Answers to your questions from our medical experts

1. Approaching a Patient with an Alcohol Problem

What is the best way to approach someone who has an alcohol problem, without him becoming defensive (as a family member and doctor)?

Submitted by: Diane Giroux, MD, Montreal, Quebec

The elements of treatment appropriate for patients with alcohol problems consist of three general steps:

- intervention,
- detoxification and
- rehabilitation.

The intervention, also called confrontation, is the most challenging step and aims at breaking through feelings of denial, to help the patient recognize the adverse consequences likely to occur if the disorder is not treated.

The physician often takes advantage of the patient's chief presenting complaint, whether it is:

- insomnia,
- difficulties with sexual performance,
- an inability to cope with life stresses,
- depression, or
- anxiety.

The physician will then explain to the patient how alcohol has either created or contributed to these problems and can reassure the patient that abstinence can be achieved with a minimum of discomfort and will most likely reduce many of these presenting symptoms.

The physician should also acknowledge to the patient that alcohol abuse is often used to self-medicate for depression and anxiety. If the patient agrees to achieve abstinence, proper treatment for the depression and/or anxiety is readily available and will resolve the need for self-medication with alcohol.

A physician intervening with a patient should remain nonjudgmental while maintaining a persistent approach each time an alcohol-related impairment is identified. It is the level of persistence rather than exceptional interpersonal skills that usually gets results. A single intervention is rarely enough. Most alcoholic persons need a series of reminders of how alcohol contributed to each developing crisis before they seriously consider abstinence as a long-term option.

Family members are encouraged to join support groups, such as Alanon. They meet many times a week and help family members see that they are not alone in their fears, worry and feelings of guilt. Family members are also encouraged not to protect the patient from the problems caused by alcohol; otherwise, the patient may not be able to gather the motivation necessary to stop drinking. Finally, family members can be of great help during the intervention stage by encouraging the patient to join AA meetings to meet and hear from people who are themselves recovering from alcoholism.

Answered by: Dr. Hany Bissada

2. Commonality of Brugada Syndrome

How often could I expect to see Brugada syndrome working in an ER?

Submitted by: Anonymous

Sudden cardiac death (SCD) without structural heart disease is rare, accounting for 5% of all cases of SCD. Causes include:

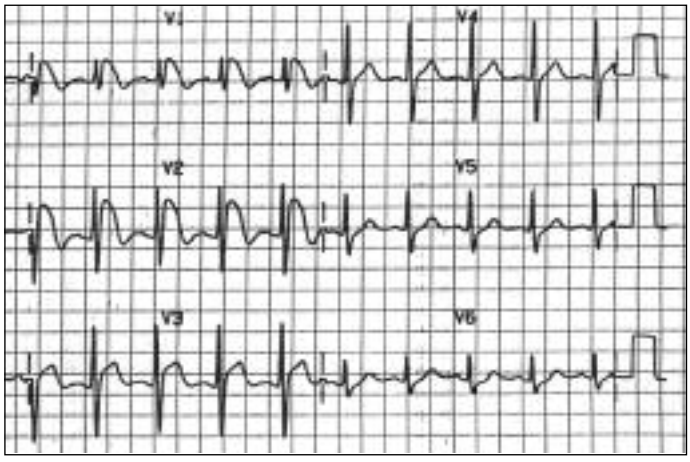
- Brugada syndrome,
- long QT,
- ventricular pre-excitation and
- *commotio cordis*.

Brugada syndrome is a cause of ventricular fibrillation in young people, particularly in young Asian men. It is recognized on the ECG by an incomplete right bundle branch block (RBBB) pattern with ST elevation in V_1 to V_2 (Figure 1). Of note, the Brugada pattern may be intermittently present and is enhanced by the administration of procainamide. The Brugada syndrome is caused by an abnormality in myocardial sodium channels.

In a large urban teaching hospital in the US, the prevalence of a Brugada ECG pattern was 0.4%.[1] The prevalence of the Brugada pattern is much greater in patients who present with apparent idiopathic ventricular fibrillation (3% to 24% in one series of 37 patients, depending upon the diagnostic criteria used).[2]

References
1. Monroe MH, Littmann L: Two-Year Case Collection of the Brugada Syndrome Electrocardiogram Pattern at a Large Teaching Hospital. Clin Cardiol 2000; 23(11):849-51.
2. Remme CA, Wever EF, Wilde AA, et al: Diagnosis and Long-Term Follow-Up of the Brugada Syndrome in Patients with Idiopathic Ventricular Fibrillation. Eur Heart J 2001; 22(5):400-9.

