1. ASA for Patients over Forty

Should everyone > 40 years of age receive ASA 81 mg daily?

Submitted by: John Dawson, MD, Richmond Hill, Ontario

No, ASA should be reserved for patients with known vascular disease (coronary artery disease, peripheral arterial disease or stroke) or those at high risk for vascular events (10 year Framingham risk of coronary event > 20%). The decision to recommend ASA to patients who are at an intermediate risk (10 to 20% 10 year coronary event rate) should be individualized. Data on patients with diabetes are conflicting — diabetes is a major risk factor for vascular events but may not be a coronary artery disease equivalent, especially if the diabetes is of recent onset.

There is a small increase in GI bleeding and intracranial bleeding even with low dose ASA (< 0.2% annual risk). The beneficial effect of ASA in primary prevention is mainly to decrease non-fatal myocardial infarction (0.18% per year in patients on ASA versus 0.23% per year with placebo). When the risk of a vascular event is low, the potential benefit of low dose ASA is outweighed by the increased risk of bleeding. The evidence is much less compelling for ASA in primary prevention than in secondary prevention.[1]

Reference

1. Antithrombotic Trialists (ATT) Collaboration, Baigent C, Blackwell L, Collins R, *et al*: Aspirin in the Primary and Secondary Prevention of Vascular Disease: Collaborative Meta-analysis of Individual Participant Data from Randomised Trials. Lancet 2009; 373(9678): 1849–1860.

Answered by: Dr. Bibiana Cujec

2. Iron Supplements

What kinds of oral iron supplements are best tolerated and most efficacious?

Submitted by: Marie-Dominique Dzineku, MD, Sherbrooke, Québec

One of the main drawbacks of oral iron supplementation is GI intolerance. Unfortunately, there is no iron formulation that seems to be consistenly better tolerated or more efficacious than the other.

Answered by: Dr. Richmond Sy

3. Treating Postpartum Depression

What is the best treatment for postpartum depression? Which antidepressants could help if the patient is breastfeeding her baby?

Submitted by: Anonymous

For postpartum depression, antidepressant medication is indicated. However, only one placebo-controlled trial and three open trials that specifically address postpartum depression have been published. Fluoxetine was significantly more effective than placebo. In open trials, sertraline, venlafaxine, and drugs grouped according to class (SSRIs and tricyclic antidepressants) were also effective.

Women who have given birth recently are often sensitive to the side-effects of medications. Treatment should be initiated at half of the recommended starting dose for four days, and doses should be increased by small increments as tolerated until full remission is achieved. If the patient has a response to an initial trial of medication lasting six to eight weeks, the same dose should be continued for a minimum of six months after full remission has been achieved to prevent a relapse.

An Expert Consensus Guideline Series recommended that the SSRI sertraline be used as a first-line treatment for breastfeeding mothers because of its low risk, although sporadic high levels have been observed in some infants. One report observed minimal changes in infants who were exposed to small amounts of sertraline through breast milk.

Taking medication immediately after breastfeeding minimizes the amount present in milk and maximizes clearance before the next feeding.

Electroconvulsive therapy (ECT) is an effective treatment for postpartum depression, when there is poor response to antidepressant medications and/or there is a serious risk for suicide. A referral to a psychiatrist in these situations is imperative.

Resource

1. Chapter 28: Special Areas of Interest. In: Sadock BJ, Sadock VA, Ruiz P (eds.): Kaplan & Sadock's Comprehensive Textbook of Psychiatry. 9th edition. Volume two. Lippincott, Williams, & Wilkins, Philadelphia, 2009, 2556–2558.

Answered by: Dr. Hany Bissada

4. Foul Taste Caused by PPIs

Why do patients on PPIs (*e.g.*, pantoprazole) get a foul taste in their mouth that seems to emanate from the stomach? Can anything be done about it?

