Letters to the Editor

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This series is coordinated by Kenny Lin, MD, MPH, Associate Deputy Editor for *AFP* Online.

Optimal Technique for Application of Corticosteroid Nasal Spray

Original Article: Treatment of Allergic Rhinitis

Issue Date: December 1, 2015

See additional reader comments at: http://www.aafp.org/afp/2015/1201/p985.html

TO THE EDITOR: I would like to point out an error implied by the cover art in the December 1, 2015, issue. The illustration demonstrates occlusion of the opposite nostril and implies a strong sniffing of the nasal corticosteroid spray. Because deposition of medication in the throat is ineffective and increases systemic absorption, strong sniffing with nasal occlusion is likely less effective than application to the turbinates without sniffing or with gentle sniffing, avoiding occlusion.[1]

Also, a common recommendation is to use the hand opposite the nostril being treated to aim the spray up and outward, away from the septum, in the direction of the tear duct or medial canthus.[2] This effectively applies spray to the more reactive surface of the turbinates and avoids the highly vascular septum, a source of first-pass absorption and a major site of epistaxis.

The best recommendations for corticosteroid nasal spray application are: (1) gently blow nose, (2) lean forward with the nasal spray aiming nearly vertical, (3) using the hand opposite the nostril being treated, aim the nozzle slightly up and outward (lateral and cephalad) toward the tear duct or medial canthus, (4) spray without sniffing or while sniffing very gently, (5) gargle and rinse the mouth and throat.

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IN REPLY: All of the studies we could find concerning this issue concluded that there is no optimal technique for administering intranasal sprays given individual anatomical differences and differences in devices. They agree that air movement helps improve medication distribution (sniffing vs. not sniffing) but cannot agree on an optimal head position, although it seems that head position may make a difference if there is no airflow present.

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EDITOR'S NOTE: Although Dr. Kelleher endorses a recommended technique, the one illustrated on the cover of the December 1, 2015, issue is the one generally illustrated in patient instructions by the manufacturers of intranasal corticosteroids: https://www.flonase.com/about/how-to-use-flonase/, http://nasacort.com/how-to-use.aspx?, and https://www.merck.com/product/usa/pi_circulars/n/nasonex/nasonex_ppi.pdf.—JAY SIWEK, MD, EDITOR

Responsiveness to Adenosine Does Not Confirm SVT Diagnosis

Original Article: Diagnosis and Management of Common Types of Supraventricular Tachycardia

Issue Date: November 1, 2015

Available online at: http://www.aafp.org/afp/2015/1101/p793.html

TO THE EDITOR: This article was well written and provided the right amount of detail for primary care clinicians. However, the assertion that adenosine can be used to reliably distinguish ventricular tachycardia (VT) ▶

from supraventricular tachycardia (SVT) with aberrancy is simply not true. Guidelines from the American Heart Association endorse the use of adenosine when a wide complex tachycardia is suspected to be the result of SVT with aberrancy, but arrhythmia termination with adenosine does not prove that the patient has SVT.[1]

VT may also be responsive to adenosine in some situations. Although VT can be ruled in or made more or less likely using the Brugada criteria mentioned in the article, there is no way to rule it out. Adenosine likely is safe for treating a patient with VT acutely and may work to break the arrhythmia, but labeling a patient with adenosine-responsive VT as having SVT with aberrancy and sending him or her home could result in disastrous outcomes. VT is a deadly arrhythmia with serious underlying causes, such as acute coronary syndrome, dilated cardiomyopathy, myocarditis, and valvular heart disease.

Because the consequences are so grave, my practice is to treat all regular wide complex tachycardias as VT, unless I have a cardiologist immediately available to review both the old and new electrocardiographs. More information about adenosine-responsive VT is available.[2-4]

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IN REPLY: Adenosine-sensitive VT, sometimes known as ventricular outflow tract VT or idiopathic VT, is a form of VT that occurs in otherwise structurally normal hearts. It is believed to occur in 10% of patients with VT.[1] It can present as exercise-induced sustained VT. Although adenosine has diverse electrophysiologic effects in supraventricular myocardium, its effect in ventricular myocardium is based solely on its inhibitory effects on adenylyl cyclase and cyclic adenosine monophosphate (cAMP).[2] This means that only cAMP-mediated VT is sensitive to adenosine, and that VT that originates from a focus within the ventricular outflow tract (usually the right) is caused by cAMP-mediated activity. Adenosine has no antiarrhythmic effect in catecholamine-dependent reentry or other types of VT. Accordingly, adenosine termination of VT is diagnostic of cAMP-mediated triggered activity, which is what accounts for most forms of right and left ventricular outflow tract tachycardia.

Until recently, outflow tract ventricular arrhythmias (including premature ventricular contractions) were considered benign, and this remains the case for most patients. However, it is now appreciated that some outflow tract arrhythmias trigger polymorphic VT, ventricular fibrillation, or sudden cardiac death, or result in cardiomyopathy.[3]

Dr. Firth is correct in asserting that this condition may be overlooked and deprive the patient of the opportunity to have the malignant arrhythmia managed with radiofrequency catheter ablation or an internal defibrillator. At present, there is not an agreed-upon parameter to distinguish between benign and malignant VT in these patients. Certainly the expertise of a cardiac electrophysiologist is indicated for management. Most experts and guidelines agree with the recommendation that all regular wide complex tachycardia should be managed as VT.

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Correction

Incorrect Drug Classification/SORT rating. The article "Sexual Dysfunction in Women: A Practical Approach" (August 15, 2015, p. 281) contained an error in *Table 2* (p. 283), which incorrectly identified trazodone and venlafaxine as examples of monoamine oxidase inhibitors. Trazodone, venlafaxine, and monoamine oxidase inhibitors should have all been listed under the heading Psychotropics. Also, the sixth recommendation in the SORT table (p. 287), "Directed masturbation is recommended for lifelong anorgasmia," should have received an A rating (consistent, good-quality patient-oriented evidence) rather than a C rating (consensus, disease-oriented evidence, usual practice, expert opinion, or case series). The online version of this article has been corrected. ■