



*Answers to your questions
from our medical experts*

1. Whispering During Laryngitis



Is it really true that whispering is as bad as trying to talk when one has laryngitis?

Submitted by: [Esther Libman, MD](#), Thornhill, Ontario

Laryngitis (inflammation of the laryngeal mucosa) is common in adults and can be devastating to a professional vocalist. Inflammation of the vocal folds leads to irritation and edema of the vocal cords. This inflammation hinders the pliable motion of the mucosa overlying the vocal ligament, leading to dysphonia (difficulty phonating).

Phonation is a complex and multi-step process. Generally, phonation requires adequate breath support to provide power. The sound of the voice is provided by apposition of the vibratory mucosal edges of the true vocal cords separated by an appropriately small gap. The produced sound is further modulated by resonance of the upper airway and articulation in the oral cavity.

During whispering, the true vocal cords are kept open to prevent vocal fold vibration

and the supraglottal structures (false vocal cords) are constricted. Though the vocal cords do not vibrate during a soft whisper, the effort required to make a whisper loud enough to be heard may still damage the vocal cords. Loud whispering is not the normal use of the voice.

Therefore, whispering during an episode of laryngitis forces the swollen vocal cords nearly together without letting them meet, requiring even more effort than normal speech.

Laryngitis will usually resolve spontaneously. The best treatment is silence. If necessary, one can speak but should not whisper in order to avoid further strain on the larynx.

Answered by: [Dr. Gideon Bachar](#); [Dr. Vitaly Kisilevsky](#); and [Dr. Jonathan Irish](#)

2. Schizophrenia and Diabetes



How often (and beginning at what age) would you recommend screening a patient with schizophrenia for diabetes?

Submitted by: [Ben Addleman, MD](#), Calgary, Alberta

Schizophrenia is a risk factor for Type 2 diabetes. A fasting plasma glucose (FPG) would be the recommended initial screening test. If the FPG is > 7.0 mmol/L, then this individual has diabetes (confirmed at least on one other occasion). If the FPG is between 5.6 mmol/L and 6.9 mmol/L, it is recommended that a 75 g oral glucose tolerance test be performed.

If the FPG is < 5.6 mmol/L, then an annual FPG can be performed.

Screening in patients without risk factors begins at age 40 and it is recommended that earlier screening be done with risk factors.

Answered by: [Dr. Vincent Woo](#)

3. Understanding Halitosis



What is the diagnosis, prognosis and treatment of halitosis?

Submitted by: **Claude Roberge, MD**, Sherbrooke, Quebec

Halitosis or bad breath is the exhalation of unpleasant odors from the mouth. It often originates from the mouth, but can also represent:

- gum disease,
- post nasal drip,
- nasal infection,
- tonsillar infection, or
- food.

Proteins trapped in the mouth are broken down to amino acids by oral bacteria usually found on the tongue. Other areas in the mouth that can cause halitosis are:

- the food particles trapped between teeth,
- faulty dental work, or
- unclean dentures.

Foul breath can also originate from disease states, such as:

- liver failure (fetor hepaticus),
- lung abscess or infections,
- renal failure and
- diabetes (ketoacidosis).

Care must be taken to exclude the benign nature of halitosis from these systemic illnesses.

The treatment of halitosis is to maintain proper oral hygiene. Brushing and flossing your teeth are essential. Cleaning the back of your tongue with a toothbrush or tongue scraper will help eliminate any remaining food debris. Maintaining clean dentures and regular visits to the dentist will help achieve good hygiene. Drinking plenty of water to prevent dryness of the mouth and stasis of saliva will also help prevent halitosis.

Answered by: **Dr. Richmond Sy**

Foul breath can also originate from disease states, such as liver failure (fetor hepaticus), lung abscess or infections, renal failure and diabetes (ketoacidosis).

4. Do Antidepressants Cause Vivid Dreams?



Many patients complain of vivid dreams while taking SSRIs or SNRIs. What causes this?

Submitted by: **Janice Van Kampen, MD**, Toronto, Ontario

Vivid dreams are occasionally reported by patients taking selective serotonin reuptake inhibitors (SSRIs) or selective norepinephrine reuptake inhibitors (SNRIs). It is usually dose-related and it is believed to be related to the increased serotonin activity caused by the medication. However, its exact mechanism remains unknown. If the vivid dreams cause a serious discomfort to the patient,

then dose reduction may help to improve the problem, otherwise switching to a different class of antidepressant, such as mirtazapine, may be tried.

Answered by: **Dr. Hany Bissada**

5. Decreasing Microalbuminuria Levels



Other than controlling BP, what can be done to decrease an increased level of microalbuminuria?

Submitted by: **Larry Bobyn**, Kelowna, British Columbia

Two things to remember in the setting of persistent microalbuminuria are that:

- its presence increases the risk of progression of renal disease in settings, such as diabetes and
- there is a growing body of evidence showing a relationship between microalbuminuria and CVD.¹

However, a correlation does not equal causation and while therapy targeting microalbuminuria in diabetes has proven to be effective in retarding the progression to macroalbuminuria and worsening renal dysfunction, we do not yet know whether mortality related to CVD is similarly reduced as a direct consequence of albuminuria reduction and control.