Submitted by: R.B. Harris, MD, Penticton, British Columbia

Proton pump inhibitors (PPIs) have become one of the most commonly prescribed medications. According to IMS health, a health care market research firm, PPIs are the third most commonly prescribed drugs in the US, with nearly 110 million prescriptions per year. They act by irreversibly blocking the H+/K+ ATPase ("proton pump") of the gastric parietal cell and are used in the treatment of gastroesophageal reflux disease.

Blockade of the "proton pump" leads to an increase in gastric, as well as oral, pH. This increase in oral pH could affect oral microbial growth.[1] In addition, PPIs could enhance oral microbial growth by decreasing oral saliva production. A change in oral microbial flora can then lead to dysgeusia or a distortion in the sense of taste. While this seems possible in theory, it has not borne out in practice. Dysgeusia is not a common side effect of PPI use. A review of the literature turned up only scattered case reports, including one by the Netherlands pharmaceutical company Lareb, which reported 10 patients with dysgeusia attributed to pantoprazole.

While PPI use may lead to "foul breath," there are certainly many other causes that are more likely.[3] These could include oral causes (poor dentition, orodental infection), respiratory causes (upper respiratory tract infection), and gastric causes (including reflux disease and delayed gastric emptying). If the "foul taste" causes the patient distress one could try an alternative PPI or switch to an alternative form of acid inhibition. Persistence of dysgeusia likely points to an alternative cause.

Reference
1. Bradshaw DJ, Marsh PD: Analysis of pH-driven Disruption of Oral Microbial Communities in Vitro. Caries Res 1998; 32(6):456–462.
2. Cems DA, Yen DM, Kreshak A, *et al*: Spontaneous Resolution of Dysgeusia. Arch Otolaryngol Head Neck Surg 1996; 122(9):961–963.

Resource
1. Lareb. http://www.lareb.nl

Answered by: Dr. Robert Bailey and Dr. Mike McCall

5. Burning in the Legs

What medication should you recommend to a patient living with a paraplegic disease (D3 level), who complains of feeling a burning in his legs (Gabapentin at 600 mg, q.i.d. is not efficient)?

Submitted by: Louis Roy, MD, Québec, Québec

If a patient is on a certain medication for symptomatic control and does not seem to derive benefit at one dose, the first step would be to increase the dose to the maximum level or until side-effects occur. Gabapentin, in this case, may be increased to 2,700 mg total daily dose. If there is no benefit, then other medications, such as pregabalin, may be used starting at a dose of 75 mg b.i.d. and gradually increased to the maximum dose of 300 mg b.i.d. Other options include tricyclic antidepressants, such as amitriptyline or nortriptyline.

Answered by: Dr. Abdul Qayyum Rana

6. Anticonvulsants and Bipolar Disorder

Please advise on which of the modern anticonvulsants are also useful in the management of bipolar illness.

Submitted by: Alexander Shearer, MD, Surrey, British Columbia

A series of newer anticonvulsants have been marketed over the past decade; they include gabapentin, pregabalin, and topiramate. Although some uncontrolled case reports and case series have suggested that these newer anticonvulsants may have some efficacy in treating patients with bipolar disorders, adequately powered controlled studies have failed to support these findings and have concluded that these newer anticonvulsants are no better than placebo as either monotherapy or as adjunctive therapy in bipolar disorders.

So far, carbamezapine, valproate, and lamotrigine are the anticonvulsants with proven efficacy in bipolar disorders.

Resource
1. Sadock BJ, Sadock VA, Ruiz P (eds.)Kaplan and Sadock's Comprehensive Textbook of Psychiatry. 9th edition. Lippincott, Williams, & Wilkins, Philadelphia, 2009.
2. Handbook of Bipolar Disorders. Terence A Ketter (ed.). American Psychiatric Publishing Inc., Arlington, Virginia, 2010.

Answered by: Dr. Hany Bissada

7. Morphea

What is morphea and how is it treated?

