

Postoperative Epistaxis Following Dental Treatment With Nitrous Oxide/Oxygen Sedation

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A 12-year-old Caucasian male undergoing a dental extraction for a grossly carious mandibular molar under inhalational sedation with nitrous oxide/oxygen experienced an episode of anterior epistaxis postoperatively that was controlled well with local measures. Epistaxis following inhalational sedation with nitrous oxide/oxygen in the dental setting is a very rare complication but has been previously reported in the literature. This case report provides a review of the existing literature regarding cases of epistaxis associated with inhalational sedation using nitrous oxide/oxygen and discusses the potential etiology of epistaxis associated with inhalational sedation. Patients at higher risk of epistaxis should be properly informed of the risks prior to inhalational sedation with nitrous oxide/oxygen, and dentists should also be familiar with epistaxis management in the dental setting.

Key Words: Epistaxis; Inhalational sedation; Nitrous oxide; Dental extraction, Case report.

Epistaxis is fairly common in adults and children as the blood vessels supplying the nasal mucosa have little anatomic support or inherent protection. The most common causes of epistaxis in children include nasal mucosal dryness, trauma, foreign body, and rhinitis. Other important potential causes that are less common include systemic conditions such as bleeding or inflammatory disorders, medications that impact hemostasis (eg, NSAIDs, anticoagulants) and tumors.¹ Congestion of nasal blood vessels or irritation and drying of the nasal mucosa are also thought to increase the risk of epistaxis.¹

Although a variety of classification schemes exist, epistaxis is often classified anatomically based on the site of bleeding: anterior or posterior epistaxis. Anterior epistaxis is most common, accounting for more than 95% of cases,² and is normally caused by bleeding from Kiesselbach plexus. It is commonly controlled mainly by local measures like compression of the nasal soft tissues or application of hemostatic dressings. Posterior epistaxis is much rarer, accounting for ~5% of all epistaxis cases,³ and it may be associated with bleeding from

Woodruff plexus. It is more commonly associated with increased age and medical comorbidities such as hypertension, thrombocytopenia, atherosclerosis, and clotting disorders. Treatment of posterior epistaxis is also more likely to require medical care and hospitalization.⁴ Complications of severe epistaxis may include hypovolemia and anemia, and in posterior epistaxis, there is an increased risk of airway difficulty, aspiration, and death.⁵

Epistaxis may occur during the provision of sedation or general anesthesia for dental care as a result of nasotracheal intubation,⁶ insertion of a nasopharyngeal airway,⁷ use of a nasal cannula,⁸ or as a complication from intranasal drug administration. This case report describes postoperative epistaxis attributed to the use of inhalational sedation with nitrous oxide/oxygen and discusses the likely etiology and potential precautions.

CASE PRESENTATION

A healthy, American Society of Anesthesiologists (ASA) I 12-year-old Caucasian male (height, 62 inches; weight, 53.1 kg; BMI, 21 kg/m²) was seen at Midlands Partnership University NHS Foundation Trust for the planned extraction of a symptomatic grossly carious mandibular right first molar under inhalational sedation with nitrous oxide/oxygen, following 2 separate unsuccessful attempts with only local anesthesia 1 week prior. He had no known allergies and was not taking any

Received August 2, 2022; accepted for publication January 16, 2023.

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Anesth Prog 70:75–79 2023 | DOI 10.2344/anpr-70-01-04
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medications. The patient had no previous history of sedation or general anesthesia and had difficulty accessing dental care due to the COVID-19 pandemic.

General anesthesia was offered but not preferred by the parent, and consent was confirmed with the patient's mother (who was also a health care professional) to arrange a future appointment 1 week later to attempt extraction under inhalational sedation with nitrous oxide/oxygen. The risks and benefits of dental extraction and inhalational sedation with nitrous oxide/oxygen, which included nausea, vomiting, headaches, and lethargy, were reviewed with the patient and his mother. Preoperative instructions given at that time included restriction to a light nonfatty meal 2 hours prior to the procedure and to contact our clinic if there were any significant changes to his medical history that may affect the provision of inhalational sedation with nitrous oxide/oxygen.