Answered by: Dr. Bibiana Cujec



Fgure 1. ECG precordial leads in patient with Brugada syndrome. Note the ST elevation in V_1 to V_3.

3. Toxoplasmosis Screening During Pregnancy

Would toxoplasmosis screening be appropriate in pregnant women who own cats?

Submitted by: Theo Kemp, MD, Blackfalds, Alberta

The incidence of fetal toxoplasmosis infection is rare in Canada (one to 10 per 10,000 births, or 40 to 400 babies infected per year); therefore, screening is not a routine part of prenatal care. However, screening for toxoplasmosis may be appropriate in pregnancy or pre-conceptual stages if concerned about exposure or if avoidance to exposure is possible. Recommendations to reduce exposure include:

- Wash fruits and vegetables thoroughly
- Wash hands and utensils after handling unwashed vegetables or uncooked meats
- Avoid direct contact with soil and sand
- Avoid changing cat litter. If necessary use gloves and wash hands
- Eat only well cooked meat

Screening includes antibodies to toxoplasmosis, if IgM alone is detected (present in approximately 25% of Canadians) then previous exposure and immunity exists; if both IgG and IgM are detected then further investigation is required (see reading).

Recommended reading
1. Many A, Koren G: Toxoplasmosis During Pregnancy. Can Fam Phys 2006; 52:29-30, 32.

Answered by: Dr. Victoria Davis

4. Targeted Oncolytic Viruses

Are there any advances in the use of viruses to target cancer cells specifically?

Submitted by: Janna Bentley, MD, Kelowna, British Columbia

Targeted oncolytic viruses have been the subject of much study in cancer therapeutics. The primary strategy is the use of therapeutic viruses which selectively replicate in and destroy cancer cells through induction of tumour-specific immunity. In addition, viruses can be primed with a therapeutic which may induce direct cell kill. Selectivity for cancer cells may be inherent to specific viruses or genetically engineered into a virus. While this novel mechanism of action may hold much promise, clinical evidence of systemic efficacy remains lacking and limited. This remains a highly investigational treatment approach and further clinical data is awaited.

Answered by: Dr. Sharlene Gill

5. Office-Based Therapies for Genital Warts

If liquid nitrogen and imiquimod are not effective for genital warts, are there any effective office-based therapies?

Submitted by: Dennis Neufeld, MD, Kelowna, British Columbia

Treatment of genital warts can be challenging because of poor host immunity to HPV and recurrence rates after treatment can be high.

If liquid nitrogen and imiquimod have been used properly and the genital warts have not responded, other options include:

- topical podophyllin applied in the office,
- podophyllotoxin which can be prescribed to the patient for home treatment,
- topical trichloroacetic acid as an office treatment,
- topical 5-fluorouracil cream,
- electrosurgery,
- cold steel surgery including curettage and
- laser therapy.

Many of these treatments can cause severe irritation as well as scarring and should be used by clinicians experienced in their use.

Answered by: Dr. Richard Haber

6. Warfarin and Pulmonary Hypertension

What is the role of warfarin in pulmonary hypertension?

Submitted by: Y. Nikahn, MD, Newmarket, Ontario

Pulmonary hypertension (PH) includes a number of disorders that result in increased pulmonary arterial pressure and pulmonary vascular resistance. The most recent ("Venice") classification scheme separates causes of PH into:

- pulmonary arterial hypertension (PAH),
- PH related to left heart disease,
- PH associated with lung disease or hypoxemia,
- PH related to thromboembolic disease and
- a miscellaneous category.[1]

Chronic anticoagulation with warfarin is currently recommended only for idiopathic PAH (previously called primary pulmonary hypertension) and PH related to thromboembolic disease.[1]

Reference

1. Langleben D, Archer S, Granton J, et al: Canadian Cardiovascular Society and Canadian Thoracic Society Position Statement on Pulmonary Arterial Hypertension. Can Respir J 2005; 12(6):303-8.