Submitted by: Denise Wexler, MD, London, Ontario

Morphea (also referred to as localized scleroderma) is a skin disorder characterized by thickening and induration of the skin and subcutaneous tissue due to excess collagen deposition. Clincally, it presents as an ivory coloured firm plaque, often round or oval, surrounded by an erythematous or purplish halo.

Morphea can be classified into several subtypes, including plaque, guttate, generalized, linear, and deep.

Morphea needs to be differentiated from progressive systemic sclerosis, which often presents in the skin with Raynaud's phenomenon and sclerodactyly.

There is no definitive therapy for morphea, and most treatments are not based on randomized controlled trials. Plaque morphea may not need to be treated, as it often resolves spontaneously over three to five years.

Treatment is necessary for cosmetically disfiguring cases, generalized cases, and cases likely to produce contractures.

Treatment of plaque morphea usually consists of a superpotent topical corticosteroid. Treatment with calcipotriol may also be helpful. There are also reports of topical tacrolimus 0.1% ointment and imiquimod 5% cream being helpful.

Treatment of more widespread and aggressive cases is best left to physicians experienced in treating these conditions, but it often includes systemic corticosteroids (both oral and pulsed intravenous steroids) in conjunction with weekly oral methotrexate. Oral hydroxychloroquine and penicillamine may rarely be helpful. Ultraviolet therapy may also be useful, especially UVA1 (340 to 400 nm), but PUVA therapy, narrowband UVB, and even broadband UVA may also be of benefit.

Answered by: Dr. Richard Haber

8. Causes of Alopecia

What factors may play a role in the causation of alopecia? Stress? Malnutrition?

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

To answer this question, one must characterize alopecias, which are divided into nonscarring (noncicatricial) and scarring (cicatricial). This question suggests the physician is asking about factors that play a role in telogen effluvium, a common type of nonscarring alopecia.

Telogen effluvium is characterized by diffuse hair shedding, often with an acute onset. The diffuse hair loss often starts three months after the precipitating event, as an increased number of hairs change from the anagen to the telogen phase; the telogen phase lasts approximately three months and those hairs are then shed.

Precipitating causes of telogen effluvium include postpregnancy (especially after delivery), severe weight loss (especially crash dieting), major illnesses (including febrile illnesses), surgery and general anesthetic, post trauma, and severe psychological events. Other causes include drugs (especially anticoagulants, beta blockers, oral retinoids, and propylthiouracil), hypo- or hyper-thyroidism, and iron deficiency.

Therefore severe stress can precipitate a telogen effluvium. Severe protein deficiency states, such as kwashiorkor and marasmus, can cause hair loss but this is not normally seen with patients in North America on normal diets. Iron deficiency, especially with iron deficiency anemia (but even without anemia), may contribute to a telogen effluvium.

When faced with a patient with diffuse non-scarring hair loss with increased shedding, telogen effluvium is the most likely diagnosis (although androgenetic alopecia is still a consideration).

In cases where a definite precipitating factor cannot be elicited in the history, I would suggest checking CBC, TSH, serum iron and TIBC, and serum ferritin to rule out metabolic causes of telogen effluvium.

Answered by: Dr. Richard Haber

9. DVT Risk after Surgery

How long, post-operatively, is a person "at risk" of having a DVT following surgery?

Submitted by: David Hawkins, MD, Kelowna, British Columbia

Patients are at risk of both venous and arterial thromboembolic events in the post-operative setting. Post-operative DVT risk depends on many factors, including the type of surgery and baseline patient comorbidities and risk factors. In general, major surgery leading to prolonged immobilization has a higher risk of post-operative thrombosis. Orthopedic surgery in particular confers a very high risk. Patients with cancer, prior thrombosis, or who have an underlying hypercoagulable state are also at a higher risk of thrombosis in this setting. To date, there are no validated studies to guide management in the duration of DVT prophylaxis in most surgical patients. The most evidence is in orthopedic surgery where the American College of Chest Physicians guidelines by Geerts, Bergqvist, Pineo, *et al* have made recommendations.[1] DVT prophylaxis for post-operative major orthopedic surgery should be at least 10 days and up to 35 days. It is recognized that current hospital stays for these patients may be less than a week, and, thus, prophylaxis should be continued with outpatients.

