doi:10.1016/j.adaj.2017.11.005

28. Chahine J, Khoudary MN, Nasr S. Anticoagulation use prior to common dental procedures: a systematic review. *Cardiol Res Pract*. 2019;2019:9308631. doi:10.1155/2019/9308631

29. Wahl MJ. Dental surgery in anticoagulated patients. *Arch Intern Med*. 1998;158(15):1610–1616. doi:10.1001/archinte.158.15.1610

30. Manfredini M, Poli PP, Creminelli L, Porro A, Maiorana C, Beretta M. Comparative risk of bleeding of anticoagulant therapy with vitamin K antagonists (VKAs) and with non-vitamin K antagonists in patients undergoing dental surgery. *J Clin Med*. 2021;10(23):5526. doi:10.3390/jcm10235526

31. Lababidi E, Breik O, Savage J, Engelbrecht H, Kumar R, Crossley CW. Assessing an oral surgery specific protocol for patients on direct oral anticoagulants: a retrospective controlled cohort study. *Int J Oral Maxillofac Surg*. 2018;47(7):940–946. doi:10.1016/j.ijom.2018.03.009

32. Zeevi I, Allon D, Rosenfeld E, et al. Four-year cross-sectional study of bleeding risk in dental patients on direct oral anticoagulants. *Quintessence Int*. 2017;48(6):503–509. doi:10.3290/j.qi.a38103

33. Yagyuu T, Kawakami M, Ueyama Y, et al. Risks of postextraction bleeding after receiving direct oral anticoagulants or warfarin: a retrospective cohort study. *BMJ Open*. 2017;7(8):e015952. doi:10.1136/bmjopen-2017-015952

34. Manfredi M, Dave B, Percudani D, et al. World workshop on oral medicine VII: Direct anticoagulant agents management for invasive oral procedures: a systematic review and meta-analysis. *Oral Dis*. 2019;25(suppl 1):157–173. doi:10.1111/odi.13086

35. Costantinides F, Rizzo R, Pascazio L, Maglione M. Managing patients taking novel oral anticoagulants (NOAs) in dentistry: a discussion paper on clinical implications. *BMC Oral Health*. 2016;16:5. doi:10.1186/s12903-016-0170-7

36. Doganay O, Atalay B, Karadag E, Aga U, Tugrul M. Bleeding frequency of patients taking ticagrelor, aspirin, clopidogrel, and dual antiplatelet therapy after tooth extraction and minor oral surgery. *J Am Dent Assoc*. 2018;149(2):132–138. doi:10.1016/j.adaj.2017.09.052

37. Dézsi BB, Koritsánszky L, Braunitzer G, Hangyási DB, Dézsi CA. Prasugrel versus Clopidogrel: a comparative examination of local bleeding after dental extraction in patients receiving dual antiplatelet therapy. *J Oral Maxillofac Surg*. 2015;73(10):1894–1900. doi:10.1016/j.joms.2015.06.158

38. Management of dental patients taking anticoagulants or antiplatelet drugs. Scottish Dental Clinical Effectiveness Programme. Updated March 2022. Accessed March 21, 2022. https://www.sdcep.org.uk/media/ypnl2cpz/sdcep-management-of-dental-patients-taking-anticoagulants-or-antiplatelet-drugs-2nd-edition.pdf

39. Kaplovitch E, Dounaevskaia V. Treatment in the dental practice of the patient receiving anticoagulation therapy. *J Am Dent Assoc*. 2019;150(7):602–608. doi:10.1016/j.adaj.2019.02.011

40. Napeñas JJ, Oost FC, DeGroot A, et al. Review of postoperative bleeding risk in dental patients on antiplatelet therapy. *Oral Surg Oral Med Oral Pathol Oral Radiol*. 2013;115(4):491–499. doi:10.1016/j.oooo.2012.11.001

41. Morimoto Y, Niwa H, Minematsu K. Hemostatic management for periodontal treatments in patients on oral antithrombotic therapy: a retrospective study. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2009;108(6):889–896. doi:10.1016/j.tripleo.2009.07.048

42. Capodanno D, Angiolillo DJ. Management of antiplatelet therapy in patients with coronary artery disease requiring cardiac and noncardiac surgery. *Circulation*. 2013;128(25):2785–2798. doi:10.1161/circulationaha.113.003675