Answered by: Dr. Paul Hernandez

7. Swimming During Treatment for Otitis Externa

When someone is being treated for external otitis with ear drops, does it matter if they continue to swim?

Submitted by: Robert Dickson, MD, Hamilton, Ontario

Otitis externa is inflammation of the skin of the external auditory canal. It can involve any point from the meatus to the eardrum. Otitis media can be localized, or generalized involving the whole canal. Predisposing factors include swimming, trauma (secondary to cotton tips) and narrow canals. Pathogens include bacterial, viral and fungal agents. Regular aural toilet, splinting the meatus (with either ribbon gauze or pope wicks) and application of topical eardrops are the mainstay of treatment. During treatment, the ear should be kept meticulously dry. During washing, care must be taken to avoid water coming into contact with the canal and occlusion of the ear canal with Vaseline on cotton wool is recommended. Swimming is inadvisable for patients with otitis externa.

Treatment should resolve the problem over the course of a week or two. Following resolution, the patient should be discouraged from using cotton tips, *etc*. When the inflammation has resolved, the patient can again go swimming.

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

8. Asymptomatic Elevation of Creatine Kinase

How do you respond to an asymptomatic creatine kinase (CK) elevation in a patient on statins or fibrates?

Submitted by: James Krahn, MD, Winnipeg, Manitoba

Elevation of CK may occur in patients receiving statin/fibrate therapy and may be associated with symptoms of myositis. When significant elevations of CK (> 10 times normal) occur in the absence of symptoms, one can usually stop therapy and start an alternative therapy such as non-statin or non-fibrate cholesterol medication. If the elevation is three to 10 times normal, then one can continue therapy and monitor for symptoms of myositis or more severe elevations of CK. Careful monitoring of the CK levels should be considered when there are factors that may increase the risk of myopathy, such as advanced age, small body size and frailty, in addition to a number of medications/toxins (such as macrolides antibiotics, alcohol and even large quantities [over one quart] of grapefruit juice).

Answered by: Dr. Richard Sheppard

9. Benign Positional Vertigo

What is the best recommendation to give a patient with benign positional vertigo?

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

Benign positional vertigo (BPV) is thought to be caused by abnormal movement of the canaliths out of their usual position in the semicircular canals. It is often associated with a minor head injury, an upper respiratory infection, or can be spontaneous.

First, I would do a Dix-Hallpike test, where patients are suddenly moved from sitting to a supine position with the head hanging to either the right or left side. After a delay of a few seconds, the patient should get a sense of spinning vertigo and rotatory nystagmus towards the direction of the affected ear. Once the affected ear has been determined, I would do an Epley maneuver. This has been shown to be more effective than drugs or vestibular physiotherapy in the short term.

If there is no improvement in one week, then I would have the patient start vestibular physiotherapy. The purpose of this is to essentially desensitize the brain stem to the abnormal signals coming in from the ear.

If this is not effective in improving the symptoms (which also improve spontaneously in most people within a few weeks), or if there is a question of diagnosis of the vertigo, I would do a neurologic and otolaryngological exam and send the patient for an electronystagmogram to clarify if the dizziness is central or peripheral. Based on this information, I would then refer on to an otolaryngologist or neurologist.

Resources

1. Hillier SL, Hollohan V: Vestibular Rehabilitation for Unilateral Peripheral Vestibular Dysfunction. Cochrane Database Syst Rev 2007; (4):CD005397.
2. Hilton M, Pinder D: The Epley (Canalith Repositioning) Manoeuvre For Benign Paroxysmal Positional Vertigo. Cochrane Database Syst Rev 2004; (2):CD0003162.

Answered by: Dr. Inge Loy-English

10. *H. Pylori* and Gastric Cancer

How strong is the association between *H. pylori* and gastric cancer? I have a Japanese patient with a strong family history of gastric cancer. No symptoms. Tests are negative for *H. pylori*. How reassuring is this?

Submitted by: John Miller, MD, Victoria, British Columbia

The association of *H. pylori* and gastric cancer is well established. There is a two- to 16-fold increase of gastric adenocarcinoma in infected individuals compared to healthy controls. The increase risk of developing gastric cancer due to *H. pylori* infections depends on multiple factors including:

- strain of bacteria,
- length of time of infection and
- other factors including the host immune response.