On the day of treatment, the patient presented to the dental clinic where all preoperative checks for inhalational sedation were completed, including obtaining a full medical and pharmacological history to confirm no changes since the last appointment, confirming the time of last oral intake (which was 2 hours prior to the sedation appointment), and ensuring nasal patency. As there were no contraindications for inhalational sedation with nitrous oxide/oxygen noted, we proceeded with the planned dental treatment using our standard technique for inhalational sedation. To start, 100% oxygen at 6 L/min was administered for 1 minute after fitting the nasal hood. The initial flow rate of 6 L/min was determined after assessing the patient's respiratory rate and breathing depth and observing the reservoir bag. Following this, nitrous oxide was concurrently administered in 10% increments up to 20% after which 5% increments were given (ie, 10%, 20%, 25%, 30%, 35%...) until the patient was considered suitably sedated. The patient was given up to 50/50% nitrous oxide/oxygen by titration but did not feel relaxed, so additional nitrous oxide was given. At this point (60/40% nitrous oxide/oxygen), the patient appeared much more relaxed; the patient was giggling and at one point started joking with his mother. However, in retrospect this may have been a sign of oversedation or disinhibition.

The patient permitted administration of local anesthesia consisting of a right lingual and inferior alveolar nerve block with 2.2 mL of 2% lidocaine 1:80,000 epinephrine (44 mg lidocaine with 27.5 µg epinephrine) that produced anesthesia of the tongue and lip. However, the patient was still reporting pain, and therefore additional local anesthesia was given via intrapulpal, intraligamentary, and buccal infiltration injections. A total of 4.4 mL of 4% articaine with

1:100,000 epinephrine was administered: 2.2 mL for the intraligamentary (88 mg articaine with 22 µg epinephrine), 1.1 mL for the intrapulpal (44 mg articaine with 11 µg epinephrine), and 1.1 mL for the buccal infiltration (44 mg articaine with 11 µg epinephrine) injections. The local anesthetic was given ~15 minutes to take effect from the first injection.

Despite this, the patient did not permit the extraction due to the sensation of pressure, and the procedure was subsequently abandoned. The patient was given 5 minutes of 100% oxygen to prevent diffusion hypoxia. Postoperative Eve test (patient instructed to extend their arm and touch the tip of their nose while having their eyes closed) and Romberg checks were satisfactory, and the patient was deemed suitable for discharge. The patient's total treatment time under sedation lasted over 60 minutes (including the 5 minutes of oxygen to prevent diffusion hypoxia).

The patient was then referred to Royal Stoke University Hospital for the planned tooth extraction under general anesthesia. To avoid further delay in treatment, a request for a panoramic radiograph for the preanesthetic and surgical assessment and orthodontic treatment planning was made ~3 to 5 minutes after the patient was deemed fit for discharge. However, the patient reported spontaneous bleeding from the right naris while the panoramic radiograph was being taken. The patient denied any inadvertent trauma to his nose (ie, excessive rubbing of his nose) and gave no previous history of epistaxis. The acute epistaxis episode was treated with local measures (compression of the nasal alae and paper towels) and eventually stopped after ~10 minutes.

The patient reported feeling lightheaded while being treated for epistaxis as he was sitting upright, and therefore, supplemental oxygen was administered through a face mask (avoiding the scavenging nasal hood due to a recent episode of epistaxis). After ~5 minutes, Eve test and Romberg checks were satisfactorily repeated, and the patient was discharged home in the care of his mother. We contacted the patient later that day, and his mother confirmed no further incidences of epistaxis and that the patient was feeling well. He had no history of bleeding issues, and it was his first episode of epistaxis that the patient and mother could recall.

