
CME review

This feature is supported in part by an educational grant from AstraZeneca LP

Pathophysiology and progression of nasal septal perforation

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Objective: To review the prevalence, causes, and treatments of nasal septal perforation (NSP).

Data Sources: A literature search was conducted in MEDLINE to identify peer-reviewed articles related to NSP using the keywords *nasal septal perforation* and *septal perforation* for articles published between January 1, 1969, and December 31, 2006, and references cited therein.

Study Selection: Articles were selected based on their direct applicability to the subject matter.

Results: Causes of NSPs include piercings, exposure to industrial chemicals, illicit drug use, intranasal steroid use, surgical trauma, bilateral cautery, and possibly improper use of nasal applicators. Prevalence is poorly reported. Mechanisms of substance-induced NSP formation are not understood. Progression from epistaxis to ulceration to NSP could not be substantiated by the literature.

Conclusion: Depending on the patient, NSP may be viewed as desirable (nose rings), problematic (whistling, congestion), or inconsequential. Understanding the pathogenesis of NSP is important for the practicing physician required to make decisions about whether to recommend surgical correction or medical treatment. Although the etiology of NSP is overwhelmingly iatrogenic, there is an association with a number of medical diseases in addition to use of illicit drugs and/or prescription nasal sprays.

Ann Allergy Asthma Immunol. 2007;99:473–480.

Off-label disclosure: Drs Lanier, Kai, Marple, and Wall have indicated that this article does not include the discussion of unapproved/investigative use of a commercial product/device.

Financial disclosure: Drs Lanier and Marple have indicated that in the last 12 months they have worked as consultants for Alcon Labs.

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INTRODUCTION

The objective of this article is to review the prevalence, causes, and treatments of nasal septal perforation (NSP). NSP is defined as a pathologic or iatrogenically produced direct communication between the 2 nasal cavities that occurs as the result of a traumatic, inflammatory, neoplastic, or autoimmune process. NSP can be entirely asymptomatic, be accompanied by bleeding, crusting, and discomfort, or may herald more significant systemic disease. NSP may escape clinical discovery in the patient without decongestion, because a healed, smooth, well-circumscribed opening produces no

symptoms in up to 39% of patients.¹ Risks associated with the use of nasal sprays are discussed. Using a question and answer format, this review focuses on 2 common questions for the clinician: (1) how often does epistaxis lead to ulceration and perforation and (2) how great is the risk of topical vasoconstrictors and steroids in the creation and progression of NSP? In reviewing the prevalence, causes, and treatments of NSP, the medical literature (MEDLINE) was searched for the keywords *nasal septal perforation* and *septal perforation*. A total of 81 articles were retrieved, which seemed to be a surprisingly low total number of related articles on this subject, considering its perceived frequency. To meet the publication requirements of the *Annals* (ie, no more than 50 references), the most important articles, in the opinions of the authors, were chosen for discussion in this review.

ANATOMY, PATHOGENESIS, AND MECHANISM

The nose receives its rich arterial blood from a number of branches derived from both the external and internal carotid arteries. Of importance is the superior labial artery providing

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Received for publication January 8, 2007.

Received in revised form August 1, 2007.

Accepted for publication August 6, 2007.

the blood and nourishment to the anterior portion of the nasal septum, where most bleeding and perforation occur. This artery along with branches from the sphenopalatine and greater palatine artery feed the Kiesselbach plexus, or the Little's area, located on the anterior cartilaginous septum where many of the smaller arteries supplying the nasal septal mucoperichondria anastomose.²