To answer the question, unlike the non-renin-angiotensin-aldosterone system antihypertensives, the addition of an ACE inhibitor (or ARB) will provide the benefit of reducing microalbuminuria beyond the effect that is obtained from the BP lowering contribution alone. Quitting smoking, optimization of glycemic control and lowering lipid levels have all been associated with reductions in microalbuminuria as well.

Reference

1. Schmieider RE, Schrader J, Zidek W, et al: Low-Grade Albuminuria and Cardiovascular Risk: What is the Evidence? *Clin Res Cardiol* 2007; 96(5):247-57.

Answered by: **Dr. Chris Sathianathan; and Dr. Manish M. Sood**

6. β -Blockers for Heart Failure



Is there a preferred β -blocker for heart failure (e.g., bisoprolol) and why?

Submitted by: **David Wiseman, MD**, Hornby Island, British Columbia

There are only three β -blockers that have been used in large clinical trials of heart failure with documented beneficial effects on survival (Table 1). These are:

- Carvedilol (The US Carvedilol Heart Failure Study Group)
- Metoprolol (Metoprolol CR/XL Randomized Intervention Trial in Congestive Heart Failure [MERIT-HF])
- Bisoprolol (Cardiac Insufficiency Bisoprolol [CIBIS] studies)

In addition to its non-selective β -blocker effect, carvedilol also has α -blocking and antioxidant effects.

Metoprolol is a β_1 -selective blocker (and does not have a large effect on the airway).

Bisoprolol is even more highly selective for β_1 -receptors (and is preferred in patients with chronic obstructive pulmonary disease who have some airway reactivity).

Patients with left ventricular (LV) ejection fractions < 40%, either secondary to coronary artery disease or nonischemic etiologies, have a significant benefit from β -blockers, both in terms of:

- reverse LV remodelling (*i.e.*, smaller LV with an average of 8% points

- improvement in LV ejection fraction),
- improved exercise performance and
- improved survival.

Only carvedilol, metoprolol and bisoprolol have documented beneficial effects on heart failure survival.

Some patients will even have a normalization of their LV dysfunction after a β -blocker is added to an ACE inhibitor. It is important to adjust the dose of the β -blocker to achieve a resting heart rate of < 65 bpm, avoiding symptomatic hypotension and significant side-effects (mainly fatigue).

Answered by: **Dr. Bibiana Cujec**

Table 1

β -blockers used in heart failure

β -blocker	Starting dose	Target dose
Carvedilol	3.125 mg b.i.d.	25 mg b.i.d.
Metoprolol	12.5 mg b.i.d.	100 mg b.i.d.
Bisoprolol	2.5 mg q.d.	10 mg q.d.

7. Hair Dye Use During Pregnancy



Can you comment on hair dye or bleaching during pregnancy?

Submitted by: Adam Kayumi, MD, Mississauga, Ontario

Most studies, though limited, indicate that it is safe to colour hair during pregnancy. A very small amount of dye is absorbed into the body; however, it is unclear how much, if any, reaches the fetus. Experts say that neither permanent nor semi-permanent hair dyes are highly toxic and exposure is extremely low. Many women decide to wait to dye their hair until after 12 weeks gestation, when the risk of chemical substances harming the fetus is much lower. The risk can be reduced further by making sure that:

- gloves are worn (when self-applied),
- the dye is left on for the minimum amount of time and
- one works in a well ventilated room.

Highlighting hair with bleach also reduces the risk as the chemicals used are only absorbed by the hair.

Pregnancy can affect the normal condition of hair and it may react differently to dyes; therefore, a test strip is recommended.

Answered by: Dr. Victoria Davis

8.

High Folic Acid Intake and Bowel Cancer



Does high folic acid intake predispose one to bowel cancer?

Submitted by: E. J. Franczak, MD, Scarborough, Ontario

Until recently, epidemiologic data had suggested that a diet low in folic acid was associated with a higher risk of colorectal cancer. However, the findings of a recent prospective randomized trial of folic acid 1 mg q.d. vs. placebo in > 1,000 subjects, with a prior history of colorectal adenomas, failed to confirm this. In fact, folic acid use was associated with a trend towards a higher risk of colorectal adenomas although no increase in invasive cancers was observed.

While this single study does not definitively prove that folic acid intake is associated with a higher risk of bowel cancer, it does demonstrate that folic acid supplementation should not be recommended for colorectal cancer chemoprophylaxis.

Resource

1. Cole BF, Baron JA, Sandler RS, et al: Folic Acid for the Prevention of Colorectal Adenomas. JAMA 2007; 297(21):2408-9.

Answered by: Dr. Sharlene Gill

9. Prevalence of Lyme Disease in Canada



What is the prevalence of Lyme disease in Canada?

Submitted by: **P. E. Frangon, MD**, Calgary, Alberta

Lyme disease is a multisystem illness caused by the spirochete *Borrelia burgdorferi* which can be spread through the bite of specific ticks. Lyme disease is not a nationally reportable disease in Canada. The Public Health Agency of Canada (PHAC) conducts surveys of the provinces and territories to determine the number of cases in distribution of Lyme disease in the country. This approach may underestimate the actual number of cases. The risk for exposure is highest where specific ticks may be found, specifically in southern and eastern Ontario, southeastern Manitoba and Nova Scotia, as well as much of southern British Columbia.