43. Patatanian E, Fugate SE. Hemostatic mouthwashes in anticoagulated patients undergoing dental extraction. *Ann Pharmacother*. 2006;40(12):2205–2210. doi:10.1345/aph.1H295

44. Devereaux PJ, Marcucci M, Painter TW, et al. Tranexamic acid in patients undergoing noncardiac surgery. *N Engl J Med*. 2022;386(21):1986–1997. doi:10.1056/NEJMoa2201171

45. Sindet-Pedersen S, Ramström G, Bernvil S, Blombäck M. Hemostatic effect of tranexamic acid mouthwash in anticoagulant-treated patients undergoing oral surgery. *N Engl J Med*. 1989;320(13):840–843. doi:10.1056/nejm198903303201305

46. Ockerman A, Miclotte I, Vanhaverbeke M, et al. Tranexamic acid and bleeding in patients treated with non-vitamin K oral anticoagulants undergoing dental extraction: the EXTRACT-NOAC randomized clinical trial. *PLoS Med*. 2021;18(5):e1003601. doi:10.1371/journal.pmed.1003601

47. Souto JC, Oliver A, Zuazu-Jausoro I, Vives A, Fontcuberta J. Oral surgery in anticoagulated patients without reducing the dose of oral anticoagulant: a prospective randomized study. *J Oral Maxillofac Surg*. 1996;54(1):27–32; discussion 323. doi:10.1016/s0278-2391(96)90297-9

48. Solomon DH. NSAIDs: Adverse cardiovascular effects. Post TW, ed. UpToDate. Waltham, MA: UpToDate Inc. http://www.UpToDate.com. Accessed on November 23, 2022.

49. Mackenzie IS, Coughtrie MW, MacDonald TM, Wei L. Antiplatelet drug interactions. *J Intern Med*. 2010;268(6):516–529. doi:10.1111/j.1365-2796.2010.02299.x

50. Solomon DH. Selective cyclooxygenase 2 inhibitors and cardiovascular events. *Arthritis Rheum*. 2005;52(7):1968–1978. doi:10.1002/art.21132

51. Risser A, Donovan D, Heintzman J, Page T. NSAID prescribing precautions. *Am Fam Physician*. 2009;80(12):1371–1378.

52. Lamberts M, Lip GY, Hansen ML, et al. Relation of nonsteroidal anti-inflammatory drugs to serious bleeding and thromboembolism risk in patients with atrial fibrillation receiving antithrombotic therapy: a nationwide cohort study. *Ann Intern Med*. 2014;161(10):690–698. doi:10.7326/m13-1581

53. Kent AP. Navigating NSAID use in patients receiving oral anticoagulation: is there a safe course? *Thromb Haemost*. 2020;120(7):1001–1003. doi:10.1055/s-0040-1713098

54. Silverstein FE, Faich G, Goldstein JL, et al. Gastrointestinal toxicity with celecoxib vs nonsteroidal anti-inflammatory drugs for osteoarthritis and rheumatoid arthritis: the CLASS study: a randomized controlled trial. Celecoxib Long-term Arthritis Safety Study. *Jama*. 2000;284(10):1247–1255. doi:10.1001/jama.284.10.1247

55. Ouellet M, Riendeau D, Percival MD. A high level of cyclooxygenase-2 inhibitor selectivity is associated with a reduced interference of platelet cyclooxygenase-1 inactivation by aspirin. *Proc Natl Acad Sci U S A*. 2001;98(25):14583–14588. doi:10.1073/pnas.251543298

56. MacDonald TM, Wei L. Effect of ibuprofen on cardioprotective effect of aspirin. *Lancet*. 2003;361(9357):573–574. doi:10.1016/s0140-6736(03)12509-3

57. Cramer JD, Barnett ML, Anne S, et al. Nonopioid, multimodal analgesia as first-line therapy after otolaryngology operations: primer on nonsteroidal anti-inflammatory drugs (NSAIDs). *Otolaryngol Head Neck Surg*. 2021;164(4):712–719. doi:10.1177/0194599820947013

58. FDA Drug Safety Communication: FDA strengthens warning that non-aspirin nonsteroidal anti-inflammatory drugs (NSAIDs) can cause heart attacks or strokes. US Food and Drug Administration; 2015. Accessed June 8, 2022. https://www.fda.gov/drugs/drug-safety-and-availability/fda-drug-safety-communication-fda-strengthens-warning-non-aspirin-nonsteroidal-anti-inflammatory

