99 Topics for the CCFP

Collaboratively developed, peer-reviewed study notes covering the Priority Topics for the Canadian Certification Examination in Family Medicine

Brady Bouchard
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Corrections, comments, and contributions are very gratefully welcome. Please feel free to get in touch with us via email at 99topics@drbouchard.ca.

Newer evidence is highlighted in yellow in the digital version.

99 Topics for the CCFP

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The most up-to-date version of these study notes is always available at: https://99topics.drbouchard.ca/.
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1. ACLS

This topic is unique in the Priority Topics, in that it is covered in depth through a dedicated (and for most residents, mandatory) course. We will not duplicate that content here — the ACLS algorithms are important to know, and can be found elsewhere. Instead, we discuss related topics, and take an alternative approach to some of the clinical scenarios presented in the ACLS course.

### KEY FEATURES

- Keep up to date with advanced cardiac life support (ACLS) recommendations (i.e., maintain your knowledge base).
- Promptly defibrillate a patient with ventricular fibrillation (V fib), or pulseless or symptomatic ventricular tachycardia (V tach).
- Diagnose serious arrhythmias (V tach, V fib, supraventricular tachycardia, atrial fibrillation, or second- or third-degree heart block), and treat according to ACLS protocols.
- Suspect and promptly treat reversible causes of arrhythmias (e.g., hyperkalemia, digoxin toxicity, cocaine intoxication) before confirmation of the diagnosis.
- Ensure adequate ventilation (i.e., with a bag valve mask), and secure the airway in a timely manner.
- In patients requiring resuscitation, assess their circumstances (e.g., asystole, long code times, poor pre-code prognosis, living wills) to help you decide when to stop. (Avoid inappropriate resuscitation.)
- In patients with serious medical problems or end-stage disease, discuss code status and end-of-life decisions (e.g., resuscitation, feeding tubes, levels of treatment), and readdress these issues periodically.
- Attend to family members (e.g., with counselling, presence in the code room) during and after resuscitating a patient.
- In a pediatric resuscitation, use appropriate resources (e.g., Braeslow tape, the patient's weight) to determine the correct drug doses and tube sizes.

An initial approach to the critically ill patient for the occasional resuscitationist

- **Airway:** open or closed?
- **Breathing:** yes or no?
- **Circulation:** is there a pulse or not? — if not, immediately start chest compressions.
- **Diabetes:** a spot sugar should be part of your vital signs, especially in pediatric patients.
- Ask the nurse to add supplementary oxygen (set to flush/full open, check the tubing from wall to patient, use a non-rebreathing mask).
- Ask for the patient to be hooked up to your monitors (continuous telemetry with pads, end tidal CO₂, pulse oximetry, and blood pressure (cycling fast)), and ask for a full set of vitals.

### PRIMARY SURVEY

**ABCD-OMIP** — Airway, Breathing, Circulation (pulse), Diabetes (spot sugar) then Oxygen, Monitors, IV Access, Pulse (recheck)

**Patients without a pulse — CPR**

- Good chest compressions: at least 100/min, 1/3rd depth of chest wall.
- Pulse check for a maximum of 10 seconds: if you don't feel it in 10 seconds, assume it isn't there.
- If the patient doesn't have a pulse, regardless of cardiac rhythm, they get adrenaline 1mg IV push. New in 2016: Vasopressin 40 IU IV push has been removed as an alternative to adrenaline in CPR.
- also consider when to stop: see Cessation of CPR.

**Patients with a pulse — take a second and breath, then think**

Stable VT, SVT, AF, or 2nd or 3rd degree heart block will usually give you a moment to check your algorithms.

- 3 questions to ask yourself:
  - Is the patient stable or unstable?
  - Is the rhythm regular or irregular?
  - Is the QRS complex wide or narrow?
Reversible causes of arrhythmias

HYPERKALEMIA

- Think of ↓K in renal patients.
- ECG changes: think of a string attached to the top of the T wave, pulling up: a tall, peaked T wave, widened QRS, which can progress to sine wave and PEA.
- Confirm the hyperkalemia if ECG normal and no reason for the patient to be hyperkalemic; think hemolysis (pseudohyperkalemia).
- Immediate treatment only needed if ECG changes or $K^+ > 7.0$:
  - Give calcium gluconate 10ml (1 amp) infused over 3 minutes to stabilize the myocardium.
  - Give 10 units of regular/rapid-acting insulin over 60 minutes in 500ml of D5.
  - If severe, can give salbutamol 10mg (double neb), although short-acting.
- If no ECG changes and $K^+ < 7.0$, no immediate treatment necessary: look for the cause and fix (missed dialysis? ACE-i? NSAIDs?)
- Immediate treatment only shifts $K^+$; you need to reduce total body potassium:
  - Lasix 40-160mg IV (if they are not anuric).
  - There is no role ✅ for Kayexalate in the acute setting, but reasonable for total body potassium excretion as an in-patient.
  - Continue short-acting treatments until you can get them to dialysis.

DIGOXIN TOXICITY

Digoxin has a very narrow therapeutic index.

- Gastrointestinal distress.
- Hyperkalemia.
- Cardiac arrhythmias and/or AV nodal blockade.
- Increased automaticity.
- Definitive treatment is Digibind. Indicated when:
  - Cardiac arrest.
  - Life-threatening dysrrhythmia.
  - $K^+ > 5.0$.
  - 10mg ingested in an adult, >4mg in a child.
  - Serum level at 6 hours of $>12ng/ml$ (need to measure at least 6 hours after ingestion, may be falsely elevated earlier due to the time it takes for digoxin to migrate into tissues).

COCAINE

- Causes MI/angina due to strong and unpredictable vasoconstrictive, vasospastic effects (even if patients without risk factors).
- Sympathomimetic effects.
- Sodium channel blockade (anesthetic properties, conduction abnormalities).
- Presynaptic catecholamine uptake blockade (catecholamine excess).
- Effects on $K^+$, $Ca^{2+}$ channels.
- Can prolong QT interval.
- Manage cocaine-induced MI the same, except no beta blockers (?theoretical risk of worsening vasoconstriction due to unopposed alpha stimulation).
CESSATION OF CPR

A general approach is to stop CPR after 20 minutes if there is no ROSC or viable cardiac rhythm re-established, and no reversible factors present that would potentially alter outcome.

- Survival is highly dependent on time to defibrillation and time to return of spontaneous circulation (ROSC)
- Survival to discharge from out-of-hospital arrest (OOHA) is minimal if the initial rhythm on arrival in ED is asystole or agonal (if asystole, double-check leads!)
- Questions to ask:
  - Was arrest observed?
  - What was the initial rhythm?
  - Cause known? reversible?
  - Time to CPR? (how many shocks, how many drugs)
  - Time to defibrillation?
  - Advanced directive?
  - Non-survivable injuries? (i.e. 100% burns)
  - Premorbid state? (dementia, cancer)
  - Talk to family if possible!
- Some special cases where you can continue CPR longer:
  - Hypothermia ("not dead until warm and dead").
  - Asthma (correct the dynamic hyperinflation and they may come back).
  - Toxicological arrest (full neurological recovery after >4 hours CPR is possible).
  - Thrombolytics given (should continue up to 2 hours post-administration).
  - Pregnancy (if fetus is at a viable age), to allow for Caesarean section.

RECOMMENDED #FOAMED RESOURCES

Weingart, S. Is Kayexalate Useless? http://emcrit.org/misc/is-kayexalate-useless/
Nickson, C. Cessation of CPR. http://lifeinthefastlane.com/ccc/cessation-of-cpr/

REFERENCES

2. Abdominal Pain

### KEY FEATURES

1. Given a patient with abdominal pain, paying particular attention to its location and chronicity:
   a) Distinguish between acute and chronic pain.
   b) Generate a complete differential diagnosis (ddx).
   c) Investigate in an appropriate and timely fashion.

2. In a patient with diagnosed abdominal pain (e.g., gastroesophageal reflux disease, peptic ulcer disease, ulcerative colitis, Crohn’s disease), manage specific pathology appropriately (e.g., with medication, lifestyle modifications).

3. In a woman with abdominal pain:
   a) Always rule out pregnancy if she is of reproductive age.
   b) Suspect gynecologic etiology for abdominal pain.
   c) Do a pelvic examination, if appropriate.

4. In a patient with acute abdominal pain, differentiate between a surgical and a non-surgical abdomen.

5. In specific patient groups (e.g., children, pregnant women, the elderly), include group-specific surgical causes of acute abdominal pain in the ddx.

6. Given a patient with a life-threatening cause of acute abdominal pain (e.g., a ruptured abdominal aortic aneurysm or a ruptured ectopic pregnancy):
   a) Recognize the life-threatening situation.
   b) Make the diagnosis.
   c) Stabilize the patient.
   d) Promptly refer the patient for definitive treatment.

7. In a patient with chronic or recurrent abdominal pain:
   a) Ensure adequate follow-up to monitor new or changing symptoms or signs.
   b) Manage symptomatically with medication and lifestyle modification (e.g., for irritable bowel syndrome).
   c) Always consider cancer in a patient at risk.

8. Given a patient with a diagnosis of inflammatory bowel disease (IBD) recognize an extra-intestinal manifestation.

### Causes

#### Pain By Region

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<thead>
<tr>
<th>RUQ</th>
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<th>LUQ</th>
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<tr>
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<td>Biliary colic</td>
<td>Dyspepsia</td>
<td>Splenic infarction (think: AF)</td>
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<td>Ascending choleangitis</td>
<td>Pneumonia</td>
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<td>Bowel obstruction</td>
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12 — Last peer reviewed: November 13, 2015
Abdominal Pain:
It’s a spectrum

- Acute
  - Pain for hours or days
  - Pain worsening, often severe

- Chronic
  - Pain for months to years
  - Pain steady (may wax or wane), or slowly worsening

- It’s important to try to differentiate acute from chronic pain as it can narrow our differential and focus your physical exam and investigations, and can give an indication of the required timeliness of interventions (a.k.a. sending the patient to Emergency if not already there).

- Pain with both acute and chronic features requires looking at a wider differential.

- The ‘surgical abdomen’ rarely presents as chronic pain.

- In both acute and chronic presentations of abdominal pain, consider cancer:
  - Does the patient have constitutional symptoms?
  - Does the patient have risk factors? Hereditary CRC syndromes, familial adenomatous polyposis, Lynch (nonpolyposis) syndrome, alcohol use/abuse, obesity, advancing age, a history of IBD.

THE ACUTE ABDOMEN WITHOUT PAIN
Be wary of sick-looking patients with minimal pain — some patients with an acute abdomen present with minimal or no pain: the elderly, the immunocompromised, children, and pregnant women in their 3rd trimester.

SPECIFIC PRESENTATIONS

- Obstruction: sudden onset of generalized, diffused abdominal pain, anorexia, bloating, nausea and vomiting (depending on the level may be bilious or feculent, shorter time between meals and vomiting usually means more proximal obstruction).

- Pancreatitis: severe abdominal pain that lasts for days, banding pain radiating to the back, severely dehydrated due to 3rd spacing, and often severe nausea and frequent vomiting.

- Ascending Cholangitis: fever, jaundice, RUQ pain.

ACUTE CHOLANGITIS
Charcot’s Triad — jaundice, fever, RUQ pain
Reynaud’s Pentad — jaundice, fever, RUQ pain, shock, ↓ LOC

SPECIFIC POPULATIONS

- Children: see Acute abdominal pain in children.

- Women: ectopic pregnancy until proven otherwise! Also think of endometriosis (cyclical pain), PID (at risk patients, previous STIs, cervical motion tenderness, vaginal discharge)

- Older patients: have a low threshold for investigation. They often present with non-specific or generalized symptoms — the frequency of misdiagnosis is high. Common things in this population: appendicitis, aneurysmal disease, mesenteric ischemia, diverticulitis, and small bowel obstruction.

- Trauma patients: hemorrhage from solid organ laceration or fluid loss, organ ischemia from vascular injury, and infection from perforated hollow viscus

- In neonates: volvulus (as a complication of malrotation) and necrotizing enterocolitis. Intussusception (invagination of a part of the intestine into itself, causing obstruction.

- Among children of all ages: appendicitis can cause peritoneal irritation and focal tenderness.

- In patients with multiple previous surgeries: Obstruction as the result of adhesions from previous surgery or inflammation.

- Immunocompromised: These patients generally appear sicker than immunocompetent patients. Consider spontaneous bacterial peritonitis (SBP).
THE SURGICAL ABDOMEN

A condition that is likely to rapidly deteriorate without surgical intervention.

- Pain is often severe.
- Pain may be refractory to analgesia.
- Patients often lie prostrate, and "look sick".
- Symptoms typically evolve rapidly.
- Patient may be vitals unstable, dehydrated.
- Signs of peritonitis: abdominal wall rigidity, percussion/rebound tenderness, involuntary guarding, absent or diminished bowel sounds, and tenderness to light palpation.
- Children with peritoneal irritation remain still or resist movement, while patients with visceral pain change position frequently, often writhing with discomfort. Test children for peritonism by asking them to hop or run (the "hopping test") [Kim].
- It’s important to consider the surgical abdomen in the elderly, who may present with more chronic pain course, and who may be minimally symptomatic: bowel obstruction, bowel ischemia.

Physical exam

The physical examination for abdominal pain in a childbearing woman is not complete unless a pelvic examination is completed.

The physical examination for abdominal pain in a male patient is not complete unless the genitals and inguinal regions are examined.

Investigations

- Investigate in an appropriate and timely fashion.
- Should include basic labs, as well as specific tests based on your differential:
  - CBC, U/A, creatinine, glucose. Add LFTs, bilirubin (direct and indirect), for suspected hepatic or biliary disease. Lipase/amylase for pancreatitis.
- Pregnancy test in all women of childbearing potential.
- Celiac disease: Anti-tissue transglutaminase (TTGA) IgA, total serum IgA levels, deamidated gliadin peptides (DGP). These tests are approx. 90% sensitive, but only if the patient is still on a diet containing gluten. High clinical suspicion should prompt referral for duodenal biopsy without antibody testing (likely to be a false negative in this case).
- Plain film 3 views of abdomen/CT (reasonable screen for bowel obstruction, ileus, free air (upright)), although often not helpful.
- Ultrasound (gallbladder disease, gynecological problems, AAA, appendicitis, renal stones). Should generally be the first-line imaging choice in the acute abdomen.
- CT Abdo (~20 times the radiation): may be used as an alternative to U/S for most U/S problems if U/S tech not available, or if U/S done but equivocal, but large radiation dose. Better than plain films for ileus/obstruction. If you suspect a bowel obstruction, don’t use barium.
- Gastroscopy/colonoscopy (PR blood; if you’re thinking of IBD or Celiac).

Management

GERD

- Lifestyle modifications:
  - weight loss.
  - reduce stress (changes at work or at home, meditation, exercise).
  - elevation of the head of bed.
  - avoidance of reflux-inducing foods (fatty foods, chocolate, peppermint, and excessive alcohol).
  - avoidance of meals before bedtime “no eating after 6pm”.
  - H. pylori testing and eradication.
- Consider hiatus hernia or malignancy (i.e. send for gastroscopy if persistent symptoms).
- Medications: Proton pump inhibitors, H2 Antagonist (Ranitidine, Cimetidine), Prokinetic agents (bethanechol, metoclopramide).
Chapter 2. Abdominal Pain

PUD
- Gastroscopy (useful to rule out more serious pathology, confirm diagnosis, and *H. pylori* testing).
- Eradication of *H. pylori* (after positive serology or Urea Breath Test).
- Antisecretory therapy after *H. pylori* eradication with PPIs and/or H2 antagonist.

INFLAMMATORY BOWEL DISEASE (CROHN’S / UC)
*Also see Autoimmune disorders – Inflammatory bowel disease.*
- Oral 5-aminosalicylates (eg, sulfasalazine, mesalamine).
- Antibiotics (eg, ciprofloxacin, metronidazole).
- Conventional glucocorticoids (eg, prednisone).
- Non-systemic glucocorticoids (eg, budesonide).
- Immunomodulators (eg, azathioprine, 6-mercaptopurine, methotrexate).
- Biologic therapies (eg, infliximab, adalimumab).
- Step-up versus step-down therapy.
- Warning signs that they may need gastroscopy +/- biopsy: age over 50 at first presentation, weight loss, persistent vomiting, dysphagia, unexplained anemia, hematemesis, palpable abdo mass, FHx of GI cancer.
- **Surgery is often curative for UC**, less so for Crohn’s.
- Think of *extra-intestinal manifestations*:
  - MSK: peripheral arthritis, sacroilitis, ankylosing spondylitis, osteoporosis.
  - Derm: erythema nodosum, pyoderma gangrenosum, aphthous stomatitis.
  - Eyes: uveitis, scleritis, episcleritis.
  - Hepatobiliary: primary sclerosing cholangitis (PSC).
  - Vascular: thromboembolic risk/events.
  - Renal: nephrolithiasis.

CHRONIC ABDOMINAL PAIN / IBS
- This can be difficult to diagnose and manage!
- Dietary modification (trialling an elimination diet): lactose free diet, exclusion of gas-producing foods, avoiding food allergies, testing for Cellic (important not to miss!), carbohydrate malabsorption or sensitivies (*The FODMAP Diet*), and can be as simple as increasing the intake of fiber.
- Psychosocial therapies.
- Medications: Antispasmodic agents (hyoscine, cimetropium, pinaverium, dicyclomine) Antidepressants (amitriptyline, imipramine, nortriptyline, and desipramine; paroxetine, fluoxetine, sertraline), 5-HT3 (serotonin) receptor antagonists (alostron, cilansetron, ondansetron and granisetron).

ABDOMINAL PAIN IN PREGNANCY
*See Pregnancy.*

Acute abdominal pain in children

COMMON PRESENTATIONS
- **Gastroenteritis**: Diarrhea, vomits, positive recent family history.
- **Infantile colic**: Young healthy infant with episodes of inconsolable cry and drawing up of knees, flatus.
- **Appendicitis**: Fever, anorexia, nausea/vomiting, migration of pain from central to RIF (see *Alvarado Score* or *Paediatric Appendicitis Score*).
- **Mesenteric adenitis**: Fever, peripheral lymphadenopathy (in 20%), pain more diffuse that in appendicitis, concomitant or antecedent URTI.
- **Intussusception**: Mostly <2 yrs, pain intermittent with increasing frequency, vomits (sometimes with bile), drawing up of knees, “red currant jelly” stool.
- **Meckel’s diverticulum**: Usually painless rectal bleeding. Symptoms may mimic obstruction or appendicitis.
• **Constipation:** history of same, pain mainly left-sided and suprapubic, if acute look for organic causes (i.e. obstruction).
• **UTI:** Fever, dysuria, flank/suprapubic pain, U/A shows nitrites and/or leukocytes +.
• **Testicular torsion:** Sudden onset, swollen tender testis with negative Prehn’s sign (no relief/increase of pain after lifting testicle).
• **Irreducible hernia:** Painful enlargement of previously reducible hernia +/- signs of bowel obstruction.
• **HSP:** Diffuse/colicky abdominal pain, non-blanching rash (obligatory sign), swollen ankles/knees, hematuria/proteinuria.
• **HUS:** Unwell child with bloody diarrhoea and triad of: anemia, thrombocytopenia and renal failure.
• **Lower lobe pneumonia:** Referred abdominal pain and triad of: fever, cough and tachypnoea.
• **Diabetic ketoacidosis:** Known diabetic or hx of polydipsia/polyuria and weight loss, metabolic acidosis and urine or serum ketosis
• **Sickle cell crisis:** Nearly exclusively in black children.
• **Trauma:** Always consider NAI, surgical review necessary.
• **Psychogenic:** Older child with excluded organic causes.

**PEDiatric RED FLAGS (CONSIDER FURTHER INVESTIGATIONS, ADMISSION, OR REFERRAL)**

• Septic appearance (fever, tachycardia, mottled skin, child looks generally unwell).
• Respiratory symptoms (tachypnoea is a very sensitive but not specific sign with abdominal pain; respiratory distress, barking cough).
• Generalized oedema (suspect nephrotic syndrome).
• Significant dehydration (>5% clinical dehydration and/or or >5% weight loss).
• Purpuric rash (suspect sepsis if febrile, or HSP if afebrile).
• Jaundice.
• Polyuria / polydipsia (suspect Diabetes).
• Peritoneal pain (guarding, generalised or localized rebound tenderness and/or abnormal bowel sounds).
• Feculent or bilious vomits.
• History of recent significant abdominal trauma.
• History of recent abdominal surgery.
• Irreducible hernia (or palpable abdominal mass).
• Suspected testicular torsion.
• Severe or increasing abdominal pain.
• Non-mobile or change in gait pattern due to pain.
• Bloody or “red currant jelly” stool.
• Abdominal distension.

**RECOMMENDED #FOAMED RESOURCES**


**REFERENCES**

Flasar, M. H., Cross, R., & Goldberg, E. Acute abdominal pain. Primary care, 33(3), 659–84, vi. doi:10.1016/j.pop.2006.06.004
3. Allergy & Anaphylaxis

**KEY FEATURES**

1. In all patients, always inquire about any allergy and clearly document it in the chart. Re-evaluate this periodically.
2. Clarify the manifestations of a reaction in order to try to diagnose a true allergic reaction (e.g., do not misdiagnose viral rashes as antibiotic allergy, or medication intolerance as true allergy).
3. In a patient reporting allergy (e.g., to food, to medications, environmental), ensure that the patient has the appropriate medication to control symptoms (e.g., antihistamines, bronchodilators, steroids, an EpiPen).
4. Prescribe an EpiPen to every patient who has a history of, or is at risk for, anaphylaxis.
5. Educate appropriate patients with allergy (e.g., to food, medications, insect stings) and their families about the symptoms of anaphylaxis and the self-administration of the EpiPen, and advise them to return for immediate reassessment and treatment if those symptoms develop or if the EpiPen has been used.
6. Advise patients with any known drug allergy or previous major allergic reaction to get a MedicAlert bracelet.
7. In a patient presenting with an anaphylactic reaction:
   a) Recognize the symptoms and signs.
   b) Treat immediately and aggressively.
   c) Prevent a delayed hypersensitivity reaction through observation and adequate treatment (e.g., with steroids).
8. In patients with anaphylaxis of unclear etiology refer to an allergist for clarification of the cause.
9. In the particular case of a child with an anaphylactic reaction to food:
   a) Prescribe an EpiPen for the house, car, school, and daycare.
   b) Advise the family to educate the child, teachers, and caretakers about signs and symptoms of anaphylaxis, and about when and how to use the EpiPen.
10. In a patient with unexplained recurrent respiratory symptoms, include allergy (e.g., sick building syndrome, seasonal allergy) in the differential diagnosis.

**Type 1 (Immediate) Immune hypersensitivity mediated by IgE.**

**Anaphylaxis**

- A severe, life-threatening, generalised or systemic hypersensitivity reaction (i.e. allergy).
- Characterized by rapidly developing life-threatening airway (airway edema) and/or breathing (bronchospasm especially in children) and tachypnea and/or circulation (hypotension and tachycardia) problems usually associated with skin and mucosal changes (urticaria).
- But, a patient can be anaphylactic with NO skin changes!
- Other physical signs: stridor, wheeze, hoarseness, skin erythema, pruritus, swollen edematous eyes, sub-conjunctival injection, syncope, nausea and vomiting.
- Anaphylaxis is a common masquerade: always consider the diagnosis in patients with any of the symptoms mentioned above.
- Common triggers: foods (younger patients), medications (older patients).
- Younger patients with anaphylaxis usually have a history of atopy.

**DIAGNOSTIC CRITERIA**

*Highly likely if 1 of the following 3 criteria: (Sampson et al.)*

1. Acute onset of an illness (minutes to several hours) involving the skin, mucosal tissue, or both (eg, generalized hives, pruritus or flushing, swollen lips-tongue-uvula) and at least one of the following:
   - Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced peak expiratory flow, hypoxemia), OR
   - Reduced blood pressure (BP) or associated symptoms and signs of end-organ dysfunction (eg, hypotonia [collapse] syncope, incontinence). (See criterion 3 below.)
2. Two or more of the following that occur rapidly after exposure to a LIKELY allergen for that patient (minutes to several hours):
   - Involvement of the skin-mucosal tissue (eg, generalized hives, itch-flush, swollen lips-tongue-uvula).
   - Respiratory compromise (eg, dyspnea, wheeze-bronchospasm, stridor, reduced peak expiratory flow, hypoxemia).
• Reduced BP or associated symptoms and signs (eg, hypotonia [collapse], syncope, incontinence).
• Persistent gastrointestinal symptoms and signs (eg, crampy abdominal pain, vomiting).

3. Reduced BP after exposure to a **KNOWN allergen for that patient** (minutes to several hours):
• Reduced BP in adults is defined as a systolic BP of less than 90 mmHg or greater than 30 percent decrease from that person’s baseline.
• In infants and children, reduced BP is defined as low systolic BP (age specific) or greater than 30 percent decrease in systolic BP.
• **Low systolic BP for children is defined as:**
  - Less than 70 mmHg from 1 month up to 1 year.
  - Less than (70 mmHg + [2 x age]) from 1 to 10 years.
  - Less than 90 mmHg from 11 to 17 years.

**DIAGNOSTIC CRITERIA**

It’s useful to know the diagnostic criteria for the exam, but in clinical practice, it’s perhaps more useful to consider anaphylaxis (and treat as such) in any patient with systemic symptoms or hypotension after exposure to a known or suspected allergen.

**MANAGEMENT**

• **Have a low threshold to diagnosis and treat** — the risk of death greatly outweighs any risk of side effects from giving epinephrine promptly after diagnosis.
• **Treatment includes addressing the ABCs while concurrently giving epinephrine IM:**
  - Adults: **0.5mg IM into the lateral thigh** (0.5mL of 1:1000 (1mg/mL) — **do not** attempt to inject 5mL of a 1:10000 cardiac amp IM!)
  - Pediatrics: 0.01mg/kg 1:1000 (1 mg/mL) to a maximum of 0.3-0.5 mg IM.
    - **Warn patients of side effects of administration:** tachycardia, headache, anxiety, dizziness, N & V.
  - **Epipen or Allerject** autoinjectors has a similar dose (0.3mg IM, 0.15mg IM for pediatrics), and can be used by patients and in the pre-hospital setting.
  - **Epinephrine can be given at 5 minute intervals** as required. After a second injection is required, consider prepping an epinephrine infusion (may be required if refractory) or asking for specialist advice.
  - **Lie patient in reverse Trendelenburg position** to maximize perfusion of vital organs.
  - **Give supplemental oxygen** (full-open (15L) O2 by non-rebreather mask).
  - **Two large-bore IVs** should be sited — consider N/S boluses if hypotensive.
• **Adjunctive treatments (typically given, but without proven benefit):**
  - Diphenhydramine (Benadryl) 50mg IM or IV (H1 blocker).
  - Ranitidine 50mg IV or cimetidine 200mg IV (H2 blocker).
  - Systemic corticosteroids (can be given PO: give IV if severe reaction or nausea and vomiting) — methylprednisolone 50-100mg IV, then 1-2mg/kg/day PO for 3 days (all reported biphasic reactions to date have occurred within 72 hours).
  - Salbutamol 5mg neb can be given if wheeze.
  - **Observe for a period of time typically at least 4-8 hours** to watch for a biphasic reaction. Longer periods of observation for patients at risk for severe anaphylaxis (i.e. asthma, history of previous biphasic reaction, severe or protracted reaction).
• **Education is key!**
  - After initial treatment is successful, discuss the patient’s likely identified trigger.
  - If no trigger identified, they should have allergist followup for testing.
  - Inform the patient that they have experienced anaphylaxis, and have a potentially deadly allergy.
  - Inform them that symptoms may recur up to 72 hours later.
  - **Every patient should leave with a prescription for an autoinjector x 2** (or preferably, dispense autoinjector x 2 directly from the ED/office).
  - Inform the patient that if symptoms recur, they need to use their autoinjector. **If they use their autoinjector, they must present to the nearest ED.**
  - Ideally, every patient should have an anaphylaxis action plan (example at the AAAAI)

**ANAPHYLAXIS**

The only proven treatment is **epinephrine** — give it early, and give it as often as required. **DO** give it IM, **DO NOT** give it IV (unless starting an infusion for refractory cases).
Pediatric Allergy

- There is no longer a recommendation to avoid or delay the introduction of potentially allergenic foods in pediatrics (Abrams and Becker).
- Current advice to prevent the development of allergies is to introduce allergenic foods at four to six months of age. Regular exposure is important for the maintenance of tolerance.

RECOMMENDED FOAMED RESOURCES


REFERENCES


4. Anxiety

**KEY FEATURES**

1. Do not attribute acute symptoms of panic (e.g., shortness of breath, palpitations, hyperventilation) to anxiety without first excluding serious medical pathology (e.g., pulmonary embolism, myocardial infarction) from the differential diagnosis (especially in patients with established anxiety disorder).

2. When working up a patient with symptoms of anxiety, and before making the diagnosis of an anxiety disorder:
   - a) Exclude serious medical pathology.
   - b) Identify:
     - other co-morbid psychiatric conditions.
     - abuse.
     - substance abuse.

