



*Answers to your questions
from our medical experts*

1. Prevention of Androgenetic Alopecia

? If saw palmetto's mode of action is similar to finasterides, can it be used to prevent androgenic alopecia?

Submitted by: **Ken Armstrong, MD**, Niagara Falls, Ontario

Finasteride is an androgen inhibitor. It inhibits type II 5- α reductase (most common skin locations are hair follicles on the top of the scalp, frontal to vertex, root sheaths and dermal papilla, sebaceous gland ducts). It is FDA-approved for treating male pattern androgenetic alopecia. Other androgen inhibitors also inhibit or block enzymes leading to dihydrotestosterone production and include:

- Dutasteride
- Ketoconazole
- Progestin hormone agents
- Leuprolide
- Nafarelin
- Herbal products (e.g., saw palmetto, green tea)

Finasteride has been shown to cause a

20% to 30% decrease in PSA levels in clinical trials with a 1 mg dose in men 18- to 41-years-old and a 50% drop in patients > 50-years-old. Consider checking PSA in men \geq 50-years-old before receiving the first dose. If a PSA is done while taking finasteride and the patient is at least 41-years-old, then double this number to estimate the "true" PSA value.

Similar precautions may be prudent if patients are taking saw palmetto. Although there is a plausible theoretical mechanism of action, there is no good evidence to support the use of saw palmetto to prevent androgenetic alopecia.

Answered by: **Dr. John Kraft**; and
Dr. Charles Lynde

2. Nitro Spray for Anal Fissures

? Does nitro spray help with the pain of anal fissures?

Submitted by: **Steve Choi, MD**, Oakville, Ontario

Treatment of chronic anal fissures with nitroglycerin has been evaluated in numerous clinical trials. Nitric oxide can cause relaxation of the anal sphincter leading to decreases in anal resting pressure and increases in blood flow to the region. The most common preparation is a nitroglycerin ointment or topical patch. To my knowledge, nitroglycerin sprays have not been evaluated for anal fissures.

Resource

1. Dhawan S, Chopra S: Nonsurgical Approaches for the Treatment of Anal Fissures. *Am J Gastroenterol* 2007; 102(6):1312-21.

Answered by: **Dr. Richmond Sy**

3. Transcranial Magnet in Depression



What is the latest info on transcranial magnet treatment in depression?

Submitted by: **Anonymous**

Transcranial magnetic stimulation (TMS) is a noninvasive method to excite neurons in the brain. The excitation is caused by weak electric currents induced in the tissue by rapidly changing magnetic fields (electromagnetic induction). This way, brain activity can be triggered or modulated without the need for external electrodes as is required in electroconvulsive therapy (ECT). Repetitive transcranial magnetic stimulation is known as rTMS.

Although research in this area is in its infancy, there is now some evidence that TMS is an effective treatment for depression, obsessive-compulsive disorder, generalized anxiety disorder and auditory hallucinations. There is reason to believe that rTMS could replace some ECT treatments currently used for severely depressed patients. In 2002, Health Canada

had approved rTMS therapy for drug-resistant depression.

Generally, TMS appears to be free from harmful effects. Research using animals and human volunteers showed little effect on the body in general as a result of stimulation and examination of brain tissue submitted to thousands of TMS pulses has shown no detectable structural changes. It is possible in unusual circumstances to trigger a seizure in normal patients, but a set of guidelines which virtually eliminate this risk are available. Research continues, but TMS is certainly free of obvious side-effects, particularly when compared to those of ECT.

Answered by: **Dr. Hany Bissada**

4. Remedies for Sore Nipples Due to Breastfeeding



Are there any remedies for sore nipples due to breastfeeding?

Submitted by: **D. Eustace, MD**, Saskatoon, Saskatchewan

Applying modified anhydrous lanolin after nursing (*i.e.*, Lansinoh® or PureLan™) may aid in healing. Surface wetness can contribute to soreness and cracking if the nipple remains moist after nursing—air dry with a hair dryer on low will help. Applying lanolin can help keep the skin soft and pliable, which helps breaks in the skin heal without forming a hard scab that can break open with nursing. Other creams such as Bag Balm and vitamin E should be avoided. Soaps are drying so they

should be avoided and bathing with clear water is all that is needed to keep nipples clean. Other causes should be excluded (*i.e.*, yeast, eczema, impetigo, Staph infections, improper use of breast pumps and allergies). By far the most common cause of nipple soreness is improper latch on and positioning. If there are concerns with the latch a lactation specialist should be consulted.

Answered by: **Dr. Victoria Davis**

5. How to Diagnose Celiac Disease

? How is celiac disease diagnosed? At what age does celiac disease surface and what are the most common signs and symptoms?