The effect of eradication of *H. pylori* and the subsequent risk of gastric cancer is not entirely clear. This issue is clouded because of lack of prospective studies. Animal models confirm reversibility of metaplasia and prevention of gastric cancer with early eradication. Published human trials looking at surrogate endpoints for gastric cancer such as biomarkers for gastric atrophy and intestinal metaplasia demonstrate a beneficial effect as well. In a non-randomized study with Japanese subjects with dyspepia undergoing endoscopy, the patients who were followed-up after receiving eradication therapy for *H. pylori* did not develop gastric cancer.[1] The group that had not received *H. pylori* treatment developed gastric cancer in 3% of the patients. Unfortunately, the follow-up periods were variable during the study. It is recommended that all patients with positive *H. pylori* on testing be treated.

Reference

1. Uemura N, Okamoto S, Yamamoto S, et al: Effect of Helicobacter Pylori Infection and the Development of Gastric Cancer. NEJM 2001; 345(11):784-9.

Answered by: Dr. Richmond Sy

There is a two- to 16-fold increase of gastric adenocarcinoma in infected individuals compared to healthy controls.

11. Treatment of Tinea Versicolor

Comment on specific treatment for tinea versicolor. Topical medications do not often work. What are specific doses for oral medications?

Submitted by: Julie Bihun, MD, Ottawa, Ontario

Tinea versicolor is more properly termed pityriasis versicolor (PV). It is caused by a *Malassezia (M.)* yeast and although reported in the past to be due to *M. furfur*, after the *Malassezia* yeasts underwent a revision of their taxonomy in 1996, it was found that the most common cause of PV is *M. globosa*.

Topical medications can be effective in PV especially in cases with localized lesions. Often, when patients state the treatment did not work, it is because of the high relapse rate of the lesions (one does not catch PV but is colonized by it and it causes clinical lesions when the yeast enters the stratum corneum in its filamentous form, often in warm weather). Patients (and physicians) may consider that the hypopigmented form of PV did not respond to therapy because it can take months for the hypopigmentation to repigment after treatment.

Topical therapies include:

- propylene glycol,
- sodium thiosulphate,
- selenium sulfide 2.5%,
- zinc pyrithione 1%,
- topical azoles (clotrimazole, miconazole, econazole, ketoconazole, ciclopirox cream or lotion) and
- terbinafine cream or spray.

Systemic therapy is indicated for extensive or refractory cases. It is very important to recognize that although topical terbinafine can be effective in treating PV, oral terbinafine is not (likely because it does not obtain fungicidal levels in the stratum corneum).

The most effective oral therapies are:

1. Oral ketoconazole 200 mg q.d. for five to seven days
2. Oral itraconazole 200 mg q.d. for five to seven days
3. Oral fluconazole 400 mg (single dose)

Physicians should be aware of potential drug interactions when using these oral treatments and that these drugs should not be used in pregnant or lactating patients.

Answered by: Dr. Richard Haber

12. Estimated Glomerular Filtration Rate

Please explain estimated glomerular filtration rate and what to do about low results in well-controlled diabetes.

Submitted by: Peter Loveless, MD, Hamilton, Ontario

The estimated glomerular filtration rate (eGFR) is a sensitive method of identifying impaired renal function. In Canada, the eGFR is most often calculated using the abbreviated Modification of Diet in Renal Disease equation, which takes into account the person's serum creatinine, age and sex. When finding a low eGFR, the cause should be sought and tests may include a urinalysis, albumin:creatinine ratio and imaging studies.

Medications such as metformin may need to be adjusted when the eGFR is low.

Answered by: Dr. Vincent Woo

13. Pickwickian Syndrome

What is Pickwickian syndrome?

Submitted by: Vasse Moodley, MD, Etobicoke, Ontario

Pickwickian syndrome is obesity hypoventilation syndrome. This syndrome is defined by morbid obesity with alveolar hypoventilation (hypercarbia and hypoxemia). In addition to hypersomnolence and cyanosis with polycythemia, there is often right heart failure and peripheral edema secondary to pulmonary hypertension. The name derives from a character in Charles Dickens' book, *The Posthumous Papers of the Pickwick Club*. Joe was a "wonderfully fat boy, standing upright with his eyes closed" who was hypersomnolent, edematous and a very loud snorer.