The nasal septal mucoperichondrium (mucosal and perichondrial vasculature) covers the relatively avascular nasal septal cartilage and provides the tissue and blood supply to the septal quadrangular cartilage on both sides like a sandwich. Any insult (chemical or physical) producing damage to this normal anatomy can lead to ischemic necrosis of the septal cartilage, resulting in perforation. Perforations usually occur when the blood supply is disrupted on both sides in approximately the same area. Disruptions of the septal mucosa are called erosion or ulceration. Excoriation refers to a superficial erosion through the mucosa or submucosa but not through the perichondrial layers. Ulceration is defined as deeper erosion to the perichondrial layers exposing cartilage, whereas NSP represents a complete breach through all layers and the cartilage. The cartilage may survive and the septal tissue heal if only 1 side of the septum is deprived of its blood supply (mechanical trauma such as "picking"), but when the blood supply is cut off on both sides at approximately the same location, as in bilateral nasal cautery and septal repair, ischemic necrosis may occur, producing NSP. Any resultant healing around the edges of a perforation over a 3-layered structure (2 sides of the mucoperichondrium and cartilage) is likely to be thin and atrophic. If the edges of an NSP fail to heal normally, they are covered with an atrophic layer of mucosa, leading to ongoing crusting and a tendency toward bleeding because of the friction of abnormal airflow forces. The friction of the turbulent abnormal air forces and mechanical picking to relieve a sensation of congestion may result in ongoing crusting, bleeding, and enlargement.

PREVALENCE

The prevalence of NSP is less than 1% in a study of healthy Scandinavians³ contrasted to 35% in workers of electroplat-

ing factories.⁴ Treatment for epistaxis with cautery or surgery is often implicated, but the true incidence of NSP with regard to surgical procedures has not been reported.^{5,6}

SYMPTOMS AND PRESENTATION

The location and size of an NSP may influence patient perception of symptoms.⁷ Patients have reported bleeding (58%), by far the most common resulting symptom, followed by crusting (43%), obstruction (39%), pain (17%), and whistling (10%), with 15% being entirely asymptomatic.^{5,6} Whistling is more often associated with a smaller NSP. The larger an NSP, the greater the disturbances in laminar airflow and resulting turbulence, which can further damage respiratory epithelia. The more turbulence and drying, the more rhinorrhea occurs as the nose attempts to improve the humidity. The dried fluid is the material seen in crusting. Turbulence may also create a sensation of obstruction, which may lead to complications as the patient attempts to remove crusting by manual picking to improve congestion.⁷ NSP has been observed within a few days of employment in potash workers for whom the first symptoms were soreness and irritation followed by crusting and bleeding.⁸

LOCATION

In a review of 74 cases, Diamantopoulos and Jones¹ found that 92% of NSPs and ulcerations were seen anteriorly and 8% were located posteriorly or superiorly in the more difficult area to examine. These posterior lesions may be more commonly associated with trauma or systemic disease such as neoplasia, connective tissue disorders, and syphilis, whereas the more anterior ones may be more associated with trauma and more symptomatic with bleeding and crusting.

WHAT ARE THE CAUSES OF NSP?

Although few causes of NSP have been unequivocally proven, those that are thought to be common causes are listed in Table 1.

Piercings

An NSP may be the result of a deliberate act with cultural roots extending back thousands of years. The Bible mentions

Table 1. Causes of Nasal Septal Perforation

Traumatic	Inflammatory or infectious	Neoplastic	Other
Piercing for nose ring	Sarcoidosis	Carcinoma	Intranasal steroid sprays
Nasal surgery involving septum	Wegener granulomatosis	T-cell lymphomas	Illicit drugs
Cauterization for epistaxis	Systemic lupus erythematosus	Cryoglobulinemia	Industrial exposure (chromic acid, potash fumes)
Nose picking	Tuberculosis		
Nasogastric or oxygen tube placement	Syphilis		Lime dust exposure
Button battery or foreign body in nose	Acquired immunodeficiency syndrome		Renal failure
	Diphtheria		
	Crohn disease		
	Dermatomyositis		
	Rheumatoid arthritis		
	Leishmaniasis		

nose rings in Genesis 24:22, when Abraham awarded Rebekah a "gold ring weighing half a shekel which he fastened on her nose." By the 16th century, the practice was common in the Hindu religion in India. NSP and nose rings are also seen in the Muslim religion, where women wear "nathunis" as a sign of wealth. Internet sites reflect a growing trend of nasal piercing to complement tattoos, scarification, and other body enhancements. Some of these iatrogenic NSPs are large enough to admit objects up to 2 cm in diameter.