The role of DVT prophylaxis must be weighed against the potential risk of bleeding. DVT prophylaxis is more complicated in patients who are already on antiplatelet agents or anticoagulants, who require perioperative management of their anticoagulation. However, further guidance from the American College of Chest Physicians is available on this topic.

Reference

1. Geerts WH, Bergqvist D, Pineo GF, *et al*: Prevention of Venous Thromboembolism: American College of Chest Physicians Evidence-based Clinical Practice Guidelines. Chest 2008;133(6 Suppl): 381S–453S.

Answered by: Dr. Cyrus Hsia and Dr. Leonard Minuk

10. Twitching Eyelid

A patient presents with a chronic twitching eyelid. What do you check? How do you treat it?

Submitted by: Shahbegum Meghji, MD, Edmonton, Alberta

Eyelid twitching can be due to several causes; some of these include myokymia, hemifacial spasm, aberrant regenerations after facial nerve palsy, and blepharospasm.

Myokymia involves spontaneous, fine fascicular contractions of the orbicularis oculi muscle of one of the lower eyelids. However, occasionally upper eyelids can be affected as well. There is no weakness of the orbicularis occuli muscle. Myokymia is often benign or self-limited and does not require any treatment. Rarely, eyelid myokymia may be a sign of brainstem lesions or an initial manifestation of hemifacial spasm.

Hemifacial spasm is usually unilateral and causes intermittant clonic movements that affect the upper face and also involve the lower face in most cases.

Blepharospasm is mostly bilateral and is characterized by tonic spasms of orbicularis occuli muscles of the eyelids. Patients with blepharospasm have a history of spontaneous eye closure, which may be aggravated by light, wind, or emotional stresss leading to functional blindness in some cases.

Eyelid twiching after facial nerve palsy is due to abberrant regeneration of the facial nerve. The abnormal movements are triggered by chewing or swollowing and are referred to as synkinesis. Brain imaging such as MRI may be performed if CNS pathology is suspected, as in cases of hemifacial spasm or brainstem lesions. Treatment depends upon the underlying cause. Botulinum toxin injections are helpful in the treatment of eyelid twiching in hemifacial spasm, blepharospasm, myokymia, and synkinesis due to facial nerve palsy.

Reference
1. Rana AQ: An Aid to Neuro-ophthalmology. Authorhouse, Bloomington, Indiana, 2009, 7–10.

Answered by: Dr. Abdul Qayyum Rana

11. Treating Anal Fissures

Do intra-anal inserts of nifedipine gel successfully treat anal fissures, as calcium channel blocking prevents muscle contraction?

Submitted by: Rajen Ramgoolam, MD, Winnipeg, Manitoba

Anal fissures are frequently encountered by primary care physicians and gastroenterologists. Trauma to the anal canal by the passage of hard, bulky stools or explosive diarrhea results in a longitudinal tear in the anorectum extending from the anal verge to the dentate line. Decreased blood flow to the posterior area of the anoderm and increased tone with spasm in the internal sphincter muscle result in ischemic ulceration and contribute to chronic fissure formation. The fissure causes severe pain during and after defecation. Acute fissures usually resolve with conservative management including stool softners, high fiber diet, topical lidocaine, and sitz baths. If symptoms persist past four weeks, it becomes chronic and usually requires additional therapy for resolution.

No single medical therapy has been demonstrated to be superior to others in chronic anal fissures. There are three classes of medical therapy, including nitrates, calcium channel blockers, and botulinum toxin A, that have been proven effective in treating chronic anal fissures.