Submitted by: [Peter Palma, MD](#), Fort McMurray, Alberta

Celiac disease is a small bowel disorder characterized by mucosal inflammation, villous atrophy and crypt hyperplasia during exposure to gluten and demonstrates improvement with withdrawal of gluten from the diet.

Diagnosis of celiac disease depends on clinical suspicion, serological testing and histopathological features.

The most sensitive and specific tests are IgA anti-tissue transglutaminase (90% sensitive and 97% specific) and IgA endomysial antibody (85% sensitive and 97% specific). These tests should be done while the patient is on a gluten containing diet. If the patient already started on a gluten free diet he needs to go back on the gluten containing diet for at least four to six weeks before the serological titers increase.

Patients with a positive IgA endomysial or transglutaminase antibody tests should undergo a small bowel biopsy.

Endoscopic features suggesting celiac disease include mucosa atrophy with loss of folds, presence of visible fissures and scalloping.

Multiple biopsies should be obtained from the second and third portion of the duodenum. The exact minimal number is uncertain but at least six to eight biopsies are suggested.

Histological features include villous atrophy and lymphocytic infiltration of the lamina propria.

The disease usually presents in late childhood after 10-years-of-age but the disease can be latent and present in adulthood.

Typical symptoms and signs include diarrhea, foul-smelling stool, steatorrhea, weight loss, anemia and osteopenia due to malabsorption. Other atypical symptoms include unexplained fatigue especially with the presence of other autoimmune diseases.

Other signs and symptoms include infertility, arthritis, depression, anxiety, vitamin D and calcium deficiency.

Answered by: [Dr. Robert J. Bailey](#); and [Dr. Akram Aljahdali](#)

6. Bone Mass Protection Regimen



What is the best bone mass protection regimen for patients on long-term oral steroids?

Submitted by: **Charles Lynde, MD**, Markham, Ontario

Glucocorticoid therapy is associated with an appreciable risk of bone loss, which is most pronounced in the first few months of use. In addition, glucocorticoids increase fracture risk and fractures occur at higher BMD values than in post-menopausal osteoporosis.

All patients should receive calcium and vitamin D supplementations of at least 1,000 mg to 1,200 mg and 800 IU respectively, in daily divided doses.

The Osteoporosis Society of Canada (OSC) recommends that all people receiving ≥ 7.5 mg of prednisone (or equivalent) daily for more than three months should be on bone-sparing therapy. Bisphosphonates, preferably alendronate (70 mg per week) or risedronate (35 mg per week) are the first-line bone-sparing agents. IV bisphosphonate, calcitonin or parathyroid

hormone are alternative options if oral bisphosphonate cannot be tolerated or contraindicated. Replacement of testosterone may be indicated in men who develop hypogonadism. The OSC also recommends that those who are receiving > 2.5 mg of prednisone daily for more than three months should be screened for osteoporosis by BMD testing.

Resource

1. Brown JP, Josse RG: Scientific Advisory Council of the Osteoporosis Society of Canada: 2002 Clinical Practice Guidelines For The Diagnosis And Management Of Osteoporosis In Canada. *CMAJ* 2002; 167(10 Suppl):S1-34.

Answered by: **Dr. Michael Starr**; and **Dr. Ahmad Al-Enizi**

7.

Relationship Between CHF and OSA



What is the relationship between congestive heart failure (CHF) and obstructive sleep apnea (OSA)? Should all patients with CHF have a sleep study?

Submitted by: **Gerard Hamilton, MD**, Belleville, Ontario

OSA is a risk factor for increased morbidity and mortality from CV conditions, such as hypertension.¹ Sleep apnea (either central or obstructive) is present in approximately 10% to 40% of patients with CHF.¹ However, it is not practical to do level I sleep studies on all patients with CHF. Rather, patients with CHF should be screened by history for symptoms that suggest superimposed sleep disordered breathing (e.g., snoring, witnessed nocturnal apneas and choking, recurrent awakening and excessive daytime somnolence).² Those patients with a history that suggests sleep disordered breathing could then have a screening

level III ambulatory home sleep study. If respiratory disturbances are detected, a level I polysomnography in a sleep laboratory should be performed to characterize the type (e.g., central vs. OSA) and severity of the sleep disorder.

References

1. Sin DD, Fitzgerald F, Parker JD, et al: Risk Factors For Central And Obstructive Sleep Apnea In 450 Men And Women With Congestive Heart Failure. *Am J Respir Crit Care Med* 1999; 160(4):1101-6.
2. Fleetham J, Ayas N, Bradley D, et al: Canadian Thoracic Society Guidelines: Diagnosis And Treatment Of Sleep Disordered Breathing In Adults. *Can Respir J* 2006; 13(7):387-92.