Disease

NSP has been associated with sarcoidosis, Wegener granulomatosis, and infectious disease such as tuberculosis, syphilis, viral disease, and diphtheria.⁹⁻¹² Other reported associations with NSP have included connective tissue disease such as systemic lupus erythematosus,¹³ Crohn disease,¹⁴ dermatomyositis, rheumatoid arthritis, human immunodeficiency virus and acquired immunodeficiency syndrome.¹⁵ Fungal association has been widely reported with NSP, as have parasitic infections in immunocompromised patients.^{16,17} Neoplasia is also an infrequent but important cause of NSP.^{18,19} Cryoglobulinemia has also been reported as a cause.²⁰

Industrial Exposure

NSP is strongly associated with industrial exposure to metal plating solutions. A cross-sectional survey of 71 platers at 20 chrome plating companies in the West Midlands, United Kingdom, between 1993 and 1995 conducted by questionnaire and clinical examination revealed that 23% were found to have dermatitis at the time of the visit, 45% of companies had at least 1 case of dermatitis in their plating workforce, 23% of platers had evidence of old "chrome ulceration," and 13% had evidence of new and healing chrome ulcers. Examination of the nasal passages revealed that 17% had inflammation and 14% had NSP. Those with perforations were younger than 35 years at the time of perforation, which had usually occurred in the first 10 years of exposure.²¹

Medical Procedures

NSP may result from surgical trauma in deviated septal repair or cautery for epistaxis.^{5,22} The time from surgery to NSP may be immediate, where perforations are created and not appreciated, but the overall incidence of NSP after either cautery or septal repair is not reported. A variety of trauma-produced NSPs include nasogastric and oxygen tube placement and foreign objects such as button batteries and small magnets.²³⁻²⁵

Illicit Drug Use

Illicit drugs, particularly cocaine, have been associated with NSP, which deserves special consideration. During the cocaine epidemic in the 1980s, Vilensky²⁶ reported that at least 22 million Americans used illicit cocaine, with 4 million using cocaine at least once a month, and an incidence rate of 4.8% for cocaine-induced nasal and sinus problems, including NSP. The prevailing theory of causation concerns the potency and the duration of the vasoconstriction caused by

inhibition of noradrenalin reuptake, but there may be more important issues. Cocaine appears to compete with calcium ions and controls the flux of sodium ions. This inhibition stops the generation of nerve impulses and produces local anesthesia so the user does not feel pain and trauma when inserting spoons and other objects into the nose. Also, illegal suppliers extend or "cut" cocaine with known irritants such as borax and talcum powder. These adulterants may contribute more to mucosal destruction than vasoconstriction. More recently, Greene²⁷ reported that excipients may also be responsible for NSP in drug abusers using crushed OxyContin by inhalation, since OxyContin is not a vasoconstrictor at all.

Intranasal Steroids

The first topical intranasal steroid spray approved in the United States was dexamethasone aerosol (Dexacort) in 1965.²⁸ Since then, a number of other steroid nasal sprays have been approved, including beclomethasone, budesonide, flunisolide, fluticasone propionate, mometasone, and triamcinolone and, most recently, ciclesonide and fluticasone furoate. The initial rationale for the use of these agents was to provide a high concentration of steroid to receptor sites in the nasal mucosa, avoiding systemic adverse effects associated with steroids.

NSP has been associated with intranasal steroids since 1975 with the use of dexamethasone aerosol,²⁹ the prevalent theory in those initial observations being that vasoconstriction leads to necrosis. However, there have been widely diverse alternative explanations, including the possibility of preservatives such as benzalkonium chloride in animal studies.³⁰ Squamous metaplasia produced within 21 days in rats indicated a potentially toxic role for this additive found in commercial preparations of beclomethasone and flunisolide.