Calcium channel blockers (CCB) work by relaxing the internal anal sphincter, since the maintenance of the internal sphincter tone is mediated by calcium dependent mechanisms. Topical therapy with 0.2% nifedipine and diltiazem ointments have been shown to heal 47 to 89% of patients in various studies. Rescue therapy after failure of treatment with nitrates resulted in a 49% healing rate after eight weeks of use in one study. Oral nifidepine therapy was used in a pilot study; 20 mg b.i.d. resulted in healing of 60% of fissures at 10 weeks. Common adverse events include flushing, minor headache, and perianal itching in 10% of patients. Two randomized controlled trials evaluating diltiazem versus nitroglycerine did not show a difference in efficacy of both the agents; however, topical CCB were tolerated better than nitroglycerine.

Topical nitroglycerine ointment, in strengths from 0.1 to 0.4% have been used with gradual dose escalation up to three times daily dosing. Response rates vary from 40% to 86% with nitroglycerine with a dose dependent effect.

Botulinum toxin A is being used with increasing popularity. It inhibits acetylcholine release and subsequently causes decreased internal anal sphincter tone. Studies show that 73% of chronic anal fissures heal; however, recurrence is common. At 42 weeks post therapy, only 41% were free from symptoms. Transient incontinence to flatus and stool soilage is common initially.

Although no one medical therapy has been consistently shown to be superior in treating anal fissures, our approach for chronic anal fissures includes the use of 2% diltiazem gel two or three times daily for four weeks. We avoid nitrates initially because of the increased rates of headaches that can decrease compliance with therapy. Moreover, botulinium toxin A injection is an invasive procedure that is as effective as CCB's, and, as such, our preference is to use the least invasive treatment available with a favourable side-effect profile. If these measures fail, or if the fissure recurs, we discuss retreatment with medical therapy or surgical management with a lateral sphinterotomy.

Answered by: Dr. Robert Bailey and Dr. Vijay Selvarajah

12. Sucralfate in Peptic Ulcer Management

What is the use of sucralfate in the management of peptic ulcer disease?

Submitted by: Peter Palma, MD, Fort McMurray, Alberta

Sucralfate is a medication that acts as a chemical barrier for prevention of peptic ulcer disease. Unfortunately, the clinical trials of sucralfate have had mixed results. With the introduction of proton pump inhibitors demonstrating superior clinical efficacy, there does not seem to be any role for sucralfate for this indication.

Answered by: Dr. Richmond Sy

13. Potassium and Diuretic Use

How often should potassium be checked with stable diuretic use?

Submitted by: Roger Hamilton, MD, Wolfville, Nova Scotia

Hypokalemia is a relatively common dose-dependent side-effect of diuretics, and life-threatening hyperkalemia can occur with potassium sparing diuretics, such as spironolactone.

Serum potassium should generally be checked two to three weeks after initiation of diuretics. Thereafter, a steady state is achieved, and repeat monitoring of electrolytes is indicated only if there is a dosage change, if extrarenal potassium loss occurs (*e.g.*, diarrhea), or if there is a decrease in potassium intake.

Patients require more frequent electrolyte monitoring if they are on large doses of diuretics (*e.g.*, furosemide 80 to 240 mg daily or metolazone), are on spironolactone, have moderate renal dysfunction (GFR < 60 mL/min), have heart failure, or have cirrhosis. Hypokalemia may precipitate arrhythmias in the setting of heart disease and the serum potassium should be maintained in the 4 to 5 mmol/L range. Hypokalemia may also increase blood pressure in patients with hypertension and precipitate encephalopathy in patients with cirrhosis.

Answered by: Dr. Bibiana Cujec

14. HbA1c as an Indicator of Glycemic Control

Are there factors that cause HbA1c, now better known as A1c, to be an inaccurate reflection of long-term glycemic control (*i.e.*, false elevations or falsely low values)?