This syndrome primarily results from two factors: an increase in the work of breathing because of obesity and a decrease in the "drive" to breathe with resetting of brain chemoreceptors to a higher partial pressure of CO_2 resulting in hypoventilation even while awake. Obstructive sleep apnea with intermittent airway obstruction is a common contributory factor (about 90% of patients). Noninvasive positive pressure ventilation is the mainstay of therapy. Weight loss is an obvious goal of therapy but is often very difficult to achieve. Bariatric surgery may be helpful but some of these patients continue to have hypoventilation even after successful weight reduction.

Answered by: Dr. Bibiana Cujec

14. Stem Cell Transplant and Hodgkin's Lymphoma

How effective is a stem cell transplant for Hodgkin's lymphoma (Type 3A) (*i.e.*, survival rates?)

Submitted by: Maury O'Neil, MD, Collinwood, Ontario

Consideration for high-dose chemotherapy followed by autologous hematopoeitic stem cell transplantation is a strategy in the management of primary refractory Hodgkin's lymphoma (failure to achieve a response from primary chemotherapy and/or radiation) and in recurrent Hodgkin's lymphoma. Based upon non-randomized evidence, 40% to 50% of patients may relapse after auto-stem cell transplantation. Risk factors for relapse after stem cell transplant include:

- chemotherapy-resistant disease,
- duration of complete response < 12 months,
- bulky residual disease at transplantation,
- extranodal disease,
- B symptoms and
- poor performance status.

Issues related to patient selection, optimal cytoreductive regimens and role of allogenic transplantation are subjects of ongoing study.

Answered by: Dr. Sharlene Gill

15. Treatment of Type 2 Diabetes

Should all individuals with Type 2 diabetes be on statins with ACE inhibitors and ASA, as well as glucose-lowering agents?

Submitted by: M. Rajami, MD, Toronto, Ontario

Most individuals with Type 2 diabetes should be considered to be at high CV risk. Exceptions are the younger individual with recently diagnosed diabetes who has no other cardiac risk factors or diabetes complications. It must be remembered however that many of these individuals may have had diabetes for some years. It is recommended that statins be used to get the LDL-C level < 2.0 mmol/L as the primary target. The secondary target is a total cholesterol (TC):HDL-C ratio of < 4.0. For vascular protection, ACE inhibitors are recommended at appropriate doses. ARBs are likely equivalent for vascular protection according to the recently released ON-TARGET study. BP goals for individuals with Type 2 diabetes are ≤ 130/80 mmHg. Multiple antihypertensives may be needed. ASA is recommended for vascular protection as well, although their efficacy seems to be less in people with diabetes. Finally, if lifestyle does not control glucose levels adequately to achieve a HgA1C < 7% then antihyperglycemic agents should be considered with metformin being the medication of first choice if it is not contraindicated or tolerated.

Answered by: Dr. Vincent Woo

16. Imiquimod Treatment for Warts

Is imiquimod effective in treating plantar and other non-genital warts?

Submitted by: Charles Cheng, Vancouver, British Columbia

Imiquimod is a topical immunomodulating agent approved for treating genital warts, actinic keratoses and superficial basal cell carcinoma. It has anti-viral and anti-tumour effects by stimulating the production of various cytokines (*e.g.*, interferon-γ, interleukin-one, interleukin-six, TNF-α) via the activation of toll-like receptor seven.

Imiquimod was first approved for treating genital warts. It is not approved for treating non-genital warts, but has been used "off-label" for this indication. Small studies have looked at its effectiveness after other therapies have failed or in immunocompromised patients. One study of 50 immunocompetent patients with previously resistant cutaneous warts achieved a response rate of 30% when applied five nights weekly.[1] A study of 12 immunosuppressed patients with palmo-plantar warts resistant to other therapies obtained a 36% response rate.[2]

Its effectiveness on non-genital warts is limited by poor cutaneous absorption. It may be more effective when used as an adjunct with destructive modalities (*e.g.*, salicylic acid, cryotherapy) and/or occlusion.

The main drawbacks to using imiquimod include local reactions (erythema, pain, scaling) and cost compared to alternatives.