Delayed or contact sensitivity to nasal steroids, particularly budesonide, has also been associated with NSP.³¹⁻³⁴ Allergic dermatitis from topical steroids has been associated with the substitution pattern on the steroid D-ring, with budesonide as the prototype for the most allergenic group. However, other authors have suggested that contact allergy or delayed hypersensitivity may be a possible explanation for septal perforations in selected patients, especially if paranasal eczema develops, but in general cannot explain why topical glucocorticosteroid use is associated with NSPs.³⁵

Corticosteroids create an anti-inflammatory effect by binding to an intracellular glucocorticoid receptor. They switch off multiple activated inflammatory genes through inhibition of histone acetyltransferase and recruitment of histone deacetylase activity to the inflammatory gene transcriptional complex. In addition, corticosteroids activate several anti-inflammatory genes and increase the degradation of messenger RNA encoding certain inflammatory proteins.³⁶⁻³⁸ Nelson³⁹ looked specifically at the action of nasal steroids and divided the mechanisms into effector and director categories. He noted that intranasal steroids reduce effector cells (eg, eosinophils) in number but not their chemotaxis or degranulation. Conversely, he noted that inhaled steroids decrease the

influx of basophils and mast cells more associated with the delayed aspect of inflammation. Decreased permeability factors, reduction of adhesion molecules, and reduced inducing cytokines were also noted. The result may be a reduction in angiogenesis and a decrease in perfusion and permeability. He described director mechanisms as the inhibition of epithelial cells from producing chemotactic and proinflammatory cytokines, including interleukin (IL) 1, IL-8, IL-6, granulocyte-macrophage colony-stimulating factor, and stem cell factor. Antigen-presenting cells are decreased, as is the production and secretion of cytokines by T_H2 CD4⁺ T lymphocytes. Wanner et al⁴⁰ divided the actions on airway vasculature into genomic and nongenomic actions, with emphasis on the properties of corticosteroids that do not involve gene transcription. Since vasodilation is considered part of inflammation, vasoconstriction can be considered anti-inflammatory. Topical intranasal steroids produce vasoconstriction by potentiating noradrenergic neurotransmission, which is rapid but transient in the vasculature. Intranasal steroids may therefore potentially be involved in the production of perforation by both decreasing vessel density and perfusion.

WHAT IS THE RISK OF NSP WITH THE USE OF APPROVED NASAL SPRAYS?

Mechanical Abrasion

Initial epistaxis seen early in the use of any nasal spray may be largely due to a mechanical trauma from touching or abrading the nasal septum with the hard plastic tip of the applicator. It is possible that erosions and sometimes even ulcerations occur with the simple mechanical trauma attendant to using a hard plastic object regularly in the nose, or even the repeated force of the liquid spray impacting the septum, especially if the septum is repeatedly damaged. Patient instruction for proper use is essential to reduce trauma and the potential for septal abrasion.⁴¹

Vasoconstrictors

Topical vasoconstriction is often mentioned as a potential cause of NSP, although the literature is scant with human observations and proof. DeBernadis et al⁴² found that rats exposed to 10 to 30 times the marketed dose of oxymetazoline experienced microscopic ulcerations with exudates at the end of 15 days of exposure. Such animal trials are often

extrapolated to human experience but may not correlate. For example, the Food and Drug Administration's (FDA's) reports of NSP with over-the-counter vasoconstrictor sprays reveal an extremely small incidence compared with a large and consistent utilization. Market research reveals that in 2006 approximately \$60 million worth of Afrin (Schering-Plough, Kenilworth, New Jersey) was sold in the United States, representing approximately 150,000,000 mL (calculation based in part on data reported by ACNielsen Inc, New York, New York, through its Strategic Planner Service for the Nasal Product Internal Category for the 52-week period ending December 30, 2006, total US excluding Wal-Mart market). When the generic brands of oxymetazoline are added in, the overall exposure to the US population is enormous, but there is no sense in the clinical community of an association of topical vasoconstrictors and NSP.