3. In patients with known anxiety disorders, do not assume all new symptoms are attributable to the anxiety disorder.

4. Offer appropriate treatment for anxiety:
   - benzodiazepines (e.g., deal with fear of them, avoid doses that are too low or too high, consider dependence, other anxiolytics).
   - non-pharmacologic treatment.

5. In a patient with symptoms of anxiety, take and interpret an appropriate history to differentiate clearly between agoraphobia, social phobia, generalized anxiety disorder, and panic disorder.

Always ask about suicidal ideation in any mental health presentation!

**Common Anxiety Disorders**

**GENERALIZED ANXIETY DISORDER (GAD)**

- Persistent, excessive, and unrealistic worry about everyday things.
- Patients experience excessive anxiety and worry, often expecting the worst even when there is no apparent reason for concern. They anticipate disaster and may be overly concerned about money, health, family, work, or other issues. GAD is diagnosed when a person finds it difficult to control worry on more days than not for at least six months and has three or more symptoms ("BESKIM").

**GENERALIZED ANXIETY DISORDER**

**BE SKIM** — Blank mind (and difficulty concentrating), Easily fatigued, Sleep disturbance, Keyed up (restless), Irritability, Muscle tension

**AGORAPHOBIA & PANIC DISORDER**

- Patients with panic disorder experience spontaneous seemingly out-of-the-blue panic attacks and are preoccupied with the fear of a recurring attack. Panic attacks occur unexpectedly, sometimes even during sleep.
- Some people stop going into situations or places in which they’ve previously had a panic attack in anticipation of it happening again — these people have agoraphobia, and they typically avoid public places where they feel immediate escape might be difficult. ~30% of patients with panic disorder will develop agoraphobia.

**PANIC DISORDER**

**STUDENTS FEAR the 3 Cs** — Sweating, Trembling, Unsteadiness/dizziness, Derealization/depersonalization, Elevated heart rate (tachycardia), Nausea, Tingling, Shortness of breath, FEAR of dying, losing control, going crazy, 3 C’s — Choking, Chest pain, Chills

Typically subsides within several minutes.

**POST-TRAUMATIC STRESS DISORDER (PTSD)**

- May occur after a life-altering or life-threatening event (war, serious accident, sudden death of a loved one, rape).
- Symptoms occur for at least one month, but need not occur until months/years after the event.
- Symptoms include spontaneous, recurrent, intrusive dreams or memories, flashbacks or other dissociative reactions, and physiological reactions to reminders of traumatic events.
OBSESSIVE-COMPULSIVE DISORDER (OCD)

- Patients have either or both of obsessions (intrusive, recurrent, undesired thoughts) and compulsions (repetitive behaviours or rituals).
- Patients are generally aware that their compulsions or obsessions are irrational.
- Patients may feel compelled to spend hours at a time performing complicated rituals involving hand-washing, counting, or checking to ward off persistent, unwelcome thoughts, feelings, or images.
- These activities interfere with the patient’s normal routine (schoolwork, job, family, social activities).

Risk factors

- Comorbid psychiatric disorder (especially anxiety or depression).
- Family history of mental disorders (especially anxiety and depression).
- Stressful life event(s) and/or abuse.
- Female sex.

HISTORY

Anxious/agitated patients are commonly difficult to assess: acknowledge any counter-transference early to aid your history-taking.

- What brings them into the clinic/ED today?
- Get collateral history from caregivers / authority figures who accompanied them.
- Current social circumstances:
  - Where are they living?
  - Who are they living with? What’s their relationship?
  - Are they working? Where?
  - Financial situation?
- Any history of mental illness?
- Any substance or EtOH misuse/abuse?
- Mental state examination (MSE) — assess suicidality (see below).

MENTAL STATE EXAMINATION

ABC STAMP LICKER — Appearance, Behavior, Cooperation, Speech, Thought (Process, Content), Affect, Mood, Perception (Hallucinations), Level of consciousness, Insight, Cognition, Knowledge base, Endings (Suicidal, homicidal), Reliability

Management

De-escalating the situation is essential before you can proceed.

- Management goals in the ED or in the clinic are the same, although availability of resources may affect your decision-making:
- Attempt to exclude a medical cause for the anxiety first.
  - Beware the “frequent flyer” who presents with his/her “usual” symptoms: they could still have a medical cause!
  - Keep an open mind as you initiate management for anxiety, as significant features of a medical comorbidity could present late.
  - Identify any co-morbid medical illness through history, physical examination and baseline labs.
  - Identify any substance misuse or intoxication that may be causative.
  - Baseline labs should include CBC, fasting glucose, TSH, urine toxicology (if recent drug abuse suspected).
- Determine if admission is required acutely (voluntary or involuntary) if the patient is a risk to themself or others (see History).
- Treat acute symptoms if necessary (i.e. benzodiazepines, antipsychotics).
- Arrange referral or followup.
- Consider psychological (i.e. CBT) and/or pharmacological treatment.
Chapter 4. Anxiety

Treatment

- First-line: SSRIs (citalopram, paroxetine, sertraline, escitalopram have RCT evidence), SNRIs (venlafaxine and duloxetine have RCT evidence). Substitute SSRI for SSRI or SNRI if ineffective, before trying second-line agents.
- Second-line: TCAs, benzodiazepines (imipramine, or choose a longer-acting benzo).
- Psychotherapy: CBT — can be first-line, second-line, or used concurrently (insufficient evidence to recommend pharmacotherapy or psychotherapy as first-line).

RECOMMENDED #FOAMED RESOURCES

REFERENCES
5. Behavioural Problems

### KEY FEATURES

1. Because behavioural problems in children are often multifactorial, maintain a broad differential diagnosis and assess all factors when concern has been raised about a child’s behaviour:
   - Look for medical conditions (e.g., hearing impairment, depression, other psychiatric diagnoses, other medical problems).
   - Look for psychosocial factors (e.g., abuse, substance use, family chaos, peer issues, parental expectations).
   - Recognize when the cause is not attention deficit disorder (ADD) (e.g., learning disorders, autism spectrum disorder, conduct disorder).

2. When obtaining a history about behavioural problems in a child:
   - Ask the child about her or his perception of the situation.
   - Use multiple sources of information (e.g., school, daycare).

3. When treating behavioural problems in children for whom medication is indicated, do not limit treatment to medication; address other dimensions (e.g., do not just use amphetamines to treat ADD, but add social skills teaching, time management, etc.).

4. In assessing behavioural problems in adolescents, use a systematic, structured approach to make an appropriate diagnosis:
   - Specifically look for substance abuse, peer issues, and other stressors.
   - Look for medical problems (bipolar disorder, schizophrenia).
   - Do not say the problem is “just adolescence”.

5. In elderly patients known to have dementia, do not attribute behavioural problems to dementia without assessing for other possible factors (e.g., medication side effects or interactions, treatable medical conditions such as sepsis or depression).

### Attention deficit / hyperactivity disorder (ADHD)

- Syndrome with two types of symptoms: hyperactivity (impulsivity) and inattention.
- Boys > girls (2:1 ~ 4:1).
  - Hyperactivity/impulsivity: inability to sit still or inhibit behavior.
  - Inattention: inability or reduced ability to focus, and slower cognitive processing (“daydreaming”).
- DSM-V diagnostic criteria:
  - ≥6 symptoms of either hyperactivity/impulsivity or inattention. Symptoms must:
    - Occur often.
    - Be present in more than one setting (i.e. school and home).
    - Persist for at least six months.
    - Be present before the age of 12 years.
    - Impair function in academic, social, or occupational activities.
    - Be excessive for the developmental level of the child.
- Pathogenesis is unknown.
- Affects 8-11% of school-aged children.
- Consider co-morbid diagnoses: oppositional defiant disorder (ODD) (up to 50% of cases), conduct disorder, anxiety disorder, depression, or learning disabilities.

### HISTORY AND EXAMINATION

- Interview parents and then child alone: age of onset, duration of symptoms, settings where symptoms occur, degree of functional impairment.
- Schools are generally equipped to do a classroom assessment, and a classroom teacher will common be the one to alert parents to a possible ADHD diagnosis, but you can request the parents and teacher to separately fill out a questionnaire (SNAP-IV is commonly used).
- Physical examination should look for dysmorphic features (i.e. fetal alcohol syndrome), growth rate, BP, cardiac exam (if medications considered), and looking for other potential causes of behaviour: vision/hearing loss, enlarged tonsils (OSA).
Chapter 5. Behavioural Problems

TREATMENT

- ADHD is a chronic condition, and should be treated as such: goal-setting with the child’s family, regular follow-up to gauge effectiveness.
- Treatment should start with behavioural interventions rather than medications, but can also include medication, or psychotherapy.
- The Canadian ADHD Resource Alliance (CADDRA) has excellent resources for physicians, patients and families.
- Examples of behavioural interventions:
  - Maintaining a routine / daily schedule.
  - Using a reward system for good behaviour.
  - Removing distractions at home and at school.
- Treatment should be aimed at realistic target outcomes (i.e. improved school performance, improved relationships, improved behaviour).
- Be sure to consider and treat co-morbid conditions.

Medication

- ADHD medications have had black box warnings in U.S. if cardiac risk factors / strong family history, but adverse events are rare (Habel et al.)
- Medications should be used only in addition to behavioural interventions.
- Switch medications during holidays if not effective (all first-line similarly efficacy).

First line – stimulants (see CADDRA’s Children’s Medical Treatment Options (6-12 years):

- Amphetamine (Adderall XR) 5mg qAM up to max of 30mg qAM, titrated ↑ 5mg qweekly.
- Methylphenidate (Biphentin) 10mg qAM up to max of 60mg qAM, titrated ↑ 5mg qweekly.
- Methylphenidate (Concerta) 18mg qAM up to max of 72mg qAM, titrated ↑ 9mg qweekly.
- Lisdexamfetamine (Vyvanse) 20mg qAM up to max of 60mg qAM, titrated ↑ 10mg qweekly.

Second-line – SNRI:
- Atomoxetine (Strattera) 0.5mg/kg/day daily up to max of 60mg daily, titrated↑ 0.8mg/kg/day qweekly.
- Consider adding an SSRI if several first-line options not effective.

Oppositional defiant disorder (ODD)

- Negativistic, defiant, disobedient, hostile behaviour towards authority figures.
- Contrasted to conduct disorder, children with ODD are not aggressive or destructive.

Conduct disorder

- Violates basic rights of others (physical or verbal aggression, destruction of property).
- Violates age-appropriate social norms (i.e. “antisocial behaviour”).

Behavioural problems in the elderly

See Dementia and Dementia — Delirium.

REFERENCES

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorder (DSM-V) (5th ed.).
doi:10.1176/appi.books.9780890425596.744053


6. Chest Pain

**KEY FEATURES**

1. Given a patient with undefined chest pain, take an adequate history to make a specific diagnosis (e.g., determine risk factors, whether the pain is pleuritic or sharp, pressure, etc.).
2. Given a clinical scenario suggestive of life-threatening conditions (e.g., pulmonary embolism, tamponade, dissection, pneumothorax), begin timely treatment (before the diagnosis is confirmed, while doing an appropriate work-up).
3. In a patient with unexplained chest pain, rule out Ischemic Heart Disease.
4. Given an appropriate history of chest pain suggestive of herpes zoster infection, hiatal hernia, reflux, esophageal spasm, infections, or peptic ulcer disease:
   - Propose the diagnosis.
   - Do an appropriate work-up/follow-up to confirm the suspected diagnosis.
5. Given a suspected diagnosis of pulmonary embolism:
   - Do not rule out the diagnosis solely on the basis of a test with low sensitivity and specificity.
   - Begin appropriate treatment immediately.

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**Causes**

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<tr>
<th>HEART</th>
<th>LUNGS</th>
<th>ESOPHAGUS</th>
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<tr>
<td>MI</td>
<td>Pneumonia</td>
<td>Esophagitis</td>
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<td>Angina</td>
<td>Pneumothorax</td>
<td>GERD</td>
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<td>Pericarditis</td>
<td>Pulmonary embolism</td>
<td>Esophageal spasm</td>
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<td>Myocarditis</td>
<td>COPD exacerbation</td>
<td>Foreign body</td>
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<tr>
<td>Valve disease (i.e. aortic stenosis)</td>
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<td>Rupture (Boerhaave syndrome)</td>
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**AORTA | UPPER ABDOMEN | CHEST WALL**

| Dissection | Biliary colic / cholecystitis | Costochondritis / Tietze's disease |
| Aneurysm | Pancreatitis | Contusion |
| Aortitis | Duodenal / peptic ulcer | Rib fracture |
| | Hepatitis | Muscle strain / tear |

| | | Herpes zoster |

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Remember that a psychogenic cause to chest pain is not uncommon, and must be considered after organic illness has been excluded.

**Risk factors**

- MI: see Ischemic Heart Disease.
- PE: ↑ age (risk doubles each decade after 60), previous DVT/PE (most significant risk factor), exogenous estrogen (i.e. OCP), coagulopathy (i.e. factor V Leiden, ATIII deficiency, malignancy), immobility, recent surgery, pregnancy, obesity, previous stroke (esp. with hemiplegia), CVD, hypertension.
- Cardiac tamponade: malignancy, penetrating/blunt chest trauma, MI, iatrogenic.
- Aortic dissection: hypertension, atherosclerosis, “pipe defects” (pre-existing aortic aneurysm, bicuspid aortic valve, coarctation of the aorta).
- Pneumothorax:
  - Primary spontaneous: trauma, smoking, family history, connective tissue disorder (i.e. Marfan), iatrogenic (i.e. subclavian central line insertion).
  - Secondary: underlying lung disease (esp. COPD).

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**Initial management**

Initially, start with the ABCs, conduct your primary survey, obtain a focused history, conduct a focused physical exam, and obtain an ECG (goal is within 10 minutes of arrival).

**PRIMARY SURVEY**

- Give them oxygen by mask (only if hypoxic), get a set of vitals (i.e. hook up to monitors), and obtain IV access. If not responding, check if they have a pulse (mnemonic: OMIP - see ACLS).

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25 — Last peer reviewed: December 11, 2015
Chapter 6. Chest Pain

FOCUSED HISTORY AND PHYSICAL EXAMINATION

- While taking a history, also include a focused pain history, including these specific questions:
  - Pleuritic?: Is the pain worse with coughing or taking a deep breath?
  - Palpable?: If I press on your chest does that reproduce the pain?
  - Positional?: Is the pain worse or better in certain positions or with certain movements?

- These specific questions come from a systematic review (Swap and Nagurney), that help risk stratify patients with suspected ACS into 4 groups just based on history alone:
  - Low risk: pain that is pleuritic, positional, or reproducible with palpation or is described as stabbing.
  - Probable low risk: pain not related to exertion or that occurs in a small inframammary area of the chest wall.
  - Probable high risk: pain described as pressure, is similar to that of prior myocardial infarction or worse than prior anginal pain, or is accompanied by nausea, vomiting, or diaphoresis.
  - High risk: pain that radiates to one or both shoulders or arms or is related to exertion.

Although history and physical examination are important, remember that there are limitations (a.k.a. patients with atypical presentations, especially women and diabetics).

6 DEADLY CHEST PAIN DIAGNOSES
3 in the heart — ACS, myocarditis/pericarditis/pericardial effusion, aortic dissection
2 in the lungs — pulmonary embolism, pneumothorax
1 in the throat — Boerhaave syndrome (esophageal rupture)

ACS (STEMI, NSTEMI, and UA)

PRESENTATION

- Retrosternal, crushing/squeezing chest pain.
- Pain may radiate to the shoulder, arm, or jaw.
- Dyspnea, diaphoresis.
- Diabetics and women especially can present atypically.

INVESTIGATIONS

- STEMI: ST elevation ≥1mm in at least 2 consecutive leads, or ≥2mm in V2 and V3.
  - In patients with LBBB: use Sgarbossa criteria for diagnosis of STEMI.
- UA and NSTEMI (a.k.a. non-ST elevation ACS or NSTEMI): may be indistinguishable on initial presentation.
  - NSTEMI: an ischemic insult to the myocardium severe enough to cause the release of detectable levels of a biomarker (i.e. troponin) — a.k.a. troponin positive. May have no ECG changes.
  - UA: by definition, no elevation in biomarkers.

CLINICAL FINDINGS HELPFUL FOR RULING IN OR OUT ACS

The Rational Clinical Examination Systematic Review (Fanaroff et al.) found the following significant clinical findings in undifferentiated patients presenting with suspected ACS.

Findings most suggestive of ACS (helpful to rule in the diagnosis):
- Prior abnormal stress test (specificity = 96%; LR = 3.1).
- Peripheral arterial disease (specificity = 97%; LR = 2.7).
- Pain radiation to both arms (specificity = 96%; LR = 2.6).
- ST-segment depression (specificity = 95%; LR = 5.3).
- Any evidence of ischemia (specificity = 91%; LR = 3.6).
- HEART Score of 7-10 (high-risk) (LR = 13).
- TIMI Score of 5-7 (high-risk) (LR = 6.8).

Findings least suggestive of ACS (helpful to rule out the diagnosis):
- HEART score of 0-3 (low-risk) (LR = 0.20).
- TIMI score of 0-1 (low-risk) (LR = 0.31).
MANAGEMENT

Your facility should have a chest pain / ACS protocol.

- Initial management as above.
- Give ASA 325mg (non-enteric coated), chew and swallow.
- Obtain labs if not already done: troponin, electrolytes, Hgb, Hct (at a minimum).
- Give sublingual nitroglycerin spray or tabs (0.4mg) every 5 minutes x 3 doses if the patient has ongoing chest discomfort or severe hypertension. Give cautiously (if at all) if hemodynamically compromised (hypotensive, or likely to become hypotensive (i.e. hearts with right-sided infarcts are preload dependent)).
- Give a beta blocker (i.e. metoprolol 25mg PO) if no signs of (or significant risk factors for) heart failure.
- After nitroglycerin, give morphine 5-10mg (or fentanyl 50-100mcg) every 10-15 minutes for ongoing pain/discomfort.
- Consider starting a statin (if patient is not already on one) during the acute episode.

STEMI

- Consult cardiology: all STEMI patients should have close cardiology followup.
- With cardiology, decide on PCI versus fibrinolysis: treat with fibrinolysis if PCI unavailable within 120 minutes unless contraindications (see your facility’s ACS/fibrinolytic protocol). Generally risks outweigh benefits if >12 hours from symptom onset.
- If fibrinolytic therapy given, give clopidogrel 300mg (age ≤75 yrs) or 75mg (age >75 yrs) (alternative newer agents: ticagrelor, prasugrel). The FDA has recently concluded that clopidogrel does not change the risk of death, and recommends newer agents.
- Start anticoagulant therapy (i.e. enoxaparin, tinzaparin, or UFH).
- Lifelong antiplatelet therapy should be initiated (i.e. ASA 81mg daily, or clopidogrel 75mg daily).

NSTEMI/UA

The primary goal in NSTEMI/UA is to prevent recurrence (Fitchett et al.).

- Consult cardiology: all NSTEMI/UA patients should have close cardiology followup.
- Give clopidogrel 300mg (age ≤75 yrs) or 75mg (age >75 yrs), then start 75mg daily (or newer agents: ticagrelor, prasugrel).
- Start anticoagulant therapy (i.e. enoxaparin 1mg/kg SC every 12 hours, UFH 60 units/kg).
- Fibrinolytics are contraindicated in NSTEMI patients.
- Invasive management should be considered in consultation with cardiology; high-risk patients benefit from early catheterization and revascularization (within 24-48 hours).
- High-risk features:
  - ST depression >0.5-1mm.
  - Transient ST elevation.
  - Deep (>2mm) symmetrical T-wave inversion.
  - Troponin >99th reference level (assay specific – check with your local lab).
  - Unstable patients (heart failure, hypotension, ventricular arrhythmias).
  - Refractory ischemia with ECG changes despite treatment.
  - Calculating a TIMI Score may help with risk stratification.
- Long-term prevention strategies are just as important in NSTEMI/UA as in STEMI (vascular protection, lifestyle modification, smoking cessation, lipid lowering, blood pressure control and glycemic control).
- Lifelong antiplatelet therapy should be initiated (i.e. ASA 81mg daily, or clopidogrel 75mg daily).

Pulmonary embolism

Also see Deep venous thrombosis (DVT).

PRESENTATION

- Wide spectrum of presentations, from minimally symptomatic to critically unwell.
- Pleuritic chest pain.
- Dyspneic, with \(\text{O}_2\) sats, tachycardia.
- May be anxious, feeling of impending doom.
- (See the PERC rule and Well’s criteria for a hint to typical presentation).
- A massive PE if systolic <90, profound bradycardia (HR <40), or pulseless.
INVESTIGATIONS

- ECG: **sinus tachycardia (most common)**, right ventricular strain pattern (T-wave inversions in V1–V4, RBBB, S1Q3T3 pattern).
- CXR: non-diagnostic.
- D-dimer: useful to **rule out low-risk** patients.
- CTPA and/or V/Q scan: definitive imaging modalities, although not 100% sensitive — if very high clinical suspicion and investigations are negative, consider presumptive treatment and/or specialist consultation.

DIAGNOSIS

- If you have a low clinical suspicion of PE, attempt to apply the **PERC Rule** to rule out without any further investigations being required.
- Calculate a pre-test probability (**Well's Criteria for PE** or Revised Geneva Score).
- If the patient is low-risk (≤4 points by Well’s criteria), **order a D-dimer:**
  - If <500ng/mL, PE excluded.
  - If ≥500ng/mL, proceed to definitive imaging (CTPA or V/Q scan).
  - **New evidence:** Can consider the use of the age-adjusted D-dimer thresholds from the ADJUST-PE trial: cutoff is then age x 10.
- If the patient is high-risk proceed directly to CTPA (or V/Q scan if contraindications).

MANAGEMENT

- If PE confirmed, consult a specialist (hematologist and/or respirologist):
  - In hemodynamically stable patients without contraindications, **anticoagulation** may be initiated acutely (i.e. LMWH), and then bridged to longer-term anticoagulation (i.e. warfarin, or the novel anticoagulants (NOACs) such as dabigatran, rivaroxaban, apixaban).
  - In hemodynamically unstable patients, **thrombolytic therapy** may be considered.
- Consider the possibility of **undiagnosed malignancy** if PE is unprovoked (although a Canadian RCT (Carrier et al.) seemed to indicate this is probably not required).

Aortic dissection

PRESENTATION

- Aortic pain with immediate onset, a tearing or ripping character, or both.
- CXR: mediastinal widening, aortic widening, or both.
- Pulse differentials, blood pressure differentials, or both (comparing right vs. left).
  All 3 above features = **96% sensitive for diagnosis** (von Kodolitsch et al.).

INVESTIGATIONS

- **D-dimer**: useful as a rule-out test (cutoff of 500ng/mL, 96% sensitive).
- CXR: poorly sensitive, but specific if mediastinal or aortic widening.
- CT or TEE are the imaging methods of choice; TEE is ↓ in popularity.
- MRI: accurate but ↓ availability and ↑ cost.

MANAGEMENT

- Consult cardiothoracics ASAP.
- Until definitive care (surgical repair) or ICU admission available, **blood pressure control** and **pain control** are important.
- Fast-onset antihypertensive (propranolol, labetalol, esmolol, verapamil, diltiazem) to a target **BP of 100-120mmHg**.
- Nitroprusside infusion if not at target BP after beta blockade.
- **Morphine** for pain control / anxiolytic.
Pneumothorax

PRESENTATION
- As with PE, a wide spectrum of presentations, from minimally symptomatic to critically unwell.
- Primary (spontaneous) pneumothorax:
  - Occurs at rest. Typical patient is 20-30yo, tall, male.
- Secondary pneumothorax:
  - By definition, due to underlying lung disease (COPD, CF, malignancy).
- Sudden onset of dypsnea and/or pleuritic chest pain, but small pneumothoraces can have minimal/no symptoms.

PHYSICAL EXAMINATION AND INVESTIGATIONS
- ↓/absent breath sounds affected side.
- Hypoxemia (common due to well-perfused, poorly-ventilated collapsed lung).
- If tensioned: tracheal deviation away from affected side, mediastinal deviation away on CXR.
- CXR: lung markings that don’t extend to the periphery, with a visible visceral pleural line (the lung/air interface).

MANAGEMENT
- Definitive management: chest tube insertion with Heimlich or underwater valve.
- Primary pneumothorax:
  - If pneumothorax is small anduntensioned (<2cm at the lung apices) and patient asymptomatic, can consider watchful waiting (i.e. six hours in ED with supplemental oxygen — if no progression on CXR, can be discharged to close followup).
  - If the pneumothorax is >2cm but the patient is clinically stable, needle decompression should be performed. If this fails to resolve the pneumothorax, a chest tube should be inserted.
  - Patients with recurrent pneumothorax should undergo thoracoscopy.
- All patients with secondary pneumothorax should be admitted with specialist consultation.
- Tension pneumothorax is a clinical diagnosis! Emergent needle decompression (2nd intercostal space midclavicular line), then chest tube insertion.

Cardiac tamponade

PRESENTATION
- Acute tamponade develops in minutes (trauma), and presents as cardiogenic shock with severe hypotension.
- Subacute tamponade develops over days to weeks (neoplastic, uremic, idiopathic): dyspnea, hypotension, tachycardia, peripheral edema, fatigue, and chest pain or “fullness”.

PHYSICAL EXAMINATION
- Sinus tachycardia.
- Elevated JVP (due to poor expansion of right ventricle under pressure).
- Pulsus paradoxus (abnormally large decrease in systolic BP on inspiration).
- Possibly, pericardial rub (inflammatory pericarditis associated with tamponade).

INVESTIGATIONS
Cardiac tamponade is a clinical diagnosis, although echo is important in assessing cardiac function / hemodynamic significance.
- ECG (may show low voltage).
- CXR (enlarged cardiac silhouette).
- Echocardiogram (looking for diastolic collapse of RA/RV due to ↑ intrapericardial pressure).
**MANAGEMENT**

- Specialist consult to consider pericardial drainage, although may be treated conservatively if early and not hemodynamically compromising.
- Either percutaneous or surgical drainage (pericardial window removal, or pericardiectomy).

**Pain of esophageal origin (spasm/motility disorders, esophagitis)**

**PRESENTATION**

- Can have classical ACS-type pain presenting acutely: crushing, central, retrosternal pain — **ACS should be ruled out as a first step**.
- Dysphagia (liquids and/or solids).
- Diffuse esophageal spasm (DES) can present with a globus sensation seconds after swallowing.
- Alarm signs/symptoms that require urgent (within two weeks) endoscopy:
  - Dysphagia.
  - Odynophagia.
  - GI bleeding.
  - Iron deficiency anemia.
  - Unexpected weight loss.
  - Recurrent vomiting.

**INVESTIGATIONS**

- Barium swallow: neither sensitive nor specific for spasm; may show “corkscrew” appearance.
- Endoscopy: biopsy to rule out malignancy and eosinophilic esophagitis.

**MANAGEMENT**

- If an acute presentation and ACS clinically suspected, investigate and rule out concurrently.
- Ask about recent medications known to cause medication-induced esophagitis (tetracycline, aspirin, NSAIDs, potassium chloride).
- If endoscopy is normal, trial a high-dose PPI for 8 weeks (i.e. omeprazole 40mg BID).
- If no resolution of symptoms, consult GI (possibilities include repeating endoscopy, esophageal impedance and pH monitoring, or esophageal manometry (if a motility disorder is suspected)).

**Pericarditis**

**PRESENTATION**

- Sudden onset of anterior, pleuritic, superficial/precordial chest pain.
- Pain classically better when leaning forward.

**PHYSICAL EXAMINATION**

- Pericardial friction rub is highly specific (but not very sensitive).

**INVESTIGATIONS**

- ECG: if changes, signifies involvement of the epicardium. Classically, diffuse ST elevation (concave up) ECGpedia — Pericarditis. ST changes typically resolves within a few days.
- Echo: typically normal in acute pericarditis without effusion.
- CXR: non-diagnostic.
- Troponin may be elevated, in which case the diagnosis is myopericarditis (by definition, involves inflammation of the heart muscle).
MANAGEMENT

- Consult cardiology.
- If high-risk, patient should be hospitalized for specialist evaluation and initiation of treatment.
  - High-risk criteria: fever and leukocytosis, evidence of cardiac tamponade, large pericardial effusion, immunosuppressed, anticoagulated (a.k.a. patient on warfarin/NOACs), history of acute trauma, failure to respond after seven (7) days of NSAIDs, or elevated troponin.
- NSAIDs alone are effective in 70-80%. Regimens include: ibuprofen 600-800mg TID, aspirin 650-1000mg TID, indomethacin 50mg TID.
- Consider the addition of a PPI for short-term GI protection.
- Colchicine added to NSAIDs reduces symptoms and reduces recurrence rates. Dose is 0.5mg daily (0.5mg BID if >70kg) for 2-3 months.
- Corticosteroids can be considered as a third-line option if no response to treatment (seek specialist advice).