References

1. Hengge UR, Esser S, Schultewolter T, et al: Self-Administered Topical 5% Imiquimod for the Treatment of Common Warts and Molluscum Contagiosum. Br J Dermatol 2000; 143(5):1026-31.
2. Harwood CA, Perrett CM, Brown VL, et al: Imiquimod Cream 5% for Recalcitrant Warts in Immunosuppressed Individuals. Br J Dermatol 2005; 152(1):122-9.

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

17. Treating Low HDL-C

How can I effectively treat isolated low HDL-C? What about flaxseed oil or omega-3-6-9 supplements? Do they work to lower HDL-C and by how much?

Submitted by: Katherine Phillips, MD, Hagersville, Ontario

There is an inverse relationship between HDL-C levels and atherosclerosis. There are numerous therapies available to treat low HDL-C levels, including aggressive lifestyle modification with weight loss, improved diet and smoking cessation. Medical therapies can include niacin, fibrates and, to some extent, statins may raise HDL-C levels. Flaxseed has not been shown to consistently raise HDL-C concentrations and in some studies has actually lowered them.[1] Omega-3 supplements have been shown in some studies to lower triglyceride levels, but their impact on HDL-C is more variable when examining the current literature. Always consult with your FP or your cardiologist before consuming any of the above-mentioned supplements.

Reference
1. Bloedon LT, Balikai S, Chittams J, et al: Flaxseed and Cardiovascular Risk Factors: Results From a Double Blind, Randomized, Controlled Clinical Trial. J Am Coll Nutr 2008; 27(1):65-74.

Answered by: Dr. Richard Sheppard

18. X-Rays in Low Back Pain

How useful are x-rays in low back pain?

Submitted by: Ann Vaidya, MD, Calgary, Alberta

The majority of patients with uncomplicated low back pain of less than four weeks duration and no risk factors do not require imaging. X-rays should only be ordered if the pain persists, or if there are any "red flags" of a serious underlying condition, such as fevers, weight loss, other constitutional symptoms and inflammatory symptoms. Plain x-rays are not considered high yield tests and they infrequently detect abnormalities that change management, but rather often do reveal findings unrelated to symptoms.

In addition, plain x-rays have a low sensitivity for many conditions. They do not detect herniated discs, but can show evidence of infection, fracture, malignancy, spondylolisthesis, degenerative changes, disc space narrowing and prior surgery. Normal plain films do not exclude malignancy or infection in patients with a suspicious history. In one review, plain radiography was 60% sensitive and 95% specific for malignancy and 82% sensitive and 57% specific for infection.[1] If there is high clinical suspicion, then investigations should proceed beyond a plain x-ray and patients should be referred for MRI, bone scan, or CT scan depending on history.

Reference
1. Jarvik JG, Deyo RA: Diagnostic Evaluation of Low Back Pain with Emphasis on Imaging. Ann Intern Med 2002; 137(7):586-97.

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

19. Differences in the Treatment of Otitis Media

Why do we in North America treat otitis media and pharyngitis while many Northern European physicians do not?

Submitted by: Adam Steacie, MD, Brockville, Ontario

Otitis media (glue ear) is fluid within the middle ear cleft, caused by Eustachian tube dysfunction. The etiology remains obscure, but it is believed to be related to recurrent upper respiratory tract infections, parental smoking and allergy. Large adenoids are thought to be involved in the etiology, not due to a mechanical effect of blocking the Eustachian tube, but more of a potential source of pathogenic bacteria, if and when the fluid becomes infected.

It is a common condition affecting 40% of children at two years. The natural course of the condition is to spontaneously resolve, such that only 1% of children are affected at 11 years. Fluid within the middle ear cleft results in a conductive deafness. In addition, the fluid may become infected.

In Europe, glue ear is treated conservatively, with a great deal of emphasis on getting the parents to stop smoking, or not smoke in close proximity to the child. Acute infections, if the child were systemically unwell, would be treated with antibiotics. Recurrent attacks are treated with myringotomy and ventilation tube insertion. In North America, there is a lower threshold for treatment with antibiotics.

Pharyngitis is a painful inflammation of the pharyngeal mucosa (either nasopharynx, oropharynx or laryngopharynx). It can be caused by infection or contact with chemical irritants, such as acid reflux. Approximately 90% of the infected cases are as a result of viral infection, the remaining majority by bacterial pathogens. In rare cases, fungal pathogens (oral thrush) may be the cause.