DISCUSSION

The vestibule, the respiratory region, and the olfactory region are the 3 simple divisions of the nasal cavity. The incisive canal also forms a strong association with the nasal cavity. The olfactory, nasopalatine, and nasociliary nerves all provide innervation to the nasal cavity. Both the internal carotid (through the anterior and posterior

Table 1. Epistaxis Cases Associated with Nitrous Oxide Sedation.*

Study	Patient age (years)	ASA class	Nitrous oxide concentration (%)	Potential etiology/risk factors associated with nitrous oxide/oxygen sedation
Faulks et al. 2007	3–81†	Study group consisted of “patients with intellectual disability”†	50	Anxiety
Baygin et al. 2010	5–8†	I or II†	40	No explanation given
Mathur et al. 2020	8	I	40	Irritation of nasal cavity Thin nasal lining Previous injury/trauma to nose
This case report	12	I	60	Excessive/aggressive nose breathing Drying and/or irritation of the nasal cavity Self-inflicted trauma (from nasal hood or on removal, nose rubbing etc) White coat syndrome (hypertension)

* Literature review of epistaxis cases associated with inhalational sedation using nitrous oxide/oxygen.

† Specific information for patient with epistaxis not detailed in study.

ethmoidal arteries) and external carotid (through the lateral nasal, greater palatine, sphenopalatine, and superior labial arteries) arteries provide its vascular supply. Furthermore, epistaxis may occur as a result of injury/trauma to Kiesselbach plexus and Woodruff plexus, 2 highly vascular regions in the nasal cavity.

The potential etiology of epistaxis following inhalational sedation with nitrous oxide/oxygen in this case report is still not definitively determined. A literature review using the medical subject headings (MeSH) terms: (((“Anesthesia, Inhalation”[Mesh])) OR “Nitrous Oxide”[Mesh]) AND “Epistaxis”[Mesh] in PubMed and the free terms ‘inhalation sedation’, ‘inhalational sedation’, ‘nitrous oxide’, and ‘epistaxis’ using Google Scholar and the Cochrane Library was carried out. In addition, the reference lists of identified articles were also assessed and further complemented by an internet free search. Only 3 previous reports regarding epistaxis associated with inhalational sedation with nitrous oxide/oxygen were identified (Table 1).

Faulks et al⁹ reported 1 case of epistaxis in over 605 sessions of inhalational sedation with 50/50% nitrous oxide/oxygen used alone in patients with intellectual difficulties. The authors concluded that epistaxis was linked to anxiety rather than the use of inhalational sedation. However, this conclusion seems rather unlikely considering virtually all dental patients who receive inhalational sedation have some degree of anxiety, and as such, anxiety-induced epistaxis would be expected to have a much higher incidence. In addition, there is no conclusive evidence of anxiety being a risk factor for epistaxis.¹⁰ It is not clear from this study if any of the patients with intellectual disabilities also had any risk factors for epistaxis.