Answered by: **Dr. Paul Hernandez**

8. Fibromyalgia and Depression



Is fibromyalgia always associated with depression, or is it at least a precursor to depression?

Submitted by: **Raouf Dimitry, MD**, Edmonton, Alberta

Fibromyalgia is a nonspecific disorder characterized by many diffuse complaints including:

- pain,
- stiffness,
- tender muscles and joints,
- overwhelming fatigue,
- distress and
- sleep disturbances.

The etiology and pathogenesis of fibromyalgia are unknown. However, fibromyalgia is currently considered a complete hyperalgesic pain syndrome characterized by reduced pain threshold, an increased response to painful stimuli (hyperalgesia) and an increase in the duration of pain after nociceptor stimulation.

The literature on the relationship between fibromyalgia and psychiatric disorders suggests that patients with fibromyalgia have a higher lifetime prevalence of major depression, panic disorder, obsessive-compulsive disorder and somatization disorder than the general population. However, the literature does not suggest that fibromyalgia is a precursor to depression or to any other psychiatric disorder.

The natural history of the syndrome is not well delineated. Therefore, the course and prognosis are quite variable. Reports range from complete remission to total disability, with no identifiable factors differentiating those who will improve from those who will have a chronic disabling condition. No single medical or psychiatric intervention has been shown to be uniformly effective for fibromyalgia.

The current approach to the patient with fibromyalgia combines supportive counselling, behavioural modification, education, physical conditioning and limited pharmacological interventions. Regular evaluations to identify and address any concomitant aggravating conditions such as sleep disturbance and depression should be performed routinely. Controlled trials of pharmacological interventions have shown beneficial effects with muscle relaxants such as cyclobenzaprine and antidepressant medications, especially tricyclic antidepressants such as amitriptyline and clomipramine.

Answered by: **Dr. Hany Bissada**

9. Excessive Diaphoresis

? What is the best treatment for excessive diaphoresis?

Submitted by: **Wendy Rosenthal, MD**, Mississauga, Ontario

Diaphoresis is excessive sweating commonly associated with shock and other medical emergency conditions.

I interpret this question to ask what is the best treatment for hyperhidrosis, which is excessive sweating and it can be classified as generalized or localized.

For generalized hyperhidrosis, it is necessary to rule out medical conditions such as hyperthyroidism, underlying infections or rarer conditions such as pheochromocytoma. The best treatment for the hyperhidrosis would be treatment of the underlying condition.

For localized hyperhidrosis affecting the axillae, treatment options include potent topical antiperspirants such as 20% aluminum chloride, iontophoresis and Botox® injections. In rare instances, surgical removal of sweat glands or liposuction of sweat glands

could be considered.

For hyperhidrosis of the palms and soles, treatment options include 20% aluminum chloride, iontophoresis, Botox® and sympathectomy which can be done endoscopically.

Oral agents such as glycopyrrolate and propantheline which are anticholinergic agents can be tried but are usually limited by side-effects, especially blurred vision and dry mouth.

Botox® injections have emerged as the best treatment for axillary and palmar hyperhidrosis unresponsive to topical therapy. This treatment is usually effective for six to 12 months but is limited by the need for repeat painful injections, cost and occasional weakness of the muscles of the injected hand.

Answered by: **Dr. Richard Haber**

10. Familial Adenomatous Polyposis

? How many members of a patient's family must have colonic polyps before one would say that there is "familial polyposis?"

Submitted by: **Anonymous**

Familial adenomatous polyposis (FAP) is the most common of the inherited polyposis syndromes consisting of hundreds or thousands of polyps in the colon. The genetic disorder is an autosomal dominant inheritance of the adenomatous polyposis coli (APC) gene. There is an 80% to 100% penetrance and is prevalent in one in 5,000 to 7,500. The patient inherits one mutated APC gene from the affected parent and adenomas develop when the second APC allele from the unaffected parent gets lost or mutated. Adenomas in patients with FAP develop at 10- or 12-years-of-age. Over time, the colon will become studded with polyps

and colon cancer will eventually develop 10 to 15 years after the onset of the first polyp. The diagnosis is made with the confirmation of > 100 polyps in the colon that are adenomatous in nature. In families with a known mutation, screening should begin at 10- to 12-years-of-age and should consist of sigmoidoscopy every year. Genetic testing for at risk individuals is also available.

Resource

1. Feldman M, Friedman LS, Brandt LJ: Sleisenger and Fordtran's Gastrointestinal and Liver Disease. Eighth Edition. Elsevier, 2006. p.2738-44.