Intranasal Steroids

One source for reported cases of NSP associated with the use of prescription intranasal steroid sprays is the FDA's Adverse Event Reporting System (AERS). The AERS computer database contains reports from foreign countries, clinical studies, literature, health professionals, consumers, and other various sources. Many think that the AERS woefully underreports adverse events. Although AERS includes mandatory reports from healthcare-related companies, noncompany reports are voluntary and neither is tied to meaningful incidence rates, since they are not correlated with actual units of use. Nevertheless, a query of the AERS conducted in May 2007 surveyed spontaneous adverse event reports of all types received between January 1, 1969, and December 31, 2006. The number of reported cases of NSP reflected by the AERS with regard to several FDA-approved intranasal sprays was low (Table 2).

Another source for the prevalence of NSP in conjunction with the use of intranasal sprays is the FDA-approved label (package insert or prescribing information). FDA-approved labels include incidence rates when available through controlled clinical trial data. When adverse events occur at a low rate in controlled clinical trials or are sporadically reported after a product is approved (eg, AERS), the FDA-approved label typically states the incidence as "rare" (no clearly defined incidence rate is associated with this term). The labeled

Table 2. Reported Cases of Nasal Septal Perforations Due to Intranasal Sprays^a

Intranasal spray	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006
Azelastine		1				1	1		2	
Budesonide		2	4	7	1		2			
Cromolyn										
Fluticasone	1			1	2	1	4	2	2	3
Ipratropium			1					1	1	1
Mometasone		3				2		1	1	
Oxymetazoline						1	1			

^a Data are from the Food and Drug Administration's Adverse Event Reporting System database for January 1, 1997, through December 31, 2006 (no nasal septal perforations were noted in 1969–1996).

incidence rate for NSP is “rare” for fluticasone propionate (Flonase), mometasone (Nasonex), budesonide (Rhinocort Aqua), beclomethasone (Beconase AQ), flunisolide (Nasarel), and triamcinolone acetonide (Nasacort AQ) steroidal sprays and “none” for ciclesonide (Omnaris) or fluticasone furoate (Veramyst).

DOES THE BLEEDING SEEN WITH NASAL SPRAYS PROGRESS TO ULCERATION AND ULTIMATELY TO PERFORATION?

The potential for progression of a toxic nasal process to initially produce epistaxis as its manifestation, followed by ulceration then perforation, is a logical but an entirely unproven sequence given the common rate of spontaneous epistaxis. Epistaxis may be the most common presenting symptom of NSP, but its role as a long-term predictor is much less certain since epistaxis as an isolated problem is extremely common.^{43,44}

The time course from exposure to perforation with industrial exposure may be a matter of days. The time course of NSP from initiation of topical intranasal steroid drug to observable perforation has been as short as several months to as long as several years. Young females may be affected more than males.³⁶ In one of the first cases reported, the sequence was irritation, crusting, and ulceration after 2 months, progressing to perforation after mechanical trauma and continued use of the drug at 3 months.⁴³ In some case reports, crusting and bleeding may have preceded the diagnosis, but a link in recent literature from bleeding to ulceration to perforation is weak,⁴⁵ especially in view of the rates of spontaneous and autoresolving epistaxis seen in clinical experience. Furthermore, although epistaxis rates consistently range from 6% to 14%, the incidence of perforation is extremely low despite continued use. In clinical trials of the existing approved nasal steroid sprays, NSPs were seldom reported. However, there is some question of preexisting lesions, since it is not common to decongest the nose for full examination before study. A standardized procedure for examination of the nose is presented in Table 3.

A NSP can be easily missed in physical examinations using an otoscope for inspection of the nose. The typical speculum used for otic inspection narrowly focuses the physician to a fairly restricted depth with the inferior turbinates as the primary inspection area. If an NSP is large and anterior in a patient with acute allergic disease, the field of view is often obscured and compromised by large and tender turbinates, forcing the examiner to stand off the usual technique. When appropriate equipment (eg, nasal illuminator and large speculum) is used in a decongested nose, a better examination can be accomplished.