GERD and hiatal hernia

See Dyspepsia.

Peptic ulcer disease

See Dyspepsia.

Also see Gastro-intestinal Bleed.

Boerhaave syndrome (esophageal rupture)

PRESENTATION

- Excruciating retrosternal chest pain.
- Classically, a history of severe retching & vomiting.
- Dysphonia and dysphagia if cervical perforation.
- Epigastric pain radiating to the shoulder if intra-abdominal perforation.
- Within hours: septic shock with cyanosis (fever, tachycardia, tachypnea, hypotension).

PHYSICAL EXAMINATION

- Chest wall crepitis (due to subcutaneous emphysema).
- Crackles on cardiac auscultation (mediastinal emphysema).

INVESTIGATIONS

- CXR (not sensitive): mediastinal or subcutaneous emphysema.
- Contrast esophagram (gastrografin or barium study): usually diagnostic, but may not be if the perforation is small.
- CT chest: in all patients with peritoneal free air, or if esophagram is non-diagnostic.
- Endoscopy: controversial (may worsen a perforation).

MANAGEMENT

- Boerhaave syndrome is rare.
- Specialist consultation if suspected concurrent with investigations.
- Management is controversial: may include NPO, parenteral nutrition, IV antibiotics or PPIs, and/or endoscopic or surgical repair.
Herpes zoster

See Skin disorders — Herpes zoster.

REFERENCES

Lalani, N. Risk Stratification of Chest Pain Patients, In Saskatchewan Emergency Medicine Annual Conference (SEMAC)
7. Contraception

**KEY FEATURES**

1. With all patients, especially adolescents, young men, postpartum women, and perimenopausal women, advise about adequate contraception when opportunities arise.
2. In patients using specific contraceptives, advise of specific factors that may reduce efficacy (e.g., delayed initiation of method, illness, medications, specific lubricants).
3. In aiding decision-making to ensure adequate contraception:
   - Look for and identify risks (relative and absolute contraindications).
   - Assess (look for) sexually transmitted disease exposure.
   - Identify barriers to specific methods (e.g., cost, cultural concerns).
4. Advise of efficacy and side effects, especially short-term side effects that may result in discontinuation.
5. In patients using hormonal contraceptives, manage side effects appropriately (i.e., recommend an appropriate length of trial, discuss estrogens in progestin acetate [Depo-Provera]).
6. In all patients, especially those using barrier methods or when efficacy of hormonal methods is decreased, advise about post-coital contraception.

All sexually active young people should receive opportunistic STI testing & contraception counselling.

We recommend the Canadian Contraception Consensus (SOGC 2015) as a succinct review of guidelines for the exam.

**Be opportunistic**

“Despite many contraceptive options, Canadian women continue to use a narrow range of contraceptive methods and to use contraception inconsistently. Consistent contraceptive use is influenced by a number of independent social variables. Future public health initiatives should focus on raising awareness of contraception options, increased access to a variety of contraceptive methods, and assisting with contraceptive adherence.” — (Black, Yang, et al.)

- **Raising awareness:**
  - At every appropriate opportunity, broach the subject of contraception with your patients.
  - Without contraception, 85% of women can expect to become pregnant after one year of regular intercourse.
  - Discuss benefits of using contraception, and risks of not using (including the risk of STIs with non-barrier contraception).

- **Where possible, increase access** by lowering the barrier for patients to obtain effective contraception:
  - Provide free condoms in your examination rooms (supplies often available from public health groups).
  - Request and post information on available contraception methods in your office (sexualityandu.ca has excellent posters).
  - Where possible, advise patients on OTC options for contraception (i.e. some COCPs are currently OTC in the U.S., and will likely become OTC in Canada in the near future).
  - Proactively discuss OTC access to emergency contraception (including how and when to use it) when discussing regular contraception methods.

- **Assist** with contraceptive adherence:
  - Consider a range of patient factors when deciding on a contraceptive choice (discussed below).
  - Where appropriate, extend prescriptions with longer courses and refills (i.e. patients who are stable on an OCP should generally be prescribed for the longest period that your local pharmacies will accept (typically 1-2 years), preferably with 3-6 months dispensed at a time rather than monthly).
  - Test for STIs as per screening guidelines.
Chapter 7. Contraception

Choosing a method

All else being equal, long-acting reversible contraceptives (LARCs: IUDs and implants) are the most effective forms of reversible contraception, have high continuation rates, and should be considered in all women presenting for contraception. Unfortunately in Canada, only the IUDs are currently available. With the exception of NIHB coverage (discussed below), most Canadian women bear the costs of contraception themselves.

- Every patient is unique, and a discussion about contraception should cover:
  - Patient’s expressed preference (i.e. “I just want the patch, doc.”).
  - Sexual behaviour/activities including number of partners, risk of STIs (i.e. high-risk sexual lifestyle should use at least a barrier method).
  - Likely adherence (i.e. forgetfulness with daily medication) — weekly patch, IUD or progesterone injection may be more appropriate.
  - Costs (i.e. upfront cost for IUDs can be prohibitive) — barrier methods are cheap and effective if used appropriately.

- Contraindications (see reproductiveaccess.org’s excellent chart). In general, consider / ask about:
  - Past/current breast cancer (hormonal methods CI’ed), endometrial or cervical cancer (IUDs CI’ed).
  - DM with end-organ damage.
  - Drug interactions (esp. HIV meds, anticonvulsants).
  - Symptomatic gallstones, pregnancy-related cholestasis, active viral hepatitis (COCP CI’ed).
  - Cirrhosis, HCC or other hepatic malignancy (all hormonal methods CI’ed).
  - Migraine (COCPs are CI’ed if >35yo, or any age with aura).
  - Hypertension (COCP CI’ed, progesterone injection CI’ed if severe (≥160 systolic)).
  - Ischemic heart disease (COCP and progesterone injection CI’ed).
  - Postpartum (COCP CI’ed until 3-6 weeks PP due to VTE risk; if intending to breastfeed, avoid until lactation and feeding well established. Can use progesterin-only methods immediately PP).
  - Smoking status (COCP CI’ed if >35yo).
  - History of stroke (COCP CI’ed).
  - Recent or upcoming major surgery with prolonged immobilization (COCP CI’ed).
  - Complicated valvular heart disease (COCP CI’ed).
  - Past or current history of DVT, or known thrombophilia (COCP CI’ed).
  - SLE (hormonal methods CI’ed if antiphospholipid Ab positive, copper IUD CI’ed if thrombocytopenia).
  - Other factors (i.e. some women require clandestine contraception (progesterone injection, etc.) for a partner who does not approve of contraception use).

ESTROGEN AND PROGESTERONE EFFECTS

All hormonal contraceptives use one or a combination of estrogens and progesterones. The hormones used (or ratio of hormones) can have side effects that influence the choice of contraceptive (Radovick and MacGillivray).

<table>
<thead>
<tr>
<th>ESTROGEN EXCESS</th>
<th>PROGESTIN EXCESS</th>
<th>ANDROGEN EXCESS</th>
</tr>
</thead>
<tbody>
<tr>
<td>menstrual bleeding</td>
<td>BMD (especially in young women; clinical significance? — no good long-term followup studies to date)</td>
<td>acne</td>
</tr>
<tr>
<td>dysmenorrhea</td>
<td>mood (depression)</td>
<td>Hirsutism</td>
</tr>
<tr>
<td>breast size</td>
<td>energy (fatigue)</td>
<td>libido</td>
</tr>
<tr>
<td>size of uterine fibroids</td>
<td></td>
<td>oily skin</td>
</tr>
<tr>
<td>risk of VTE</td>
<td></td>
<td>Cholestatic jaundice</td>
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<tr>
<td>BP</td>
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</tbody>
</table>

- Estrogen deficiency can cause continuous or early-cycle bleeding, or absence of withdrawal bleeding.
- Progestin deficiency can cause mid-cycle bleeding, or delayed withdrawal bleeding, or menorrhagia.

METHODS

Hormonal IUD/IUS (“Mirena,” “Jaydess”)

- 0.2% of women will become pregnant after one year of typical use.
- Approximately 2% of sexually active women using this method in Canada.
- Pros:
  - Lasts up to 5 years.
Chapter 7. Contraception

- No contraceptive routine required.
- Progestin only (no estrogen).
- May ↓ menstrual flow (i.e. can be useful for AUB), up to 20% of women amenorrheic at 1 year.

**Cons:**
- Expensive (Mirena: $365) up-front for patients who do not have cost covered.
- Commonly causes irregular bleeding/periods for the first few months.
- May fall out of the uterus (6% of women over 5 years).
- Can be an uncomfortable insertion (better with based on provider experience, more difficult if nulliparous).
- Rare risk of uterine perforation with insertion.
- Must be inserted and removed by a trained healthcare professional.
- Should not be used if current or recent STI in the previous 3 months.
- Does not protect against STIs.

**Copper IUD**

- 0.8% of women will become pregnant after one year of typical use.
- Approximately 2.3% of sexually active women using this method in Canada.
  - Lasts up to 5 years.
  - No contraceptive routine required.
  - Non-hormonal.
  - Maybe ↓ risk of endometrial cancer.
  - Most effective form of emergency contraception.

**Cons:**
- Commonly causes irregular bleeding/periods for the first few months.
- May increase menstrual bleeding or cramping in some women.
- May fall out of the uterus (7% of women over 5 years).
- Can be an uncomfortable insertion (better with based on provider experience, more difficult if nulliparous).
- Rare risk of uterine perforation with insertion.
- Must be inserted and removed by a trained healthcare professional.
- Should not be used if current or recent STI in the previous 3 months.
- Does not protect against STIs.

**Subdermal implantable rod (“Implanon”, “Nexplanon”)**

- Not currently available in Canada, but available in 85 other countries.
- 0.05% of women will become pregnant after one year of typical use.
- Approximately 0.1% of sexually active women using this method in Canada.

**Vasectomy**

- 0.5% of women will become pregnant after one year of typical sexual activity.
- Approximately 7.4% of sexually active women using this method (a.k.a. rely on their sexual partner to have had a vasectomy) in Canada.
- Only appropriate if family is complete; most practitioners reluctant to perform on young men.

**Pros:**
- No contraceptive routine required.
- No significant long-term side-effects.
- Less invasive, more cost effective, and fewer complications compared to tubal ligation.

**Cons:**
- Usually permanent.
- Possibility of regret.
- Not effective immediately — backup contraception until followup semen analysis (2-3 months).
- Does not protect against STIs.
Chapter 7. Contraception

Tubal ligation
- 0.15% of women will become pregnant after one year of typical use.
- Approximately 6.0% of sexually active women using this method in Canada.
- Only appropriate if family is complete; most practitioners reluctant to perform on young women.
- Pros:
  - No contraceptive routine required.
  - No significant long-term side-effects.
- Cons:
  - Usually permanent.
  - Possibility of regret.
  - Possible short-term complications related to intra-abdominal surgery.
  - Failure rates vary based on type of procedure (i.e. ligation, clip, cauterization).
  - Does not protect against STIs.

Progesterone injection (“Depo-Provera”)
- 6% of women will become pregnant after one year of typical use.
- Approximately 2.4% of sexually active women using this method in Canada.
- Pros:
  - No contraceptive routine required.
  - Most women will stop having periods.
  - No estrogen — useful in women who should not take estrogen (breastfeeding; >35yo and smoker).
- Cons:
  - Likely saturates binding sites for progesterone, androgens and glucocorticoid receptors, so common side effects include acne, weight, hair growth, BMD, insomnia, and can worsen DM.
  - Requires a healthcare visit for injection.
  - Delay in return of ovulation (thus, fertility) on average of 9 months after stopping.

Combined oral contraceptive pill (COCP)
- 9% of women will become pregnant after one year of typical use.
- Approximately 45.5% of sexually active women using this method in Canada.
- Simplified advice for missed pills:
  - If 1 pill missed, take as soon as possible, and continue with current pack.
  - If 2 pills missed, do not take the missed pills: start a new pack and use alternative birth control methods (i.e. condom) until next menstruation.
- Pros:
  - Regulates menstrual cycle, can reduce heavy flow (useful in AUB).
  - Often reduces PMS symptoms.
- Cons:
  - Daily routine required.
  - Estrogen is contraindicated in some women; increases the risk of DVT.
  - Effectiveness can be reduced by other medications.
  - All hormonal contraception options can worsen (or improve) acne.

Diaphragm
- 12% of women will become pregnant after one year of typical use.
- Approximately 0.2% of sexually active women using this method in Canada.
- Pros:
  - Non-hormonal.
  - Protects against some STIs.
- Cons:
  - Reduces spontaneity of intercourse.
  - Requires proper insertion technique.
  - Needs to be left in the vagina for 6-8 hours after intercourse.
Male condom

- **18%** of women will become pregnant after one year of typical use.
- Approximately **54.3%** of sexually active women using this method in Canada.
- **Pros:**
  - Cheap.
  - Non-hormonal.
  - Both partners can participate in, and verify use.
  - Protect against STIs.
- **Cons:**
  - Reduces spontaneity of intercourse.
  - May slip or break during intercourse.

Female condom

- **21%** of women will become pregnant after one year of typical use.
- Approximately **0.3%** of sexually active women using this method in Canada.
- **Pros:**
  - Non-hormonal.
  - Protect against some STIs.
- **Cons:**
  - Reduces spontaneity of intercourse.
  - Requires proper insertion technique.

Sponge/spermicide

- **12-28%** of women will become pregnant after one year of typical use.
- Approximately **0.8%** of sexually active women using this method in Canada.
- **Pros:**
  - Non-hormonal.
  - Not very effective with typical use.
- **Cons:**
  - Reduces spontaneity of intercourse.
  - Requires proper insertion technique, and must be inserted into vagina ahead of time.
  - Some may have allergies to spermicide.

Withdrawal method (“coitus interruptus”)

- **22%** of women will become pregnant after one year of typical use.
- Approximately **11.6%** of sexually active women using this method in Canada.
- **Pros:**
  - Non-hormonal, natural.
  - Not very effective with typical use.
  - Free.
- **Cons:**
  - Can be risky as requires self-control and practice.
  - May reduce pleasure for either partner.
  - Does not protect against STIs.

Natural family planning

- **24%** of women will become pregnant after one year of typical use.
- Approximately **2.5%** of sexually active women using this method in Canada.
- Includes **calendar method**, and/or monitoring changes in **basal temperature and cervica musuc**.
- **Pros:**
  - Non-hormonal, natural.
  - Not very effective with typical use.
  - Cheap.
- **Cons:**
  - Demands willingness, time and motivation, including a period of abstinence.
  - Affects spontaneity.
  - Does not protect against STIs.
NIHB CONTRACEPTION COVERAGE

NIHB covers the following methods of contraception:
- Male condoms.
- Copper IUD (1 device every 12 months) — Flexi-T, Liberte UT, Nova-T, Mona Lisa.
- Hormonal IUD/IUS (1 device every 2 years) — Mirena, Jaydess.
- Depo-medroxyprogesterone acetate — Depo-Provera.
- COCP.
- Progestin-only pill (POP) — Micronor.
- Vaginal contraceptive ring — Nuvaring.
- Transdermal contraceptive patch — Evra.

Emergency contraception (EC)

Consider using when (Dunn and Guilbert):
- No contraception used and there is a desire not to get pregnant.
- One missed COCP in the 1st week.
- Three or more COCPs missed in the 2nd or 3rd week.
- One or more pills missed on progestin-only pill (POP).
- When progesterone injection late by 2 weeks or more.

Canadian Contraception Consensus (Black, Guilbert, et al.):
- The copper IUD is the most effective form of EC (0.1% risk of pregnancy). Can be used up to 7 days after intercourse following a negative pregnancy test.
- Levonorgestrel (LNG) (“Plan B”, etc., available OTC) is effective up to 5 days after intercourse, but has relatively low efficacy after 72 hours (2.6% risk of pregnancy up to 72 hours).
- Ulipristal (UPA) is now available in Canada (not OTC), and is more effective than LNG (1.8% risk of pregnancy up to 72 hours). It can be used up to 5 days after intercourse, and has particularly improved efficacy over LNG after the first 72 hours.
- Hormonal EC methods (LNG and UPA):
  - Are not effective if taken on the day of ovulation or thereafter.
  - Can be less effective in overweight women (BMI >25 for LNG, >35 for UPA), but these women should not be discouraged from use.
  - If given, should be followed by starting (or continuing) regular hormonal contraception (COCP, etc.) the day of, with 7 days of backup contraception (for LNG) or 5 days after, with 14 days of backup contraception (for UPA) [ed: couldn’t be clearer, eh?].
- Follow up any use of EC with a pregnancy test within 21 days if no menstrual period, or if a copper IUD is inserted.

RECOMMENDED #FOAMED RESOURCES


REFERENCES

8. Croup

**KEY FEATURES**

1. In patients with croup,
   - Identify the need for respiratory assistance (e.g., assess ABCs, fatigue, somnolence, paradoxical breathing, in drawing)
   - Provide that assistance when indicated.
2. Before attributing stridor to croup, consider other possible causes (e.g., anaphylaxis, foreign body (airway or esophagus), retropharyngeal abscess, epiglottitis).
3. In any patient presenting with respiratory symptoms, look specifically for the signs and symptoms that differentiate upper from lower respiratory disease (e.g., stridor vs. wheeze vs. whoop).
4. In a child presenting with a clear history and physical examination compatible with mild to moderate croup, make the clinical diagnosis without further testing (e.g., do not routinely X-ray).
5. In patients with a diagnosis of croup, use steroids (do not undertreat mild-to-moderate cases of croup).
6. In a patient presenting with croup, address parental concerns (e.g., not minimizing the symptoms and their impact on the parents), acknowledging fluctuating course of the disease, providing a plan anticipating recurrence of the symptoms.

See also Differentiating respiratory symptoms in children.

**Clinical features**

- A respiratory illness characterized by inspiratory stridor, barking cough, and hoarseness resulting from inflammation of the larynx and subglottic airway.
- Often accompanied by signs of URTI: nasal discharge, congestion, coryza, fever.
- Incubation period: 3-6 days.
- Peak incidence: 6mo-3yo, during fall-winter.
- Most often viral: parainfluenza virus type 1 (most common), parainfluenza virus type 2 and type 3, RSV, adenoviruses, human coronavirus, measles, influenza, rhinovirus, enterovirus, HSV.
- Secondary bacterial infections may occur: Staph aureus, Strep pyogenes, S. pneumoniae.
- Symptoms worse at night (ED visits between 2200 and 0400), peak at 24-48 hours.
- Self-limited with resolution in 4-7 days, fluctuating course.
- Mortality rate less than 0.5%.

**Evaluation**

- Make the child as comfortable as possible as agitation is associated with increased respiratory effort.
- Rapid assessment of general appearance, vital signs, pulse oximetry, airway stability (including the presence of stridor at rest), and mental status, hydration status (consider insensible losses from fever, tachypnea, and decreased intake).
- If clinical presentation consistent with croup, further investigations (i.e. CXR, labs) is generally not indicated.
  - Neck x-ray (if done) shows a steeple sign. 

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39 — Last peer reviewed: Pending review
**Croup Severity**

<table>
<thead>
<tr>
<th>Westley Croup Score</th>
<th>MILD</th>
<th>MODERATE</th>
<th>SEVERE</th>
<th>IMPENDING RESPIRATORY FAILURE</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;2</td>
<td>3 to 7</td>
<td>&gt;8</td>
<td>&gt;12</td>
<td>Not prominent (fatigue)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Barky cough</th>
<th>Occasional</th>
<th>Frequent</th>
<th>Frequent</th>
<th>Not prominent (fatigue)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stridor</td>
<td>None or on exertion</td>
<td>Inspiratory stridor at rest</td>
<td>Prominent inspiratory +/− expiratory at rest</td>
<td>Prominent inspiratory +/− expiratory at rest</td>
</tr>
<tr>
<td>Work of breathing</td>
<td>None to mild</td>
<td>Suprasternal and/or intercostal indrawing</td>
<td>Marked suprasternal and intercostal indrawing</td>
<td>Indrawing may not be marked</td>
</tr>
<tr>
<td>Distress</td>
<td>None</td>
<td>None</td>
<td>Substantial distress/agitation</td>
<td>Lethargy</td>
</tr>
</tbody>
</table>

**Westley Croup Score**

<table>
<thead>
<tr>
<th>CLINICAL FEATURE</th>
<th>ASSIGNED SCORE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level of Consciousness</td>
<td>Normal, including sleep = 0</td>
</tr>
<tr>
<td></td>
<td>Disoriented = 5</td>
</tr>
<tr>
<td>Cyanosis</td>
<td>None = 0</td>
</tr>
<tr>
<td></td>
<td>With agitation = 4</td>
</tr>
<tr>
<td></td>
<td>At rest = 5</td>
</tr>
<tr>
<td>Stridor</td>
<td>None = 0</td>
</tr>
<tr>
<td></td>
<td>With agitation = 1</td>
</tr>
<tr>
<td></td>
<td>At rest = 2</td>
</tr>
<tr>
<td>Air Entry</td>
<td>Normal = 0</td>
</tr>
<tr>
<td></td>
<td>Decreased = 1</td>
</tr>
<tr>
<td></td>
<td>Markedly decreased = 2</td>
</tr>
<tr>
<td>Retractions</td>
<td>None = 0</td>
</tr>
<tr>
<td></td>
<td>Mild = 1</td>
</tr>
<tr>
<td></td>
<td>Moderate = 2</td>
</tr>
<tr>
<td></td>
<td>Severe = 3</td>
</tr>
</tbody>
</table>

**DIFFERENTIAL DIAGNOSIS**

- **Common:**
  - Croup
  - Anaphylaxis
  - Asthma
- **Less common:**
  - **Bacterial tracheitis:**
    - *S. aureus, H. influenzae, S. pneumo.*
    - **Airway emergency:** high fever, toxic.
    - Incidence: 1mo-6yo, fall/winter.
    - Presentation: Stridor, cough, preference for lying flat.
    - Lateral/AP XR: ragged edge, membrane spanning trachea.
    - **Differentiate from croup:** poor response to epi + dexamethasone.
    - Tx: maintain airway, IV access, clindamycin + ceftriaxone.
  - **Peritonsillar abscess:**
    - Polymicrobial.
    - Odynophagia, 'hot potato' voice, otalgia, sore neck, dysphagia, drooling, halitosis, uvula deviation, soft palate asymmetry.
Bedside US to differentiate from cellulitis.

Tx: Needle aspiration (consult ENT if not comfortable doing in ER).

Rare:

- **Epiglottitis:**
  - *H. influenzae type B* (incidence much lower with Hib vaccination), Group A/B/C strep.
  - Airway emergency.
  - Peak incidence: 2-8yo, 35-39yo.
  - Presentation: Tripoding, drooling, stridor (late sign), hoarse/muffled voice, fever, sore throat, dysphagia, cervical lymphadenopathy, co-existing AOM/meningitis/cellulitis.
  - Differentiate from croup: no barking cough, unwell appearance, copious drooling.
  - Tx: Avoid stimulating (avoid exam if high suspicion), prep for intubation (call most experienced intubator, ideally OR/ICU), IV, corticosteroids + ceftriaxone, O₂.
  - Avoid racemic epi.
  - Prevention: immunization!

- **Retropharyngeal abscess:**
  - Causative: Group A strep +/- aerobes/anaerobes of mouth flora.
  - Extension of pharyngeal infection.
  - Odynophagia, torticollis, neck swelling/pain/mass, cervical lymphadenopathy.
  - Lateral X-ray: bulging posterior pharynx >7mm at C2 and >14mm at C7.
  - CT is the imaging modality of choice for a deep neck space infection.
  - Tx: consider securing airway early, I&D, penicillin G x 10/7.
  - FB aspiration or ingestion: tracheal or esophageal.
  - Neoplasm.
  - Thermal injury.
  - Angioedema.
  - Laryngeal diphtheria.

Treatment:

- **Mild**
  - Dexamethasone 0.15-0.6mg/kg.
  - Education: anticipated course of illness, signs of respiratory distress, when to seek medical assessment.
  - Hand hygiene, contact avoidance.

- **Moderate**
  - Dexamethasone 0.15-0.6mg/kg and monitor for 4h.
  - After monitoring, if:
    - Improvement (no indrawing, no stridor at rest) educate and d/c home.
    - No improvement/worsening: admission and step up treatment.

- **Severe**
  - Dexamethasone 0.15-0.6mg/kg + nebulized racemic epinephrine 2.25% (0.5mL in 2.5mL saline) or L-epi 1:1000 (3-5mL) and monitor for 4-6h.
  - May repeat dexamethasone once.
    - If vomiting or too distressed: budesonide 2mg neb with epi.
  - After monitoring, if:
    - Improvement: educate and d/c home.
    - No improvement/worsening: admission and step up treatment.

- **Impending respiratory failure**
  - Dexamethasone 0.15-0.6mg/kg + nebulized racemic epinephrine 2.25% (0.5mL in 2.5mL saline) or L-epi 1:1000 (5mL) and monitor for 4-6h.
  - May repeat dexamethasone once.
    - If vomiting or too distressed: budesonide 2mg neb with epi.
  - Prep to intubate (airway cart at the bedside, discuss the plan).
  - After monitoring, if:
    - Improvement (persistent mild symptoms, no stridor at rest): educate and d/c home.
    - No improvement/worsening: repeat neb epi.
    - If good response: continue to observe.
    - If poor response: repeat neb epi, contact ICU.
  - Consider admission if:
• Received steroid >4h ago with ongoing respiratory distress.
• Dehydration.
• Social factors.

REFERENCES
9. Deep Venous Thrombosis (DVT)

**KEY FEATURES**

1. In patients complaining of leg pain and/or swelling, evaluate the likelihood of deep venous thrombosis (DVT) as investigation and treatment should differ according to the risk.
2. In patients with high probability for thrombotic disease (e.g., extensive leg clot, suspected pulmonary embolism) start anticoagulant therapy if tests will be delayed.
3. Identify patients likely to benefit from DVT prophylaxis.
4. Utilize investigations for DVT allowing for their limitations (e.g., Ultrasound and D-dimer).
5. In patients with established DVT use oral anticoagulation appropriately, (e.g., start promptly, watch for drug interactions, monitor lab values and adjust dose when appropriate, stop warfarin when appropriate, provide patient teaching).
6. Consider the possibility of an underlying coagulopathy in patients with DVT, especially when unexpected.
7. Use compression stockings in appropriate patients, to prevent and treat post-phlebitic syndrome.

This topic overlaps with pulmonary embolism in that a DVT may progress to a PE, but the clinical presentation, diagnosis and management can generally be considered separately.

**Presentation**

- Swollen, painful or erythematous limb (almost always lower limbs / calves).
- Clinical presentation may overlap with PE if a DVT has embolized.
- (See the Well’s criteria for DVT for a hint to typical presentation).

**Risk factors**

Also see the Padua Prediction Score.

- Previous history of DVT/clot.
- Thrombophilias (i.e. factor V Leiden, some autoimmune conditions).
- Prolonged immobility (i.e. bedridden, post-op, slight increase in risk with >6hr airplane travel).
- Pregnancy.
- Malignancy.
- ↑ age.
- Smoking.
- Obesity.
- Exogenous estrogen (i.e. OCP).

**Diagnosis**

- If you are concerned about a PE, that is the more important diagnosis — this algorithm does not apply until PE ruled out!
- Apply the Well’s criteria for DVT to arrive at a pre-test probability.
- If the patient has a low pre-test probability (<2 Well’s):
  - A negative D-dimer can safely rule out DVT.
  - A positive D-dimer should be followed by compression duplex ultrasound of the affected limb.
    - If normal, then DVT is safely ruled out.
    - If abnormal, treat.
- If the patient has a moderate or high pre-test probability (≥2 Well’s):
  - Compression duplex ultrasound of the affected limb:
    - If normal, then a D-dimer should be performed — if D-dimer negative, DVT can be safely ruled out. If D-dimer is positive, then a repeat ultrasound should be done in 3-7 days.
    - If abnormal, treat.
  - Homan’s sign is still widely done in clinical practice, but has very poor sensitivity/specificity (McGee).
A NOTE REGARDING D-DIMER

D-dimer can be a _hotly debated_ topic in Family and Emergency Medicine circles! If you think that the patient you are seeing has an _extremely low_ probability of having a DVT, then you probably shouldn’t order a D-dimer (it’s likely to be false positive!). D-dimer is not an appropriate generalized screening test for those without signs or symptoms. Similarly, D-dimer levels increase naturally with age, so that it becomes a much less useful test in older patients (age-adjusted D-dimer cutoffs _may be more useful_). 

Inpatient VTE (venous thromboembolism) prophylaxis

- Hospital accreditation standards in Canada mandate _VTE risk assessment_ when patients are admitted.
- As a result of these changes, there has been a dramatic reduction in the incidence of DVT and PE (and associated morbidity/mortality) for hospital inpatients.
- Patients who are _immobile_, who have _cancer_, and who are _post-surgery_ are at greatest risk.
- Assess all inpatients on admission using a risk scoring tool such as _Padua_ (your hospital should have one).
- _Prophylaxis can be mechanical_ (compression stockings, or intermittent pneumatic compression devices), or _chemoprophylaxis_ (with UFH, LMWH, etc.). Always _encourage mobility_ if possible!