Answered by: **Dr. Richmond Sy**

11. Pathogenesis of Syndrome X

? What is the pathogenesis of syndrome X (coronary disease) and how is the management different from classic ischemic heart disease?

Submitted by: **Elijah Musoke, MD**, Melfort, Saskatchewan

Cardiac syndrome X should be distinguished from metabolic syndrome X which consists of abdominal obesity, hypertension, insulin resistance and low HDL-C. Cardiac syndrome X is a syndrome of angina with angiographically normal coronary arteries and no evidence of coronary artery spasm. There may be evidence of ST segment depression on stress testing or a perfusion abnormality on myocardial perfusion imaging. Postulated mechanisms for syndrome X are myocardial ischemia from endothelial dysfunction or increased cardiac sympathetic tone and heightened pain sensitivity. Syndrome X is not the same as non-cardiac chest pain such as esophageal spasm, panic attacks and musculoskeletal chest wall pain.

Typically syndrome X occurs in perimenopausal or post-menopausal women. The chest pain tends to be more prolonged than typical angina and occurs at rest as well

as on exertion. Response to nitroglycerin is variable. The diagnosis of syndrome X is one of exclusion. In general, the patient should have no other cause of chest pain, have an abnormal stress test and normal coronary arteries (on CT angiography or contrast angiography).

Prognosis is excellent. Patients should be reassured that they are unlikely to have a myocardial infarct or die from the chest pain and therapy should focus on symptom relief. A β -blocker is the treatment of choice. Chest pain does not respond predictably to nitroglycerine and calcium channel blockers tend not to be as effective as β -blockers. Some patients respond to imipramine or hormone replacement therapy. I routinely advise these patients to start on an exercise program including walking for 30 minutes daily.

Answered by: **Dr. Bibiana Cujec**

12. Use of Corticosteroids in COPD

? Does the use of a corticosteroid in COPD modify the loss of lung function?

Submitted by: **Len Grbac, MD**, Etobicoke, Ontario

Inhaled corticosteroids (ICSs) have a limited role in COPD. According to the most recent Canadian Thoracic Society COPD guidelines, ICSs are indicated in combination with a long-acting β -agonist for treatment of patients with moderate to severe COPD who experience dyspnea unrelieved by long-acting bronchodilators alone or who experience frequent severe exacerbations of COPD.¹ Four studies have been published regarding the role of

ICSs in slowing disease progression in COPD. There is no evidence that ICSs slow the rate of decline of lung function, specifically the forced expired volume in one second.²⁻⁵

For references, please contact diagnosis@sta.ca

Answered by: **Dr. Paul Hernandez**

13. Treating Paronychial Infections



What is the best way to treat paronychial infections?

Submitted by: **Cecilia Siegling, MD**, Prince George, British Columbia

Paronychia is defined as an inflammation of the proximal nail fold surrounding the nail of a finger or toe. Paronychia can be classified as acute or chronic and the treatment depends on the type you are treating.

The most common precipitating episode in acute paronychia is trauma. This trauma allows secondary infection to occur which is usually *Staphylococcus aureus*. Acute paronychia commonly presents with erythema, edema and marked tenderness over the proximal and/or lateral nail fold. Treatment can be medical or surgical and is determined by the degree of inflammation. If an abscess is present, it should be incised, drained and cultured to determine bacterial sensitivities and rule out methicillin-resistant *Staphylococcus aureus*. Medical treatment includes warm water soaks, especially if there is no abscess to drain as well as antibiotic therapy. Very mild cases can be treated with topical fucidin or mupirocin creams but often oral antibiotics are necessary, usually using an oral anti-staphylococcal agent such as cloxacillin or cephalixin.

The treatment of chronic paronychia is quite different. Chronic paronychia usually presents with erythema and swelling of the proximal nail fold with absence of the cuticle and with or without a nail plate abnormality. It is much less painful than an acute paronychia. Although often felt to be associated with a *Candida albicans* infection, the role of *Candida* in chronic paronychia is controversial as it may or may not be present. Chronic paronychia is

now regarded by nail experts as a variant of a chronic irritant hand dermatitis being seen primarily in patients frequently exposed to wet environments.

Treatment of chronic paronychia involves avoiding wet work as much as possible and treating the underlying inflammation or infection. Often a topical fluorinated corticosteroid used for three to four weeks is effective in treating chronic paronychia.

Prolonged treatment with a potent topical corticosteroid is to be avoided to prevent development of skin atrophy. A topical or systemic anti-*Candida* agent should be tried only if *Candida* is cultured and topical corticosteroids are not effective.

A randomized controlled trial of 45 adults with chronic paronychia treated with either a topical steroid cream, or oral itraconazole or oral terbinafine for three weeks, revealed 85.3% of nails were improved or cured at the nine week follow-up vs. 52.7% with terbinafine and 45.3% with itraconazole. *Candida* eradication was associated with clinical cure in only two or 18 patients who grew *Candida* at the beginning of the study.