Infections can be treated with symptom control (viral, bacterial and fungal), antibiotics (bacterial) and anti-fungal agents (oral thrush). Symptom control includes gargles and analgesics. Most physicians in Europe will first treat pharyngitis symptomatically, with the premise that if they are infective, they are likely to be as a result of viral pathogens. However, those patients with a probable strep throat (systemically unwell, presence of pharyngeal exudate, purulence) would be treated with antibiotics. Again, in North America, there is a lower threshold for treatment with antibiotics. The difference in treatment is likely to be historical and over time may well become cohesive.

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

20. Prescribing an Adrenaline Auto Injector

If a patient has urticaria and angioedema, with no shortness of breath, should I use (prescribe) an adrenaline auto injector?

Submitted by: Steve Choi, MD, Oakville, Ontario

This depends upon the circumstances, both concerning the patient's history as well as the hives/swelling symptoms. In the setting of chronic idiopathic urticaria, flares of hives and angioedema obviously do not require use of an adrenaline auto injector, as this is a manifestation of a chronic recurrent condition. Likewise, in a young patient with viral associated urticaria, adrenaline is not required. In the setting of a patient with known food or venom allergy, "internal" symptoms (*i.e.*, sudden vomiting, shortness of breath, throat discomfort) necessitate immediate use of adrenaline and then a visit to the ER. The appearance of isolated hives in these patients does present some degree of judgement. The usual advice for the known allergic patient with an auto injector is to use adrenaline for the sudden appearance of diffuse hives and/or swelling, especially if contact with an offending agent is suspected. In the absence of suspected contact, the gradual appearance of only a few hives without any other symptoms may be simply observed, with adrenaline close at hand in case the hives suddenly worsen, or angioedema or internal symptoms appear. However, if a patient (without any known previous allergic history) describes a history of a sudden outbreak of diffuse hives or angioedema, which resolved over several hours (suggesting an allergic mechanism), then it would be prudent to prescribe an auto injector and refer to an allergy specialist for further evaluation for a potential allergic trigger.

Answered by: Dr. Tom Gerstner

In the setting of chronic idiopathic urticaria, flares of hives and angioedema obviously do not require use of an adrenaline auto injector, as this is a manifestation of a chronic recurrent condition.

21. Health Concerns with Bromine Use

Are there any health concerns with bromine used in hot tubs as a disinfectant?

Submitted by: Maury O'Neil, MD, Collingwood, Ontario

A number of infectious and non-infectious health hazards are associated with hot tub use. Hot tub folliculitis has been described due to skin infection with *Pseudomonas aeruginosa*. Hypersensitivity pneumonitis related to inhalation of nontuberculous mycobacteria can occur, particularly if hot tub filtration and cleaning systems are not well maintained. To reduce infection risk, halogens, such as chlorine and bromine, are available for hot tub sanitation. Bromine is typically used in one of three forms, all of which generate the bactericidal agent hypobromous acid when dissolved in water.[1] In acidic pH and particularly at usual hot tub temperatures, hazardous bromine liquids and vapors are released. Depending upon the amount of exposure, injury can range from mild skin and upper airway irritation, to more serious burns and upper and lower airway tissue damage. Acutely, chemical pneumonitis has been described; long-term sequelae include reactive airways dysfunction syndrome.[1] Illness due to bromine exposure should be considered when assessing health hazards of hot tub use.

Reference
1. Burns MJ, Linden CH: Toxicity Secondary to Bromine and Hydobromic Acid Exposure. Chest 1997; 111(3):816-9.

Answered by: Dr. Paul Hernandez

22. Investigating a First Episode of Kidney Stones

What kind of investigations will you do for a first episode of kidney stones in an 87-year-old female?

Submitted by: Michel Behamdouni, MD, Outremont, Quebec

Renal stones are common with an annual incidence of seven to 12 cases per 10,000 persons. Kidney stones can have varying compositions with the most common being calcium oxalate (75%), followed by struvite (15% to 20%), uric acid (10% to 15%) and cysteine (1%).