Management

- _Anticoagulation_ is the mainstay of treatment to allow clot resolution and prevent extension of the thrombus.
- Generally, treat acutely with LMWH (i.e. enoxaparin 1mg/kg BID) in preference to UFH for a week as a bridge until your chosen long-term agent (warfarin or a novel oral anticoagulant (NOAC)) is therapeutic. Treat with long-term anticoagulation as per the timelines below; if unsure, refer to hematology.
  - Patients with a _first, provoked_ (i.e. trauma, immobility, surgery) DVT should be _treated for three months_.
  - In patients with DVT and _an ongoing trigger_ (i.e. malignancy), _treatment should be continued until the trigger is resolved_.
  - Patients with a _first, unprovoked_ (i.e. no known trigger) should be treated for _at least three months_, _but may require longer-term therapy_ in some cases. Perhaps counterintuitively, _male_ patients with a first, unprovoked clot are at higher risk of recurrence (Douketis et al.)
  - For patients with proximal DVT (i.e. above the knee extension of thrombus), risks and benefits should be discussed with the patient, generally leaning toward indefinite anticoagulation.
- Calf-vein-only DVT may be treated with one week of LMWH and long-term compression stockings rather than long-term anticoagulation _if no thrombus extension at one week_.
- Imaging (abdo/pelvic CT) is of _no benefit_ (i.e. _looking for occult cancers_) in those with a first unprovoked DVT. DVT diagnosis only slightly increases the chance of cancer (Carrier et al.)
- _Thrombophilia screen_ may be useful (in consultation with a hematologist) in young patients (<40 yrs) with a first unprovoked DVT and a positive family history of symptomatic thrombus (>2 first degree family members).
- _A single, large RCT suggests no benefit from compression stockings on preventing post-thrombotic syndrome (or for pain relief)_ (Berntsen et al.)

RECOMMENDED #FOAMED RESOURCES

Cadogan, M. Venous Thromboembolism - Lecture Notes.
http://lifeinthefastlane.com/aftb-lecture-notes-venous-thromboembolism/
REFERENCES
10. Dementia

**KEY FEATURES**

1. In patients with early, non-specific signs of cognitive impairment:
   - Suspect dementia as a diagnosis.
   - Use the Mini-Mental State Examination and other measures of impaired cognitive function, as well as a careful history and physical examination, to make an early positive diagnosis.

2. In patients with obvious cognitive impairment, select proper laboratory investigations and neuroimaging techniques to complement the history and physical findings and to distinguish between dementia, delirium, and depression.

3. In patients with dementia, distinguish Alzheimer's disease from other dementias, as treatment and prognosis differ.

4. In patients with dementia who exhibit worsening function, look for other diagnoses (i.e., don’t assume the dementia is worsening). These diagnoses may include depression or infection.

5. Disclose the diagnosis of dementia compassionately, and respect the patient's right to autonomy, confidentiality, and safety.

6. In patients with dementia, assess competency. (Do not judge clearly competent patients as incompetent and vice versa.)

7. In following patients diagnosed with dementia:
   - Assess function and cognitive impairment on an ongoing basis.
   - Assist with and plan for appropriate interventions (e.g., deal with medication issues, behavioural disturbance management, safety issues, caregiver issues, comprehensive care plans, driving safety, and placement).

8. Assess the needs of and supports for caregivers of patients with dementia.

9. Report to the appropriate authorities patients with dementia who you suspect should not be driving.

10. In patients with dementia, look for possible genetic factors to provide preventive opportunities to other family members, and to aid in appropriate decision-making (e.g., family planning).

**Definition**

An impairment in at ≥2 of the following cognitive domains:

- Memory,
- Executive function,
- Language,
- Visuospatial ability and
- Personality (behaviour).

These deficits must cause impairment in social or occupational functioning that is a significant decline from their previous functional level and must not be able to be explained by other etiologies such as delirium and psychiatric disorders.

Mild cognitive impairment (MCI) — Decline in function without significant interference in social or occupational functioning.

**Types**

**ALZHEIMER’S DISEASE (AD)**

- Caused by deposition of beta amyloid protein and neurofibrillary tangles in the cortex.
- Gradual onset (months to year).
- First deficit is usually memory (amnestic dementia) such as difficulty learning new things, episodic memory loss and difficulty with recalling recently learned information.
- Other cognitive deficits include language (word-finding difficulty, anomia, aphasia and eventually mutism) and visuospatial (getting lost in familiar settings, unable to recognize faces).
- Eventual behavioural and personality changes (e.g. agitation, irritability, psychotic symptoms and problems with grooming/dressing).
DEMENTIA WITH LEWY BODIES (DLB)

- Fluctuating levels of cognition: pronounced changes in alertness and attention (e.g. daytime drowsiness, staring into space, disorganized speech).
- Prominent visuospatial deficits.
- Recurrent visual hallucinations (animals, mystical creatures).
- Parkinsonism (early onset, within year of dementia onset).
- Less prominent memory deficits (at least in the early stages).
- May also have autonomic dysfunction presenting as syncope, falls, transient loss of consciousness, and REM sleep disorders.

**Clinical Features of DLB**

“DDaVP” — Dementia, Delirium (fluctuating cognition), and Visual hallucinations with Parkinsonism.

VASCULAR DEMENTIA

- Rare in isolation.
- Vascular dementia often co-occurs with AD, known as “mixed dementia”.
- Due to cortical vascular disease; patients have many vascular risk factors such as atherosclerosis, smoking, hypertension and diabetes.
- Often sudden drop in cognitive functioning after a CVA, or gradual if chronic small-vessel disease.
- Symptoms generally vary and include language impairment, executive impairment or psychological and behavioural problem.
- Neurological deficits may be patchy in nature.
- Mood changes and depression are common symptoms.

FRONTOTEMPORAL DEMENTIA

- Neurodegenerative dementia.
- More often onset at <65yo.
- Insidious onset, characterized mainly by behavioural and personality changes (with relatively spared memory function) with or without aphasia.
- 3 variants of FTD exist:
  - Behavioural variant (a.k.a. Pick’s Disease): personality changes, inappropriate social behaviours (e.g. emotional blunting, loss of insight, apathy, inactivity and disinhibition, inappropriate remarks, hyperorality, stereotypic behaviours, poor hygiene).
  - Semantic dementia type: effortless but meaningless speech, agnosia, semantic paraphasia) and impaired recognition of faces.
  - Progressive nonfluent aphasia type: limited speech that is agrammatic and hesitant, phonemic paraphasia and impaired comprehension.

Complications

- Severe impairment of executive function, complete dependence on others for ADLs, impaired language and comprehension, even mutism and parkinsonism.
- Behavioural and psychological symptoms of dementia include agitation and aggression (can be very distressing to family members), psychosis (mostly visual hallucinations, delusions), wandering, sleep disturbances, disruptive vocalizations, resistance to grooming, apathy.

Diagnostic approach

- Goal is to rule out reversible causes (delirium and other reversible causes such as polypharmacy, hypothyroidism, B12 deficiency, syphilis), and then assess the type of dementia (don’t assume AD).
  - Suspect dementia if your patient is repeating stories/questions, missing appointments that if out of character, have previously well-controlled chronic conditions that are now decompensating (as this may indicate patient is forgetting to take their medication), accidents, getting lost, change in behaviour, etc.
- Use a combination of effective history-taking from the patient and a “knowledgeable informant” such as a family member or caregiver, and a formal cognitive test such as the MOCA/MMSE. They can be referred for neuropsychiatric testing if suspicion is high but history or cognitive tests are ambiguous.
HISTORY

1. Obtain time course: onset (sudden vs. gradual), progressive or stepwise, worsening or stable.
2. Elicit cognitive deficits in each of the cognitive categories:
   a) Memory: episodic memory impairment, difficulty learning new things, difficulty recalling familiar names/episodes.
   b) Language: agnosia, word-finding difficulties, aphasia, mutism.
   c) Visuospatial: getting lost in familiar places, difficulty recognizing faces.
   d) Executive: reason, judgement, problem solving, planning, calculations, multistep tasks.
   e) Personality/behaviour: agitation, disinhibition, aggression, apathy.
3. Review of symptoms: psychosis (delusion, hallucinations), neurological deficits, depression screening, delirium risk factors, thyroid symptoms.
4. Medications: new meds? medications that put the patient at risk for delirium? (See STOPP Criteria (O’Mahony et al.).)
5. Risk Factors: delirium risk factors (such as hospitalization, recent surgery, narcotics), dementia risk factors.
6. Substance use and dependence.
7. Functional assessment: ask about basic and instrumental ADLs. These include grooming, feeding, bathing, dressing and balancing check book, caring for finances, transportation, medication management, respectively.

PHYSICAL EXAMINATION

Limited utility, useful for specific purposes.

- Neurological exam – neurosensory deficits, gait assessment.
- Parkinsonism screen.
- Audiology and visual acuity exams.

COGNITIVE TESTING

- **Mini-Cog** — three-word recall and clock drawing test. Total score is out of 5 (1 point for each word recalled correctly during the delayed recall and 2 points to draw clock correctly). 0-2 is highly suggestive of dementia. 2-4 minutes to complete and has minimal education and language bias.
- **Verbal fluency test** — patient recalls as many words as they can in a category e.g. animals in 60 seconds. Score less than 15 is a positive screen for dementia or MCI. It is very dependent on language and educational level so scores should be interpreted with caution and used in association with another test such as mini-cog.
- **MMSE (Mini Mental State Exam)** — 30-point scale that tests orientation, recall, attention, calculation, language and visuospatial abilities. Scores less than 24 indicate dementia, 24-26 indicate MCI and greater than 26 are normal, usually. However, the MMSE has cultural and language bias so score must be interpreted in full clinical and occupational context. MMSE can also be used to detect changes every 6-12 months in patients, which would indicate cognitive decline. In normal aging, scores should not deteriorate.
- **Montreal Cognitive Assessment (MOCA)** — 30-point cognitive screening tool that assesses the same functions as the MMSE with a cut-off score of 26. It is better able to detect MCI than MMSE. However it is available for free and in multiple languages. The educational level bias still exists.
- **Rapidly progressive dementia (RPD)** (less than 12 months from first symptoms to clinically significant impairment) should be referred to physicians with experience in managing RPD in order to mount an organized and comprehensive diagnosis process.

LABORATORY INVESTIGATIONS


NEUROIMAGING

- Neuroimaging in dementia is controversial: the AAN recommends structural neuroimaging with either a non-contrast head CT or MRI in the routine initial evaluation of all patients with dementia (Knopman et al.) (Moore et al.) recommends neuroimaging if it will change clinical management.
Management

What is the purpose/objective of treatment? Management of dementia includes non-pharmacologic (supportive) and pharmacologic treatment. The goal of treatment is to preserve or improve cognition and function for as long as possible and ensure good quality of life.

PHARMACOLOGIC TREATMENT

- RXFiles — Behavioural management in Dementia.
- Behavioural and Psychological Symptoms of Dementia — Algorithm.
- Cholinesterase inhibitors (rivastigmine, donepezil and galantamine) and NMDA receptor antagonists (memantine):
  - Evidence for use in AD (any stage), DLB, dementia of Parkinson’s and mixed dementia.
  - Variable symptomatic improvement and may delay the decline of cognitive function, but does not cure dementia or prevent its natural progress to its end stage.
  - All 3 ChEIs have similar efficacy in terms of improving cognitive function and delaying functional decline. Combination with memantine is a rational option.
  - Adverse effects: nausea, vomiting, diarrhea and sometimes syncope. Memantine may cause headaches and dizziness. Stable or improving cognitive scores indicate effectiveness of these drugs.
  - Indefinite therapy until there is no function left to lose or if patient declines it (e.g. due to side effects). Therapy may be discontinued if patient feels there is no improvement, is non-adherent, risks outweigh benefits or if side effects are overbearing.
- Behavioural and psychological symptoms of dementia (BPSD) — treat cautiously with risperidone, olanzapine and/or aripiprazole. BPSD usually resolves spontaneously so any medications started for its treatment should be discontinued at least every 6 months in order to assess baseline.
- Dementia with Lewy bodies — ChEIs have great evidence for success in DLB for the dementia as well as psychotic symptoms. Levodopa/carbidopa can also be used to treat Parkinsonian symptoms. Anticholinergics and antipsychotics (especially typical antipsychotics) must be avoided (autonomic dysfunction and EPS side effects).

SUPPORTIVE MANAGEMENT

- Caregiver stress and assistance — most common complications of dementia and may lead to elder neglect or abuse. Physician must be able to identify and offer respite options for caregivers.
- Occupational therapy — patients should be offered referral to an occupational therapist if there are any concerns from the patient or family about a decline in independence (interventions include: environmental assessment and modification to aid independent functioning; prescription of assistive technology; and tailored intervention to promote independence in activities of daily living).
- Driving safety — physicians are professionally and/or legally required to report patients who they deem unfit to drive in most provinces.
  - Good resources for determining medical fitness to drive are available at Alzheimer Society of Canada including a free e-module and patient resources.
- Home safety — periodically assess patient’s environmental supports such as availability of MedicAlert bracelets, alarm bells, bathroom railings.
- Placement issues — periodically monitor for changing dependency needs for basic and instrumental ADLs as this is an important aspect of progressive dementia. Placement must be arranged as appropriate if the need arises.

RECOMMENDED #FOAMED RESOURCES

REFERENCES


11. Depression

**KEY FEATURES**

1. In a patient with a diagnosis of depression:
   - Assess the patient for the risk of suicide.
   - Decide on appropriate management (i.e., hospitalization or close follow-up, which will depend, for example, on severity of symptoms, psychotic features, and suicide risk).

2. Screen for depression and diagnose it in high-risk groups (e.g., certain socio-economic groups, those who suffer from substance abuse, postpartum women, people with chronic pain).

3. In a patient presenting with multiple somatic complaints for which no organic cause is found after appropriate investigations, consider the diagnosis of depression and explore this possibility with the patient.

4. After a diagnosis of depression is made, look for and diagnose other co-morbid psychiatric conditions (e.g., anxiety, bipolar disorder, personality disorder).

5. In a patient diagnosed with depression, treat appropriately:
   - drugs, psychotherapy.
   - monitor response to therapy.
   - active modification (e.g., augmentation, dose changes, drug changes).
   - referral as necessary.

6. In a patient presenting with symptoms consistent with depression, consider and rule out serious organic pathology, using a targeted history, physical examination, and investigations (especially in elderly or difficult patients).

7. In patients presenting with depression, inquire about abuse:
   - sexual, physical, and emotional abuse (past and current, witnessed or inflicted).
   - substance abuse.

8. In a patient with depression, differentiate major depression from adjustment disorder, dysthymia, and a grief reaction.

9. Following failure of an appropriate treatment in a patient with depression, consider other diagnoses (e.g., bipolar disorder, schizoaffective disorder, organic disease).

10. In the very young and elderly presenting with changes in behaviour, consider the diagnosis of depression (as they may not present with classic features).

Always ask about suicidal ideation in any mental health presentation!

Depression is common: 5.4% of the Canadian population aged 15 years and over reported symptoms that met the criteria for a mood disorder in the previous 12 months, including 4.7% for major depression and 1.5% for bipolar disorder (Pearson et al.)

Assessing suicidality and risk

See Suicide — Assessing risk.

Risk factors

Certain demographics are much more likely to suffer from depression, including patients with:

- Chronic pain, especially those diagnosed with psychogenic pain.
- Post-partum patients.
- Substance abuse issues.
- Chronic disease (i.e. cancer, stroke, heart disease, diabetes).
- Other psychiatric diagnoses including anxiety, eating disorders, or PTSD.
- A family history of psychiatric illness.
- A history of trauma, esp. in childhood (also, refugees).
- Elderly patients who live alone.

Widespread screening doesn’t appear to be effective, except in high risk groups (i.e. children 12 to 18yo) (Siu).
Chapter 11. Depression

Diagnosis

DSM-V depressive diagnoses:

- Disruptive mood dysregulation disorder.
- Major depressive disorder (and major depressive episode).
- Persistent depressive disorder (dysthymia).
- Premenstrual dysphoric disorder.
- Substance/medication-induced depressive disorder.
- Depressive disorder due to another medical condition.

It is important to rule out organic disease if suspected before diagnosis and treatment: i.e. hypothyroidism, hypoandrogenism.

MAJOR DEPRESSIVE DISORDER

In the DSM-IV, grief was considered separately from major depression. In the updated DSM-V, this distinction has been eliminated — “...the decision about whether a major depressive episode (or just a normal response to loss) is present 'inevitably requires the exercise of clinical judgment based on what the clinician knows about the individual in question and the individual's cultural norms for the expression of distress in the context of loss'” (Maj).

- In order to be a disorder, it must impair function (social, occupational, educational, etc.).
- Five (5) of the following nine (9) symptoms, present almost every day (they must have at least one of the first two):
  - Depressed mood most of the day, nearly every day.
  - ↓ interest or pleasure in most activities.
  - Significant weight change (↑ or ↓ 5%), or significant change in appetite.
  - Change in sleep.
  - Psychomotor agitation or depression.
  - Fatigue / loss of energy.
  - Feelings of guilt or worthlessness.
  - ↓ concentration.
  - Suicidality (thoughts or plans).

Subtypes

- Melancholic depression.
- Atypical depression — mood reactivity and positivity, significant weight gain or increased appetite (comfort eating), etc.
- Catatonic depression — rare; mute and almost stuporous, remains immobile or exhibits purposeless or even bizarre movements.
- Postpartum depression — onset within one month after delivery; an incidence rate of 10–15% among new mothers.
- Seasonal affective disorder (SAD) — depression with seasonal variability; at least two episodes have occurred in colder months with none at other times, over a two-year period or longer.

MAJOR DEPRESSIVE DISORDER

SIGECAPS — Sleep changes, Interest (loss), Guilt (worthlessness), Energy (↑, fatigue), Concentration (↓), Appetite (↓, weight loss), Psychomotor: agitation or retardation, Suicidality (thoughts or plans).

Functional assessment / screening tools

In the acute stage after diagnosis, close followup (~weekly) with sequential functional assessments can be invaluable in guiding management.

- PHQ-9 — self-reported nine questions, based on the DSM-IV criteria.
- Beck Depression Inventory — self-reported 21 questions.
Management

Clinical pearl: **utilize peer support workers/groups if available and suitable** (i.e. people with personal experience of mental illness/depression or chronic disease), and **don’t forget the impact and input from family** in the management process.

**MAJOR DEPRESSIVE DISORDER**

- Non-pharmacological:
  - Interpersonal therapy.
  - Cognitive behavioural therapy (CBT).
  - There is Level 1 evidence to support **light therapy** in seasonal MDD and **St. John’s wort** in mild to moderate MDD. There is also some evidence for the use of **exercise, yoga and sleep deprivation**. *(Ravindran et al.)*
- Pharmacological:
  - SSRIs, SNRIs, TCAs, MAOs, melatonin agonists.

**PERSISTENT DEPRESSIVE DISORDER (DYSTHYMIA)**

The management of dysthymia is similar to MDD, although the duration of treatment may be much longer due to the much longer duration of symptoms (i.e. 2+ years) that lead to the diagnosis.

**PREMENSTRUAL DYSPHORIC DISORDER**

- Non-pharmacological (for mild symptoms): **regular exercise, stress reduction techniques**.
- Pharmacological (moderate/severe symptoms): **SSRIs** (either continuous or as **luteal phase therapy** (i.e. medication is started on day 14 of the menstrual cycle and discontinued on the first day of menses).

“**Medically clearing**” psychiatric patients in the ED

Patients presenting to ED with acute symptoms **have a causative or concomitant medical disorder in 63% of cases** *(Szpakowicz and Herd)*. After the decision is made to seek specialist help, psychiatrists will often ask for the patient to be “medically cleared” before they are admitted or seen.

The goals of medical clearance in the ED are to rule out *(Guthrie)*:  
- toxidrome or intoxication; if intoxicated, the patient should not be “cleared” until sober enough for a thorough history and adequate physical examination.  
- underlying (and contributing) medical illness.

When seeing acutely agitated psychiatric patients in the ED, it is most important to **ensure the safety of yourself and the ED staff** (see the Violent/Aggressive Patient).

If the patient is acute unwell, history taking may be difficult, underscoring the utility of a thorough physical examination. Significant points to cover:
- Presenting problem / presenting circumstances.  
- Current social circumstances / social supports / home situation.  
- Previous psychiatric contact / diagnosis / treatment.  
- Current medications.  
- Previous or current alcohol or drug use.  
- Social stressors: any recent relationship breakdown, family issues, financial issues, homelessness.  
- Medical history, especially chronic disease.  
- **Thorough** physical examination, including vital signs and **mental state examination**.

**MENTAL STATE EXAMINATION** — Appearance, Behavior, Cooperation, Speech, Thought (Process, Content), Affect, Mood, Perception (Hallucinations), Level of consciousness, Insight, Cognition, Knowledge base, Endings (Suicidal, homicidal), Reliability.
Chapter 11. Depression

Bipolar disorder

See Bipolar disorder.

RECOMMENDED #FOAMED RESOURCES


REFERENCES


Szpakowicz, M., & Herd, A. "Medically cleared": how well are patients with psychiatric presentations examined by emergency physicians? The Journal of emergency medicine, 35(4), 369–72. doi:10.1016/j.jemermed.2007.11.082


Pearson, C., Janz, T., & Ali, J. Mental and substance use disorders in Canada (tech. rep.). Statistics Canada
12. Diabetes

**KEY FEATURES**

1. Given a symptomatic or asymptomatic patient at high risk for diabetes (e.g., patients with gestational diabetes, obese, certain ethnic groups, and those with a strong family history), screen at appropriate intervals with the right tests to confirm the diagnosis.

2. Given a patient diagnosed with diabetes, either new-onset or established, treat and modify treatment according to disease status (e.g., use oral hypoglycemic agents, insulin, diet, and/or lifestyle changes).

3. Given a patient with established diabetes, advise about signs and treatment of hypoglycemia/hyperglycemia during an acute illness or stress (i.e., gastroenteritis, physiologic stress, decreased intake).

4. In a patient with poorly controlled diabetes, use effective educational techniques to advise about the importance of optimal glycemic control through compliance, lifestyle modification, and appropriate follow-up and treatment.

5. In patients with established diabetes:
   - Look for complications (e.g., proteinuria).
   - Refer them as necessary to deal with these complications.

6. In the acutely ill diabetic patient, diagnose the underlying cause of the illness and investigate for diabetic ketoacidosis and hyperglycemia.

7. Given a patient with diabetic ketoacidosis, manage the problem appropriately and advise about preventing future episodes.

Here, we primarily discuss type 2 diabetes, as it is the most common type encountered in Family Medicine, and many of the concepts are common to both type 1 and type 2. Type 1 diabetes will be discussed separately.

**Screening**

- 2013 Canadian Diabetes Association (CDA) Guidelines (Canadian Diabetes Association):
  - Screen every 3 years if >40yo.
  - Screen every 3 years if high risk (according to CANRISK risk calculator [?]).
  - Screen earlier and/or more frequently if presence of other risk factors (see below).

- Which test to use?
  - Any of: fasting glucose (FPG), 2hPG after 75g glucose drink (OGTT), A1c, or random glucose (random PG).
  - The Canadian Task Force on Preventive Health Care (CTFPHC) recommends screening with A1c.

### Diagnosis of Prediabetes & Diabetes

<table>
<thead>
<tr>
<th>TEST</th>
<th>RESULT</th>
<th>DYSGLYCEMIA CATEGORY</th>
</tr>
</thead>
<tbody>
<tr>
<td>FPG (mmol/L) — no caloric intake for at least 8 hours</td>
<td>6.1 - 6.9</td>
<td>IFG</td>
</tr>
<tr>
<td></td>
<td>≥7.0</td>
<td>Diabetes</td>
</tr>
<tr>
<td>2hPG in a 75g OGTT (mmol/L)</td>
<td>7.8 - 11.0</td>
<td>IGT</td>
</tr>
<tr>
<td></td>
<td>≥11.1</td>
<td>Diabetes</td>
</tr>
<tr>
<td>A1C (%) Standardized, validated assay, in the absence of factors that affect the accuracy of A1C &amp; not for suspected type 1 diabetes</td>
<td>6.0 - 6.4</td>
<td>Prediabetes</td>
</tr>
<tr>
<td></td>
<td>≥6.5</td>
<td>Diabetes</td>
</tr>
<tr>
<td>Random PG (mmol/L)</td>
<td>≥11.1</td>
<td>Diabetes</td>
</tr>
</tbody>
</table>

If asymptomatic, a repeat confirmatory test (FPG, A1C, or a 2hPG in a 75 g OGTT) must be done. If symptomatic, diagnosis made and begin treatment.

You can get a falsely elevated/lowered A1c with hemoglobinopathies, hemolytic anemia, iron deficiency, or severe renal/hepatic impairment.

**REMEMBER**

Remember, screening by definition doesn't include symptomatic patients and those who you have clinical suspicion of disease — those patients should be tested in a timely fashion.
RISK FACTORS FOR THE DEVELOPMENT OF TYPE 2 DIABETES

- Age >40 years.
- First-degree relative with type 2 diabetes.
- Member of high-risk population (i.e. Aboriginal, African, Asian, Hispanic or South Asian descent).
- History of prediabetes (IGT, IFG or A1c 6.0%-6.4%).
- History of gestational diabetes mellitus.
- History of delivery of a macrosomic infant.
- Presence of end organ damage associated with diabetes:
  - Microvascular (retinopathy, neuropathy, nephropathy).
  - Macrovascular (coronary, cerebrovascular, peripheral).
- Presence of vascular risk factors:
  - HDL cholesterol level <1.0 mmol/L in males, <1.3 mmol/L in females.
  - Triglycerides ≥1.7 mmol/L.
  - Hypertension.
  - Overweight.
  - Abdominal obesity.
- Presence of associated diseases:
  - Polycystic ovary syndrome.
  - Acanthosis nigricans.
  - Psychiatric disorders (bipolar disorder, depression, schizophrenia).
  - HIV infection.
  - Obstructive sleep apnea.
- Use of drugs associated with diabetes:
  - Glucocorticoids.
  - Atypical antipsychotics.
  - Highly active antiretroviral therapy (HAART).
  - Other (see Appendix 1 in the guidelines).
- Other secondary causes (see Appendix 1 in the guidelines).

Prevention

Target prevention (a.k.a. lifestyle interventions) in patients at high-risk: prediabetics, obese, or other risk factors (see above).

- Healthy diet (Mediterranean, vegetarian, DASH)
- Moderate weight loss (aim for 5% of initial body weight) until at BMI target (<25)
- Regular physical activity: aerobic exercise ≥150 min/week, resistance training 3x/week

Management

Lifestyle interventions and/or metformin for all type 2 diabetes patients.

GLYCEMIC CONTROL

Glycemic control is often easiest to achieve with insulin early in the disease course — consider starting insulin early in willing patients.

- If A1c <8.5%:
  1. Trial lifestyle interventions and/or metformin for 2-3 months.
  2. If A1c target not met, start or increase metformin.
  3. If A1c target still not met, increase dose of metformin (max 2.5g/day), or add another agent.
- If A1c >8.5%:
  1. Start metformin, consider adding another initial agent.
  2. If A1c target still not met, increase dose of metformin (max 2.5g/day), or add another agent.
- If symptomatic hyperglycemia with metabolic decompensation:
  1. Start metformin and/or insulin.
  2. If A1c target still not met, start/increase dose of metformin (max 2.5g/day) or insulin.
Type 2 diabetes glucose lowering therapies

Consider both patient and agent factors when choosing an antihyperglycemia agent: degree of hyperglycemia, risk of future hypoglycemia, overweight or obese, comorbidities (renal, cardiac, hepatic) and preferences and access to treatment. Agent factors: glucose lowering efficacy and durability, risk of inducing hypoglycemia, effect on weight, contraindications and side effects, cost and coverage.