If a potential diagnosis of Lyme disease is being considered, the PHAC recommends that any blood test be interpreted in terms of the clinical context, specifically, the presence (or absence) of the characteristic rash, erythema, chronicum migrans. The PHAC also cautions against the use of serologic tests that have not been validated.

Resources

1. Public Health Agency of Canada: Lyme Disease Fact Sheet. 2006. http://www.phac-aspc.gc.ca/id-mi/pdf/lyme-fs_e.pdf.
2. Ogden NH, Lindsay LR, Morshed M, et al: The Rising Challenge of Lyme Borreliosis in Canada. *Can Commun Dis Rep* 2008; 34(1):1-19.

Answered by: **Dr. John M. Embil**

10. Following-Up on Mildly Elevated PSA



How often should you check the PSA in a 60-year-old male who has had mildly elevated, unstable PSA for two years? (Biopsy negative for prostate cancer).

Submitted by: **Katherine Allen, MD**, Belleville, Ontario

There is no consensus on how often a PSA test should be done in these circumstances but two times a year can be recommended and sometimes more if the degree of suspicion is high. Patients should be made aware that sexual intercourse can elevate PSA, so they should avoid sexual activity for four days before PSA testing.

If PSA velocity is > 0.75 ng/ml per year, then a second biopsy should be discussed.

Another option can be the use of finasteride

for a period of six months. This should lower PSA by half if the elevated PSA is caused by benign prostatic hyperplasia. A decrease to less than half of initial PSA should alert the physician to the possibility of prostate cancer.

Answered by: **Dr. Hugues Widmer**

11. Treating Cradle Cap



What is the latest prescription for cradle cap? Is treatment with antifungals of any use?

Submitted by: **J. V. Patidar, MD**, Edmonton, Alberta

Cradle cap refers to infantile seborrheic dermatitis of the scalp. It starts one week after birth and lasts for months. Greasy scales encapsulate the entire scalp, with inflammation and oozing.

Treatment involves reassuring parents that this condition is self-resolving and that it is not associated with any increased risk of seborrheic dermatitis as an adult. They should avoid picking at the scales.

Affected infants should be bathed daily with mild shampoos. Harsh keratolytics should be avoided. Moisturize the scalp daily. Scales can be gently removed with daily use of 3% to 5% salicylic acid in olive

oil or a water base or with warm olive oil compresses alone.

Treatment with antifungals is reserved for more severe or persistent cases. As in adults, infantile seborrheic dermatitis is due to the proliferation of a commensal lipophilic yeast, *Malassezia sp.* Therefore, topical azoles (e.g., ketoconazole 2% cream or shampoo) can be used for challenging cases. Low potency topical corticosteroids (e.g., 1% hydrocortisone cream) can be added for several days to treat inflammation.

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

12. Lubricating Gel and Pap Smears



Does the use of lubricating gel affect the interpretation of Pap smears? Should we be using lubricating gel in Pap tests?

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

The use of lubricating gel makes the Pap smear less uncomfortable for the patient. In infertility, lubricating gel has been recognized as spermicidal so that an effect on Pap smears may be anticipated.

Evidence shows that if used appropriately, vaginal speculum lubrication does not impact on the quality of either standard cervical cytology or the newer liquid-based Pap testing.

Resources

1. Griffith WF, Stuart GS, Gluck KL, et al: Vaginal Speculum Lubrication and its Effects on Cervical Cytology and Microbiology. *Contraception* 2005; 72(1):60-4.
2. Hathaway JK, Pathak PK, Maney R: Is Liquid-Based Pap Testing Affected by Water-Based Lubricant? *Obstet Gynecol* 2006; 107(1):66-70.

Answered by: **Dr. David Cumming**

13. Is Strontium Better than a Bisphosphonate?



In your opinion, will strontium prove to be more efficacious than bisphosphonates in treating osteopenia/osteoporosis?

Submitted by: **V. Moody, MD**, Mississauga, Ontario

Strontium has been shown to increase bone formation and inhibit bone resorption in animal studies. In humans, there are three main clinical trials of strontium in osteoporosis (OP).

The first was a two-year clinical trial of 353 post-menopausal women with OP and at least one vertebral fracture who were randomly assigned to receive strontium or placebo. Bone density increased and the risk of vertebral fracture decreased in a dose-dependant manner at two years time.¹

In a second larger trial, the same investigators randomized 1,649 post-menopausal women with OP and at least one vertebral fracture to receive strontium or placebo for three years. The risk of new vertebral fractures was decreased significantly by 41% and BMD increased by 8%.²

The largest trial was one of 5,091 post-menopausal women with OP randomly assigned to receive strontium (2 g q.d.) or placebo. In this trial, the relative risk of non-vertebral fracture was decreased by 16% in the strontium group compared to placebo. In high-risk patients with T scores < -3 at baseline, there was an even greater reduction in the risk of vertebral and hip fracture at 39% and 36%, respectively, with strontium compared to placebo.

There is no data comparing strontium head-to-head with bisphosphonates and its side-effect profile has not sufficiently been established. Although it was well tolerated in clinical trials, its use has been associated with an increased annual incidence of venous thromboembolism. In addition, although the data for vertebral fracture reduction is good, strontium has not yet been shown to reduce hip fractures in post-menopausal women with OP.