Resources

1. Rigopoulos D, Larios G, Gregoriou S, et al: Acute And Chronic Paronychia. *Am Fam Physician* 2008; 77(3):339-46.
2. Tosti A, Piraccini BM, Ghetti E, et al: Topical Steroids Versus Systemic Antifungals In The Treatment Of Chronic Paronychia: An Open, Randomized Double-Blind And Double Dummy Study. *J Am Acad Dermatol* 2002; 47(1):73-6.

Answered by: **Dr. Richard Haber**

Your patients should know
when they are dangerously low.

For your patients with diabetes.

Trademarks are owned by Johnson & Johnson and used under license. ©2009 LifeScan Canada Ltd., Burnaby, BC V5C 6C6 AW 094-207A 05/09



14. New Treatments for Gout



Are there any new treatments for gout on the horizon?

Submitted by: **David Hawkins, MD**, Westbank, British Columbia

Allopurinol is still the current agent of choice for long-term management of gout. However, the FDA recently approved febuxostat, a novel xanthine oxidase inhibitor, at doses of 40 mg and 80 mg, administered orally once daily for the chronic management of hyperuricemia in gout patients.

Uricase (urate oxidase) is an enzyme present in mammals, which catalyzes conversion of urate to a more soluble purine degradation product, allantoin. Polyethylene-glycol (PEG-uricase) is currently being studied as an IV infusion or subcutaneous injections every two to four weeks. In preliminary studies when used in patients failing to respond to standard urate-lowering agents, there was profound and prolonged (several weeks) reduction in serum urate levels and in acute gout attacks. Although results are promising, further studies

are needed to assess the long-term safety.

For the management of acute episodes, in a pilot, open-labelled study, anakinra (IL-1 inhibitor) was given to 10 patients with gout who could not tolerate or had failed standard anti-inflammatory therapies. All patients responded rapidly and no adverse effects were observed. These clinical findings need to be confirmed in a controlled study.

Resources

1. Sundy JS, Ganson NJ, Kelly SJ, et al: Pharmacokinetics and Pharmacodynamics of Intravenous PEGylated Recombinant Mammalian Urate Oxidase In Patients With Refractory Gout. *Arthritis Rheum* 2007; 56(3):1021-8.
2. Ganson NJ, Kelly SJ, Scarlett E, et al: Control Of Hyperuricemia In Subjects With Refractory Gout, And Induction Of Antibody Against Poly(Ethylene Glycol) (PEG), In A Phase I Trial Of Subcutaneous PEGylated Urate Oxidase. *Arthritis Res Ther* 2006; 8(1):R12.
3. So A, De Smedt T, Revaz S, et al: A Pilot Study of IL-1 Inhibition By Anakinra In Acute Gout. *Arthritis Res Ther* 2007; 9(2):R28.

Answered by: **Dr. Michael Starr**; and **Dr. Ahmad Al-Enizi**

15. Recommendations for SBE Prophylaxis



What are the newest recommendations for subacute bacterial endocarditis (SBE) prophylaxis?

Submitted by: **Pierre Juéry, MD**, Ottawa, Ontario

The guidelines for antibiotic prophylaxis have been recently updated and published.¹ They have been simplified based on literature suggesting the risk of developing infective endocarditis (IE) is minimal in certain conditions previously considered high risk for development of IE. Cardiac conditions considered at high risk for the development of IE include: prosthetic heart valve, previous IE, certain forms of congenital heart disease and cardiac transplantation patients who develop valvulopathy. Note that for patients with mitral valve prolapse, prophylaxis for IE is no longer recommended. All dental procedures that manipulate the gingiva and may perforate oral

mucosal surfaces should require antibiotic prophylaxis (including routine teeth cleaning). The usual oral regimen for dental procedures is an antibiotic 30 to 60 minutes prior to the procedure (but up to two hours after the procedure if there was no dose given pre-procedure). The antibiotic chosen should be individualized based on medication history.

Reference

1. Wilson W, Taubert KA, Gewitz M, et al: Prevention of Infective Endocarditis Guidelines From the American Heart Association A Guideline From the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee, Council on Cardiovascular Disease in the Young, and the Council on Clinical Cardiology, Council on Cardiovascular Surgery and Anesthesia, and the Quality of Care and Outcomes Research Interdisciplinary Working Group. *J Am Dent Assoc* 2007; 138(6):739-45,747-60.

Answered by: **Dr. Richard Sheppard**

16. Risk of Recurrence of Depressive Disorder



What is the risk of recurrence of depressive disorder?