The value of biopsy of an NSP has been debated^{5,46} because of the possibility of a diagnosis of neoplasia, connective tissue disease, or an odd inflammatory picture. However, the prebiopsy and postbiopsy diagnosis agreement in uncomplicated cases is so good that most authors believe that biopsy, particularly for anteriorly placed NSP, adds little to

Table 3. Standard Examination of the Nose for the Presence of Nasal Septal Perforations

Equipment:

Nasal speculum, light source, otoscope with speculum no smaller than 1 cm, oxymetazoline spray

Nondecongested examination: to rate congestion only

Decongested examination: use 2 sprays of oxymetazoline on each side 10 minutes before examination. Inspect from 3 different positions: head up 30°, head neutral, and head down 30°. Use either nasal speculum or large otoscope speculum.

Related observations:

1. Perforations:

- Appearance: 85% of perforations are smooth and well circumscribed, 77% are less than 2 cm in diameter.
- Location: Little's area 84% and posterior 9%
- Important history: 60% epistaxis, 43% crusting, 17% pain, 10% whistling. 95% will have had nasal surgery.

2. Excoriation/Ulcerations: Thought by some to be the precursor to perforation because they are found in the same location and pattern of perforations, they may be obscured by crusting. Irrigation of the area with a saline nasal spray may facilitate the inspection.

the overall clinical management except in the case of suspected Wegener granulomatosis where biopsy is indicated. There are no grossly observable characteristics for NSP that provide a clue for systemic disease.

ONCE PERFORATION IS DIAGNOSED, WHAT IS THE DANGER OF CONTINUED DRUG USE?

A clinical dilemma may occur when an NSP is diagnosed in a patient who needs ongoing treatment for allergy, including nasal steroid sprays. If the margins of a smaller perforation are well healed and the patient can be instructed to avoid trauma from the applicator tip or direct spray, nasal sprays including steroids may be used with some discretion if followed closely by a physician. It is possible that enlargement might be minimized if the proper technique of directing the sprays to the lateral wall is used, but caution should be exercised and considerations for surgical correction made. However, enlargement of preexisting NSP has been reported even when the steroid was used in ocular application.⁴⁷

Selecting the appropriate treatment for an NSP depends on a number of factors that must be individualized. Symptoms are related not only to the size of the perforation but also to its location. In vitro modeling reveals that perforations located along the anterior aspect of the nasal septum cause abrupt disruption of laminar airflow patterns.⁴⁸ Abrupt transition to turbulent airflow at the posterior edge of the perforation gives rise to symptoms of drying, crusting, whistling, and subjective obstruction. Given that physiologic turbulence is normally present along the posterior nasal cavity, posterior perforations are less likely to result in similar symptoms. Another factor that can lead to patient complaints is the size of the perforation. Although larger anterior perforations are more likely to result in crusting, bleeding, and nasal airway

obstruction, smaller perforations are more typically associated with “whistling.”

Asymptomatic NSPs or those giving rise to only mild, intermittent symptoms should be treated conservatively. Many asymptomatic patients require no specific treatment.⁴⁷ Others who experience only mild symptoms can be managed simply through the use of nasal saline sprays, irrigations, and emollients. Regular use of these methods in this population of patients is intended to decrease crusting and attenuate local inflammation.

Another nonsurgical option for the treatment of symptomatic NSP is placement of a septal button.⁴⁹ The basic design of the septal button is rather simple and intended to correct the discontinuity in the nasal septum. Shaped roughly like a sewing bobbin, the button is designed to straddle the perforation. Most commercially available septal buttons are constructed from medical grade Silastic to decrease crusting, irritation, and extension of the existing perforation. Placement of a septal button can easily be performed under topical anesthesia in the clinic setting. Once in place, the button will remain without the need for significant maintenance for long periods. Longitudinal studies have confirmed the effectiveness of this form of treatment for control of epistaxis, nasal airway obstruction, and whistling. Unfortunately, many patients will continue to experience some crusting at the margin of the button.