With regards to the preferential use of strontium over bisphosphonates, more studies are needed comparing both drugs in terms of efficacy and side-effects.

References

1. Meunier PJ, Slosman DO, Delmas PD, et al: Strontium Ranelate: Dose-Dependent Effects in Established Post-Menopausal Vertebral Osteoporosis: A 2-Year Randomized Placebo Controlled Trial. *J Clin Endocrinol Metab* 2002; 87(5):2060-6.
2. Meunier PJ, Roux C, Seeman E, et al: The Effects of Strontium Ranelate on the Risk of Vertebral Fracture in Women with Post-Menopausal Osteoporosis. *N Engl J Med* 2004; 350(5):459-68.

Resource

1. Reginster JY, Seeman E, De Vernejoul MC, et al: Strontium Ranelate Reduces the Risk of Nonvertebral Fractures in Post-Menopausal Women with Osteoporosis: Treatment of Peripheral Osteoporosis (TROPOS) Study. *J Clin Endocrinol Metab* 2005; 90(5):2816-22.

Answered by: **Dr. Sabrina Fallavollita**; and **Dr. Michael Starr**



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The most commonly reported vaccine-related injection-site adverse experiences in clinical trials with GARDASIL® in females (n=5,088), aluminum-containing placebo (n=3,470) and saline placebo (n=320), respectively, were pain (83.9%, 75.4%, 48.6%), swelling (25.4%, 15.8%, 7.3%), erythema (24.6%, 18.4%, 12.1%) and pruritus (3.1%, 2.8%, 0.6%). The most commonly reported vaccine-related systemic adverse experience in females was fever: 10.3% for GARDASIL® (n=5,088) vs 8.6% for aluminum and non-aluminum containing placebo (n=3,790).

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Experts on Call

14. Using Statins in Adolescents



Is it safe to use statins in a 15-year-old male patient recently diagnosed with diabetes, hypertension, elevated cholesterol and fatty liver on abdominal ultrasound?

Submitted by: **Sameera Benjamen, MD,**
Stellarton, Nova Scotia

The long-term safety and efficacy of statins in children and adolescents has not generally been established for CVD prevention. In a small study of 140 patients aged 10 to 17 years with heterozygous familial hypercholesterolemia, treatment with atorvastatin at a dose of up to 20 mg showed safety and tolerability comparable to placebo over a six month follow-up period. Statins have not been studied in pre-pubertal children and adolescent females taking statins require effective contraception to avoid potential fetal teratogenic effects.

In light of the limited data available, a 15-year-old with multiple CV risk factors, including diabetes, hypertension and dyslipidemia initially requires aggressive counselling regarding lifestyle measures to optimize body weight, reduce abdominal obesity and increase physical activity. Tobacco use is, of course, contraindicated. If BP remains elevated despite these measures, antihypertensive drug therapy should be considered. The use of dyslipidemic drug therapy, including statins, should be reserved for selected cases with severe dyslipidemia and a family history of premature coronary artery disease in first-degree relatives, generally < 50-years-of-age.

Answered by: **Dr. George N. Honos**

15. Can Traumatic Experiences be Erased from Memory?



Can traumatic experiences be erased from affective memory with β -blocker drugs in inpatients with post-traumatic stress disorder?

Submitted by: **Tim Seipp, MD**, Penticton, British Columbia

No, traumatic experiences cannot be erased from affective memory with β -blockers. However, β -blockers (e.g., propranolol) are sometimes used in post-traumatic stress disorders to reduce the increased physiological arousal (caused by flashbacks of traumatic experiences) manifested by:

- sleep disturbance,
- irritability,
- poor concentration,
- hyper-vigilance and
- increased startle.

Answered by: **Dr. Hany Bissada**

...the diseases they cause:

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See prescribing summary and study parameters on page 144

16. Risk of Prednisone Use for Poison Ivy



Does risk/benefit justify the use of prednisone for poison ivy?

Submitted by: **Bill Beck, MD**, Kingston, Ontario

Poison ivy is a member of the plant genus *Toxicodendron* which includes poison oak and sumac. Poison ivy dermatitis is an allergic contact dermatitis to urushiol, an oleoresin found on the poison ivy plant.

The best treatment for poison ivy is recognition of the plant and avoidance.

Localized poison ivy can be treated with potent topical steroids such as betamethasone 17-valerate 0.1% cream or even clobetasol propionate 0.05% cream twice daily. As well, sedating antihistamines, such as diphenhydramine or hydroxyzine, can be used for the symptomatic relief of pruritus.

The risk of avascular necrosis with short courses of oral corticosteroids is extremely small but has been reported and patients need to be warned about this rare but potentially serious side-effect.

However, when poison ivy dermatitis is widespread or involves large areas of the face or around the eyes, systemic corticosteroids are often necessary to treat the dermatitis and control the severe pruritus.

As well, poison ivy dermatitis often causes an id (autoeczematization) reaction with new dermatitic areas developing at distant sites from the initial areas of contact sensitization. In this scenario, systemic corticosteroids are necessary to treat the severe dermatitis and to prevent development of new distant dermatitic lesions.