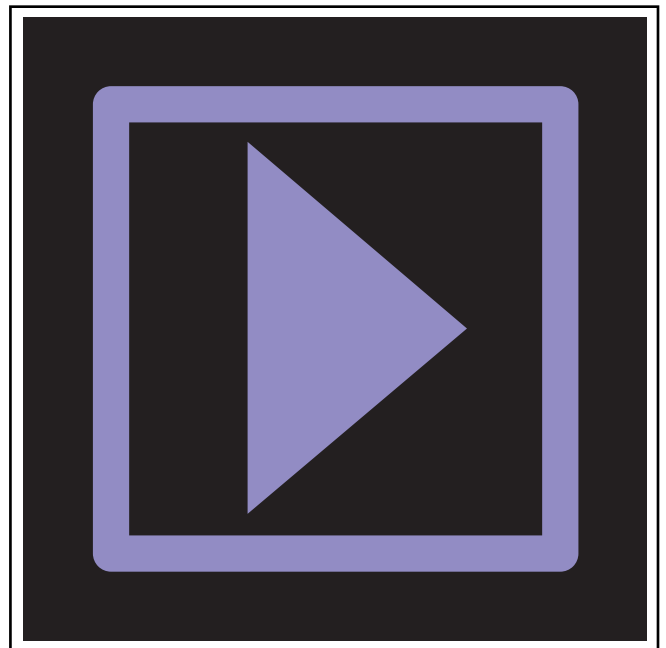
Submitted by: [Sylvie Hudon, MD](#), Ottawa, Ontario

About one-third of all major depressive episodes do not recur. Such patients tend to be older and less likely to have a positive family history for mood disorders. Patients who are experiencing their first episodes of major depressive disorder and are likely to experience recurrent episodes of major depression in the future, tend to be younger, have a positive family history for depression and their first episode of major depression is more likely to have been preceded by a depressive temperament or a dysthymic disorder.

The average length of episodes is six months, whereas the mean interval between episodes tends to vary (typically years). The mean number of recurrent major depressive episodes over a lifetime, according to retrospective and prospective studies, is five to

six. Strong evidence indicates that patients with three or more depressive episodes should receive maintenance medication treatment. Patients with only two major depressive episodes should receive maintenance treatment if there was poor recovery between the two episodes, the two episodes occurred within the last three years, or if there is a positive family history for affective disorders.

Answered by: [Dr. Hany Bissada](#)



17.

Best Treatment for Sinus Pain



What is the best treatment for sinus pain?

Submitted by: [Janna Bentley, MD](#), Kelowna, British Columbia

Sinus pain is caused by irritation of sensory nerves lining the sinuses within the head. There are three pairs of sinuses within the head including the frontal, maxillary and ethmoid sinuses. In addition, there is a midline, sphenoid sinus that is divided into left and right with a bony septum, although this septum is usually asymmetrical. Each sinus is lined with mucosa that can become inflamed by local irritants (allergens) or infected by either viral or bacterial pathogens. Irrespective of the initiating event, the membranes of the sinuses become swollen and congested, resulting in pressure and pain. In addition, the membranes contain mucus glands which over-secrete and result in fluid in the sinuses (leading to pressure) and rhinorrhea. Trapped secretions predispose to further bacterial infections, due to an ideal “culture medium” for bacterial growth.

Treatment depends on the cause, but is generally amenable to OTC medications. Many OTC medications combine analgesics with either decongestants or antihistamines.

In addition, non-medicated nasal saline sprays can provide good relief of symptoms, without any risk of side-effects. If the case is related to allergy, antihistamines will provide good relief (either intranasal or oral medication). Nasal decongestant sprays shrink down oedematous mucosa, clearing the nasal passages, affording relief of pressure. However, they are an easy “drug” to become addicted to, resulting in significantly worse (rebound) congestion (rhinitis medicamentosa). With this in mind, they should only be used for three to five days at a time. Some patients develop a bacterial sinusitis, generally as a result of a preceding viral illness. In this situation, antibiotics may be appropriate.

Answered by: [Dr. Jonathan Irish](#); and [Dr. Emma Barker](#)

18. Vitamin D Recommendations



In light of the new evidence regarding vitamin D, what dose should we be recommending and how about need for exposure to sun?

Submitted by: **Lorna D'Silva, MD**, Oakville, Ontario

Several recent studies have identified a surprisingly high prevalence of vitamin D deficiency in otherwise healthy adults living in Canada and the US.

Although the daily intake of vitamin D required to minimize fracture risk is uncertain, the Osteoporosis Society of Canada (OSC) recommends a daily dose of 400 IU for individuals between 19- to 50-years-old and 800 IU for those > 50-years-old. However, some patients may need higher doses. Doses are also available on a weekly or monthly basis (e.g., 10,000 IU or 50,000 IU) for certain indications.