59. Concomitant use of ibuprofen and aspirin: potential for attenuation of the anti-platelet effect of aspirin. US Food and Drug Administration. September 8, 2006. Accessed February 16, 2023. https://www.fda.gov/media/76636/download

60. Fernando H, Shaw JA, Myles PS, Peter K, Stub D. The opioid-P2Y(12) inhibitor interaction: Potential strategies to mitigate the interaction and consideration of alternative analgesic agents in myocardial infarction. *Pharmacol Ther*. 2021;217:107665. doi:10.1016/j.pharmthera.2020.107665

61. Tavenier AH, Hermanides RS, Ottervanger JP, et al. Impact of opioids on P2Y12 receptor inhibition in patients with ST-elevation myocardial infarction who are pre-treated with crushed ticagrelor: Opioids aNd crushed Ticagrelor In Myocardial infarction Evaluation (ON-TIME 3) trial. *Eur Heart J Cardiovasc Pharmacother*. 2022;8(1):4–12. doi:10.1093/ehjcvp/pvaa095

62. Roberts J. Procedures in the setting of anticoagulation. In: Roberts JR, Custalow CB, eds. *Roberts and Hedges' Clinical Procedures in Emergency Medicine and Acute Care*. Elsevier; 2017;1520–1528.

63. Fox E, Misko J, Rawlins M, Manning L. The risk of intramuscular haematoma is low following injection of benzathine penicillin G in patients receiving concomitant anticoagulant therapy. *J Thromb Thrombolysis*. 2020;50(1):237–238. doi:10.1007/s11239-019-02013-6

64. Casajuana J, Iglesias B, Fàbregas M, et al. Safety of intramuscular influenza vaccine in patients receiving oral anticoagulation therapy: a single blinded multi-centre randomized controlled clinical trial. *BMC Blood Disord*. 2008;8:1. doi:10.1186/1471-2326-8-1

65. Prevaccination checklist for COVID-19 vaccination. US Centers for Disease Control and Prevention. Accessed December 15, 2021. https://www.cdc.gov/vaccines/covid-19/downloads/pre-vaccination-screening-form.pdf

66. LaVallee J, Royer R, Smith G. Prevalence of bleeding complications following ultrasound-guided botulinum toxin injections in patients on anticoagulation or antiplatelet therapy. *PM R*. 2017;9(12):1217–1224. doi:10.1016/j.pmrj.2017.06.002

67. Beyer-Westendorf J, Gelbricht V, Förster K, et al. Peri-interventional management of novel oral anticoagulants in daily care: results from the prospective Dresden NOAC registry. *Eur Heart J*. 2014;35(28):1888–1896. doi:10.1093/eurheartj/eht557

68. Perrin G, Beller CL, Darnige L, et al. Intramuscular vaccination in adults with therapeutic anticoagulation in the era of COVID-19 vaccines outbreak: a practical review. *TH Open*. 2021;5(2):e166–e170. doi:10.1055/s-0041-1729627

69. General best practice guidelines for immunization: special situations. US Centers for Disease Control and Prevention. Updated March 15, 2022. Accessed March 22, 2022. https://www.cdc.gov/vaccines/hcp/acip-recs/general-recs/special-situations.html#bleeding

70. Hatzakorzian R, Shan WL, Backman SB. Epiglottic hematoma: a rare occurrence after tracheal intubation. *Can J Anaesth*. 2006;53(5):526–527. doi:10.1007/bf03022630

71. Hsueh WD, Hwang PH, Abuzeid WM. Perioperative management of antithrombotic therapy in common otolaryngologic surgical procedures: state of the art review. *Otolaryngol Head Neck Surg*. 2015;153(4):493–503. doi:10.1177/0194599815600409

72. Saba ES, Higgins MK, Heyes R, Lott DG. Is continued perioperative antithrombotic therapy safe when performing direct microlaryngoscopy? *Laryngoscope*. 2020;130(12):2853–2858. doi:10.1002/lary.28600

73. Watt S, Pickhardt D, Lerman J, Armstrong J, Creighton PR, Feldman L. Telescoping tracheal tubes into catheters minimizes epistaxis during nasotracheal intubation in children. *Anesthesiology*. 2007;106(2):238–242. doi:10.1097/00000542-200702000-00010