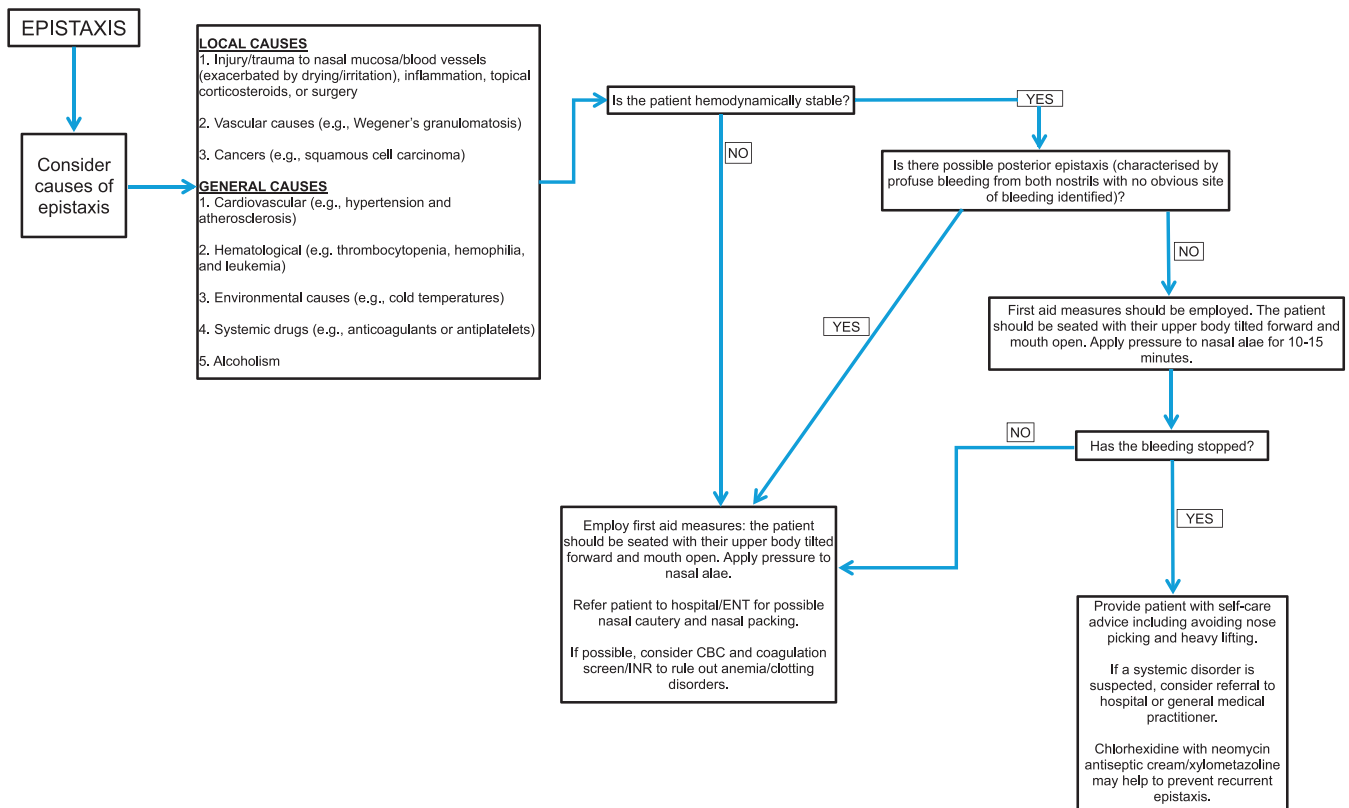
Baygin et al¹¹ also reported 1 case of epistaxis following treatment with inhalational sedation with 40/60% nitrous oxide/oxygen. This study involved children between 5 and

8 years of age and ASA I or II, but there was no specific information provided about the patient, and no explanation was given for the potential cause of epistaxis.

Most recently, Mathur et al¹² also reported in 2020 a rare case of epistaxis in an 8-year-old male during dental treatment with 40/60% nitrous oxide/oxygen inhalational sedation. Similar to this case report, that patient had no risk factors for epistaxis and was scheduled for dental extractions, but the epistaxis episode occurred during the administration of inhalational sedation in contrast with this case. Mathur et al¹² suggested the etiology of epistaxis associated with inhalational sedation could be due to irritation of the nasal cavity mucosa, an overly thin nasal mucosal lining, or a pre-existing or previous nasal injury (although this was not proven).

Considering the risk factors for epistaxis, it is likely that the potential causes of epistaxis for the patient in this case report were excessive and overly enthusiastic nasal breathing as the inhalational sedation was being administered, which may have caused some trauma (eg, rupture of vessels), and the prolonged administration of nitrous oxide/oxygen. These factors may have contributed to excessive drying of the nasal mucosa and increased the risk of epistaxis. Although the patient denied rubbing his nose, another potential cause could have been self-inflicted trauma following removal of the nasal hood. Nitrous oxide is not known to cause irritation of mucosal membranes¹³; however, oxygen therapy can cause irritation, this may relate to the duration of exposure or concentrated episodes (ie, high oxygen flow rates as occurs with use of the oxygen flush button). In addition, increased arterial blood pressure at the onset of epistaxis may be associated with white coat syndrome¹⁴ in patients receiving dental sedation. Anxiety is related to a rise in heart rate, blood pressure, and increased respiratory rate, which may explain why it could be considered a risk factor for epistaxis.