### Glucose lowering therapies

<table>
<thead>
<tr>
<th>CLASS</th>
<th>A1C LOWERING</th>
<th>HYPOGLYCEMIA</th>
<th>WEIGHT</th>
<th>OTHER CONSIDERATIONS</th>
<th>COST</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha-glucosidase inhibitor (acarbose)</td>
<td>↓</td>
<td>Rare</td>
<td>neutral to ↓</td>
<td>Improved postprandial control, GI side effects</td>
<td>$$</td>
</tr>
<tr>
<td>Incretin agents: DPP-4 Inhibitors (sitagliptin) GLP-1 receptor agonists (liraglutide)</td>
<td>↓↓</td>
<td>Rare</td>
<td>neutral to ↓</td>
<td>GI side effects</td>
<td>$$$</td>
</tr>
<tr>
<td>Insulin</td>
<td>↓↓↓</td>
<td>Yes</td>
<td>↑↑</td>
<td>No dose ceiling, flexible regimen</td>
<td>$-$$$$</td>
</tr>
<tr>
<td>Insulin secretagogue: Sulfonylurea (gliclazide, glyburide)</td>
<td>↓↓</td>
<td>Yes</td>
<td>↑</td>
<td>Gliclazide and glimepiride associated with less hypoglycemia than glyburide</td>
<td>$</td>
</tr>
<tr>
<td>Insulin secretagogue: Meglitinides (repaglinide)</td>
<td>↓↓</td>
<td>Yes</td>
<td>↑</td>
<td>Less hypoglycemia</td>
<td>$$</td>
</tr>
</tbody>
</table>

Also see from the CDA:
- **Highly recommended**: [Sample Diabetes Patient Care Flow Sheet for Adults](#)
- [Approximate Cost Reference List for Antihyperglycemic Agents](#)
- [Therapeutic Considerations for Renal Impairment](#)

### Starting insulin

There are multiple algorithms (Strange) that can achieve glycemic control, although more aggressive strategies increase the risk of hypoglycemia. A straightforward algorithm is detailed below:

1. Stop oral antihyperglycemics (except metformin).
2. Start with a long-acting insulin (insulin glargine, insulin detemir) at a dose of 10 units QHS.
3. Titrate the QHS dose in the evenings twice a week, increasing by 2 units each time, until morning (fasting) glucose is at target (4-7).
4. Once fasting glucose is at target, if A1c is not at target after 3 months, consider adding in postprandial self-monitoring of glucose and mealtime short-acting insulin (once/day, working up to 3x/day if necessary).

### Self-monitoring of blood glucose (SMBG)

- Patient adherence is an important factor as frequent testing can have a significant quality of life impact.
- Generally, there is no benefit to starting SMBG in patients who are solely on oral antihyperglycemics (a.k.a. no insulin).
- A large, nonrandomized study of individuals with stable type 2 diabetes using insulin, testing at least 3 times a day was associated with improved glycemic control (Sheppard et al.)
- Patient education is essential:
Chapter 12. Diabetes

1. How and when to perform SMBG.
2. How to record the results in an organized fashion.
3. The meaning of various BG levels.
4. How behaviour and actions affect SMBG results.

- SMBG targets:
  - New diagnosis of type 2 diabetes: test once/day at different times for six months.
  - Patients who are taking oral antihyperglyemics and at target: 1-2x/week or less.
  - Patients who are starting a new medication that can cause hypo/hyperglycemia, or are acutely unwell: 1-2x/day.

Glycemic targets

- In most people, target A1c is ≤7.0%.
- Target A1c of 7.1-8.5% (or occasionally higher) in those who are at risk of hypoglycemia and falls from tighter control, or who are not likely to benefit from tighter sugar control: the elderly, frail, those with cognitive impairment, those who are highly functionally dependent, those with limited life expectancy, extensive coronary artery disease, or multiple co-morbidities.
  *Multiple RCTs have confirmed that tighter glucose control improves microvascular (but not necessarily macrovascular) outcomes.*
- If patient is SMBG, target preprandial BSL of 4-7mmol/L, and 2hr postprandial BSL of 5-10mmol/L.

VASCULAR PROTECTION

See the flowchart: CDA: Vascular protective medications.

- Does the patient have evidence of macrovascular disease? *(Cardiac ischemia (silent or overt), peripheral arterial disease, or cerebrovascular / carotid disease)*
  - Consider starting ASA + ACE-i/ARB + statin.
- Does the patient have evidence of microvascular disease? *(Retinopathy, nephropathy (ACR ≥2.0), or neuropathy)*
  - Consider starting ACE-i/ARB + statin.
- Is the patient ≥55yo?
  - Consider ACE-i/ARB + statin.
- Is the patient ≥40yo or is >30yo with diabetes for >15 years, OR warrants statin therapy according to the CCS lipid guidelines?
  - Consider starting statin.
- ASA is not recommended for primary prevention of cardiovascular disease, even in diabetics.
- ACE-i/ARB doses should be at studied target doses if possible: perindopril 8mg daily (EUROPA), ramipril 10mg daily (HOPE), telmisartan 80mg daily (ONTARGET).

*ABCD*ES FOR DIABETICS

A1c — optimal glycemic control (usually ≤7%)
BP — optimal blood pressure control (<130/80 mmHg)
Cholesterol — LDL-C ≤2.0 mmol/L if decision made to treat (see algorithm and Risk Assessment Tool)
Drugs (cardioprotective medications: ASA, ACE-i, statins)
Exercise / Eating — Discuss regular physical activity, healthy eating, achievement and maintenance of healthy body weight (BMI <25)
Smoking cessation

PATIENT SELF-MANAGEMENT

*Specifically discuss self-management with patients who have a new diagnosis, or are poorly controlled.*
Discuss signs, symptoms and treatment of hyperglycemia and hypoglycemia.

Hyperglycemia

- polyuria, polydipsia, polyphagia, dry mouth, unexplained weight loss.
- blurred vision, fatigue, pruritus, paresthesia, arrhythmias, coma.

Hypoglycemia

- Neurogenic: diaphoresis, anxiety, tremo, palpitations, hunger, nausea, tingling.
- Neuroglycopenic: dizziness, fatigue, weakness, confusion, behavioral changes, convulsions, coma.
- Effect of glucagon: hunger, rumblings (↑ stomach contractions), N/V, abdominal pain.
- Treatment (if blood glucose ≤4 mmol/L):
  - Conscious patient:
    - 3 teaspoons of table sugar dissolved in water, or
    - ¼ cup of juice or soft drink, or
    - 6 lifesaver candies, or
    - 1 tablespoon of honey.
  - Unconscious patient:
    - 1mg glucagon IM/SC (at-risk patients should carry a glucagon kit for carers/family to use while awaiting medical aid).
    - D50W 20-50ml IV, slow push over 1-3 minutes.
  - If significantly symptomatic, unwell, unresponsive to initial treatment or otherwise concerning:
    - Repeat fingerprick glucose 15 minutes later.
    - If blood glucose is ≤4 mmol/L, repeat treatment.
  - Once corrected and patient is conscious, alert, and without significant ongoing symptoms:
    - Instruct patient to be sure to eat a meal or snack consumed at the usual time of day.
    - If the next meal is >1 hour wait, eat a carbohydrate- and/or protein-heavy snack.
    - Consider adjusting medications.

Goals of self-management

The goal of self-management in diabetes is to enable patients to function effectively in managing symptoms, preventing future complications, and maintaining and improving their quality of life.

5 elements to achieve successful self-management in chronic disease:

- Assess,
  - Assess patient’s willingness and ability to achieve change. (i.e. “How important to you is your diet, and how can we work on helping to manage your diabetes through changes to your diet?”)
- Educate,
  - Patients require education about their disease, on how to manage symptoms, and how prevent complications. Consider referral to a DNE (diabetic nurse educator) if one is available.
- Collaborate,
  - Chronic disease management requires a collaborative approach that includes strong physician listening skills, and takes into account the patient’s wishes and goals.
- Set goals, and
  - Follow the S.M.A.R.T. algorithm for effective goalsetting with patients: goals should be specific, measurable, achievable, realistic, and time-limited (i.e. “I am going to check and record my blood sugar once every day for the next 2 weeks and bring that record to my followup appointment.”).
- Followup regularly.
  - Frequent, brief patient-physician interaction improves chronic disease outcomes as compared to longer but less frequent contact.

Ongoing followup and investigations

Most provincial chronic disease management (CDM) programs expect diabetic patients to have A1c monitoring and clinic followup every ~3 months.
INITIAL VISIT

- Fasting blood glucose.
- A1c.
- Lipid profile.
- Albumin/creatinine ratio (ACR) and eGFR.
- Ophthalmology referral.
- Consider podiatry referral.
- Erectile dysfunction questionnaire (i.e. IIEF questionnaire).
- Discuss smoking cessation.
- Fill out and provide a customized diabetes plan (i.e. Managing My Diabetes - My Action Plan).

AT EVERY THREE-MONTHLY VISIT

- BMI / waist circumference.
- BP (target and treatment threshold for diabetics are both <130/80 — see Hypertension).
- A1c.
- Mental health screening PRN (i.e. PHQ-9) — depression is more common in those with any chronic disease.
- Discuss symptoms of erectile dysfunction.

AT EVERY YEARLY VISIT

- Lipids (may not need to treat to target or monitor lipid levels once therapy is initiated! (Allan et al.)).
- ACR (albumin-creatinine ratio).
  - If negative: ACR and eGFR yearly.
  - If positive: ACR and eGFR every 6 months.
- 10g monofilament / foot examination.

WHEN TO REFER

- Nephrology
  - Chronic progressive loss of renal function.
  - ACR consistently ≥60mg/mmol.
  - eGFR <30mL/min (i.e. new Stage 3 CKD).
  - Persistent hyperkalemia.
  - Increase in Cr of ≥30% within 3 months of initiation of ACE-i or ARB (up to 30% rise is acceptable), or inability to tolerate an ACE-i/ARB for renal protection.
- Internal Medicine
  - Hypertension refractory to treatment.
  - No response to PDE-5 inhibitors.
  - Contraindication to PDE-5 inhibitors.
- Podiatrist
  - Ulcers and foot complications.
- Ophthalmology (should be routinely referred and/or have regular optometrist followup if diabetic retinopathy screening offered.)
  - Suspected vision changes, new retinopathy, vitreous hemorrhage or macular edema.
Acute unwell diabetic patients: Diabetic ketoacidosis (DKA) and Hyperglycemic hyperosmolar state (HHS)

Acute unwell diabetic patients may present with:

- **History:**
  - Classic diabetic symptoms: polyuria, polyphagia, polydipsia
  - Neuro symptoms: H/A, fatigue, lethargy, ↓ LOC (confusion, coma)
  - GI symptoms: N&V, abdominal pain, decreased appetite

- **Physical examination:**
  - Dehydration: dry skin (no sweating) with ↓ turgor
  - Postural hypotension
  - Kussmaul breathing (DKA)
  - Generalized abdominal tenderness
  - Sickly sweet / "fruity" breath (DKA only — ketones)

- **Common precipitants:** infection (most common), insulin non-adherence, ischemia (MI, PE, stroke, mesenteric ischemia), EtOH, trauma, meds (corticosteroids, thiazides, antipsychotics, sympathomimetics), abdominal pathology (pancreatitis, cholecystitis, appendicitis, splenic injury)

<table>
<thead>
<tr>
<th>DKA versus HHS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DKA</strong></td>
</tr>
<tr>
<td>Develops fast (over hours/a day)</td>
</tr>
<tr>
<td>BG &gt;14</td>
</tr>
<tr>
<td>Ketone bodies in urine/plasma (ketotic)</td>
</tr>
<tr>
<td>pH &lt;7.3 (acidotic)</td>
</tr>
<tr>
<td>Bicarb &lt;18 (metabolic acidosis)</td>
</tr>
<tr>
<td>Significant dehydration (average: 9L total body water deficit)</td>
</tr>
<tr>
<td>Serum osmolality &gt; 320 mOsm / Kg</td>
</tr>
</tbody>
</table>

**INITIAL INVESTIGATIONS**

- Spot glucose (and serum for confirmation).
- Urinary and/or serum ketones (ketosis).
- Serum osmolality (for HHS).
- Arterial blood gas (acidosis).
- CBC.
- Urea, electrolytes, creatinine, and extended electrolytes (Ca, Mg, PO₄).
- LFTs, albumin, CK.
- Urinalysis.

Specific presentations:
- Suspected pancreatitis: lipase.
- Infection - refer to your sepsis protocol (typically serum lactate, urinalysis, urine culture, blood cultures x 2, and CXR, and early empiric antibiotics).
- Women: βhCG.
- ≥30 years of age: ECG, troponin (elevated troponin in DKA is strongly correlated with future major cardiovascular event [Eubanks et al.]).

**MANAGEMENT**

Acute management of DKA involves three important issues: correcting the (usually severe) dehydration, giving insulin to correct the serum glucose, and monitoring serum potassium (K⁺) frequently and replacing any deficit to prevent severe hypokalemia. Your facility should have a DKA/HHS protocol (especially in pediatrics).

- Ensure ABCs
- Obtain IV access with 2 large bore IVs (this allows you to run NS continiously while adjustments are made on the second line)
- Attach capnography and pulse oximetry, and consider cardiac monitoring: should *always* be monitored if K⁺ replacement ≥20mEq/hr.
• **Measure serum glucose hourly:** electrolytes, plasma osmolality, and arterial or venous pH every two to four hours until the patient is stable.

• **Determine and treat the underlying cause** of DKA (i.e. infection).

• **Replete fluid deficits:**
  - Patients with DKA have a **large total water deficit**.
  - If the patient is shocked, administer fluids as per sepsis/shock protocol, typically 20-30mg/kg bolus as rapidly as possible.
  - Administer NS IV at 10-20 mL/kg/hr (1-1.5L/hour in adults) for the first few hours (slow the rate or total volume infused in heart failure patients).
  - Switch to D5NS when serum glucose reaches 11.1 mmol/L (easy to remember: same as the random BSL level required for a diagnosis of type 2 DM).

• **Fix potassium (K⁺) deficits:**
  - Patients with DKA have a **large total body potassium deficit**, and the serum potassium does not correlate with this.
  - If initial serum K⁺ is below 3.3 mEq/L: **hold insulin** and give 20-40mEq/hr of KCl (for adults, put KCl 20-40mEq in each bag of NS; prefer premixed bags) *(Remember cardiac monitoring).*
  - If initial serum K⁺ is between 3.3 and 5.3 mEq/L: give KCl 20-30mEq/hr (20-30mEq of KCl in each litre of NS), aim to maintain serum K⁺ between 4 to 5 mEq/L.
  - If initial serum K⁺ is above 5.3 mEq/L, do not give potassium. Check serum K⁺ every 2 hours and start K⁺ if <5.3mEq/L.

• **Administer insulin:**
  - Do not give insulin if initial serum K⁺ is below 3.3 mEq/L! Fix the K⁺ and fluid deficit first. This implies that you should **not start insulin at all until initial lab investigations are reported**.
  - Administer regular insulin *(Humulin R, Novolin R)* to all patients with an adequate serum K⁺. Give **0.1 units/kg IV bolus followed by 0.1 units/kg/hr** or alternatively, **0.14 units/kg/hr with no bolus** *(Kitabchi et al.)*
  - If serum glucose does not fall by at least 10% after the first hour, double the rate of insulin infusion.
  - When the serum glucose reaches 11.1mmol/L, dextrose should be added to the IV fluid (as above).
  - Continue insulin infusion until ketoacidosis is resolved (no/trace urine ketones, venous pH >7.3, and anion gap <12 mEq/L), and serum glucose is below 11.1 mmol/L. Start subcutaneous sliding scale insulin (SSI).

### DKA CLINICAL PEARL

**DKA (and HHS) is a fluid problem:** start to correct the typically profound dehydration (total water deficit can be >9L) and this will help **all by itself** to correct the glucose through dilution, **before** starting insulin or other interventions.

### RECOMMENDED #FOAMED RESOURCES


REFERENCES
Screening for Type 1 and Type 2 Diabetes. Canadian Journal of Diabetes, 37, S295–S297. doi:10.1016/j.jccjd.2013.02.003
Screening for Type 2 Diabetes. https://canadiantaskforce.ca/guidelines/published-guidelines/type-2-diabetes/
Frendl, G. Pocket ICU. http://amzn.to/1NKw3tq
Zane, R. D., & Kosowsky, J. M. Pocket Emergency Medicine (Pocket Notebook Series). http://amzn.to/1ld3rly
13. Dysuria

**KEY FEATURES (DYSURIA)**

1. In a patient presenting with dysuria, use history and dipstick urinalysis to determine if the patient has an uncomplicated urinary tract infection.
2. When a diagnosis of uncomplicated urinary tract infection is made, treat promptly without waiting for a culture result.
3. Consider non-urinary tract infection related etiologies of dysuria (e.g., prostatitis, vaginitis, sexually transmitted disease, chemical irritation) and look for them when appropriate.
4. When assessing patients with dysuria, identify those at higher risk of complicated urinary tract infection (e.g., pregnancy, children, diabetes, urolithiasis).
5. In patients with recurrent dysuria, look for a specific underlying cause (e.g., post-coital urinary tract infection, atrophic vaginitis, retention).

**KEY FEATURES (URINARY TRACT INFECTION)**

1. Take an appropriate history and do the required testing to exclude serious complications of urinary tract infection (UTI) (e.g., sepsis, pyelonephritis, impacted infected stones).
2. Appropriately investigate all boys with urinary tract infections, and young girls with recurrences (e.g., ultrasound).
3. In diagnosing urinary tract infections, search for and/or recognize high-risk factors on history (e.g., pregnancy; immune compromise, neonate, a young male, or an elderly male with prostatic hypertrophy).
4. In a patient with a diagnosed urinary tract infection, modify the choice and duration of treatment according to risk factors (e.g., pregnancy, immunocompromise, male extremes of age); and treat before confirmation of culture results in some cases (e.g., pregnancy, sepsis, pyelonephritis).
5. Given a non-specific history (e.g., abdominal pain, fever, delirium) in elderly or very young patients, suspect the diagnosis and do an appropriate work-up.
6. In a patient with dysuria, exclude other causes (e.g., sexually transmitted diseases, vaginitis, stones, interstitial cystitis, prostatitis) through an appropriate history, physical examination, and investigation before diagnosing a urinary tract infection.

**History**

- **Cystitis**: dysuria, urinary frequency, gross hematuria, suprapubic pain/tenderness, cloudy urine (pyuria). Should be in the absence of vaginal symptoms.
- **Pyelonephritis**: cystitis symptoms, plus nausea, vomiting, fever, chills, new lower/mid back pain.
- **Vaginitis**: external (as opposed to internal) burning sensation, pruritis, change in vaginal discharge, dyspareunia.

**Physical examination**

- **If unwell**: Vital signs including temperature, BP, PR, RR. Consider checking capillary glucose.
- **If pyelonephritis suspected**: abdominal examination + CVA tenderness.
- **If vaginitis symptoms**: pelvic examination +/- vaginal swabs for microscopy and culture.
- **If male**: consider rectal examination ? prostatism ? BPH.

**Uncomplicated urinary tract infections (UTIs)**

- **Uncomplicated UTIs** occur in otherwise healthy, nonpregnant women.
- **Bottom line**: >50% of women who complain of cystitis or pyelonephritis symptoms in the absence of vaginitis-type symptoms will have a culture-positive UTI, and should be treated empirically.
  - Women with two or more cystitis symptoms in the absence of vaginitis symptoms have a culture-positive UTI in 90% of cases.
Chapter 13. Dysuria

RISK FACTORS

• Previous UTIs.
• Sex (much higher incidence in females).
• Age (women of childbearing age, incidence increases with age in men).
• Anatomical / functional abnormalities of the renal tract (i.e. BPH, congenital abnormalities).
• Sexual intercourse (incidence in sexually active women).

Complicated UTIs

• Anatomical or functional abnormality of the renal tract (i.e. stones, strictures, transplant).
• Immunosuppressed patients (i.e. poorly controlled DM).
• Pregnant patients.
• UTIs in males.

Recurrent UTIs

• Definition:
  - 2+ UTIs within six months, or 2+ positive cultures with 12 months.
  - Reinfection: infection with a new organism (culture positive) at any time, or, if the same organism, 2+ weeks after completion of treatment of the initial infection.
  - Relapse / treatment failure: return of symptoms with positive culture of the same organism within 2 weeks of completion of treatment of the initial infection.
  - Asymptomatic bacteruria: urine culture positive (>10^6 CFUs/ml) without any attributable symptoms.
• Look for a cause or predisposing factor and manage:
  - Post-coital UTIs (advise to always void after intercourse).
  - Atrophic vaginitis (consider topical estrogen therapy).
  - Urinary retention (investigate and treat cause).

Management

EMPIRIC TREATMENT

<table>
<thead>
<tr>
<th>UTI CAUSATIVE ORGANISMS</th>
</tr>
</thead>
<tbody>
<tr>
<td>KEEPS — Klebsiella pneumonia, E. coli, Enterococcus faecalis, Proteus mirabilis, Staph. saprophyticus.</td>
</tr>
<tr>
<td>In Saskatchewan, most common in order: E. coli, E. faecalis, K. pneumoniae, and P. mirabilis.</td>
</tr>
</tbody>
</table>

• Uncomplicated UTI:
  - First-line: TMP/SMX DS 1 tab BID x 3/7, Macrobid 100mg BID x 5/7.
  - Second-line: Fosfomycin 3g x 1 dose, Cephalexin 500mg QID x 7/7, Ciprofloxacin 500mg BID x 3/7.
• Recurrent:
  - First-line: TMP/SMX DS 1 tab BID x 14/7, Macrobid 100mg BID x 14/7.
  - Second-line: Amoxiclav 875mg BID x 7/7, Ciprofloxacin 500mg BID x 3/7.
• UTI in pregnancy:
  - First-line: Fosfomycin 3g x 1 dose, Amoxicillin 500mg TID x 7/7, Macrobid 100mg BID x 5/7 (avoid nitrofurantoin at 36+ weeks).
  - Second-line: TMP/SMX DS 1 tab BID x 3/7 (avoid TMP in 1st trimester and last 6 weeks).
• Pyelonephritis:
  - First-line: Ciprofloxacin 500mg BID x 7/7.
  - Second-line: Amoxiclav 875mg BID x 10-14/7, consider inpatient IV therapy.
  - Consider testing for STIs if pyelonephritis diagnosed.
• In the elderly, renal dose adjustment is often necessary.

Empiric regimens referenced from the RXFiles: UTI Treatment Options - Adult.
Chapter 13. Dysuria

URINALYSIS / DIPSTICK

- Urinalysis is useful primarily as a rule-out test: if urine is not cloudy (pyuria), NPV of 97%.
  - A positive result for leukocyte esterase or nitrites is fairly specific (82%) (Bent et al.) for a urinary tract infection, but isn’t useful for ruling-out in the presence of positive symptoms.
- Urine dipstick is neither sensitive nor specific if patient is catheterized.

URINE CULTURE

- Culture is not necessary in uncomplicated UTIs — treat empirically.
- Culture all complicated UTIs, and consider treating empirically while awaiting results if clinically indicated (i.e. suspected pyelonephritis).
- In pregnant patients, at least one urine culture should be performed at the end of the first trimester, and asymptomatic bacteruria should be treated (Allen et al.)
- In the elderly in LTC settings, and in those with indwelling urethral catheters, do not treat asymptomatic bacteruria: treat based on signs and symptoms, not testing (Diagnosis and Management of Urinary Tract Infections in Long Term Care Facilities).

INVESTIGATIONS

- Culture all complicated UTIs, and consider treating empirically while awaiting results if clinically indicated (i.e. suspected pyelonephritis).
  - In children:
    - Urine samples from children who are not toilet trained can include urethral catheterization, suprapubic aspiration (SPA), a pediatric urine collection bag or leaving the child with the diaper off and obtaining a clean-catch urine when the child voids.
    - Bag urine is most useful as a rule-out (if normal), as there is a high rate of contamination.
    - Suspected cystitis does not require imaging (Robinson et al.)
    - Renal ultrasound should be done to rule out structural abnormalities or vesicoureteral reflux (VUR):
      - <2yo with a 1st febrile UTI.
      - Any age with recurrent febrile UTIs.
      - Who do not respond as expected to antibiotic therapy.
      - Any age, with a family history of renal disease, poor growth, or hypertension.
    - Voiding cystourethrogram (VCUG) should be done to rule out vesicoureteral reflux:
      - Any age with 2+ febrile UTIs.
      - Any age with 1st febrile UTI and: (Shaikh and Hoberman)
        - Abnormalities on renal ultrasound, or
        - Temperature over 39°C and a pathogen other than E. coli, or
        - Poor growth or hypertension.
  - In elderly women with asymptomatic bacteriuria, do not culture and do not treat.

FOLLOWUP

- No need to test for cure (repeat dipstick / culture) unless symptoms recurrent or persistent.
- Hematuria is common with UTIs, but persistent hematuria following treatment requires further investigation.
- If a culture is sent and the pathogen shows resistance to your empiric antibiotic choice, an alternative antibiotic is only required if symptoms persist.

RECOMMENDED #FOAMED RESOURCES

Thoma, B. Urinalysis Voodoo. https://boringem.wordpress.com/2012/12/12/urinalysis-voodoo/
Diagnosis and Management of Urinary Tract Infections in Long Term Care Facilities. http://www.topalbertadoctors.org/cpgs/
REFERENCES
14. Earache

**KEY FEATURES**

1. Make the diagnosis of otitis media (OM) only after good visualization of the eardrum (i.e., wax must be removed), and when sufficient changes are present in the eardrum, such as bulging or distorted light reflex (i.e., not all red eardrums indicate OM).
2. Include pain referred from other sources in the differential diagnosis of an earache (eg. Tooth abscess, trigeminal Neuralgia, TMJ dysfunction, pharyngitis, etc.).
3. Consider serious causes in the differential diagnosis of an earache (eg. tumors, temporal arteritis, mastoiditis).
4. In the treatment of otitis media, explore the possibility of not giving antibiotics, thereby limiting their use (e.g., through proper patient selection and patient education because most otitis Media is of viral origin), and by ensuring good follow-up (e.g., reassessment in 48 hours).
5. Make rational drug choices when selecting antibiotic therapy for the treatment of otitis media. (Use first-line agents unless given a specific indication not to.)
6. In patients with earache (especially those with otitis media), recommend appropriate pain control (oral analgesics).
7. In a child with a fever and a red eardrum, look for other possible causes of the fever (i.e., do not assume that the red ear is causing the fever).
8. Test children with recurrent ear infections for hearing loss.

**Causes**

- Most common cause of otalgia in children is otitis media.
- Primary causes are more common in children; referred pain (secondary otalgia) from another source is more common in adults; but both populations can present with either.

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<th>SECONDARY OTALGIA</th>
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<td>Primary otologic neoplasms</td>
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<td>Herpes Zoster (Ramsay-Hunt syndrome)</td>
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<td>Acute mastoiditis</td>
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<td><strong>Trigeminal neuralgia</strong></td>
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<tr>
<td><strong>Temporal arteritis</strong></td>
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</table>

**History**

- Severity of otalgia may not reflect the seriousness of the underlying condition, so a complete history should always be taken.
- Inquire about associated symptoms of hearing loss, tinnitus, aural fullness, dizziness and vertigo, otorrhea, vision loss, voice changes, current or recent infections, and systemic symptoms including fever.
- Ask: sudden or gradual onset, precipitating events and occupation.
- Inquire about recent airplane travel or scuba diving.
- Shorter timeframe usually indicates a primary cause while longer timeframe points to secondary causes.
Physical examination

- Always examine both ears with otoscope, starting with non-painful ear.
- Painful ear might be very painful so be gentle: otitis externa may be differentiated from otitis media by pain on movement of pinna.
- To ease examination, or if a foreign body is found and needs to be removed, appropriate analgesia and local anesthetic may be helpful (i.e. 3 drops of 2% lidocaine).
- For adults, pull pinna upwards and backwards; for children, downwards and backwards.
- Bulging, erythematous tympanic membrane is indicative of otitis media.
- If ear and otoscopic examinations do not reliably reveal the cause, complete head and neck (including cranial nerves and lymph nodes) examination should be done looking for secondary causes.

RED FLAGS

- Oropharyngeal symptoms (dysphagia, dysphonia, odynophagia).
- Neurological symptoms (headaches, drowsiness).
- Hemoptyisis.
- Unexplained weight loss.
- Hearing loss (gradual or sudden).
- Any vision changes/loss.
- Immunosuppression (i.e. poorly controlled DM).
- Persistent fever.
- Offensive discharge >9 days.
- Swelling posterior to ear.
- Persistent, worsening pain unresponsive to treatment.

INVESTIGATIONS

- Rarely needed for otalgia.
- Ear swabs should be taken for recurrent otitis externa.
- Hearing tests if associated hearing loss, especially if doesn’t resolve with resolution of otalgia.
- CT head if suspicious for malignancy.

Management

OTITIS MEDIA

- A 10-day course of therapy is recommended in current Canadian guidelines for children <2 years of age (Le Saux and Robinson) (Hoberman et al.)
- First-line antibiotic for acute otitis media (adults and children) is amoxicillin 80mg/kg divided BID or TID.
- A bulging tympanic membrane, especially if yellow or hemorrhagic, has a high sensitivity for AOM.
- Watchful waiting can be considered instead of initial antibiotics for children >6mo and <2yrs who are otherwise well (not systemically unwell), with a mild fever (<39°C) and reliable caregivers.
- With appropriate treatment, should resolve within 48 hours; patient should be re-evaluated at 10 days if symptoms haven’t resolved.
- In children, chronic otitis media occurs after repeated bouts of acute otitis media. In adults, chronic otitis media tends to occur with perforation of the tympanic membrane that will heal, typically with a history of recurrent ear infections in childhood.
OTITIS EXTERNA
- "Swimmer’s ear", "surfer’s ear", and "tropical ear"; often secondary to a frequently "wet" external ear canal.
- Breakdown of skin → cerumen barrier → inflammation and edema → obstruction and pruritus → further injury from scratching → alteration in quality and amount of cerumen produced, impairment of epithelial migration, and elevated pH → ideal environment for numerous pathogens.
- Risk factors: water exposure (including swimming), trauma, hearing aids, allergic contact dermatitis.
- Symptoms: Pain and tenderness localized to external auditory meatus; other symptoms may include pruritus, discharge, hearing loss, and pain with movement of jaw.
- Tenderness when auricle is pulled or with tragal pressure are indicative of otitis externa, but may be absent in mild cases.
- Diagnosed clinically (culture may be advised in severe or recurrent cases, or in immunosuppressed patients).
- Fluid in the middle ear or a perforated tympanic membrane should raise the question of otitis media.
- Management: aural toilet including keeping ear dry, dressings, topical antimicrobials (+/- steroid combination: ciprofloxacin/dexamethasone otic), analgesia, wicks.
- Other antibiotics to consider for coverage of P. aeruginosa and S. aureus: ciprofloxacin, ofloxacin, tobramycin, gentamycin.

