What is the most effective therapy for vasomotor rhinitis?

**Evidence-Based Answer**

Symptoms of vasomotor rhinitis (VR) may be reduced with:

- Intranasal fluticasone propionate (SOR: A, based on a meta-analysis of RCTs)
- Intranasal azelastine (SOR: B, based on one RCT)
- Intranasal ipratropium bromide (SOR: B, based on one RCT)
- Acupuncture (SOR: C, based on one low-quality RCT)

A meta-analysis was performed of 3 randomized, placebo-controlled trials evaluating use of intranasal fluticasone propionate in 983 adult patients with VR.1 Patients with and without nasal eosinophilia were randomized to 3 groups: 200 mcg fluticasone per nostril daily, 400 mcg fluticasone per nostril daily, and placebo. The primary outcome was change over a 28-day treatment period in total nasal symptom score (TNSS; a 300-point symptom score of the patient’s ratings of nasal obstruction, postnasal drip, and rhinorrhea). The mean change in TNSS was –84 in the 200-mcg group, –82 in the 400-mcg group, and –64 in the placebo group (P<.002 for both treatment groups vs placebo).

Two multicenter, randomized placebo-controlled trials with 426 adult patients evaluated the efficacy of intranasal azelastine for VR. Patients were randomized to receive 1.1 mg azelastine per nostril daily or placebo nasal spray for 21 days in 2 parallel groups.2 The primary outcome, change in total VR symptom score (patient report of nasal congestion, postnasal drip, sneezing, and rhinorrhea graded as 0=none to 3=severe during the previous 12 hours), was reduced by 24% and 22% in the azelastine groups, but by only 11% and 12% in the placebo groups (P<.001 and P=.007, respectively).

Another RCT evaluated 233 adult patients randomized to ipratropium 0.03% nasal spray 2 sprays per nostril 3 times daily or placebo.3 The primary outcome measures of duration and severity of rhinorrhea decreased 34% and 30%, respectively, in the ipratropium group compared with 19% and 15% in the placebo group (P<.05 for both comparisons).

Finally, a 2009 RCT included 24 adult patients randomized to acupuncture or sham laser for treatment of VR. The primary outcome of change in nasal sickness score (NSS; a 27-point scale assessed by patient response to a questionnaire) was –5.2 points for acupuncture compared with –2.0 points for placebo (P<.01). Limitations included small study size and a significant baseline difference in NSS between groups.4

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Is a carotid bruit a good predictor of underlying carotid stenosis in an asymptomatic person?

**Evidence-Based Answer**

No. A carotid bruit is a poor predictor of carotid stenosis in asymptomatic patients. In addition, the absence of a bruit is not reassuring. (SOR: B, based on conflicting cohort studies.)

In a prospective cohort study, 2,736 apparently healthy asymptomatic patients from a preventive cardiology clinic were assessed.1 Participants were referred for an evaluation of their risk factors and a routine ambulatory cardiovascular screening. They all were examined for carotid bruits and had ultrasound examinations of the carotid arteries. Hemodynamically significant stenosis was defined as >50% stenosis. The participants’ mean age was 52 years. Nearly half had hypertension.

Overall, 95 of 114 subjects with >50% stenosis had no bruit. The positive likelihood ratio (+LR) of carotid bruit for carotid artery stenosis >50% was 0.90 (95% CI, 0.34–2.41) and the negative likelihood ratio (−LR) was 1.00 (95% CI, 0.97–1.04). Thus, the presence or absence of a carotid bruit did not affect the likelihood of significant carotid artery stenosis.1

In a prospective, multiethnic, community-based cohort study, 686 asymptomatic subjects were examined for carotid bruits and underwent carotid duplex scanning.2 The mean age was 68 years. About 60% of subjects were Hispanic, 20% African American, and 20% Caucasian. Carotid bruits were detected in 4.1% of subjects. Carotid stenosis ≥60% was found in 2.2% of subjects. Seven of 16 subjects with ≥60% stenosis...
had no bruit. Sensitivity and specificity of a bruit for stenosis were 56% and 98% (+LR 28 and −LR 0.45).

In a prospective cohort study, 153 patients undergoing coronary artery bypass grafting who had no previous history of cerebrovascular events were evaluated.³ They were all examined for cervical bruits and received carotid artery duplex scanning. The mean age was 57 years, and 94% were male. Bruits were detected in 7.8% of patients. The sensitivity and specificity of cervical bruit for detection of ≥50% ipsilateral internal carotid artery stenosis were 20% and 93.5%, respectively (+LR 3.1 and −LR 0.85).

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Do ACE inhibitors alter lung function in patients with asthma?

Evidence-Based Answer
No. Angiotensin-converting enzyme inhibitor (ACE-I) medications do not alter lung function in patients with asthma. (SOR: C, based on bench research.)

ACE-I medications increase the production of pulmonary bradykinin and frequently cause cough. Theoretically, they might alter pulmonary function or disease severity in patients with asthma.

A prospective, randomized double-blind study (n=21) evaluated the effects of oral enalapril 12 mg daily (n=10) and spirapril 12 mg daily (n=11) on airway responsiveness and symptoms.¹ Patients were included if they demonstrated bronchial responsiveness to a methacholine challenge test (defined as a reduction in FEV1 by 20%). Baseline FEV1, forced vital capacity (FVC), and FEV1/FVC ratios were obtained.

After 3 weeks, no significant changes were noted in FEV1, FVC, or FEV1/FVC with either therapy. In addition, bronchial responsiveness to methacholine did not change with the use of either ACE-I agent. Six weeks after discontinuing treatment, there were still no changes in bronchial reactivity, FVC, FEV1, or FVC/FEV1. No P values or confidence intervals were provided. Limitations of the study included the small sample size and the use of baseline measures rather than a placebo arm.¹

An older, double-blind crossover study (n=16) from Spain assessed the effect of an ACE-I on patients with asthma with increasing doses of captopril and placebo for 4 weeks.² Asthma was defined using clinical and lung function criteria (based on the American Thoracic Committee of 1962). Half the patients received captopril 25 mg twice a day, which was increased up to 75 mg twice a day; the others received a placebo. After 4 weeks, individuals were all retested and the groups were crossed over.

Neither group had significant changes in spirometric results or methacholine responsiveness. Limitations to the study included small sample size and lack of clinical symptom scoring.²

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How long does prenatal RhoGAM last?

Evidence-Based Answer
The total body load of anti-D immunoglobulin G (IgG) 12 weeks after a single 300-mcg dose of intramuscular anti-D immunoglobulin given at 28 weeks' gestational age (GA) is variable, ranging from undetectable to more than 25 mcg. Similarly variable total body loads result if 100 mcg of the immunoglobulin is given at both 28 and 34 weeks' GA. In many patients, therefore, the residual anti-D IgG will be insufficient to prevent Rh sensitization with the birth of an Rh D-positive infant. Routine administration of anti-D immunoglobulin within 72 hours of delivery of an Rh D-positive infant is therefore recommended. (SOR: B, based on consistent cohort studies.)

In 2003, a randomized, multicenter cohort study followed serum concentrations of anti-D IgG in 14 pregnant Rh D-negative women who received 300 mcg anti-D immunoglobulin at 28 weeks’ GA.¹ The serum half-life of anti-D IgG was about 17 days. Quantifiable