First episodes of renal stones require little workup aside from urinalysis, serum calcium, phosphate, uric acid, creatinine and blood urea nitrogen. If possible, urine should be strained to recover stones and a stone analysis performed. Recurrent renal colic requires a more extensive metabolic work-up including, in addition to above, 24-hour urine collections for calcium, phosphate, citrate, uric acid, creatinine, volume, sodium, magnesium, pH and urea nitrogen. Other specific investigations will be based on stone type. The 24-hour urine collection should be based on the patient's regular diet and medication regiment.

Answered by: Dr. Manish Sood

23. Causes of Hair Loss in Teenagers

What are the causes of hair loss in teenagers/young adults?

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

Hair loss is a comprehensive topic to which many textbooks are devoted. The same approach can be used in teenagers and adults, provided hair shaft abnormalities (*e.g.*, inherited conditions such as Menkes syndrome, Netherton syndrome) are ruled out. The major fork in the road is scarring vs. non-scarring and this distinction helps narrow the approach. In non-scarring alopecia, hair follicles are visible whereas in scarring alopecia, one sees only scarred scalp with the absence of follicles. Scarring alopecia is the result of permanent injury to the stem cell region of the hair follicle. Hair growth is permanently impaired.

Table 1

Causes of scarring alopecia

Developmental/hereditary disorders
- Aplasia cutis congenita
- Epidermal nevi
- Romberg's syndrome
- Generalized follicular hamartoma

Primary causes
- Group 1: Lymphocytic
  - Lupus erythematosus
  - Lichen planopilaris
  - Classic pseudopelade
- Group 2: Neutrophilic
  - Folliculitis decalvans
- Group 3: Mixed
  - Acne keloidalis nuchae

Secondary causes
- Infectious agents
  - Bacterial (*i.e.*, post-cellulitis)
  - Fungal (*i.e.*, tinea capitis)
- Neoplasms (*i.e.*, basal cell carcinoma, squamous cell carcinoma, lymphomas and metastatic tumours)
- Physical agents
  - Mechanical trauma
  - Burns
  - Radiotherapy
- Caustic chemicals

For non-scarring alopecia, consider the following entities: alopecia areata, trichotillomania, telogen effluvium, androgenic alopecia, tinea capitis, traction alopecia, chemically-induced alopecia (*i.e.*, hair relaxers). Investigations for non-scarring alopecia include:
- No routine investigation usually necessary
- Consider association with other autoimmune diseases
- Complete blood count
- TSH
- Thyroid autoantibodies
- Serum B12

Likewise, there are many causes of scarring alopecia. Here, a further distinction is whether the scarring is a genetic/congenital disorder, a primary process, or a secondary cause (Table 1).

Treatment is based on the underlying condition for most cases (*e.g.*, immunosuppressive therapy for alopecia areata) (Table 2).

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

Table 2

Approach to patient presenting with scarring alopecia

History/physical
- Factors associated with onset (*i.e.*, trauma)
- Use of hair practices (*i.e.*, hot curling irons)
- Duration (*i.e.*, developmental)
- Presence of other lesions (*i.e.*, lichen planus)
- Symptoms of collagen/vascular disease (*i.e.*, lupus)
- History of neoplasm

Investigations
- Scalp biopsy
- As indicated:
  - Cultures
  - Bacterial and fungal if clinically indicated
  - Blood test (antinuclear antibody)

24. Role of Serum Catechol in Depression

What is the role of serum catechol in major depression?

Submitted by: Achla Virmani, MD, Brossard, Quebec

The postulated role of catecholamines in depression is essentially based on two sets of pharmacological observations. First, reserpine (which decreases BP by depleting catecholamines stores) precipitates clinical depression in some patients. Second, antidepressant medications (which alleviate clinical depression) raise the functional capacity of the catecholamines in the brain.

The catecholamines are synthesized from the amino acid tyrosine, which is taken up into the brain via an active transport mechanism. Two enzymes that play major roles in the degradation of catecholamines are monoamine oxidase and catechol O-methyltransferase, hence the antidepressant effect of the monoamine oxidase inhibitors class of antidepressant medications.

Despite more than three decades of extensive research and indirect evidence, no deficiency or excess of catecholamines in specific brain structures has been shown to be necessary or sufficient for the occurrence of mood disorders. It has not been possible to confirm the putative role of central norepinephrine in depression nor to discard it altogether.

Answered by: Dr. Hany Bissada