MALIGNANT OTITIS EXTERNA
- "Necrotizing otitis externa" and "skull base osteomyelitis".
- Pseudomonas aeruginosa
- Occurs when infection spreads from the ear canal to the surrounding bones of the skull base.
- Occurs in diabetics, immunocompromised, and elderly.
- Rare in children.
- Management: immediately start empiric antibiotics (i.e. ciprofloxacin 500mg BID), urgent referral to ENT.
- Suspicious symptoms: granulation tissue on the ear canal floor at the bony cartilaginous junction, necrosis of skin, intense pain, otorrhea, systemic signs of toxicity, fever, possible tenderness at the temporal bone or facial nerve palsy.
- Investigations: CRP/ESP (should be ↑), CT/MRI skull osteomyelitis.
- Consider P. aeruginosa resistance to ciprofloxacin; patients infected with these require hospitalization for biopsy and debridement (to rule out cancer), and IV antibiotic treatment with an appropriate antipseudomonal beta-lactam agent (i.e. ceftazidime, cefepime, piperacillin-tazobactam).
- Rarely, serious/potentially fatal complications can include meningitis, brain abscess, and dural sinus thrombophlebitis.

CELLULITIS
- Cellulitis can develop post-trauma, ear piercings, or insect bites.
- Rapid onset and progression of pain, swelling, erythema, induration, tenderness.

HERPES ZOSTER OTICUS (RAMSAY HUNT SYNDROME)
- Reactivation of the varicella virus in a previously exposed patient, typically affecting the facial nerve.
- Ipsilateral facial paralysis, ear pain and vesicles on the face/ear/inside the ear canal.

FURUNCULOSIS
- Localized skin infection of the hair follicles.
- Typically S. aureus.
BAROTRAUMA

- Caused by pressure differences between middle ear and outside world distorting the tympanic membrane leading to pain and injury.
- Most common cause is **flying**, but can also be from **diving**, hyperbaric oxygen chambers, blast injuries, and decompression.
- Positive pressure can be produced via the Valsalva maneuver to attempt to correct the pressure differential, although the pressure difference usually self-resolves within a few hours.
- If the pressure differential is too great, the tympanic membrane can stretch, leading to bruising, bleeding, fluid exudate in the middle ear, and occasionally rupture.
- Management: **prevention**: medications (oral or nasal decongestants, antihistamines) or techniques such as frequent swallowing, yawning, or the Valsalva maneuver.
  - Chewing gum for adults or sucking on a bottle for babies may help.
  - Ear plugs designed to help with equalization of pressure while flying are available at the drug store, although they cannot be used for diving.
- If injury does occur, it will usually self-resolve with time. Appropriate analgesia is indicated. In extreme cases, surgical intervention is required.

MASTOIDITIS

- Complication of acute or chronic otitis media.
- Can lead to bony erosion, temporal lobe abscess, or septic thrombosis of lateral sinus.
- Presents with fever, ear pain, postauricular tenderness, localized erythema over the mastoid bone, and/or edema of the auricle.
- Imaging not always necessary (can be clinical dx), but possible imaging includes CT scan to confirm mastoiditis dx, and MRI if suspected additional intracranial pathology.
- Admit to hospital and commence IV antibiotics (coverage should include **S. aureus**, **Pseudomonas**, enteric gram negative rods, **S. pneumoniae**, **H. influenza**)
- If unresponsive to antibiotics, further intervention is warranted and may include mastoidectomy, myringotomy, and/or (if a cholesteatoma is present) tympanomastoidectomy

CHOLESTEATOMA

- Keratinized mass in the middle ear or mastoid.
- May occur as a primary lesion, or secondary to tympanic membrane perforation.
- Primary: usually occur as a result of prolonged Eustachian tube dysfunction.
- Accumulates keratinized squamous debris and grows in size, often eroding the scutum near the head of the malleus.
- Cholesteatomas may be caused iatrogenically if squamous epithelium is inserted into the middle ear space during surgery.
- May lead to erosion of the ossicles and hearing loss, and rarely lead to erosion into the inner ear.
- Patients may be asymptomatic or present with dizziness, hearing loss, +/- otorrhea.
- Clinical diagnosis, CT head if extracranial complications are suspected.
- Must be surgically removed, usually in conjunction with tympanoplasty +/- mastoidectomy if cholesteatoma extends beyond middle ear.

TEMPOROMANDIBULAR JOINT DISORDER (TMD)

- Most commonly caused by TMJ trauma, changes in dental occlusion/malocclusion, or possibly head and cervical postures.
- Presents with **pain** (unilateral facial pain that increases and decreases in intensity, radiating to ear, temporal and periorbital regions, angle of mandible, +/- posterior neck), ear discomfort/dysfunction, headache, +/- temporomandibular joint discomfort +/- dysfunction (restricted mandibular range of motion, popping/clicking noises on movement, clenching or grinding teeth, or deviation of the jaw to the affected side).
- **Treatment**:
  - Non-pharmacological: **patient education** and **self-care** to restore function and reduce pain (posture, sleeping positions).
  - Pharmacological: analgesia (trial **NSAIDs**, **muscle relaxants**): if additional pain management is required, **TCAs** (i.e. amitriptyline 10mg QHS titrating up to 50mg QHS, then down to lowest effective dose after 4 months).
- Refer to dentist if visible damage to teeth.
TRIGEMINAL NEURALGIA (TGN)

- Characterized by recurrent, sudden, (usually) brief episodes of acute, unilateral shock-like facial pain in the distribution of one (or more) of the branches of the CN V.
- Triggered by inoffensive stimuli.
- **Uncommon** overall can be **debilitating**; one of the more common neuralgias seen in older adults.
- Commonly caused by **compression of the nerve root** (possibly leading to demyelination), or an aberrant loop of artery or vein; many cases are **idiopathic** or secondary to other lesions.
- Clinical diagnosis; neuroimaging (MRI) if treatment unresponsive.
- **Diagnostic Criteria (ICHD-3)** (**Headache Classification Committee of the International Headache Society (IHS)**):
  A. At least three attacks of unilateral facial pain fulfilling criteria B and C.
  B. Occurring in one or more divisions of the trigeminal nerve, with no radiation beyond the trigeminal distribution.
  C. Pain has at least 3 of 4 characteristics:
     - Recurring in paroxysmal attacks lasting from a fraction of a second to two minutes.
     - Severe intensity.
     - Electric shock-like, shooting, stabbing, or sharp in quality.
     - At least three attacks precipitated by innocuous stimuli to the affected side of the face (some attacks may be, or appear to be, spontaneous).
  D. No clinically evident neurologic deficit.
  E. Not better accounted for by another ICHD-3 diagnosis.

- **Treatment:**
  - Pharmacologic: **carbamazepine**: start low and titrate up (start with 100-200mg BID, titrate up by 200mg increments, typical daily total maintenance dose 600-800mg.
  - Screening for **HLA-B*15:02 allele** should be performed prior to prescribing either drug in at-risk populations (i.e. Chinese ancestry); this genetic marker renders patients susceptible to developing Steven-Johnson's syndrome +/- TEN. If (+), carbamazepine C’ed (baclofen, lamotrigine as alternatives) (Dean).
  - **Surgical microvascular decompression** or ablative procedures are reserved for patients refractory to pharmacologic therapy.

When to refer

- Persistent or unexplained otalgia.
- Suspicion of malignancy.
- Refer to dental or maxillofacial specialist if odontogenic cause.
- Persistent discharge from canal.
- Incomplete resolution of otitis media or other infection, or no response to treatment.
- Persistent hearing loss.
- Frequent recurrences.
- Evidence of severe complications.
- Attic perforation/cholesteatoma.
REFERENCES


15. Epistaxis

KEY FEATURES

1. Through history and/or physical examination, assess the hemodynamic stability of patients with epistaxis.
2. While attending to active nose bleeds, recognize and manage excessive anxiety in the patient and accompanying family.
3. In a patient with an active or recent nosebleed, obtain a focused history to identify possible etiologies (e.g., recent trauma, recent upper respiratory infection, medications).
4. In a patient with an active or recent nosebleed,
   a) Look for and identify anterior bleeding sites,
   b) Stop the bleeding with appropriate methods.
5. In a patient with ongoing or recurrent bleeding in spite of treatment, consider a posterior bleeding site.
6. In a patient with a nosebleed, obtain lab work only for specific indications (e.g., unstable patient, suspicion of a bleeding diathesis, use of anticoagulation).
7. In a patient with a nosebleed, provide thorough aftercare instructions (e.g., how to stop a subsequent nose bleed, when to return, humidification, etc.).

Bleeding from the nose.

Causes & risk factors

In most cases, a specific cause is not identified.

- Nose picking (super common).
- Facial trauma.
- Foreign bodies (i.e. children).
- Sinus infections (decongestants).
- Old nasal fractures / septal deviation.
- Environmental:
  - Cold weather.
  - Low humidity.
  - High-flow unhumidified air/O₂.
- Iatrogenic:
  - NG/NJ tube insertion.
- Drugs:
  - Snorting cocaine.
  - Nasal steroids.
  - Nasal decongestants, antihistamines.
  - Anticoagulants (warfarin, NOACs).
- Coagulopathies: renal failure, vWD, hemophilia, liver failure/cirrhosis, etc.
- Hypertension is rarely a cause — bleeding profusely from the nose → anxiety → ↑ blood pressure.

Physiology

- Anterior bleeds: 90% (Kesselbach’s Plexus in Little’s area).
- Posterior bleeds: 10% — these will usually need help from ENT.

Management

Good effective first aid should stop 90% of nose bleeds.

In any bleeding patient, always start with the ABCs:

- Airway: is the patient speaking? Is their voice garbled? ("a noisy airway is an occluded airway") ☰.
- Vitals: BP, HR, SpO₂ (assess for volume loss/anemia).

HISTORY

- Duration, estimate volume (did they bring in soaked rags, clothes?).
- Has this occurred before? If so, how was it managed?.
- History of bleeding dyscrasias, previous transfusions, ask about risk factors.
- Past medical history.
- Current medications.
Chapter 15. Epistaxis

PHYSICAL EXAM

If bleeding is not severe, give basic first aid a try at stopping it before examining (physical exam is often difficult with ongoing bleeding anyway).

- Try to identify a site of bleeding — is it anterior or posterior? Bilateral bleeding without explanatory trauma is more likely to be posterior.
- Use a nasal speculum if available and if difficult to visualize without.
- Consider attempting anterior nasal cautery during examination (little evidence).

INVESTIGATIONS

Only generally needed if there is evidence of, or risk of, significant bleeding or symptoms of hypovolemia.

- CBC (Hct, Hgb).
- Blood group and hold.
- Coagulation studies including PT/INR and aPTT (if coagulopathy suspected).

BASIC FIRST AID

- Decrease anxiety: find a calm area of your office or ED,
- Have the patient lean forward, and
- Apply pressure by pinching the anterior aspect of both nares between the patient’s fingers (if the patient is unable or unwilling to do this, jerry-rigged tongue depressors work great as a nose clamp),
- for at least 15 minutes!

IF FIRST AID HAS FAILED...

- Chemical cautery with silver nitrate sticks can be attempted, although there is poor evidence that it works.
  - Only works in anterior bleeds.
  - Do not attempt to cauterize bleeding in both nares — you risk a septal perforation.
  - Suction first: silver nitrate works best when the area is moist but not flooded with blood.
  - Apply the sticks for ~5 seconds at a time, and look for a black eschar area to develop (this is usually readily evident).
- Electrocautery is effective, but leave this for the ENTs.
- Vasoconstrictive agents are highly effective!
  - Topical lidocaine, cocaine, or epinephrine sprays.
  - Cotton pledgets soaked in 1-2% lidocaine (for analgesia) mixed with 1:1000 epinephrine.
  - There is a risk of aspiration in those patients with ↓ LOC.
- Suction should be available and can be used to clear clots and aid visualization.
- If vasoconstrictors and cautery fails, anterior packing is required:
  - Vaseline-impregnated ribbon gauze, or
  - Cotton nasal tampons, or
  - Anterior epistaxis balloon.

POSTERIOR BLEEDS

- These are difficult to manage, and don't respond to normal first aid and treatment.
- Analgesia is generally required when packing the posterior nares.
- Double balloon catheters or Foley catheters can be effective (i.e. a 14/16F Foley catheter with a 30ml balloon).
- These patients need to be admitted with an ENT consult.
Follow up

- Discuss common precipitating factors with the patient: dry air, allergy (rhinitis), repeated trauma (nose picking), and coagulopathies (i.e. supertherapeutic INR) are the enemy!
- Advise patient to keep the area moist and avoid repeated trauma: avoid nose blowing/picking, consider applying vasoline topically BID, use a humidifier while sleeping, consider temporarily stopping the anticoagulant if supratherapeutic.
- Advise the patient that most will resolve with basic first aid (i.e. 15+ minutes of tamponade).
- Advise the patient to return if significant ongoing bleeding >1 hour, worrying associated symptoms (SOB, syncopal, new palpitations).
- Discontinue NSAIDs for 2-3 days.
- Consider *Staph* coverage (controversial! ☹️) if anterior packing, with packing removal in 48-36 hours.

**RECOMMENDED #FOAMED RESOURCES**


Short Sharp Scratch. ENT Basics: Epistaxis (Inserting a Nasal Tampon). [https://www.youtube.com/watch?v=eDRGz5QLwDM](https://www.youtube.com/watch?v=eDRGz5QLwDM)

**REFERENCES**


16. Fractures

KEY FEATURES

1. In a patient with multiple injuries, stabilize the patient (e.g., airway, breathing, and circulation, and life-threatening injuries) before dealing with any fractures.
2. When examining patients with a fracture, assess neurovascular status and examine the joint above and below the injury.
3. In patients with suspected fractures that are prone to have normal X-ray findings (e.g., scaphoid fractures in wrist injuries, elbow fracture, growth plate fracture in children, stress fractures), manage according to your clinical suspicion, even if X-ray equivocal.
4. In assessing elderly patients with an acute change in mobility (i.e., those who can no longer walk) and major, immediately life-threatening fractures (i.e., femur or pelvic fractures) should be dealt with during your primary survey: apply direct pressure to the fracture if open and bleeding, consider a pelvic binder and/or tourniquet, and grossly reduce and splint long bone fractures.

ORTHOPEDIC EMERGENCIES

After the primary survey, consider conditions for which you should obtain urgent orthopedic consultation:

- Evidence of vascular compromise.
- Open fractures.
- Potential or present neurological compromise (i.e. cauda equina syndrome, unstable C-spine fracture).
- Compartment syndrome:
  - The "5 P's" of Compartment syndrome: pain, pallor, paresthesia, paralysis, pulseless. The most specific sign is pain with passive stretch of compartment muscles.
- Hip dislocation.
- Osteomyelitis/septic arthritis.
- Unstable Pelvic fracture.

ORTHOPEDIC EMERGENCIES

VON CHOP — Vascular compromise, Open fracture, Neurological compromise/cauda equina syndrome, Compartment syndrome, Hip dislocation, Osteomyelitis/septic arthritis, Unstable Pelvic fracture

Fracture assessment

Note: These key features do not include technical and or psychomotor skills such as casting, reduction of dislocations, etc. Also see Procedural Skills.

Initial approach

Also see ACLS.

- In major trauma, follow ACLS first principles, starting with a primary survey: Airway, Breathing, Circulation.
- Of relevance to this topic, major, immediately life-threatening fractures (i.e. femur or pelvic fractures) should be dealt with during your primary survey: apply direct pressure to the fracture if open and bleeding, consider a pelvic binder and/or tourniquet, and grossly reduce and splint long bone fractures.
DOMESTIC/CHILD/ELDER ABUSE

Remember to consider the possibility of non-accidental injury (NAI) in all trauma/fracture presentations!

- **Age, gender, and mechanism of injury** can help clue you in to the type and severity of the fracture.
- A pathological or fragility fracture (i.e. osteoporotic hip fracture in the elderly) can happen with only minor trauma.
- Always **assess at least one joint above and below** the area of interest. Assess for:
  - Tenderness, bruising, and deformities / open wounds.
  - Range of motion.
  - Distal neurological and vascular function/compromise.

**INVESTIGATIONS**

- If at all possible, **administer analgesia prior to ordering investigations** — your radiology technician will thank you, the patient will thank you, and you’ll get better images.
- If there is a suspected or obvious open fracture and/or a contaminated wound, administer tetanus prophylaxis and consider antibiotics.
- "**Rule of twos**" for fracture imaging:
  - 2 sides (bilateral views for comparison).
  - 2 views (AP and lateral — some fractures will require more views).
  - 2 joints (imaging should include both the joint above and below).
  - 2 times (before/after reduction).
- Be wary of **common occult fractures (equivocal XR findings at initial presentation):**
  - **Scaphoid fractures** are commonly missed (or not visible) on initial XR after a fall on an outstretched hand (“FOOSH”). If you suspect a scaphoid fracture (convincing mechanism/hard fall, snuff box tenderness, or pain on axial loading of the thumb), then splint and re-XR in 7-10 days — a healing fracture line will likely be apparent.
  - Others: distal radius fractures, neck of femur fractures, radial head fractures, supracondylar fractures in children.
Chapter 16. Fractures

SPECIFIC CLINICAL DECISION RULES

Canadian C-Spine Rule
For alert (GCS=15) and stable trauma patients where cervical spine injury is a concern.

**Any high-risk factor which mandates radiography?**
- Age ≥65yo
- Dangerous mechanism*
- Parasthesias in extremities

No

**Any low-risk factor which allows safe assessment of range of motion?**
- Simple rear end MVC**
- Sitting position in ED
- Ambulatory at any time
- Delayed (non-immediate) onset of neck pain
- Absence of midline C-spine tenderness

Yes

**Rule Not Applicable If:**
- Non-trauma cases
- GCS <15
- Unstable vital signs
- Age <16yo
- Acute paralysis
- Known vertebral disease
- Previous C-spine surgery

Unable

No radiography

Able to actively rotate neck?
45 degrees left and right

Able

No radiography

Unable

Radiography

---

*Dangerous Mechanism:
- fall from elevation ≥3 feet / 5 stairs
- axial load to head, e.g. diving
- MVC high speed (>100km/hr), rollover, ejection - motorized recreational vehicles
- bicycle struck or collision

**Simple Rear end MVC excludes:**
- pushed into oncoming traffic
- hit by bus / large truck
- rollover
- hit by high speed vehicle

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Chapter 16. Fractures

Ottawa Knee Rules
Request imaging of the affected knee if:

1. Age >55yo
   OR
2. isolated tenderness of the patella
   (no bony tenderness of the knee other than the patella)
   OR
3. tenderness of the head of the fibula
   OR
4. inability to flex to 90°
   OR
5. Inability to bear weight both immediately and in the ED for 4 steps
   (unable to transfer weight twice on to each lower limb)

Ottawa Ankle Rules
Request imaging of the affected ankle (AP and lateral XR) if:

Pain in the malleolar region and ANY ONE of the following:
1. Inability to bear weight (e.g. unable to take four steps without assistance, regardless of limping) both within the first hour of injury and in the ED.
2. Bony tenderness over the posterior edge or tip of the distal 6 cm of the medial malleolus.
3. Bony tenderness over the posterior edge or tip of the distal 6 cm of the lateral malleolus.

Ottawa Foot Rules
Request imaging of the affected foot if:

Pain in the midfoot region and ANY ONE of the following:
1. Inability to bear weight both immediately and in the ED.
2. Bony tenderness over the base of the 5th metatarsal.
3. Bony tenderness over the navicular.

Management

- For open fractures: control hemorrhage (early attempts at reduction can be useful), provide adequate pain relief, and thoroughly irrigate and clean the wound.
- After adequate analgesia achieved, reduction should be attempted if warranted. Different fracture types and locations have differing criteria for adequate reduction/positioning. Reduction can be:
  - Closed: can be attempted with local nerve block, hematoma block (i.e. for distal radius fractures), or procedural sedation.
  - Open: generally attempted after a failed closed reduction, pathological fractures, or when there is evidence of improved outcomes over closed reduction.
- Immobilize the joint: either splint or cast the joint (ensuring the splint is long enough to adequately immobilize the joint).
- Consider post-reduction films (for fractures where reduction was necessary).
- Consider early re-imaging for those fractures prone to avascular necrosis (scaphoid, femoral head, talus).
- Ensure adequate followup — this will vary based on the nature and location of the fracture, and the age and reliability of the patient.
INDICATIONS FOR OPEN REDUCTION

NO CAST — Non-union, Open fracture, Neurovascular compromise, Intra-articular fracture, Salter-Harris 3,4,5, Polytrauma

REFERENCES


17. Gastro-intestinal Bleed

KEY FEATURES

1. In a patient with blood in the stools who is hemodynamically stable, use history to differentiate upper vs. lower gastrointestinal (GI) bleed as the investigation differs.
2. In a patient with suspected blood in the stool, explore other possible causes (e.g., beet ingestion, iron, Pepto-Bismol) before doing extensive investigation.
3. Look for patients at higher risk for GI bleed (e.g., previous GI bleed, intensive care unit admission, non-steroidal anti-inflammatory drugs, alcohol) so as to modify treatment to reduce risk of GI bleed (e.g. cytoprotection).
4. In a patient with obvious GI bleeding, identify patients who may require timely treatment even though they are not yet in shock.
5. In a stable patient with lower GI bleeding, look for serious causes (e.g., malignancy, inflammatory bowel disease, ulcer, varices) even when there is an apparent obvious cause for the bleeding (e.g., do not attribute a rectal bleed to hemorrhoids or to oral anticoagulation).
6. In a patient with an upper GI bleed,
   - Include variceal bleeding in your differential,
   - Use history and physical examination to assess the likelihood of a variceal bleed as its management differs.

Definition

- Upper gastro-intestinal bleeding (UGIB) occurs proximal to the ligament of Treitz (distal duodenum).
- Lower gastro-intestinal bleeding (LGIB) occurs distal to this ligament.

Upper gastro-intestinal bleed (UGIB)

Typically, hematemesis or coffee ground emesis.

RISK FACTORS

- **Peptic ulcer disease**, especially combined with any of: (Rodríguez-Hernández et al.)
  - Smoking.
  - <60yo.
  - History of previous UGIB.
  - Alcohol consumption.
- Medications:
  - Antiplatelet agents: ASA or NSAID use.
  - Vitamin K antagonists: warfarin.
  - Novel oral anticoagulants (NOACs): rivaroxaban, apixaban, dabigatran.
- Infection:
  - *H. pylori*.
- Comorbid disease:
  - Cirrhosis with varices (secondary to NASH, EtOH, etc.)

DIFFERENTIAL DIAGNOSIS

**UGIB MIMICS**

Epistaxis or hemoptysis can mimic a significant UGIB.

Esophagus

- Mallory-Weiss tear.
- Esophagitis.
- Variceal bleed.
- Esophageal cancer.
- Aortoenteric fistula (esp. in patients with known surgical history of AAA repair).
Stomach
- Gastritis: stress gastritis (post-surgical), alcohol.
- Gastric ulcer (mainly secondary to chronic NSAIDs).

Duodenum
- Duodenal ulcer (mainly secondary to H. pylori).

Iatrogenic
- Post-ERCP sphincterotomy bleed:
  - Proximal to ligament of treitz.
  - Hematemesis, melena or hematochezia in brisk UGIB.

Lower gastro-intestinal bleed (LGIB)
Includes hematochezia (without hematemesis), fecal occult blood, unexplained anemia, (rarely) malena.

RISK FACTORS
- Smoking (may contribute to ischemia and ischemic colitis).
- Family history of colon cancer or inflammatory bowel disease.
- Diverticulosis.
- Hx of radiation to abdomen or pelvis.
- Iatrogenic: post biopsy.
- Pediatric risks: cow’s milk allergy, Meckel’s diverticulum, IBD.

DIFFERENTIAL DIAGNOSIS
Small bowel
- Inflammatory bowel disease.
- Radiation enteritis.

Terminal ileum
- Crohn’s disease (may have "skip" lesions from "gum to bum")

Colon
- Neoplasms: Polyps or cancer.
- Infectious conditions: Crohn’s or ulcerative colitis (Continuous from colon).
- Infectious: bacterial colitis (i.e. Campylobacter jejuni, Clostridium difficile).
- Ischemia: known vasculopathy with occlusion of SMA or IMA leading to ischemic colitis.
- Radiation: Proctitis following radiation for prostate cancer.
- Vascular: Angiodysplasia.
- Structural: Diverticulosis (typically bleeds much more than ‘diverticulitis’).
- Benign: Hemorrhoids.

BENIGN CAUSES OF RECTAL BLEEDING
- 3 key questions to ask:
  - Are you on any medications? Look specifically for iron – can cause dark melena stools.
  - What have you eaten recently? Ask about beets – these discolor stools.
  - Have you taken any OTC medications? Any Pepto-Bismol, etc?
Chapter 17. Gastro-intestinal Bleed

IDENTIFYING HIGH-RISK PATIENTS
1. Have you had a GIB before (blood in stools, dark tarry stools, vomiting blood)?
2. Have you been admitted to hospital/ICU for GIB?
3. Have you taken recently/do you regularly take NSAIDs?
4. Ask: EtOH? Known liver disease? Do they see a gastroenterologist/hepatologist?
   • If high-risk, consider initiation of gastric cytoprotection: options include PPIs or H₂ antagonists.

VARICEAL BLEEDING
Main causes: chronic alcohol abuse, NASH, hepatitis B/C, hemochromatosis.

History
• Diet: fatty diet (Non-alcoholic steatohepatitis, NASH), Alcohol (alcoholic cirrhosis).
• IV Drug use, recent travel, high risk sexual practices (↑ risk of Hep B, Hep C).

Physical examination
• Skin: jaundice, telangiectasia, palmar erythema.
• Abdomen: Ascites (bulging flanks, fluid wave), Small firm liver, splenomegaly.
• Venous Engorgement: caput medusa, hemorrhoids, varices (not detected on history of physical exam but may be more likely if caput medusa or hemorrhoids present).

HEMODYNAMIC COMPROMISE
• Hemodynamic compromise can occur secondary to UGIB/LGIB! Look for:
  - Signs of shock.
  - Hematochezia due to brisk upper GI bleed, and/or
  - Massive hematemesis due to variceal bleeding.
  - Airway compromise due to ongoing bleeding.
• At-risk patients include those with:
  - Previous History of UGIB.
  - Known portal hypertension/cirrhosis/esophageal varices.
  - (Due to increased mortality) those with COPD and hypertension.
• Massive GI bleeds are managed differently, see First10EM: Management of the Massive GI Bleed for a good summary. Main points:
  - Wear PPE.
  - Do the basics (ABCs, baseline and frequent vitals (fast-cycling BP cuff), multiple large-bore IVs).
  - Activate your institution’s massive transfusion protocol: give blood as soon as possible (normal saline as an alternative).
  - Secure the airway: these patients aspirate!

Diverticulitis
• Episodic LLQ pain.
• Alternating diarrhea/constipation.
• Occasionally, painless PR bleeding.
• Outpatient management (may need hospital admission if treatment failure after 2-3d):
  - ↑ fiber intake.
  - Antibiotics: Ciprofloxacin 500mg BID x 7/7 + Metronidazole 500mg TID x 7/7.
  - No evidence for dietary restrictions (i.e. NPO, clear fluids only).
  - Reassess q2-3d.
  - Colonoscopy at 6wks to rule out colon cancer.
RECOMMENDED #FOAMED RESOURCES

First10EM: Management of the Massive GI Bleed 🌐

REFERENCES


Kargbo, J. *Upper GI Bleeding*. https://medskl.com/Module/Index/upper‐gi‐bleeding


Raman, M. *Lower GI Bleeding*. https://medskl.com/Module/Index/lower‐gi‐bleeding

18. Grief

**KEY FEATURES**

1. In patients who have undergone a loss, prepare them for the types of reactions (e.g., emotional, physical) that they may experience.
2. In all grieving patients, especially those with a prolonged or abnormal grief reaction, inquire about depression or suicidal ideation.
3. Recognize atypical grief reactions in the very young or the elderly (e.g., behavioral changes).
4. In patients with a presentation suggestive of a grief reaction without an obvious trigger, look for triggers that may be unique to the patient (e.g., death of a pet, loss of a job).