Submitted by: Craig Render, MD, Kelowna, British Columbia

The HbA1c (A1c) correlates with the patient's mean glucose concentration over the last two to three months. The assay is well standardized; however, there are a few caveats where the A1c may over or underestimate mean glucose control, due to biological and patient-related factors. First, in patients who have very wide swings in blood glucose levels, one has to be cautious in solely using the A1c in monitoring and adjusting treatment, especially if one has many hypoglycemic readings. In this setting, one has to also look carefully at the self monitored glucose readings. The A1c is also influenced by RBC survival, and, thus, any condition that shortens RBC turnover will falsely increase A1c, as in iron, folate, and B12 deficiency anemia. Rapid RBC turnover will lead to a falsely decreased A1c as in hemolysis, acute bleeding, and patients treated for iron, B12, or folate deficiency. Certain hemoglobinopathies may falsely elevate A1c values (Hb F and Hb S); however, this is not a problem with most assays. Chronic renal failure may also falsely elevate or decrease A1c levels. In the above situations, fructosamine may be used; however, it reflects the mean glucose control over the last one to two weeks, and, if the albumin level is low or the turnover is low, it can give inaccurate results. Furthermore, fructosamine is not as well standardized as the A1c. Despite a few sources of error, the A1c is the best test to monitor long-term glucose control.

Answered by: Dr. Ally Prebtani

15. Screening for Hemochromatosis

Should screening be done for hemochromatosis in all Caucasian patients?

Submitted by: Juliet Kwong, MD, Vancouver, British Columbia

General population-based screening for hereditary hemochromatosis (HH) is not recommended. Clinical features suggestive of HH may include a known family history, individuals in their fifth or sixth decade of life who present with darkened (or tanned-appearing) skin, arthritis, type 2 diabetes mellitus, liver function abnormalities or hepatomegaly, and/or cardiomyopathies without other underlying causes. Hereditary hemochromatosis has been well studied by the Hemochromatosis and Iron Overload Study (HEIRS) investigators.[1]

Initial screening can be done using serum ferritin and transferrin saturation when guided by clinical features. Ferritin, a macromolecule that contains thousands of iron atoms, is generally an intracellular storage protein for iron. Approximately 1% of the body's ferritin is soluble in plasma and is in equilibrium with the intracellular storage ferritin. Serum ferritin, albeit an imperfect measure of the body's iron stores, is a noninvasive and cheap test; it is generally a better screening test for iron deficiency than for iron overload. It is a nonspecific test of iron content, as ferritin is an acute phase reactant and may become falsely elevated in the presence of inflammation. A more sensitive, yet still inexpensive and noninvasive, test for iron overload is transferrin saturation (T. sat). This is a measure of the percentage of transferrin molecules, which are the body's iron transporters that are bound to iron. No absolute elevation in transferrin saturation is agreed upon as a universal standard; however, levels above 60% in males and 45 to 50% in females should raise suspicion and call for a repeat test. The gold standard to diagnose iron overload is still an invasive liver biopsy. For analysis of the most common mutations seen in the Caucasian populations with HH, samples can be sent for molecular diagnostic testing for mutations in the HFE gene.

Reference

1. Adams PC, Reboussin DM, Barton JC, *et al*: Hemochromatosis and Iron-overload Screening in a Racially Diverse Population. N Engl J Med 2005;352(17):1769–1778.

Answered by: Dr. Cyrus Hsia and Dr. Leonard Minuk

16. Treatment Options for Trigeminal Neuralgia

What other treatment options are available for trigeminal neuralgia that does not even respond to opioids?

Submitted by: Shiraz Aziz, MD, Richmond, British Columbia

Carbamazepine is the medication of choice for treatment of trigeminal neuralgia. Other medications that have proven effective are phenytoin, gabapentin, pregabalin, amitriptyline, and baclofen. With the progression of the disease, increased doses of medications may be required, which may lead to side-effects in some cases. Use of opioids is generally discourged in trigeminal neuralgia and headache management overall.