The intake at which the dose of vitamin D becomes toxic is not clear. The current recommended maximum safe dose is 2,000 IU per day. Newer data, however, indicate that higher doses may be safe, at least over a several month period. In practice, patients who require high doses of vitamin D should have periodic monitoring of serum calcium, parathyroid hormone and 25-hydroxy vitamin D levels to detect those who develop vitamin D toxicity. Although the optimal serum concentration of 25-hydroxy vitamin D has not been firmly established, many agree that 70 nmol/L to 80 nmol/L is ideal.

Regarding sun exposure, casual exposure provides adequate amounts of vitamin D, but is influenced by geographic location, season, use of sun block lotion and skin pigmentation. All these factors beside the potential risk of skin cancer precluded firm recommendations for standard sun exposure dose intervals.

Resources

1. Brown JP, Josse RG: Scientific Advisory Council of the Osteoporosis Society of Canada: 2002 Clinical Practice Guidelines For The Diagnosis And Management Of Osteoporosis In Canada. *CMAJ* 2002; 167(10 Suppl):S1-34.
2. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, FaNBioM Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D and Fluoride. In. Washington, D.C.: National Academy Press; 1997.

Answered by: **Dr. Michael Starr; and Dr. Ahmad Al-Enizi**

19. Diagnosing Rhinorrhea



How can one diagnose rhinorrhea?

Submitted by: **R. McCammon, MD**, Winnipeg, Manitoba

Rhinorrhea is nasal discharge. The mucous membrane lining of the nose contains mucous glands that may over-secrete when stimulated by both allergens and infective agents.

In addition, vasomotor rhinitis is a non-infective, non-allergic condition associated with blood vessels dilatation within the nasal mucous membranes. These vessels are under the control of the autonomic nervous system and in some patients are over sensitive to changes in weather, temperature or chemical irritants. The pathogenesis underlying this cause of rhinorrhea is poorly understood. Rhinorrhea may also be as a result of a breach in the skull base, resulting in cerebrospinal fluid rhinorrhea. There is usually, although not always, a history of trauma for the latter. However, patients complaining of spontaneous, watery, unilateral nasal discharge

should have a sample submitted for glucose detection and β -2 transferrin analysis. Children may present with a malodorous unilateral rhinorrhea. There may be no significant history of the child placing a foreign body in the nose but this should be suspected and if necessary the child may require an examination under anaesthesia. The foreign body initially results in mucosal lining irritation but if left long enough will act as a nidus for infection and unilateral coloured rhinorrhea will result. The diagnosis of rhinorrhea is generally straightforward after a thorough clinical history and examination. The specific cause may rely on tests including glucose and β -2 transferrin, but more usually on a history of either allergy or an upper respiratory tract infection.

Answered by: **Dr. Jonathan Irish; and Dr. Emma Barker**

RELPAX (eletriptan hydrobromide) is indicated for the acute treatment of migraine with or without aura in adults. RELPAX is not intended for the prophylactic therapy of migraine or for use in the management of hemiplegic, ophthalmoplegic or basilar migraine. Safety and effectiveness of RELPAX have not been established for cluster headaches, which is present in an older, predominately male population.

For complete prescribing information, please refer to the Product Monograph. The Product Monograph is available upon request from Pfizer Canada Inc., 17300 Trans-Canada Highway, Kirkland, Quebec H9J 2M5

Reference: RELPAX Product Monograph, Pfizer Canada Inc., March 2006

RELPAX[®] 40 mg
eletriptan HBr



RELPAX[®] Pfizer Products Inc., owner/Pfizer Canada Inc., Licensee
© 2009 Pfizer Canada Inc., Kirkland, Quebec H9J 2M5



20. Malaria Prophylaxis



Is malaria prophylaxis necessary if visiting an endemic area for less than one week?

Submitted by: **R. Martens, MD**, Winnipeg, Manitoba

Yes. While it may be possible to avoid mosquitos by staying in an insect proof area, avoiding the outdoors during the period of the day when mosquitos bite, using physical barriers such as clothing and bed nets to provide bites and chemical barriers such as insect repellants and insecticides, the bite of even one malaria infected mosquito could lead to the development of malaria. Therefore, prophylaxis is always required when travelling to areas endemic for malaria. The chemoprophylaxis should be based upon the antimalarial drug resistance of the malaria parasites in the region to which travellers propose. It is

important to follow the dosing recommendations for the antimalarial therapy to achieve optimal protection. Chemoprophylaxis should be started prior to entering the malarial region, during the period of exposure and for a period after leaving the malarial region. If any questions arise surrounding malaria chemoprophylaxis, a discussion should be undertaken with an expert in travel and/or tropical medicine.