**Definition**

- A normal emotional response to a significant loss of loved one (including pet), change in function, community, or social position (i.e. job, retirement, end of relationship, sickness/disability, miscarriage).
- Can manifest emotionally, physically, behaviourally, cognitively, spiritually.
- Grief can precede the loss – anticipatory grief: i.e. anticipation of a loss (including one’s own death).
- Grief is the distress that occurs after bereavement (the situation of losing a loved one).

**Clinical presentation**

- Traditionally thought of as occurring in stages (i.e. the “five stages of grief”), but no good evidence.
- Symptoms vary as bereaved people adapt to the loss and acute grief is transformed and integrated.
- Symptoms can vary amongst individuals, as the ability to cope varies.
- Usually time-limited.
- Typically: feelings of shock, denial, anger, disbelief, yearning, anxiety, sadness, helplessness.
- Also: sleep disturbance, fatigue, social withdrawal, disruption of daily life / routine.

**STAGES OF GRIEF**

Denial, Anger, Bargaining, Depression, Acceptance.

**Complications and differential diagnosis**

- Most people eventually adapt / accept the loss, typically within 6 months.
- Always consider other diagnoses, including major depression, PTSD.
- Consider major depression if:
  - Suicidal ideation is present.
  - Thoughts or feelings of sadness are not focused on the loss.
  - Feelings of guilt, if present, come from a feeling of failure or worthlessness, rather than focused on the relationship with the deceased.
  - The patient cannot imagine being happy, even if the loss did not occur.
- 10-20% may develop complicated grief. Diagnosed if all of:
  - Ruminative preoccupation with troubling aspects of the circumstances or consequences of the death.
  - Excessive avoidance of reminders of the loss.
  - Excessive difficulty regulating emotions.
  - Grief symptoms persisting for at least six months after the death and interfering with functioning.
- Risk factors for poor bereavement outcomes:
  - Women > men.
  - Pre-existing mental illness.
  - Substance abuse.
  - History of childhood trauma.
- Recent, multiple prior losses.
- Insecure attachment/dependent relationship.
- Poor social support.
- Caregiver status.
- Sudden, violent or unexpected death lack of preparation.
- Death of a child.
• Complex grief associated with higher rates cancer, cardiovascular disease; mortality increases after death of loved one.

Persistent complex bereavement disorder

• Defined in the DSM-V.
• Synonymous with complicated grief.

DIAGNOSIS

• Brief grief questionnaire [Shear et al.]
• 1 or more of following symptoms persisting on more days than not for more than 1 year:
  - Yearning/longing for deceased.
  - Intense sorrow/emotional pain.
  - Preoccupation with deceased/circumstances of death.
• 6 or more of following symptoms persisting on more days than not:
  - Disbelief/numbness.
  - Difficulty accepting death.
  - Unable to positively reminisce.
  - Bitterness/anger.
  - Self-blame.
  - Excessive avoidance of reminders of the loss.
  - Desire to die to be with deceased.
  - Difficulty trusting others.
  - Feeling alone or detached.
  - Feeling life is meaningless.
  - Diminished self-identity.
  - Reluctance to pursue interests or plan for future.
• Symptoms have been present for at least six months. However, in some social, cultural, or religious settings, a longer duration (eg, 12 months) of symptoms may be required.
• Distress causes impairment of function/out of proportion to personal or cultural norms.

Management

• Empathy, support and watchful waiting.
• Management strategies.
  - Provide safe space to talk, encourage expression of emotions.
  - Help patients identify network of support among their own friends and family.
  - Listen actively.
  - Ask “how are you coping?” “what's going on in your mind?”
  - Encourage healthy, active lifestyle.
  - Confront avoided situations.
  - Repeat stories of death.
  - Address loss/restoration – talk about events leading to loss, then how to recover back to life, plan for the future.
  - Encourage mastering concrete tasks e.g. finances.
  - Develop new relationships/routine.
  - Join support group.
• Red flags: suicidal ideation, increased smoking/substance use.
• If requiring more support, have complicated grief diagnosis, concurrent mood disorders, or suicidality consider adding psychotherapy and/or pharmacotherapy.

  - Psychotherapies:
    • **Grief counselling**: individual or group, multidisciplinary with nurse, social worker, psychologists, chaplains.
    • **Cognitive behavioral therapy (CBT)**.
    • Interpersonal therapy (IPT).
  - Pharmacotherapy:
    • **Antidepressants**: no good evidence but paroxetine or bupropion suggested (Solomon and Shear).
  - Refer to psychiatry if lack of response to medication, if significant psychiatric comorbidities or unclear diagnosis.

REFERENCES


19. Headache

### KEY FEATURES

1. Given a patient with a new-onset headache, differentiate benign from serious pathology through history and physical examination.
2. Given a patient with worrisome headache suggestive of serious pathology (e.g., meningitis, tumour, temporal arteritis, subarachnoid bleed):
   - Do the appropriate work-up (e.g., biopsy, computed tomography [CT], lumbar puncture [LP], erythrocyte sedimentation rate).
   - Make the diagnosis.
   - Begin timely appropriate treatment (i.e., treat before a diagnosis of temporal arteritis or meningitis is confirmed).
3. Do not assume that relief of symptoms with treatment excludes serious pathology.
4. In a patient with a history of chronic and/or relapsing headache (e.g., tension, migraine, cluster, narcotic-induced, medication-induced), treat appropriately, and avoid narcotic, barbiturate dependence.
5. In a patient suffering from acute migraine headache:
   - Treat the episode.
   - Assess the ongoing treatment plan. (referral when necessary, take a stepwise approach).

### Diagnosing the benign headache

On first presentation with new onset headache, it is most important to differentiate benign or “primary” headaches from serious pathology.

### BENIGN FEATURES

- Age <30 years.
- Features typical of a primary headache syndrome (i.e. tension, cluster, migraine).
- History of similar headaches previously.
- A normal screening neurological exam.
- No abrupt change in a patient’s usual headache pattern.
- No high-risk comorbid conditions (e.g., immunocompromised: HIV, uncontrolled diabetes).

### RED FLAGS REQUIRING FURTHER WORKUP

**Emergent (address immediately)**

- Thunderclap (i.e. very sudden) onset of headache.
- Associated fever and/or meningism, or "toxic" appearance.
- Papilledema with focal signs.
- ↓ LOC.
- Acute glaucoma (sudden, severe unilateral eye pain, blurred vision and a red eye).

**Urgent (address within hours/days)**

- Papilledema (without focal signs or ↓ LOC).
- Elderly patient with new headache and cognitive changes, or recent history of falls.
- Rapid onset of headache with exertion (think: carotid artery dissection, ICH).
- Suspected giant cell (temporal) arteritis (however if diagnosis suspected, start high-dose steroids (i.e. prednisone 60-100mg daily) when first seen).
Chapter 19. Headache

Physical examination

Physical examination should be tailored to the acuity of the patient and the setting, but in general should include at least:

- Vital signs (especially temperature).
- Screening neurological examination, including:
  - Level of consciousness (i.e. GCS),
  - Cranial nerves (including visual acuity),
  - Signs of meningism (photophobia, nuchal rigidity),
  - Any tenderness over the temporal arteries, and
  - Papilledema.

Common types of benign headache

TENSION HEADACHE

- Headache without nausea, and ≥2 of:
  - Bilateral, banding pain,
  - Non-pulsating, and
  - Not worsened by activity.

- Acutely:
  - NSAIDs and acetaminophen: ibuprofen 400mg, naproxen 500mg, acetaminophen 1000mg.

- Prophylaxis (consider if frequent (>3/month) attacks or acute meds ineffective):
  - 1st line: TCAs: amitriptyline 10mg QHS (typical range: 10-100mg QHS), nortriptyline 10mg QHS (typical range: 10-100mg QHS).
  - 2nd line: SSRI/SNRIs: mirtazapine 30mg daily, venlafaxine 150mg daily.

MIGRAINE HEADACHE

- Headache with ≥2 of:
  - Nausea.
  - Photophobia.
  - Interference with activities.

- Migraine is historically underdiagnosed.

- Consider migraine diagnosis for recurring "sinus" headache.

- Women to men ratio of 2:1, most common in women of childbearing age.

- Acutely:
  - 1st line: NSAIDs and acetaminophen: ibuprofen 400mg PO, naproxen 500mg PO, acetaminophen 1000mg PO.
  - 2nd line: triptans: sumatriptan 100mg PO, rizatriptan 10mg PO, zolmitriptan 2.5mg PO, etc.
  - If vomiting: antiemetics and sumatriptan 6mg SC.
  - 3rd line: combination of naproxen and a triptan.
  - Adjuncts: antiemetics (metoclopramide 10mg IM/IV, ondansetron 8mg SL/IV, dexamethasone 8mg PO/IM/IV), high-flow oxygen (some evidence), IV fluid bolus (500-1000ml) with N/S (little evidence, but commonly used).

- Prophylaxis (consider if frequent, debilitating attacks):
  - 1st line: beta-blockers and TCAs: propranolol 20mg PO BID (typical range: 40-120mg BID), metoprolol 50mg PO BID (typical range: 50-100mg BID), amitriptyline 10mg PO QHS (typical range: 10-100mg QHS), nortriptyline 10mg PO QHS (typical range: 10-100mg QHS).
  - 2nd line: topiramate 25mg PO daily (target: 50mg BID), gabapentin 300mg PO daily (typical range: 400-600mg TID).
MEDICATION-INDUCED (OR MEDICATION REBOUND) HEADACHE

- Typically caused by frequent, long-term use of analgesics.
- Consider when patient is taking:
  - Ergots, triptans, combination analgesics, or opiates (esp. codeine) ≥10 days/month.
  - Acetaminophen or NSAIDs ≥15 days/month.

- Acutely:
  - Educate patient.
  - **Consider prophylactic medication** rather than frequent acute meds.
  - Provide an effective acute medication for severe attacks with limitations on frequency of use.
  - **Begin withdrawing acute analgesics** (opiates should be withdrawn gradually, others can be stopped abruptly if possible).

Uncommon types of benign headache

CLUSTER HEADACHE

- All of:
  - Frequent, severe headache.
  - Brief (<3 hours).
  - Unilateral (always the same side).
  - Ipsilateral eye redness, tearing, or restlessness during attacks.
- **Men to women ratio of 2.5:1, most common in men 20-50yo.**

- Acutely:
  - **Sumatriptan** 6mg SC.
  - Zolmitriptan 5mg IN (intranasal).
  - **High-flow O₂** (12+ L/min through NRB mask).

- Prophylaxis (required in most cases as attacks are debilitating):
  - 1st line: **verapamil** 240-480mg daily.
  - **Consider prednisone 50mg daily for 6 days when starting verapamil as it takes ~days to start working.**
  - 2nd line: **lithium** 900-1200mg daily.
  - **Consider topiramate, melatonin as 3rd line or adjuncts.**

- Patients presenting with new onset cluster headaches should be referred for **neurology review.**

HEMICRANIA CONTINUA

- All of:
  - Unilateral (always the same side).
  - Continuous.
  - Dramatically responsive to indomethacin.

- Management:
  - Specialist referral.

Meningitis

See **Meningitis.**
**Giant cell (temporal) arteritis**

- New onset headache.
- Visual disturbances (i.e. “amaurosis fugax”, very uncommon).
- Symptoms of polymyalgia rheumatica.
- Jaw claudication (“does your jaw feel painful or weak after meals?”).
- Unexplained fever or anemia.
- ↑ESR and/or ↑CRP.

Acutely:
- **Immediate initiation of corticosteroids** (i.e. prednisone 60-100mg daily).
- Specialist referral for biopsy.

**Space-occupying lesion (i.e. tumour, abscess, ICH)**

Patients with space-occupying lesions usually present with symptoms due to increased ICP and direct compression effects:

- Severe headache, worse in the morning and with: bending, coughing, sneezing, valsalva.
- ↓LOC.
- Anisocoria (unequal pupils), due to uncal herniation.
- Papilledema.

**INVESTIGATION AND TREATMENT**

Urgent CT for diagnosis and concurrent specialist consultation. If presentation is acute and ICH suspected, patient should be seen and managed emergently in ED.
Quick reference algorithm

Red flags present

Possible indicators of secondary headache
Unexplained focal signs, atypical headaches, unusual headache precipitants, unusual aura symptoms, onset >50y, aggravation with neck movement, jaw symptoms (consider TMJ disorder)

Yes

Refer and investigate

Yes

Migraine symptoms
Headache and ≥2 of: nausea, light sensitivity, interference with activities

Treat as migraine

No

Tension-type symptoms
Headache without nausea and ≥2 of: bilateral headache, nonpulsating pain, mild/moderate pain, not worsened by activity

Treat as tension H/A

No

Uncommon headache syndromes
Cluster headache, hemicrania continua, or new persistent daily headache.

Specialist referral

Adapted with permission from CFP, and Toward Optimized Practice: “Guideline for primary care management of headache in adults”

Headache after trauma

See Loss of consciousness - minor head trauma.

RECOMMENDED #FOAMED RESOURCES

REFERENCES
### 20. Hyperlipidemia

**KEY FEATURES**

1. Screen appropriate patients for hyperlipidemia.
2. In all patients whose cardiovascular risk is being evaluated, include the assessment of lipid status.
3. When hyperlipidemia is present, take an appropriate history, and examine and test the patient for modifiable causes (e.g., alcohol abuse, thyroid disease).
4. Ensure that patients diagnosed with hyperlipidemia receive appropriate lifestyle and dietary advice. Periodically reassess compliance with this advice (especially in patients at overall low or moderate CV risk).
5. In treating hyperlipidemic patients, establish target lipid levels based on overall CV risk.

Everything you need to know about lipids in primary care is covered in the excellent Dr. Michael Allan article in the CFP journal (he also does the Best Science Medicine Podcast). For completeness’ sake, we ’summarize the summary’ below.

### Screening

- For primary prevention (i.e., patients without CVD or CVD risk equivalents), screen with lipid testing and risk estimation (using a calculator) men ≥40yo and women ≥50yo.
  - Consider testing earlier in patients with traditional CVD risk factors: hypertension, family history of premature CVD, diabetes, and smoking.
- **Repeat screening no more than every 5 years** in those not on lipid-lowering therapy.
- Patients **do not need to fast for lipid testing**. Nonfasting levels are adequate for risk estimation.

**REMEMBER**

**Primary prevention** — patients with no signs or symptoms of the disease.
**Secondary prevention** — patients with signs of the disease or a disease event (i.e. MI); you’re attempting to prevent a recurrence of the event or worsening of the disease state.

### Calculating risk

- After measuring lipids, use a validated risk calculator (i.e. Framingham) to assess risk.
  - Diabetics: as above.
  - Patients with CKD: use a risk calculator that includes CKD in its estimation (i.e. QRISK2).
  - If risk calculation is desired for patients taking lipid therapy, pretreatment lipid levels should be used and risk should be adjusted for known benefits of statin or ASA therapy.
- No need to calculate risk in patients:
  - <40yo (without additional risk factors) and those >75yo: as risk equations are not validated for patients in these age ranges.
  - Patients taking lipid therapy, as calculators are not designed to adjust for changes with lipid therapy.
  - With pre-existing CVD, as they are automatically at high risk. CVD risk equivalents include:
    - Coronary heart disease (CHD) (i.e. MI, angina, CHF, wall motion abnormalities on echo).
    - Cerebrovascular disease (i.e. stroke/TIA).
    - Peripheral artery disease (i.e. intermittent claudication, arterial ulcers).
    - AAA.
    - (Wilson) and the Canadian Diabetes Association also consider diabetics with end-organ damage (i.e. CKD) to be CVD risk equivalent.

**REMEMBER**

As physicians, we shouldn’t care about particular lipid levels, as they don’t correlate well with risk — what we should care about is what a validated risk calculator says about a patient’s particular risk of disease (a.k.a. MI, AAA, stroke), and then weighing likely treatment benefits against their risk of side effects. Treat the patient, not the numbers!
Chapter 20. Hyperlipidemia

Management

- **Lifestyle interventions! Smoking cessation, Mediterranean diet, and exercise for all patients.**
  - NNT over 2 years of intervention for high-risk patients: smoking cessation to prevent death = 11, physical activity (150min/week) to prevent any CVD event = 6, Mediterranean diet to prevent any CVD event = 12.
  - Although less common, also consider other modifiable causes of hyperlipidemia, including diabetes mellitus, hypothyroidism, alcoholism, nephrotic syndrome, hemochromatosis, etc.

- **Primary prevention:**
  - If <10% 10yr CVD risk, retest in 5yrs (no treatment).
  - If 10-19% 10yr CVD risk: discuss risks/benefits of moderate-intensity statins.
  - If >20% 10yr CVD risk: discuss risks/benefits of high-intensity statins.
  - Patients >75yr: don't test lipids, and don't start statins, however:
    - Patients who are a “really good” 75yos can be offered statins.
    - Patients already taking and tolerating a statin should not have their statin stopped or reduced just because they have aged beyond 75yo.

- **Secondary prevention:**
  - All on high-intensity statins if tolerated.
  - If >75yo, consider moderate-intensity statins.
  - If frail elderly and those with renal impairment: consider lower-intensity.

- **Statin regimens include:**
  - Moderate-intensity: lovastatin 40mg, pravastatin 40mg, simvastatin 40mg, atorvastatin 10-20mg, rosuvastatin 5-10mg.
  - High-intensity: atorvastatin 40-80mg, rosuvastatin 20-40mg.
  - Avoid pravastatin in patients ≥65yo (uncertainty around cancer risk).

- If intolerant of statins, encourage patients to keep trying, unless they’ve had a serious reaction. Try alternate statins or a lower dose, and/or a drug holiday.
  - Any statin intensity is preferred to non-statin lipid-lowering therapy.
  - Alternate daily dosing can be considered if a patient does not tolerate daily dosing.

- In primary prevention, non-statin lipid-lowering drugs should not be used as first-line monotherapy or in combination with statins.
  - Statins have a 25% relative risk reduction (RRR) in CVD, and 14% RRR on mortality.

- In secondary prevention, ezetimibe can be considered in discussion with patients as add-on therapy to statins, but owing to the higher relative benefit of statins, statin therapy should be maximized first (to high intensity).
  - Ezetimibe has a 6% RRR on CVD, but no benefit on mortality. Other medications have no benefit for either CVD or mortality.

**PRIMARY PREVENTION WITH ASA**

- **We discourage the use of ASA** for patients without previous CVD and an estimated 10yr CVD risk <20%.
  - ASA can be considered in primary prevention if the 10yr CVD risk is ≥20% and bleeding risk is low.
  - Use of ASA for primary CVD prevention should be considered only after statin therapy has been discussed and introduced.

- Patients offered ASA should be informed of the potential benefits and harms of ASA use.

**Followup**

- The use of cholesterol targets for reducing CVD is not required.
- Routine monitoring of repeat lipid levels after a patient begins lipid-lowering therapy is not required.
- Adherence to statins can be improved with patient reinforcement (this may include lipid testing for patient motivational purposes).
- Baseline CK or ALT levels is generally unnecessary. The evidence against testing baseline ALT or CK levels is poor and some clinicians might prefer to test one or both.
- No routine monitoring of CK and ALT levels (consider in symptomatic patients or those at higher risk of adverse reactions).
Chapter 20. Hyperlipidemia

RECOMMENDED #FOAMED RESOURCES

REFERENCES
Mohiuddin, S. M. Intensive Smoking Cessation Intervention Reduces Mortality in High‐Risk Smokers With Cardiovascular Disease. CHEST Journal, 131(2), 446. doi:10.1378/chest.06‐1587
21. Hypertension

**KEY FEATURES**

1. Screen for hypertension.
2. Use correct technique and equipment to measure blood pressure.
3. Make the diagnosis of hypertension only after multiple BP readings (i.e., at different times and during different visits).
4. In patients with an established diagnosis of hypertension, assess and re-evaluate periodically the overall cardiovascular risk and end-organ complications:
   - Take an appropriate history.
   - Do the appropriate physical examination.
   - Arrange appropriate laboratory investigations.
5. In appropriate patients with hypertension (e.g., young patients requiring multiple medications, patients with an abdominal bruit, patients with hypokalemia in the absence of diuretics):
   - Suspect secondary hypertension.
   - Investigate appropriately.
6. Suggest individualized lifestyle modifications to patients with hypertension. (e.g., weight loss, exercise, limit alcohol consumption, dietary changes).
7. In a patient diagnosed with hypertension, treat the hypertension with appropriate pharmacologic therapy (e.g., consider the patient's age, concomitant disorders, other cardiovascular risk factors).
8. Given a patient with the signs and symptoms of hypertensive urgency or crisis, make the diagnosis and treat promptly.
9. In all patients diagnosed with hypertension, assess response to treatment, medication compliance, and side effects at follow-up visits.

The 2015 Canadian Hypertension Education Program (CHEP) has everything you need to know for the exam and for practice; the guidelines published alongside the paper are a fairly short and recommended read, or even shorter, their one-page summary.

**New in the 2015 CHEP recommendations**

- A strong recommendation to use electronic (automated) BP measurement in the office setting instead of auscultation.
- If a patient has elevated BP in the office setting, they should have some sort of out-of-office BP measurement (either ambulatory BP monitoring, or a series of home BP measurements) to rule out white-coat hypertension.
- Out-of-office assessments should preferably be a 24-hour ambulatory BP.
- Patients with normal out-of-office readings can be diagnosed with white-coat hypertension, and should not be started on medication, with annual BP checks thereafter.

**Definitions**

- **Isolated systolic hypertension:** systolic BP >140 with diastolic <90.
- **Resistant hypertension:** BP above target despite concurrent use of three antihypertensive agents of different classes, or BP controlled with four or more medications.
- **Refractory hypertension:** uncontrolled BP despite specialist consultation and maximal medical therapy (four or more drugs with complementary mechanisms).
- **Hypertensive urgency (a.k.a. severe asymptomatic hypertension):** BP ≥180/120 without signs of target end-organ damage or acute symptoms attributable to their hypertension. **High blood pressure should be treated gradually in this group** — the weight of evidence does not indicate that acutely lowering severe asymptomatic hypertension (especially in the Emergency Department) is beneficial.
- **Hypertensive emergency:** BP ≥180/120 with signs of target end-organ damage.
- **Secondary hypertension:** broadly, renovascular and endocrine causes. Think about these causes (and investigate) if patient is not responding to typical treatment for essential hypertension, or falls outside of typical age or demographic at diagnosis.
- **Preeclampsia:** see Pregnancy — Preeclampsia.

**Screening**

For patients >18yrs: screen for hypertension with BP measurement at all appropriate primary care visits (Lindsay et al.)
Diagnosis

Elevated BP reading(s): office/home/pharmacy

Visit 1
BP ≥ 180/110
Hypertension

AOBP ≥ 135/85
OBPM ≥ 140/90

No

No Hypertension

Out of Office Assessment:
- ABPM (preferred)
- HBPM diagnostic series

Visit 2 (within 1 month)
Daytime ABPM or HBPM ≥ 135/85
24-hour ABPM ≥ 130/80

White Coat Hypertension
If the average HBPM < 135/85, consider repeating ABPM or HBPM series to confirm.

Yes

Alternate Method
(if ABPM/HBPM not available)

Visit 2 (within 1 month)
OBPM ≥ 140/90

Yes

No

Visit 3
OBPM ≥ 160/100

No

Visits 4 & 5
OBPM ≥ 140/90

Yes

Hypertension

AOBP = automated office blood pressure
OBPM = office blood pressure measurement
ABPM = ambulatory blood pressure measurement
HBPM = home blood pressure measurement

Management

- **Lifestyle interventions** are effective in hypertension!
  - **Physical exercise**: 30-60 minutes of moderate intensity exercise 4-7 days/week.
  - **Weight reduction**: to maintain or obtain a normal BMI (18.5-24.9) and waist circumference (<102cm for men, <88cm for women).
  - **Limit EtOH**: <2 drinks/day; <14/week for men, <9/week for women.
  - **DASH diet**.
  - **Limit sodium intake**: <2000mg/day (5g of salt).
  - **Stress management techniques**: if contributing to hypertension.
  - **Smoking cessation**.

- **Treat to target** in all patients:
  - <140/90 in most patients (including CKD).
  - <130/80 for diabetics (or maybe 140-150 systolic is a better target? (Brunström and Carlberg)).
  - <150 systolic in the very elderly (be careful if initiating therapy in the elderly with low diastolic pressures (<60 mmHg)).

- **Focus on adherence** (and barriers to adherence) with patients to pharmacotherapy and lifestyle changes — non-adherence is the most common cause of uncontrolled hypertension.
# Hypertension

## HYPERTENSION TARGETS

- **<140/90** for most, **<130/80** for diabetics, less aggressive in the frail/elderly.

### First-line Agents

<table>
<thead>
<tr>
<th>CI</th>
<th>SIDE EFFECTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiazide and thiazide-like diuretics</td>
<td>Rash, ↓Na+/K+, ↑Ca2+/uric acid/glucose/cholesterol</td>
</tr>
<tr>
<td>ACE-i/ARB only first-line if non-black</td>
<td>Cough (common with ACE-I, switch to ARB if occurs), hyperkalemia</td>
</tr>
<tr>
<td>Beta blockers only first-line if &lt;60yrs</td>
<td>Fatigue, insomnia, impotence, can mask hypoglycemia</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>Pedal edema, dizziness, H/A</td>
</tr>
</tbody>
</table>

### ANTIHYPERTENSIVE AGENTS

“ABCD” — ACE inhibitors / angiotensin receptor blockers, Beta-blockers, Calcium channel blockers, Diuretics (thiazide/thiazide-like).

- For most patients, start monotherapy preferably with a **thiazide** or thiazide-like diuretic.
- For patients with **cardiovascular or kidney disease (including diabetics)**, initial monotherapy should be with an **ACE-i or ARB**.
  - **Think**: does the patient have evidence of atherosclerosis? proteinuria? diabetic? Start an angiotensin med!
  - **Check** for (and avoid) hypokalemia after initiating a thiazide.
- If the patient is intolerant, trial another medication from the same class.
- If not at target, **add another first-line agent**.
  - Grade B evidence for thiazide + CCB.
  - Grade C evidence for CCB + ACE-i.
  - Grade D for all other combinations.
- If after two agents the patient is not at target, consider **reasons for poor response**:
  - Poor adherence, associated conditions (i.e. obesity, EtOH), drug interactions (i.e. NSAIDs, steroids), suboptimal treatment doses, volume overload, secondary hypertension. See more at Supplemental table S10.
- **Resistant hypertension responds best to the addition of spironolactone as a 4th agent**. (Williams et al.)
- Different from younger patients, in **elderly patients**, coronary heart disease risk varies directly with the systolic and pulse pressures and **inversely with the diastolic pressure** (i.e. lower diastolic pressures are associated with increased risk).

### INVESTIGATIONS

- After diagnosis, all patients with hypertension should have (Grades C and D evidence):
  - U/A.
  - K+, Na+, Cr.
  - Fasting sugar and/or A1c.
  - Lipids (including CV risk assessment (i.e. Framingham)).
  - ECG.
  - In diabetics: protein/creatinine ratio (to assess for microalbuminuria).

CHEP recommends repeating tests “with a frequency reflecting the clinical situation”.

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100
RECOMMENDED #FOAMED RESOURCES


REFERENCES

Brunström, M., & Carlberg, B. Effect of antihypertensive treatment at different blood pressure levels in patients with diabetes mellitus: systematic review and meta-analyses. BMJ, 352, i717. doi:10.1136/bmj.i717


22. Immigrants

KEY FEATURES

1. As part of the periodic health assessment of newly arrived immigrants:
   - Assess vaccination status (as it may not be up to date).
   - Provide the necessary vaccinations to update their status.

2. As part of the ongoing care of immigrants, modify your approach (when possible) as required by their cultural context (e.g., history given only by husband, may refuse examination by a male physician, language barriers).

3. When dealing with a language barrier, make an effort to obtain the history with the help of a medical interpreter and recognize the limitations of all interpreters (e.g., different agendas, lack of medical knowledge, something to hide).

4. As part of the ongoing care of all immigrants (particularly those who appear not to be coping):
   - Screen for depression (i.e., because they are at higher risk and frequently isolated).
   - Inquire about a past history of abuse or torture.
   - Assess patients for availability of resources for support (e.g., family, community organizations).

5. In immigrants presenting with a new or ongoing medical condition, consider in the differential diagnosis infectious diseases acquired before immigration (e.g., malaria, parasitic disease, tuberculosis).

6. As part of the ongoing care of all immigrants, inquire about the use of alternative healers, practices, and/or medications (e.g., “natural” or herbal medicines, spiritual healers, medications from different countries, moxibustion).

Culturally safe practice in healthcare

In particular, [culturally safe practice] is used to express an approach to healthcare that recognizes the contemporary conditions of Aboriginal people which result from their post-contact history. Although the principles of culturally safe practice in Canada and elsewhere have been developed in the context of Aboriginal peoples, much similarity exists for immigrants and refugees.