In the refractory cases of trigeminal neuralgia, surgical procedures may be considered. However, in some cases medications may still be required even after the surgery has been performed, although in a much lower dosage.

Several surgical procedures have been used for the treatment of trigeminal neuralgia, including microvascular decompression and rhizotomy. Each surgical procedure has its potential benefits as well as its risks. One surgical procedure may not be effective in every case. The selection of surgical procedures may vary case by case and depends upon many factors.

Microvascular decompression surgery involves placing a teflon felt implant between the offending blood vessels and trigeminal root, which helps to alleviate the neurovascular compression. Percutaneous rhizotomy can be done in several ways, such as injecting glycerol into the trigeminal nerve and gasserion ganglion, compressing the gasserion ganglion with an inflated baloon, or thermally damaging the gasserion ganglion with a heated electrode. Gamma knife radiosurgery by cobalt radiations, peripheral trigeminal nerve block, sectioning of the peripheral part of the trigeminal nerve, and microsurgical rhizotomy, which involves surgical sectioning of the trigeminal nerve root near its entry into the brain stem, may be tried in some cases.

Reference

1. Rana AQ: A Synopsis of Neurological Emergencies. Authorhouse, Bloomington, Indiana, July 2009, 51–68.

Answered by: Dr. Adul Qayyum Rana

17. Biologics and Psoriasis

In patients with psoriasis who have failed topical therapy, what is the role of biologics?

Submitted by: Mohamed Ravalia, MD, Twillingate, Newfoundland

Biologic therapy of psoriasis consists of drugs administered by injection or infusion that target the T cells or chemical mediators that have been shown to be involved in the pathogenesis of psoriasis. These drugs are either monoclonal antibodies or fusion proteins that target T cell activation, tumour necrosis factor-α, or interleukins-12 and -23. Biologic therapy for psoriasis is usually considered for moderate to severe cases, defined as a psoriasis area severity index (PASI) > 10 (range 0 to 72) or a body surface area (BSA) > 10% and dermatology life quality index (DLQI) >10 (range 0 to 30). Special consideration can be given to severe psoriasis of lesser extent but involving functionally significant sites, such as the palms, soles, or genital area.

In patients who fail topical therapy, dermatologists would then consider ultraviolet phototherapy, systemic therapies (methotrexate, cyclosporin or acitretin), and biologic therapies as potential next treatments. This would be decided after discussions with the patients regarding the risk-benefit ratio of each treatment; treatment needs to be individualized for each patient. Biologic therapies have certainly been shown to lead to significant improvement in patient's PASI and DLQI scores, and, for many patients, they have been life-altering treatments.

Answered by: Dr. Richard Haber

18. Mohs Surgery

When should a patient be referred for Mohs surgery?

Submitted by: Catherine McCuaig, MD, Montréal, Québec

Mohs micrographically controlled surgery is the gold standard for removal of non-melanoma skin cancer, especially basal cell carcinoma. In Mohs surgery, cancerous tissue is removed one layer at a time with frozen section control until the whole tumour has clear tissue margins; the residual surgical wound is then repaired.

Mohs gives the best efficacy with recurrence rates of 1% at 5 years for previously untreated basal cell carcinoma (primary basal cell carcinoma). Recurrence rates with other treatment modalities for primary basal cell carcinoma are listed in Table 1.

Table 1
Recurrence Rates by Treatment

Surgical Excision	10%
Curettage and Electrodesication	7.7%
Radiation	8.7%
Cryotherapy	7.5%

The indications to refer a patient for Mohs surgery include poorly defined tumours, morpheaform basal cell carcinoma, and high-risk or incompletely removed tumours or recurrences in sites where tissue conservation is imperative (*e.g.*, eyelids, nose, lips). Also Mohs is very useful in immunosuppressed patients with skin cancers or in sites of previous radiation.