As in all cases, the risk vs. the benefits of using systemic corticosteroids must be individually evaluated for each patient. However, the treatment is usually of short duration (two to three weeks) and the acute side-effects of the steroids (*i.e.*, hyperglycemia, hypertension, electrolyte imbalance) can be monitored. The risk of avascular necrosis with short courses of oral corticosteroids is extremely small but has been reported and patients need to be warned about this rare but potentially serious side-effect.

If there are no significant contraindications to oral steroids and the patient has a severe allergic contact dermatitis, I would treat with 0.5 mg/kg to 1.0 mg/kg p.o. prednisone in the morning for one week and taper it down to zero over the next two weeks. If too short a course is given, the patient may have a rebound worsening of the dermatitis after stopping the drug prematurely. Therefore, commercially-available dose packs should be avoided.

Answered by: **Dr. Richard Haber**

17. Are Flu Shots Safe During Pregnancy?



Is it recommended to give a flu shot during pregnancy?

Submitted by: **S. Abouna, MD**, Mississauga, Ontario

The Centre for Disease Control and the Canadian equivalent recommend the influenza vaccine for pregnant women due to their increased risk of influenza infection and its complications. No study has demonstrated an increased risk of maternal complications or adverse fetal outcomes associated with inactivated influenza vaccine.

Resource

1. Naleway AL, Smith WJ, Mullooly JP: Delivering Influenza Vaccine to Pregnant Women. *Epidemiol Rev* 2006; 28:47-53.

Answered by: **Dr. Victoria Davis**

18. Evaluating Elevated Creatinine



A healthy male, age 50, has a creatinine of 150 μ moles/L and is on no medications. He is not hypertensive and all investigations are normal. Are there any further investigations/treatments you would recommend?

Submitted by: **Ewen Mackenzie, MD**, Kingston, Ontario

The evaluation of an elevated creatinine begins with determining whether it represents an acute or chronic process. Identifying and treating a reversible cause, such as illness, volume depletion and medication(s), is the next step. For non-readily identifiable causes, the anatomical approach to acute renal failure is most useful (pre-renal, renal and post-renal). After a history/physical, the work-up should commence with a urinalysis and renal ultrasound. The need to perform a urine protein/creatinine ratio should be guided by results of the urinalysis. If a chronic process is suspected, the creatinine clearance should be determined using the estimated glomerular filtration rate (eGFR). Chronic kidney disease (CKD) is confirmed with serial testing and is defined as a GFR < 60 ml/minute/1.73m² three months apart.

The primary care physician can manage most patients with non-progressive CKD. The goal is to slow the progression of renal disease through BP (target $< 130/80$ mmHg) and proteinuria control (target urine protein/creatinine < 60 mg/mmol using an ACE inhibitor or ARB). Consider referral to a nephrologist if there is acute renal failure, an eGFR < 30 ml/minute/1.73m², the urine protein/creatinine ratio is > 100 mg/mmol, or if there is progressive loss of renal function.

Resource

1. Canadian Society of Nephrology Guidelines: Care and Referral of Adult Patients with Reduced Renal Function. Recommendations from the Canadian Society of Nephrology. <http://csnscn.ca/local/files/guidelines/implementationcommittee/CKDReferralSummary2007.doc>

Answered by: **Dr. Chris Sathianathan; and Dr. Manish M. Sood**

19. Looking at Multiple Endocrine Neoplasia



Multiple endocrine adenomas/neoplasia is a rare phenomena. Can you go over generalities, treatment and prognosis? Are there partial forms?

Submitted by: [John Sader, MD](#), Mount-Royal, Quebec

Multiple endocrine neoplasia (MEN) subclassified as MEN Type 1 (MEN1) and MEN Type 2 (MEN2), is an autosomal dominant condition. Type 1 is characterized by tumours of the parathyroid glands (near 100% penetrance), anterior pituitary and pancreatic islet cells. While hyperparathyroidism and pituitary tumours can be effectively treated, the malignant sequelae of pancreatic neuroendocrine tumours can be life-threatening and impact prognosis. The consensus definition of MEN1 is the presence of two of the three MEN1 tumours. Familial MEN1 is defined as an index MEN1 case with at least one relative who has one of the three main MEN1 tumours. MEN1 is largely a clinical diagnosis; the role of genetic testing is unclear.

MEN2 includes medullary thyroid cancers (near 100% penetrance), pheochromocytomas and primary parathyroid hyperplasia (2A) or mucosal neuromas (2B).

For MEN2, early genetic testing in suspected kindreds should be considered.

While there are no defined partial forms of MEN, the penetrance of the characteristic neoplasms within is variable. Treatment and prognosis are dictated by the neoplastic manifestations of MEN in a given individual.

Answered by: [Dr. Sharlene Gill](#)

20. Penile Engorgement: A Normal Physiologic Reaction?



A three-year-old boy develops penile engorgement when straining with a bowel movement. Is this a normal physiological reaction? What is the mechanism of this reaction?

Submitted by: [D. Petrovic, MD](#), Burnaby, British Columbia

Penile engorgement does not correspond to a known physiologic reaction. The clinician should make sure the patient does not have a phimosis causing enlargement when the patient urinates.

Penile engorgement does not correspond to a known physiologic reaction.

Answered by: [Dr. Hugues Widmer](#)

21. Drugs to Relieve Fibromyalgia Pain



Besides increased physical activity, what are the drugs of choice for relieving pain in fibromyalgia?