Figure. Epistaxis Management Flowchart.



Management of epistaxis in the primary dental care setting (adapted from National Institute for Health and Care Excellence¹⁵).

Epistaxis prevention primarily involves obtaining a thorough medical history, screening for hypertension, and using moisturizing balm prior to treatment. In patients with bleeding disorders or on anticoagulants and who are therefore at higher risk of epistaxis, preinvestigations (such as coagulation assessments) may help to reduce the risk of epistaxis. Should anterior epistaxis occur, continuous pressure should be applied to the nasal alae. Other measures may include chlorhexidine with neomycin cream, xylometazoline, and nasal packing (Figure). A small amount of moisturizing balm or cream can be applied into each nostril (pointing away from the nasal septum) and spread around interiorly by gently squeezing together the alae of the nose to spread the balm or cream around the nose. Xylometazoline normally comes as a nasal spray and used in each nostril to help prevent recurrent epistaxis. Patients with suspected posterior epistaxis or epistaxis not controlled by local measures may require referral to a hospital setting.

The majority of guidelines and studies reviewed focus on nasal oxygen therapy and how it can dry and irritate the nasal mucosa, along with the possibility that the nasal cannula prongs may directly traumatize the nasal

mucosa⁸ and increase the risk of epistaxis. The potential increased risk of epistaxis linked to the use of scavenging nasal hoods during inhalational sedation for dental surgery needs to be taken further into account. The risk of epistaxis is probably highest with a nasal cannula (or other equipment or airway devices that directly contact the nasal mucosa), somewhat lower with scavenging nasal hoods, and minimal with full face masks used during general anesthesia.

From the cases in the literature including this case report, epistaxis associated with inhalational sedation was seen in patients receiving nitrous oxide at concentrations of 40% and above, and all 4 cases presented as anterior epistaxis, the most common cause of epistaxis. One of the advantages of inhalational sedation with nitrous oxide/oxygen is that there are few medical contraindications to its use compared with intravenous sedation and general anesthesia. The author suggests that patients taking anticoagulants or those with bleeding disorders, a history of epistaxis, or other medical risk factors for epistaxis should be warned about the possible rare risk of anterior epistaxis with inhalational sedation. Most cases of anterior epistaxis should resolve quickly with local

Table 2. Summary and Recommendations.**Summary and recommendations for patients at higher risk of epistaxis requiring inhalational sedation*

All reported cases in the literature presented with anterior epistaxis and were controlled with local measures.

All patients receiving inhalational sedation should be screened for previous episodes of epistaxis or other episodes of bleeding.

Patients who are at higher risk of epistaxis (eg, previous trauma/epistaxis, inflammation, on anticoagulants, or patients with bleeding disorders) should be informed of risks beforehand.

* Summary of findings and recommendations from a literature review of epistaxis associated with inhalational sedation using nitrous oxide/oxygen.

measures, but in certain cases, such as the higher risk patient groups previously mentioned, epistaxis may require urgent medical care and/or referral to an ear, nose, and throat surgeon. A summary of the findings and recommendations from our literature review of epistaxis associated with inhalational sedation with nitrous oxide/oxygen for dental surgery are presented below (Table 2).

CONCLUSION

Inhalational sedation with nitrous oxide/oxygen is a common form of sedation used for dental treatment, and it has an excellent safety record. However, it can be very rarely associated with anterior epistaxis. Patients at higher risk of epistaxis should be warned prior to treatment to gain informed consent. Simple preventative measures for patients at higher risk of epistaxis who require inhalational sedation may include moisturizing the nasal cavity prior to treatment, screening for hypertension, reducing treatment time under sedation, avoiding direct trauma to the nasal anatomy, and asking patients to avoid aggressive and excessive nasal breathing. It may also be beneficial to assess for hemostatic stability in patients with bleeding issues and those at higher risk of epistaxis prior to treatment. Dentists should also be aware of the management of epistaxis and when to refer patients to a hospital setting.

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