Answered by: Dr. Richard Haber

19. Antidepressants for Chronic Pain

What are some new antidepressants for chronic pain?

Submitted by: Anonymous

Patients with chronic pain commonly experience different kinds of pain. The distinctions are important, because they call for different treatment strategies. In nociceptive pain, both somatic and visceral pain are more responsive to NSAIDs, if the pain is mild, and to opiates, if the pain is moderate to severe. Neuropathic pain is generally less responsive to NSAIDs and opioids but may be effectively treated with antidepressants. Serotonin-norepinephrine reuptake inhibitors (SNRIs), such as duloxetine, which has recently received FDA approval for specific chronic non-malignant pain syndromes, may be used for the treatment of chronic pain; the suggested dose is 90 mg per day for pain management. Tricyclic antidepressants (TCAs), such as nortriptyline and amitritptyline, are effective in relieving neuropathic pain, purportedly by inhibiting reuptake of norepinephrine and serotonin in descending pain-modulating systems. Amitriptyline has been a popular drug for treatment of chronic pain syndromes and migraine headache prophylaxis. During treatment of pain, the usual dose of amitriptyline is lower (*e.g.*, 75 mg per day) than that in depression, and effects occur more quickly, usually within the first two weeks.

Answered by: Dr. Hany Bissada

20. Niacin Contraindications

Other than someone with a known allergy to niacin, who would be advised not to take it?

Submitted by: John Dawson, MD, Ottawa, Ontario

Niacin is occasionally used to treat low HDL levels and high triglycerides. Niacin should not be used in patients with active liver disease, persistent, unexplained, elevated liver transaminases (AST/ALT), active peptic ulcer disease, and arterial hemorrhage. Caution needs to be exercised in treating patients with diabetes, since it may worsen glycemic control; with gout, since it may exacerbate hyperuricemia; with active gallbladder disease, since this may also be exacerbated; and with acute coronary syndrome, since this may be exacerbated due to vasodilation from the niacin.

Answered by: Dr. Ally Prebtani

21. When to Stop Bisphosphonates

When should you stop bisphosphonates in post menopausal women?

Submitted by: **Sylvie Gill, MD,** Sorel-Tracy, Québec

There is no clear consensus on how long to treat patients with bisphosphonates. The most widely referred to study that helps to answer this question is the FLEX trial, which was an extension of the FIT trial.[1] Women who received alendronate for five years in the FIT trial were randomized to continue alendronate for another five years or to take placebo. Note that women at the highest risk for fracture were excluded in this trial. At 10 years, patients who took placebo had a gradual decline in their bone mass density (BMD), as well as a gradual rise in their bone turnover markers; however, their BMD remained higher than the level 10 years earlier. Rate of non-vertebral fracture was not significantly different, but there was a slightly higher risk of clinically detected vertebral fractures (5.3 and 2.4% for placebo and alendronate respectively).

From this information, stopping alendronate after five years might be reasonable for low risk women based on their assessment of fracture risk as well as the clinical judgement of the physician. Since high risk patients were excluded from the FLEX trial, continuing alendronate in this population is recommended; however, there is no data beyond 10 years.

Current Canadian guidelines state that "there is little evidence to support any recommendation regarding duration of therapy or the use of drug holidays."[2] They recommend that patients at "high risk for fracture should continue osteoporosis therapy without a drug holiday." Dx

Reference

1. Watts NB, Chines A, Olszynski WP, *et al*: Fracture Risk Remains Reduced One Year After Discontinuation of Risedronate. Osteoporos Int 2008; 19(3):365–372.
2. Papaioannou A, Morin S, Cheung AM, *et al*: 2010 Clinical Practice Guidelines for the Diagnosis and Management of Osteoporosis in Canada: Summary. CMAJ 2010; 182(17):1864–1873.

Answered by: **Dr. Michael Starr**