Surgical options are typically reserved for those symptomatic patients who have failed or do not care for other forms of treatment. Success of repair is potentially dependent on a number of factors that should be considered before surgery. Larger perforations, use of cocaine, and active nasal manifestations of autoimmune disease are associated with high rates of failure. Of these risk factors, the size of the NSP has been most commonly correlated with failure, demonstrating that the incidence of reoperation can be as high as 70% in the case of NSP larger than 4 cm in diameter. As a result, a number of different surgical approaches and techniques have been developed to decrease the rate of failure in these more challenging cases (Table 4).

CONCLUSION

NSP is a relatively uncommon problem that is viewed by patients as desirable (nose rings), problematic (because of whistling or the sensation of congestion), or inconsequential. NSP is often viewed by physicians with alarm and concern and worthy of a sometimes difficult repair. The most common causes of NSP are thought to include piercings, industrial exposure to chemicals, use of nasal sprays, use of illicit drugs, or as a result of surgical trauma or bilateral cautery. If cautery is performed, it would be best kept to 1 side, since bilateral trauma is more likely to ultimately result in NSP. The mechanism of NSP due to nasal spray use is unclear. Evidence to support progression from epistaxis to ulceration to NSP is lacking. Notwithstanding the prevailing theory of vasoconstriction due to nasal spray use as a precursor to NSP, literature support is again lacking. Mechanical trauma from a

Table 4. Nasal Septal Perforation Repair Options

Nonsurgical	Surgical
Observation	Closed approach
Nasal irrigation	Local pedicled flaps
Emollients	Local flaps with interposed graft
Septal button	Open approach (possible staging)
	Local flaps
	Local flaps with interposed graft
	Use of composite grafts
	Tissue expander
	Graft materials
	Autograft
	Temporalis fascia
	Fascia lata
	Conchal cartilage
	Allograft
	Acellular dermis

plastic nasal applicator is also perceived as a common precursor, leading to ulceration, physical aggravation by the patient, and NSP. In this case, patients can be taught the proper method of administering a nasal spray.

ACKNOWLEDGMENTS

We thank Dr Stephen Motsko, The Degge Group Ltd, Arlington, Virginia, for providing the AERS data and Beverly Loyd for assistance with preparation of the manuscript.

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Objectives: After reading this article, participants should be able to demonstrate an increased understanding of their knowledge of allergy/asthma/immunology clinical treatment and how this new information can be applied to their own practices.

Participants: This program is designed for physicians who are involved in providing patient care and who wish to advance their current knowledge in the field of allergy/asthma/immunology.

Credits: ACAAI designates each Annals CME Review Article for a maximum of 2 category 1 credits toward the AMA Physician's Recognition Award. Each physician should claim only those credits that he/she actually spent in the activity. The American College of Allergy, Asthma and Immunology is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

CME Examination

1–5, Lanier B, Kai G, Marple B, Wall GM. 2007;99:473–480.

CME Test Questions

1. Which of the following is most commonly associated with nasal perforation?
 - a. intranasal cocaine use
 - b. bilateral septal bleeding and crusting in a 12-year-old child who picks his nose
 - c. employment at a potash factory
 - d. recurrent ulceration of the right side of the nose in a 30-year-old with epistaxis
 - e. nasal septal repair
2. Nasal septal perforations may escape casual detection with:
 - a. use of otic speculum for nasal inspection
 - b. anterior placement in Little's area
 - c. severe allergic inflammation without decongestant
 - d. a and c
 - e. b and c
3. The second most common presenting symptom of a nasal septal perforation is:
 - a. whistling
 - b. obstruction
 - c. pain
 - d. crusting
 - e. bleeding
4. The most common mechanism for nasal spray-induced nasal septal perforation is:
 - a. vasoconstriction
 - b. agenesis of vessels with decrease in perfusion and permeability
 - c. improper use of the nasal applicator
 - d. incipients and preservatives
 - e. anticoagulation
5. Treatment of nasal septal perforation should not include:
 - a. septal button
 - b. surgical reconstruction
 - c. nasal steroids
 - d. nasal saline sprays, irrigation, or emollients
 - e. no treatment at all

Answers found on page 521.