Submitted by: [Renée Kuska, MD](#), Sillery, Quebec

Patient education is a key component in managing fibromyalgia. Reassurance, lifestyle modification and addressing psychosocial factors are all important. In this light, increased physical activity is one of the best measures for these patients.

With regards to pharmacologic management of fibromyalgia, many medications have been tried with variable success.

Tricyclic antidepressants, such as amitriptyline at doses of 10 mg to 50 mg q.h.s. may be effective, particularly for sleep modulation. Although these doses are usually lower than those required to treat depression, this group of patients often experience significant side-effects, such as:

- dry mouth,
- constipation,
- fluid retention,
- weight gain and
- somnolence.

Muscle relaxants, such as cyclobenzaprine used at night, may give similar benefit as the tricyclics. Other antidepressants in the selective serotonin reuptake inhibitor family have also been effective and often better tolerated than amitriptyline and may have a pain relieving effect independent of mood alteration.

More recently, gabapentin and pregabalin have been shown to decrease pain scores in fibromyalgia. Doses required are often elevated and the dose needs to be titrated upwards as tolerated. Pregabalin has been

shown to:

- decrease pain,
- improve sleep and
- decrease fatigue.

Doses used tend to be between 75 mg to 300 mg q.d. Also, there is some preliminary data suggesting benefit with oral cannabinoids.

With regards to pharmacologic management of fibromyalgia, many medications have been tried with variable success.

It should be noted that traditional pain relieving medications like NSAIDs and narcotics are generally not helpful and should be avoided if possible.

Answered by: [Dr. Sabrina Fallavollita](#); and [Dr. Michael Starr](#)

22. Canadian Data on Anaphylaxis



Is there any Canadian data on anaphylaxis affecting children and adults?

Submitted by: **Michael Keating, MD**, Saint John, New Brunswick

The true incidence of anaphylaxis is unknown, partly because of the lack of a precise definition of the syndrome. Some clinicians reserve the term for the full-blown syndrome, while others use it to describe milder cases. The epidemiology of anaphylaxis has been described in very few published reports. There are about 100,000 episodes each year in the US, of which two-thirds are new cases and almost 1% are fatal. Some have estimated that 10% to 20% of the US population is at risk for anaphylaxis by virtue of past history or existing sensitization.¹ An anaphylaxis working group in the US estimated the frequency of anaphylaxis at approximately 50 to 2,000 episodes per 100,000 persons (lifetime prevalence 2%).²

In the UK, Pumphrey collected data on fatal and non-fatal anaphylaxis from 1994 to 2004 and found about 20 deaths identified each year (one each year for each three million population, about the same as reported in the US above).³

In Canada, we have no direct figures on the incidence of anaphylaxis, but likely ours is similar to the US. In 2000, the Canadian Pediatric Surveillance Program set about to better define the picture of anaphylaxis in Canadian infants, children and teens.⁴ The survey took place from January 2000 until June 2001, with 130 Canadian pediatricians participating. More than 700 episodes of anaphylaxis involving patients aged one month to 17 years were reported (including one fatality in a food-allergic teen). Results showed that 60% of all anaphylaxis

episodes occurred in males; 60% of all episodes occurred in children less than six-years-of-age; 64% of all episodes occurred in the child's home and a parent was present during 75% of all episodes; 31% of episodes occurred following a child's first known exposure to the trigger; 25% of episodes occurred in children who had a history of a previous reaction to the trigger; and 81% of all episodes were triggered by foods, most commonly peanuts, tree nuts, cow's milk, eggs, fish/shellfish and fruits/vegetables. Non-food triggers, such as medications (8%), insect stings (4%), latex rubber (2%), exercise (2%), immunotherapy (2%) and other (1%) were also reported.

Despite the large number of anaphylaxis episodes reported, the study likely underestimated the true occurrence rate of anaphylaxis in the pediatric population.

References

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3. Pumphrey R: Anaphylaxis: Can We Tell Who is at Risk of a Fatal Reaction? *Curr Opin Allergy Clin Immunol* 2004; 4(4):285-90.
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Answered by: **Dr. Tom Gerstner**

23. Diagnosing the Metabolic Syndrome



How to diagnose Metabolic syndrome?

Submitted by: **Daniel Solonyna, MD**, Pointe-Claire, Quebec

The Metabolic syndrome is a multifaceted condition characterized by:

- abdominal obesity,
- hypertension,
- lipid abnormalities,
- insulin resistance and
- glucose elevations.

There are multiple definitions of the Metabolic syndrome,¹⁻⁴ each with their own proponents. There is a lack of consensus regarding the most appropriate definition. There are also groups that believe that the Metabolic syndrome is not a useful concept.

References

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2. Grundy SM, Cleeman JI, Daniels SR, et al: Diagnosis and Management of the Metabolic Syndrome: An American Heart Association/National Heart, Lung and Blood Institute Scientific Statement. *Circulation* 2005;112(17):2735-52.
3. Expert Panel on Detection, Evaluation and Treatment of High Blood Cholesterol in Adults: Executive Summary of The Third Report of The National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* 2001; 285(19):2486-97.
4. International Diabetes Federation. The IDF Consensus Worldwide Definition of the Metabolic Syndrome. 2005. Available at: www.idf.org.