Answered by: **Dr. John Embil**

21. Causes of Fetal Tachycardia in Pregnancy



In a pregnant lady, what are the causes of fetal tachycardia?

Submitted by: **Anonymous**

The definition of fetal tachycardia is a baseline (mean fetal heart rate [FHR]) > 160 bpm. The causes of fetal tachycardia include: maternal fever, fetal infection (group B streptococcus, duration of ruptured membranes), maternal dehydration/ hypovolemia, maternal or fetal anemia, medication or drug response, fetal hypoxia, prematurity (premature babies have immature nervous systems resulting in an increased heart rate), maternal anxiety (maternal adrenaline crosses the placenta, resulting in an increased FHR), fetal cardiac or

congenital anomalies. If persistently severe and fetal well-being cannot be confirmed, delivery should be expedited.

Resource

1. Liston R, Sawchuck D, Young D, et al: Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline. *J Obstet Gynaecol Can* 2007; 29(9 Suppl 4):S3-56.

Answered by: **Dr. Victoria Davis**

22. Endocardial Strep Infection



If a patient is allergic to penicillin, what doses of other classes of antibiotics can be used to prevent endocardial strep infection pre-dental surgery?

Submitted by: **Paul Stephan, MD**, Scarborough, Ontario

The new American Heart Association recommendations for endocarditis prophylaxis target only high-risk patients and those in whom endocarditis would pose a greater than average risk of mortality.

The major change is that only the following patients require endocarditis prophylaxis:

- Prior endocarditis
- Prosthetic valves
- Complex congenital heart disease (cyanotic, incomplete repair or six months following complete repair with device or prosthetic patch)
- Cardiac transplant with valvular disease

The antibiotic of choice is amoxicillin 2 g p.o. 30 to 60 minutes before procedure. If the patient is allergic to penicillin then clindamycin 600 mg or azithromycin 500 mg or clarithromycin 500 mg are alternatives.

Resource

1. Wilson W, Taubert KA, Gewitz M, et al: Prevention of Infective Endocarditis: A Guideline From the American Heart Association Rheumatic Fever, Endocarditis, and Kawasaki Disease Committee. *Circulation* 2007; 116(15):1736-54.

Answered by: **Dr. Bibiana Cujec**

23. Tinea Pedis Between Toes



How do you treat tinea pedis between toes which has not responded to treatment with topical ciclopirox (twice daily) for a long period of time?

Submitted by: **France De Carufel, MD**, Laval, Quebec

If you are suspecting tinea pedis, consider performing a skin scraping (and nail clipping if the nails are involved) and sending for fungal culture. This can help determine if this is in fact a dermatophyte infection or if it is a yeast or other non-dermatophyte.

As a general rule, azoles (e.g., ketoconazole) work best for yeasts and terbinafine is best for dermatophytes. Topical ciclopirox can be effective against yeasts and dermatophytes.

Consider trying topical terbinafine applied twice daily for one to two weeks. If this is not effective, oral antifungals can be very effective. It is prudent to obtain baseline complete blood count and liver function tests prior to starting an oral agent. Treatment regimens for adults with tinea pedis include terbinafine 250 mg p.o. daily for two weeks or itraconazole 200 mg p.o. b.i.d. for one week.

Answered by: **Dr. John Kraft; and Dr. Charles Lynde**


24. Success Rate of Venom Immunotherapy With Allergy Injections



What is the success rate of (venom) immunotherapy with allergy injections? How long should one continue with allergy injections?

Submitted by: **Roshan Dheda, MD**, Bradford, Ontario

Patients with a history of a systemic reaction following a venom sting and in whom an IgE-mediated mechanism is identified (through intradermal testing and radioallergosorbent testing), are at higher risk for a future systemic reaction with a future sting (60% to 70%) and at the very least, they require an autoinjector. These patients are candidates for venom immunotherapy, which can reduce the likelihood of a systemic reaction to < 5%. Thus, this form of therapy is highly effective and strongly indicated in these patients. Following a “build-up” protocol in which injections are given usually on a weekly basis, monthly injections are given on average for about three years (frequency can sometimes be reduced to every two months during the final year). Protection from severe reactions following future stings continues for many years following immunotherapy. Patients with a history of severe anaphylaxis (*e.g.*, intubation) and those with severe forms of honeybee allergy, may require a more prolonged course of

immunotherapy. Any patient who has developed more than a local reaction following a venom sting should be referred for an allergy assessment, as immunotherapy in these patients can be life-saving. 

Answered by: **Dr. Tom Gerstner**