74. Elwood T, Stillions DM, Woo DW, Bradford HM, Ramamoorthy C. Nasotracheal intubation: a randomized trial of two methods. *Anesthesiology*. 2002;96(1):51–53. doi:10.1097/00000542-200201000-00014

75. Sanuki T, Kotani J. Thermosoftening of the Parker Flex-Tip(TM) tracheal tube in preparation for nasotracheal intubation. *Anesth Prog*. 2013;60(3):109–110. doi:10.2344/0003-3006-60.3.109

76. Hall CE, Shutt LE. Nasotracheal intubation for head and neck surgery. *Anaesthesia*. 2003;58(3):249–256. doi:10.1046/j.1365-2044.2003.03034.x

77. Thiele T, Kaftan H, Hosemann W, Greinacher A. Hemostatic management of patients undergoing ear-nose-throat surgery. *GMS Curr Top Otorhinolaryngol Head Neck Surg*. 2015;14:Doc07. doi:10.3205/cto000122

78. Peri-procedure management of anticoagulants. The University of Texas MD Anderson Cancer Center. Updated October 19, 2021. Accessed March 24, 2022. https://www.mdanderson.org/content/dam/mdanderson/documents/for-physicians/algorithms/clinical-management/clin-management-peri-procedure-anticoagulants-web-algorithm.pdf

79. Smith JE, Reid AP. Asymptomatic intranasal abnormalities influencing the choice of nostril for nasotracheal intubation. *Br J Anaesth*. 1999;83(6):882–886. doi:10.1093/bja/83.6.882

80. Thongrong C, Thaisiam P, Kasemsiri P. Validation of simple methods to select a suitable nostril for nasotracheal intubation. *Anesthesiol Res Pract*. 2018;2018:4910653. doi:10.1155/2018/4910653

81. Tong JL, Tung A. A randomized trial comparing the effect of fiberoptic selection and guidance versus random selection, blind insertion, and direct laryngoscopy, on the incidence and severity of epistaxis after nasotracheal intubation. *Anesth Analg*. 2018;127(2):485–489. doi:10.1213/ane.0000000000003396

82. Kim H, Lee JM, Lee J, et al. Influence of nasal tip lifting on the incidence of the tracheal tube pathway passing through the nostril during nasotracheal intubation: a randomized controlled trial. *Anesth Analg*. 2018;127(6):1421–1426. doi:10.1213/ane.0000000000003673

83. Kwon MA, Song J, Kim S, Ji SM, Bae J. Inspection of the nasopharynx prior to fiberoptic-guided nasotracheal intubation reduces the risk epistaxis. *J Clin Anesth*. 2016;32:7–11. doi:10.1016/j.jclinane.2015.12.016

84. O'Hanlon J, Harper KW. Epistaxis and nasotracheal intubation–prevention with vasoconstrictor spray. *Ir J Med Sci*. 1994;163(2):58–60. doi:10.1007/bf02943016

85. Katz RI, Hovagim AR, Finkelstein HS, Grinberg Y, Boccio RV, Poppers PJ. A comparison of cocaine, lidocaine with epinephrine, and oxymetazoline for prevention of epistaxis on nasotracheal intubation. *J Clin Anesth*. 1990;2(1):16–20. doi:10.1016/0952-8180(90)90043-3

86. Kim YC, Lee SH, Noh GJ, et al. Thermosoftening treatment of the nasotracheal tube before intubation can reduce epistaxis and nasal damage. *Anesth Analg*. 2000;91(3):698–701. doi:10.1097/00000539-200009000-00038

87. Takasugi Y, Futagawa K, Umeda T, Kazuhara K, Morishita S. Thermophysical properties of thermosoftening nasotracheal tubes. *Anesth Prog*. 2018;65(2):100–105. doi:10.2344/anpr-65-02-06

88. Seo KS, Kim JH, Yang SM, Kim HJ, Bahk JH, Yum KW. A new technique to reduce epistaxis and enhance navigability during nasotracheal intubation. *Anesth Analg*. 2007;105(5):1420–1424, table of contents. doi:10.1213/01.ane.0000281156.64133.bd

89. Kim EM, Chung MH, Lee MH, et al. Is tube thermosoftening helpful for videolaryngoscope-guided nasotracheal intubation? A randomized controlled trial. *Anesth Analg*. 2019;129(3):812–818. doi:10.1213/ane.0000000000003822