Answered by: **Dr. Vincent Woo**

24. Routine Specimen Collection for STIs



Should routine STI screening include serum antibody testing for herpes simplex virus Type 1 (HSV-1) and Type 2 (HSV-2), as part of the routine specimen collection?

Submitted by: **Fredericka Abcarius, MD**, Montreal, Quebec

Ultimately, it may become part of the routine battery of investigations. As a matter of clarification, the antibody response to primary infection is characterized by the appearance of IgM, followed by the appearance of IgG with a waning of the IgM antibody within several months of infection. Therefore, the presence of IgM antibody to HSV-2 is an indirect indication of "recent" infection. Most individuals will seroconvert within three to six weeks.

Once these investigations become standard practice, performing acute and convalescent-type specific serology will

allow practitioners to establish a diagnosis of primary infection and determine whether the infection is due to HSV-1 or -2. At this time, type-specific HSV antibody assays are only available in a few Canadian laboratories.

To determine whether the service is offered in your jurisdiction, contact your local provincial laboratory.

Resource

1. The Public Health Agency of Canada: Canadian Guidelines on Sexually Transmitted Infections, 2006 Edition. http://www.phac-aspc.gc.ca/std-mts/sti_2006/sti_intro2006_e.html.

Answered by: **Dr. John M. Embil**

25. The Danger of Portable Media Players



How dangerous is it to use gadgets on/over the ears, such as portable media players, etc.?

Submitted by: **Fernand Arseneau, MD**, Moncton, New Brunswick

There is a growing concern that the use of headphones or earphones with portable music players may cause significant hearing loss. The potential hearing loss is a function of several variables including the:

- volume,
- length of time the ear is exposed to the sound and
- the prior cumulative exposure to noise.

Sound is measured in units of decibels (dB). A portable sound system can reach a maximum output ranging from 50 dB to 140 dB. To put that in perspective, 140 dB is equivalent to the sound produced by a gunshot or firecracker. Generally, exposure to sound < 70 dB is not a risk factor for hearing loss. The currently accepted guidelines regarding noise exposure at work (assuming 20 years of work) are dependent on the noise intensity. Specifically, the guidelines allow for up to eight hours of exposure to sound at 90 dB, four hours of exposure to sound at 95 dB and only 30 minutes of exposure to sound at 110 dB. Generally, when listening to pop music, the sound level usually ranges between 85 dB to 115 dB but may reach up to 140 dB. This critical level may cause immediate and permanent hearing loss. Exposure to “loud” noise may cause a temporary hearing loss called a temporary threshold shift (TS). This consists of a temporary hearing loss in the higher frequencies, which manifests as a muffling of sounds and may be accompanied by tinnitus, a ringing sensation in the ears. Permanent TS often is a result of repeated exposure. This may cause permanent hearing loss due to

irreversible damage to the hair cells in the inner ear.

Hearing damage from headphones is probably more common than from loudspeakers, because many people exploit the acoustic isolation by listening at higher volumes. The use of ear buds may be more harmful than the “over the ear” headphones. The ear buds fit into the ear, effectively sealing the ear canal thereby not allowing the noise to escape. Therefore, tight-fitting ear buds tend to produce higher sound levels than other commercially-available headphones.

Early signs of transient or permanent hearing loss include:

- tinnitus,
- muffling of sounds and
- difficulties following social conversations.

To reduce the risk of noise induced from hearing loss from portable sound systems, one should limit the intensity and duration of music exposure.

Answered by: **Dr. Gideon Bachar;**
Dr. Vitaly Kisilevsky; and **Dr. Jonathan Irish**

26. Antibiotics and OC Efficacy



Do antibiotics affect the efficacy of depot-medroxyprogesterone acetate (DMPA)?

Submitted by: **Christina Fisher, MD**, Toronto, Ontario

It is well recognized that antibiotics and other medications can reduce the contraceptive effectiveness of OC pills. This effect is probably due to a reduce reabsorption of ethinyl estradiol in the entero-hepatic circulation of estrogens. Anticonvulsants also reduce the effectiveness of the OC pill, probably by induction of hepatic enzymes increasing clearance of the estrogen.

Contraceptive plasma levels are reached within 24 hours of administering DMPA and are maintained for 14 weeks. DMPA inhibits

ovulation and is highly effective when administered once every three months. Given appropriately, DMPA remains one of the most effective forms of contraception from both the theoretical and user viewpoints. Neither antibiotics nor antiseizure medicines reduce the effectiveness of DMPA.

Aminoglutethimide (used to treat some patients with Cushing's syndrome) is the only drug which reduces its effectiveness.

Answered by: **Dr. David Cumming**

27. Role of Caffeine in Hypertension



What is the role of caffeine in hypertension?

Submitted by: **W. E. Osmun, MD**, Mount Brydges, Ontario

Consumed in coffee, tea, soft drinks and small amounts in chocolate, caffeine is the most widely-used stimulant worldwide. CV effects include:

- increased contractility,
- increased heart rate and
- an acute increase in BP.

The BP increase is more pronounced in patients who are under stress or have underlying hypertension. Caffeine can acutely raise the BP by as much as 10 mmHg in patients who are infrequently exposed. However, there is little effect on BP in habitual coffee drinkers, nor does caffeine appear to increase the risk of new onset hypertension.

A meta-analysis of 18 controlled clinical trials found that coffee ingestion increased systolic and diastolic BP by 1.2 mmHg and 0.49 mmHg, respectively.¹ Similar reductions in BP may be seen when regular coffee drinkers either abstain from coffee or switch to decaffeinated coffee. However, there are individual differences in BP response to caffeine with rapid metabolizers of caffeine having more of a BP increase.

Reference

1. Noordzij M, Uiterwaal CS, Arends LR, et al: Blood Pressure Response to Chronic Intake of Coffee and Caffeine: A Meta-Analysis of Randomized Controlled Trials. *J Hypertens* 2005; 23(5):921-8.

Answered by: **Dr. Bibiana Cujec**

28. Benefit of Humidified Air in Croup



Is there any evidence-based medicine to support the use of a vaporizer (humidifier) in infants with upper respiratory infections (URIs)?

Submitted by: **Norm Blustein, MD**, Richmond Hill, Ontario

OTC medications and homeopathic remedies, such as humidified air, are commonly used for children with acute URIs. Most URIs in children are caused by acute viral infections. A severe form of URI, croup or acute laryngotracheobronchitis, characterized by hoarseness, a barking cough and inspiratory stridor is common in young children under the age of two years.

There is evidence of marginal benefit of

humidified air from three studies of infants with severe croup presenting to the ED.¹ More studies are needed to assess the role, if any, of humidified air in lesser severity croup and other forms of URIs in the community setting.

Reference

1. Moore M, Little P: Humidified Air Inhalation for Treating Croup. Cochrane Database Syst Rev 2006; 3:CD002870.

Answered by: **Dr. Paul Hernandez**

29. Belching in Adults



What causes prolonged belching in adults?

Submitted by: **I. D'Souza, MD**, Willowdale, Ontario

All people have gas in the intestinal tract. On average, people will generate one to three pints of gas a day. Gas is obtained by mainly two sources, exogenous gas from swallowed air or endogenous gas produced from colonic bacteria. Gas derived from swallowed air is composed of oxygen, nitrogen and carbon dioxide. Gas rich in hydrogen and methane is usually derived from colonic bacteria acting on residual food particles. On average, the content of intestinal gas is 90% ingested air and 10% formed by the bacteria in the colon.

Gas that is swallowed can be retained in the stomach and then released by belching. Normally, swallowed air can remain in the stomach for a period of time. Peristalsis will

normally pass small amounts of air into the small intestines and eventually to the colon where it can be released in small amounts via the rectum as flatus.

Factors that increase the intake of air include gum chewing, poor fitting dentures, post nasal drip, smoking, gulping down food, talking while eating, sipping beverages (especially with a straw), drinking carbonated beverages and excessive chewing of food.

Many normal individuals are concerned about having too much gas. This is rarely the case. Intestinal gas is not dangerous and is part of the normal physiological functioning of the digestive tract.

Answered by: **Dr. Richmond Sy**




Antidepressant

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30. Consequences of Asbestos Exposure



A 70-year-old asymptomatic man asks for a chest radiograph because of concern over asbestos exposure. A co-worker recently died of an asbestos-related illness. What should be done?


Submitted by: **Patricia Menard, MD**, Antigonish, Nova Scotia

Asbestos refers to a group of naturally occurring, heat-resistant, flexible, fibrous hydrous silicate minerals. Worldwide production of asbestos has declined over the past two decades as concern about the negative health consequences of this mineral has increased. Asbestos exposure can result from a number of occupations, such as:

- mining the raw material,
- direct application of the mineral (e.g., insulation) and
- disturbing finished products containing asbestos (e.g., construction worker, mechanic).¹

Asbestos exposure can result in a variety of pneumoconioses, or occupational lung diseases caused by the inhalation of mineral dusts. Pleural disease is common and can range from benign, self-limited pleurisy to benign pleural plaques to malignant mesothelioma. Lung disease can include asbestosis (i.e., diffuse interstitial lung fibrosis), round atelectasis and lung cancer.

There is a long latency between asbestos exposure and the onset of symptoms; additionally, asbestos-related diseases can progress even after exposure has ceased.

Earliest detection of significant exposure is often, but not always, associated with recognition of pleural plaques on chest radiograph. Early, inspiratory crackles found on a physical examination of the chest are an early finding in asbestosis. Pulmonary function tests (e.g., spirometry, lung volumes, diffusion capacity) usually correlate with abnormalities on chest radiographs. A high-resolution CT scan of the lungs can detect pleural or parenchymal lung involvement by fibrosis prior to plain chest radiographs. Asymptomatic individuals should have a thorough baseline assessment and regular follow-up at least annually. If disease is detected, the patient should be encouraged to initiate a disability claim with the worker's compensation board. Importantly, all workers should follow proper workplace practices to limit their asbestos exposure. 

Reference

1. Becklake MR, Cowie RL: Pneumoconiosis. In: Murray JF, Nadel JA (eds.): *Textbook of Respiratory Medicine*. Third Edition. W.B. Saunders Co. Philadelphia, Pennsylvania 2000, pp. 1811-52.

Answered by: **Dr. Paul Hernandez**

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