90. Tong JL. Xylometazoline pretreatment reduces nasotracheal intubation-related epistaxis in paediatric dental surgery. *Br J Anaesth*. 2011;106(2):279; author reply 279-280. doi:10.1093/bja/aeq395

91. Enk D, Palmes AM, Van Aken H, Westphal M. Nasotracheal intubation: a simple and effective technique to reduce nasopharyngeal trauma and tube contamination. *Anesth Analg*. 2002;95(5):1432–1436, table of contents. doi:10.1097/00000539-200211000-00061

92. Delgado AV, Sanders JC. A simple technique to reduce epistaxis and nasopharyngeal trauma during nasotracheal intubation in a child with factor IX deficiency having dental restoration. *Anesth Analg*. 2004;99(4):1056–1057. doi:10.1213/01.Ane.0000133914.26066.99

93. Mahajan R, Gupta R. Another method to avoid trauma during nasotracheal intubation. *Anesth Analg*. 2005;101(3):928–929. doi:10.1213/01.Ane.0000173679.77321.A8

94. Adamson DN, Theisen FC, Barrett KC. Effect of mechanical dilation on nasotracheal intubation. *J Oral Maxillofac Surg*. 1988;46(5):372–375. doi:10.1016/0278-2391(88)90220-0

95. Dhakate VR, Singam AP, Bharadwaj HS. Evaluation of nasopharyngeal airway to facilitate nasotracheal intubation. *Ann Maxillofac Surg*. 2020;10(1):57–60. doi:10.4103/ams.ams_190_19

96. Canpolat DG, Orbay Yasli S. Does a nasal airway facilitate nasotracheal intubation or not? A prospective, randomized, and controlled study. *J Oral Maxillofac Surg*. 2021;79(1):89.e1–89.e9. doi:10.1016/j.joms.2020.08.029

97. Anderson M. Peri-procedure management of antiplatelet therapy. The University of Texas MD Anderson Cancer Center. Updated 2021. Accessed June 19, 2022. https://www.mdanderson.org/content/dam/mdanderson/documents/for-physicians/algorithms/clinical-management/clin-management-antiplatelet-therapy-web-algorithm.pdf

98. Jourdi G, Godier A, Lordkipanidzé M, Marquis-Gravel G, Gaussem P. Antiplatelet therapy for atherothrombotic disease in 2022—From population to patient-centered approaches. *Front Cardiovasc Med*. 2022;9:805525. doi:10.3389/fcvm.2022.805525

99. Muluk V, Cohn SL, Whinney C. Perioperative medication management. Post TW, ed. UpToDate. Waltham, MA: UpToDate Inc. http://www.UpToDate.com. Accessed on June 5, 2022.

Continuing Education Questions

This continuing education (CE) program is designed for dentists who desire to advance their understanding of pain and anxiety control in clinical practice. After reading the designated article, the participant should be able to evaluate and utilize the information appropriately in providing patient care.

The American Dental Society of Anesthesiology (ADSA) is accredited by the American Dental Association and Academy of General Dentistry to sponsor CE for dentists and will award CE credit for each article completed. You must answer 3 of the 4 questions correctly to receive credit.

Submit your answers online at www.adsahome.org. Click on "On Demand CE."

CE questions must be completed within 3 months and prior to the next issue.

1) Clinical recommendations for direct-acting oral anticoagulants (DOACs), such as apixaban or dabigatran, include which of the following?

   a. Consider a longer DOAC withholding period preoperatively.
   b. Do not interrupt DOAC therapy if bleeding risk is anticipated to be minimal for dental surgery.
   c. Immediately restart DOAC therapy postoperatively.
   d. Utilize bridging therapy for most major and minor dental procedures.

2) For patients on warfarin and currently within therapeutic anticoagulation range, how long should warfarin be held preoperatively to reduce their International Normalized Ratio (INR) value?

   a. $\leq$1 day
   b. 2 days
   c. 4 days
   d. 5 days

3) Surgical extraction of more than 3 teeth falls into which bleeding risk category?

   a. Minimal
   b. Low
   c. Medium
   d. High

4) Which of the following local measures for controlling bleeding are dependent upon antifibrinolytic lysine analogues?

   a. 2-octyl cyanoacrylate
   b. Kaolin-impregnated gauze
   c. Oxidized cellulose
   d. Tranexamic acid