The 5 P principles necessary for cultural safety (Brascoupé and Catherine Waters BA):

- Protocols – respect for cultural forms of engagement.
- Personal knowledge – understanding one’s own cultural identity and sharing information about oneself to create a sense of equity and trust.
- Process – engaging in mutual learning, checking on cultural safety of the service recipient.
- Positive purpose – ensuring the process yields the right outcome for the service recipient according to that recipient’s values, preferences and lifestyle.
- Partnerships – promoting collaborative practice.

Newly arrived immigrants/refugees

There are some excellent third-party resources available to help you work through the first few visits in your clinic with newly arrived immigrants, especially from disparate cultures. Be mindful that you may need to arrange an interpreter (either in-person or via telephone) prior to the first visit.

- Strongly recommended: CCIRH’s Evidence-Based Preventative Care Checklists
- CFPC’s Refugee Health Care: Resources to Assist Family Physicians
- CAMH (Centre for Addiction and Mental Health) Refugee Mental Health Project

IMMIGRATION MEDICAL EXAMINATION

All new permanent residents or refugees, as part of their visa application process, are required to undergo an immigration medical examination, which includes:

- CXR (screen for TB).
- Syphilis serology.
- Urinalysis.
- HIV testing.
- Complete physical (inc. vision and hearing).
THE FIRST CLINIC VISIT

Region-specific information from the CCIRH’s Evidence-Based Preventative Care Checklists. In general:

- Remain alert to (but do not specifically screen for) PTSD.
- Remain alert to child neglect and intimate partner violence.
- Screen for depression only if linked to an integrated treatment program.
- Screen for contraceptive needs / emergency contraception.
- Conduct age-appropriate screening and testing as you would for other Canadians.
- Refer for (or perform yourself) age-appropriate vaccinations as you would for other Canadians, including “catch-up” vaccinations as necessary.
- Advise or arrange for a first dental visit.
- Test: hepatitis B, varicella and vaccinate if appropriate or refer if hepatitis B positive.
- Test: CBC (females/children).
- Test: for region-specific disease (i.e. malaria) only if signs or symptoms.

Poverty and social determinants of health

- Strongly recommended: Ontario College of Family Physicians — Primary Care Interventions into Poverty.

RECOMMENDED #FOAMED RESOURCES

CCIRH. Evidence-Based Preventive Care Checklist for New Immigrants and Refugees. http://www.ccirhken.ca/ccirh/checklist_website/

Ontario College of Family Physicians. Primary Care Interventions into Poverty. http://ocfp.on.ca/cpd/povertytool


REFERENCES


23. Infertility

**KEY FEATURES**

1. When a patient consults you with concerns about difficulties becoming pregnant:
   - Take an appropriate history (e.g., ask how long they have been trying, assess menstrual history, determine coital frequency and timing) before providing reassurance or investigating further.
   - Ensure follow-up at an appropriate time (e.g., after one to two years of trying; in general, do not investigate infertility too early).

2. In patients with fertility concerns, provide advice that accurately describes the likelihood of fertility.

3. With older couples who have fertility concerns, refer earlier for investigation and treatment, as their likelihood of infertility is higher.

4. When choosing to investigate primary or secondary infertility, ensure that both partners are assessed.

5. In couples who are likely infertile, discuss adoption when the time is right. (Remember that adoption often takes a long time.)

6. In evaluating female patients with fertility concerns and menstrual abnormalities, look for specific signs and symptoms of certain conditions (e.g., polycystic ovarian syndrome, hyperprolactinemia, thyroid disease) to direct further investigations (e.g., prolactin, thyroid-stimulating hormone, and luteal phase progesterone testing).

**Definition**

- No conception after 12 months of unprotected and frequent intercourse.
  - Primary — no previous pregnancy.
  - Secondary — after previous pregnancy.

**Risk factors**

- Female:
  - Age ≥ 35.
  - Oligo/amenorrhea.
  - History of STI/PID.
  - Previous cancer treatment.
  - Previous abdo/pelvic surgery.
  - Extremes of body weight.
  - Family history of early menopause (<45yo).
  - Smoking.

- Male:
  - Age ≥ 40.
  - History of cryptorchidism.
  - Previous cancer treatment.
  - Previous genitourinary tract surgery.
  - Smoking.

**History**

Ask about:

1. Ovulatory dysfunction (pre-menstrual/menstrual symptoms, regularity of menses).
2. Risk factors for tubal infertility (STIs/PID, previous ectopic, abdominal/pelvic surgery).
3. Sexual factors (timing/frequency of intercourse, lubricants).
4. Male (sperm) factors (children/pregnancy with previous partner, genitourinary issues).
Preconception counselling

See Pregnancy — Preconception counselling.

- If >40yo: screen for concurrent conditions (i.e. hypertension, diabetes, ultrasound for PCOS).
- Ovulation tracking (ovulation generally occurs 14 days before first day of period, i.e. for 30 day cycle, ovulation occurs on day 30 - 14 = 16). Can also use ovulation predictor kits, changes in basal body temperature, or changes in cervical mucus.
- Chances are dramatically higher if intercourse occurs in the 3 days preceding ovulation. Optimal timing for intercourse is 5 days before until 2 days after the predicted day of ovulation.
- If no particular effort to time intercourse around ovulation, then regular frequency of intercourse every 2 or 3 days (i.e. 2-3 times/week) should be encouraged.
- Weight loss if obese (BMI > 30).
- Avoid alcohol and drugs (particularly marijuana: blocks action of GnRH).
- Avoid NSAIDs: can block oocyte release.
- Avoid lubricants (can slow sperm).

Investigation

- 1st line:
  - Ovulation:
    - Hormone profile (age >35, or if irregular cycles): day 3 FSH, LH, estradiol, TSH, prolactin.
    - If regular cycles: day 21 (mid-luteal) progesterone (i.e. 7 days before expected period).
  - Anatomy:
    - Pelvic examination.
    - Pelvic U/S: if abnormal or unreliable exam.
  - General:
    - Pap and cervical cultures (STI).
    - Rubella and varicella serology (vaccinate if not immune).
    - Genetic testing (if indicated based on history or exam).
  - Semen analysis (repeat if abnormal).
- 2nd line:
  - Referral!
  - Female:
    - Karyotype.
    - Pelvic ultrasound (rule out structural abnormalities, if not already done).
    - Hysterosalpingogram (HSG) to assess tubal patency +/- hysteroscopy (visualize uterine cavity).
  - Male:
    - Karyotype.
    - Free testosterone.

When to refer

- Female age <35yo: after 1 year of trying.
- >35yo: 6-12 months of trying.
- >40yo: refer after 6 months of trying.
- Also refer if:
  - Female: irregular menses, risk factors for tubal disease (STIs, PID, previous ectopic, abdominal surgery), or history of chemotherapy/radiation.
  - Male: history of cryptorchidism, pelvic/groin surgery, or previous chemotherapy/radiation.
  - Investigations: day 3 FSH > 10, pelvic pathology on examination or U/S, or abnormal semen analysis x 2.
3rd party reproduction

- Use of eggs, sperm, or uterus outside of the couple trying to conceive: egg donation, sperm donation, gestational carriers (non-biological mother), surrogacy (biological mother).
- Payment for any of sperm, eggs, or gestational carrier is illegal.
- Sex selection is also illegal in Canada.

RECOMMENDED #FOAMED RESOURCES
Fertility for Family Physicians — Kimberly Liu, Mount Sinai Centre for Fertility & Reproductive Health

REFERENCES
24. Low-back Pain

**KEY FEATURES**

1. In a patient with undefined acute low-back pain (LBP):
   - Rule out serious causes (e.g., cauda equina syndrome, pyelonephritis, ruptured abdominal aortic aneurysm, cancer) through appropriate history and physical examination.
   - Make a positive diagnosis of musculoskeletal pain (not a diagnosis of exclusion) through an appropriate history and physical examination.

2. In a patient with confirmed mechanical low back pain:
   - Do not over-investigate in the acute phase.
   - Advise the patient: that symptoms can evolve, and ensure adequate follow-up care.
   - that the prognosis is positive (i.e., the overwhelming majority of cases will get better).

3. In a patient with mechanical low back pain, whether it is acute or chronic, give appropriate analgesia and titrate it to the patient’s pain.

4. Advise the patient with mechanical low back pain to return if new or progressive neurologic symptoms develop.

5. In all patients with mechanical low back pain, discuss exercises and posture strategies to prevent recurrences.

**Classification**

- **Acute:** within 12 weeks of pain onset.
- **Chronic:** more than 12 weeks since pain onset.

**Etiology**

- **98% are due to "mechanical" causes:**
  - Pain is typically exacerbated by movement, relieved by rest.
  - Differential to consider includes: sprain/strain injury, disc herniation or degenerative disc disease, facet joint degeneration, spinal stenosis, spondylolysis, spondylolisthesis, pregnancy.

- **2% are due to "non-mechanical" causes:**
  - Pain is worse at rest and does not change with position.
  - Non-mechanical causes are more concerning: differential includes: cauda equina syndrome, abdominal aortic aneurysm (AAA), ankylosing spondylitis, malignancy, tuberculosis, osteomyelitis, spinal abscess, spinal stenosis.
  - Note that pain can be referred (e.g. perforated ulcer, pyelonephritis, ectopic pregnancy), or related to a psychiatric illness.

**Initial assessment**

- Assessment in primary care should include a comprehensive history and physical examination (including neurological examination).
- Evaluate for the presence of red and/or yellow flags.

**SPECIFIC HISTORY FOR BACK PAIN**

- Is the pain worse with flexion or extension?
- Is the pain above or below the gluteal fold?
  - If the patient poorly differentiates, ask: "if you could pick only one point where the pain is the worst, where would that be?”, or “if we could remove the pain in only one spot, what spot would you rather get rid of?”
- Is the pain constant or intermittent?
  - “Is there ever a time when you are in your best position or at the best time of your day when your pain stops, and I know it comes right back, but is there even just a moment?”
- What movements or positions make it worse?
- What movements or positions make it better?
Chapter 24. Low-back Pain

- What has changed since the patient was last seen for the back pain (if ever)?
- What treatment have been tried? Have they worked?
- Impact: what can the patient not do now that they could before?
- New concerns: does the patient have any new symptoms or concerns since last seen (for example, new or worsening numbness or tingling in your legs, difficulty walking, or fever, or problems with your bowels or bladder)?

EXAMINING FOR LOW BACK PAIN

- Gait and posture.
- Range of motion of upper torso: pain worse with flexion or extension?
- Palpation of lumbar spine: point tenderness can be concerning for fracture or infection.
- Palpation of the paraspinous regions: may be able to localize spasm.
- Straight leg raise (SLR) test: testing for disc herniation.
- Reflexes and motor: can help localize nerve root dysfunction.
  - Limited neurological examination should include (at least): ankle and great toe dorsiflexion, ankle reflexes, SLR and light touch over the foot.

CONCERNING FEATURES OF LBP

| BACK PAIN | Bowel or bladder dysfunction, Anaesthesia (saddle), Constitutional Sx / malignancy, K - Chronic disease, Parasthesias, Age >50 (and first episode of pain), IV drug use (infection) OR alcohol, Neurological deficits |

<table>
<thead>
<tr>
<th>Red Flags</th>
<th>Yellow Flags</th>
</tr>
</thead>
<tbody>
<tr>
<td>Features of Cauda Equina Syndrome (loss of sphincter tone / fecal incontinence, acute urinary retention / overflow incontinence, saddle anesthesia, leg weakness) - emergent.</td>
<td>Belief that pain and activity are harmful.</td>
</tr>
<tr>
<td>Severe worsening pain, especially at night or when lying down - urgent.</td>
<td>“Sickness behaviours”: (i.e. extended periods of rest), lack of support, overprotective families.</td>
</tr>
<tr>
<td>Significant trauma - urgent.</td>
<td>Low or negative mood, social withdrawal.</td>
</tr>
<tr>
<td>Fever, unintended weight loss, or personal history of cancer - urgent.</td>
<td>Treatment expectations that do not fit with best practice.</td>
</tr>
<tr>
<td>Use of steroids or intravenous drugs - urgent.</td>
<td>Problems with claim and compensation.</td>
</tr>
<tr>
<td>Patient with first episode over 50 years old, especially over 65 - soon.</td>
<td>History of back pain, time-off, other claims.</td>
</tr>
<tr>
<td>Widespread neurological signs - soon.</td>
<td>Heavy work, unsociable hours (i.e. shift work).</td>
</tr>
</tbody>
</table>

- emergent: immediate referral (within hours).
- urgent: referral within 24-48 hours.
- soon: referral within weeks.
## PATTERNS OF BACK PAIN

<table>
<thead>
<tr>
<th>History</th>
<th>Pattern 1:</th>
<th>Pattern 2:</th>
<th>Pattern 3:</th>
<th>Pattern 4:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>* Pain worse with sitting or bending forward, may be eased with extension</td>
<td>* Pain is worse with extension</td>
<td>* Pain changes with position</td>
<td>* Pain is worse with activity (often walking)</td>
</tr>
<tr>
<td></td>
<td>* Can be constant or intermittent</td>
<td>* Never worse with flexion (may improve with flexion)</td>
<td>* Pain is constant</td>
<td>* Improves with rest and posture change (usually flexion)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>* Intermittent or short in duration</td>
</tr>
</tbody>
</table>

### Back dominant pain
(pain is greatest above the gluteal fold)

### Leg dominant pain
(pain is greatest below the gluteal fold)

<table>
<thead>
<tr>
<th>Physical Examination</th>
<th>Likely Pathology</th>
<th>Likely Pathology</th>
<th>Likely Pathology</th>
<th>Likely Pathology</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Discogenic or adjacent ligaments</td>
<td>Posterior joint complex (associated ligaments and capsular structures)</td>
<td>Sciatica</td>
<td>Neurogenic claudication (e.g. spinal stenosis)</td>
</tr>
<tr>
<td></td>
<td>Unremarkable neurological exam</td>
<td>Unremarkable neurological exam</td>
<td>Leg pain can improve but will not fully disappear</td>
<td>No irritative findings</td>
</tr>
<tr>
<td></td>
<td>Fast responder - Improves with extension</td>
<td></td>
<td>Positive straight leg raise</td>
<td>May have conduction loss</td>
</tr>
<tr>
<td></td>
<td>Slow responder - No change or worsens with extension</td>
<td></td>
<td>May have conduction loss</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fast responder - Improves with specific back position</td>
<td>No improvement with position</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Slow responder - No improvement with position</td>
<td></td>
</tr>
</tbody>
</table>

### Do I Investigate?

- **Indication for lumbar spine x-ray is as follows:**
  - No improvement after 6 weeks of conservative management.
  - Recent onset back pain with fever >38°.
  - "New" back pain in age > 50 (**could this be osteoporosis / osteoporotic fracture?**).
  - Systemic symptoms (i.e. looks unwell).
  - Unexplained neurological deficits.
  - Significant trauma.
  - Unexplained weight loss.
  - Suspicion of ankylosing spondylitis.
  - History of cancer (rule out metastases) – also consider CBC, ESR, SPEP if cancer suspected.
  - Alcohol/drug abuse (due to increased risk of osteomyelitis, trauma, fracture).
- **Otherwise, plain films are not recommended in the initial evaluation of low back pain.**

### Management and followup

- **If any red flags are present, consider referring for further evaluation and treatment (i.e. emergency department, relevant specialist).**
- **If no red flags are present, the management is dependent on whether the patient presents with acute or chronic LBP.**

### ACUTE LOW BACK PAIN

*Low back pain of <12 weeks duration, LBP without presence of red flags.*

- **Patient education:** in the absence of red flags, reassure. Reinforce that pain typically is self-limiting, and should resolve in 2-3 weeks (mean: 8 weeks) without intervention.
- **Offer conservative management** including self-care strategies:
  - Alternating cold and heat.
  - Continuation of usual activities as tolerated.
- AVOID bed rest.
- Encourage exercises and positioning as per diagnosed pattern.

- Encourage early return to work.
- If necessary, analgesia should be prescribed in the following order, titrating to adequate response:
  - Acetaminophen (1st line, likely not better than placebo (Machado et al.)).
  - NSAIDs (2nd line) [consider adding PPI for gastric protection].
  - **Opiates and muscle relaxants have no role in acute low back pain** (Friedman et al.)
- Re-assess the patient in 2-4 weeks time, re: red flags or symptom progression. Further management should be based on patient function.
- If pain persists at the 6 week mark with no improvement, consider referral at this time:
  - Spinal pathway program (if available in your province).
  - Physiotherapist.
  - Chiropractor (poor evidence).
  - Physiatry.
  - Spinal surgeon (for resolving radicular symptoms).
  - Multidisciplinary pain program (if not returning to work).

**CHRONIC LOW BACK PAIN**

Low back pain of >12 weeks duration, LBP without presence of red flags.

- Physical or therapeutic exercise.
- Analgesic options (in this order):
  - Acetaminophen (1st line, likely not better than placebo).
  - NSAIDs (2nd line) [consider adding a PPI for gastric protection]
  - Low dose tricyclic antidepressants.
- Referral options:
  - Community-based active rehabilitation program.
  - Community-based self management/CBT program.
- Additional options:
  - Progressive muscle relaxation.
  - Acupuncture.
  - Aqua therapy, yoga.
  - Massage and transcutaneous electrical nerve stimulation (TENS) can be used as an adjunct to active therapy.

**SPECIFIC EXERCISES FOR LOW BACK PAIN**

- **Pattern 1**: Supine lie, prone lie, “Z” lie, knees to chest, lumbar roll (sitting), lumbar roll (night), sloppy push-up.
- **Pattern 2**: “Z” lie, knees to chest, sitting flexion, trunk flexion stretch, knee to chest stretch (single leg or double leg).
- **Pattern 3**: “Z” lie, prone lie, prone lie on elbows, rest on hands and knees, lumbar night roll, lumbar support when sitting.
- **Pattern 4**: Single leg abdominal press, pelvic tilt, sitting flexion, cat and camel.

*For exercise descriptions, refer to the Saskatchewan Spine Pathway - Information for Patients.*

---

**RECOMMENDED #FOAMED RESOURCES**

- Video for Patients: Dr. Mike Evans – Low Back Pain
  [http://www.topalbertadoctors.org/cpgs/](http://www.topalbertadoctors.org/cpgs/)  
REFERENCES
Amer Academy of Orthopaedic
# 25. Meningitis

## KEY FEATURES

1. In the patient with a non-specific febrile illness, look for meningitis, especially in patients at higher risk (e.g., immuno-compromised individuals, alcoholism, recent neurosurgery, head injury, recent abdominal surgery, neonates, aboriginal groups, students living in residence).
2. When meningitis is suspected ensure a timely lumbar puncture.
3. In the differentiation between viral and bacterial meningitis, adjust the interpretation of the data in light of recent antibiotic use.
4. For suspected bacterial meningitis, initiate urgent empiric IV antibiotic therapy (i.e., even before investigations are complete).
5. Contact public health to ensure appropriate prophylaxis for family, friends and other contacts of each person with meningitis.

Also see [Sepsis](#).

## Risk factors

- **Community-acquired bacterial meningitis:**
  - Acute or chronic otitis media.
  - Sinusitis.
  - Pneumonia.
  - Endocarditis.
  - Alcohol abuse.
  - Diabetes.
  - Immunosuppressed (asplenia, glucocorticoid excess, complement deficiency, HIV etc.).
  - Recent contact with patient known to have bacterial meningitis.
  - Recent travel to regions with endemic meningococcal disease (sub-Saharan Africa).
- **Nosocomial bacterial meningitis:**
  - Recent head injury.
  - Recent neurosurgery.
  - CSF leak.
  - Intracranial medical devices (intracerebral pressure monitor, CSF shunt, cochlear implant).

## Pathogenesis

- **Bacterial:** *pneumococcus* (60%), *meningococcus* (15%), GBS (15%), *H. flu*, *Listeria*.
- **Viral:** HSV, CMV, Herpes, Coxsackie, Enteroviruses.
- Either spreads **hematogenously** (from respiratory tract) or is **locally invasive** (i.e. otitis media, sinusitis, brain abscess).

## Presentation

- In adults, 95% will have at least two of: **fever, nuchal rigidity, altered mental status** (more likely in bacterial vs. aseptic), **headache** (van de Beek et al.).
- Other associated symptoms:
  - Nausea/vomiting.
  - Photophobia.
  - Focal neurological deficits (including new cranial nerve palsies).
  - Seizures.
  - Rash.
  - Arthritis.
  - Symptoms proximal infection (i.e. otitis media, sinusitis).
EXAMINATION

- Most described physical examination findings in meningitis come from an era when late-stage disease was much more common. In the modern era, physical examination is a poor predictor in suspected meningitis.
- Kernig’s and Brudzinski’s signs are traditionally taught, but very poor clinical tests (Thomas et al.) (Nakao et al.)
- Jolt accentuation: positive test defined as a headache accentuation with horizontal head rotation at a frequency of 2-3 times/second. Poor sensitivity and specificity (Tamune et al.) (Nakao et al.)
- Nuchal rigidity: poor sensitivity and specificity (Nakao et al.)

IN CHILDREN

History and physical examination in children can be more reliable than in adults (Curtis et al.):

- **History:**
  - Bulging fontanelle [positive likelihood ratio (LR): 8.00].
  - Neck stiffness [LR: 7.70].
  - Seizures [LR: 4.40 when outside age range for febrile-induced convulsions].
  - Reduced feeds [LR: 2.00].

- **Exam:**
  - Jaundice [LR: 5.90].
  - Toxic-looking [LR: 5.80].
  - Neck stiffness [LR: 4.00].
  - Bulging fontanelle [LR: 3.50].
  - Kerning’s sign [LR: 3.50].
  - Fever >40 degrees Celsius [LR: 2.90].
  - Brudzinski’s sign [LR: 2.50].

Differential diagnosis

- Common and benign: migraine, cluster, tension headaches.
- Emergent causes of headache: intracranial/subarachnoid hemorrhage (ICH/SAH), carbon monoxide poisoning, hypertensive emergency, space-occupying lesions, basilar artery dissection, central venous thrombosis.
- Urgent causes of headache: temporal arteritis, idiopathic intracranial hypertension (i.e. pseudotumor cerebri), acute glaucoma.
- Less serious causes of headache: trigeminal neuralgia, dehydration, analgesia overuse headaches.
- Other causes of seizure: pre-eclampsia, epilepsy.

Investigation

- CBC with differential (looking for elevated white count and a left shift).
- Blood and CSF cultures (blood cultures are positive in most patients with bacterial meningitis (Kanegaye et al.), and can be useful if lumbar puncture is delayed, as empiric parenteral antibiotics should not be delayed for lumbar puncture).
- Serum glucose (to calculate CSF/blood glucose ratio).
- Renal function (as part of fluid resuscitation and general sepsis protocol).
- Coagulation profile (if petechia/purpura and DIC suspected).
- Electrolytes.
LUMBAR PUNCTURE

CT prior to LP

- Note that ↑ intracranial pressure (ICP) is a relative contraindication to LP in meningitis, due to the risk of cerebral herniation. Signs that have been shown to correlate with the need for head CT prior to LP (Hasbun et al.):
  - >60 yo.
  - Immunocompromised (i.e. HIV, transplant patient, or immunosuppressed).
  - Seizure within 1 week of presentation.
  - Neurological findings (i.e. ↓LOC, palsies, speech deficits).
  - History of CNS disease (i.e. mass lesion, stroke, or focal infection).
- If ↑ ICP is a consideration, LP opening pressure should be measured (LP should be performed with patient lying horizontal for an accurate pressure reading).

<table>
<thead>
<tr>
<th></th>
<th>NORMAL</th>
<th>BACTERIAL</th>
<th>ASEPTIC (VIRAL)</th>
<th>FUNGAL</th>
<th>TUBERCULOSIS</th>
<th>SUBARACHNOID HEMORRHAGE</th>
<th>NEOPLASTIC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appearance</td>
<td>Clear</td>
<td>Clear, cloudy, or purulent</td>
<td>Clear</td>
<td>Clear or opaque</td>
<td>Clear or opaque</td>
<td>Xanthochromia, bloody, or clear</td>
<td></td>
</tr>
<tr>
<td>Opening Pressure (cmH₂O)</td>
<td>10-20</td>
<td>&gt;25</td>
<td>Normal or ↑</td>
<td>&gt;25</td>
<td>&gt;25</td>
<td>&gt;25</td>
<td>Normal or ↑</td>
</tr>
<tr>
<td>WBC Count (10⁶/L)</td>
<td>0-5</td>
<td>&gt;500</td>
<td>&lt;1000</td>
<td>&lt;100-500</td>
<td>50-500</td>
<td>0-5 (see correction section)</td>
<td>&lt;500</td>
</tr>
<tr>
<td>% PMNs</td>
<td>&gt;80-90%</td>
<td>1-50%</td>
<td>1-50%</td>
<td>Early PMN then lymph</td>
<td>1-50%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glucose</td>
<td>&gt;60% of serum glucose</td>
<td>Low</td>
<td>Normal</td>
<td>Low</td>
<td>Normal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>Protein (g/L)</td>
<td>0.18-0.45</td>
<td>&gt;1</td>
<td>&lt;1</td>
<td>N</td>
<td>↑</td>
<td>↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Gram Stain</td>
<td>Negative (-)</td>
<td>Positive (+)</td>
<td>Negative (-)</td>
<td>N/A</td>
<td>Requires specific stain</td>
<td>N/A</td>
<td></td>
</tr>
</tbody>
</table>

Adapted from lifeinthefastlane.com and wikiem.org.

Gram Stain

- Sensitivity of Gram stained CSF for the diagnosis of bacterial meningitis is 60 to 90%, while the specificity approaches 100%. If (+), can allow targeted therapy pending culture:
  - Gram (+) diplococci: *S. pneumoniae*.
  - Gram (+) cocci (neonate): Group B Streptococcus.
  - Gram (+) bacilli: *L. monocytogenes*.
  - Gram (-) diplococci: *N. meningitides*.
  - Small pleomorphic Gram (-) bacilli: *H. influenzae*.
  - Larger Gram (-) (especially in neonate): *E. coli*.
Clinical suspicion of bacterial meningitis

Immunocompromise, history of CNS disease, new onset seizure, papilledema, altered LOC, or focal neuro deficit

Blood cultures and LP

Empiric antimicrobial therapy (consider adding dex*) 
without delay!

CSF findings consistent with bacterial meningitis?

No

Consider alternative diagnosis

 Continue empiric antimicrobial therapy (+/- dex)

Yes

(+ ) Gram stain

 Continue targeted antibiotic therapy (+/- dex)

Blood cultures

Empiric antimicrobial therapy (consider adding dex*) 
without delay!

CT Head

Contraindication for LP?

No

Perform LP

Yes

Continue antibiotic therapy +/- consider alternative diagnosis

ANTIMICROBIAL REGIMENS

• Follow your local sepsis protocols for empiric therapy choices for meningitis (if available).

• Meningitis antibiotic regimens must:
  – Use agents that cross the blood-brain barrier,
  – Be effective against the most likely pathogens, and
  – Must be structured to optimize efficacy based on the pharmacodynamics of the drugs selected.

* Dexamethasone therapy is recommended as an adjunct in adults in developed countries with known or suspected pneumococcal meningitis.
PREDISPOSING FACTOR | COMMON BACTERIAL PATHOGENS | ANTIMICROBIAL THERAPY OPTIONS
--- | --- | ---
Age | | |
<1 month | Streptococcus agalactiae (GBS), Escherichia coli, Listeria monocytogenes | Ampicillin + cefotaxime
 | | Ampicillin + an aminoglycoside
1 to 23 months | Streptococcus pneumoniae, Neisseria meningitidis, S. agalactiae (GBS), Haemophilus influenzae, E. coli | Vancomycin + a third-generation cephalosporin
2 to 50 years | N. meningitidis, S. pneumoniae | Vancomycin + a third-generation cephalosporin
>50 years | S. pneumoniae, N. meningitidis, L. monocytogenes, aerobic gram-negative bacilli | Vancomycin + ampicillin + a third-generation cephalosporin

Head trauma
Basilar skull fracture | S. pneumoniae, H. influenzae, group A beta-hemolytic streptococci | Vancomycin + a third-generation cephalosporin

Penetrating trauma
Staphylococcus aureus, coagulase-negative staphylococci (especially Staphylococcus epidermidis), aerobic gram-negative bacilli (including Pseudomonas aeruginosa) | Vancomycin + cefepime
 | Vancomycin + ceftazidime
 | Vancomycin + meropenem

Postneurosurgery
Aerobic gram-negative bacilli (including P. aeruginosa), S. aureus, coagulase-negative staphylococci (especially S. epidermidis) | Vancomycin + cefepime
 | Vancomycin + ceftazidime
 | Vancomycin + meropenem

Immunocompromised state
S. pneumoniae, N. meningitidis, L. monocytogenes, aerobic gram-negative bacilli (including P. aeruginosa) | Vancomycin + ampicillin + cefepime
 | Vancomycin + ampicillin + meropenem


Contact tracing
- The cornerstone of prevention of secondary cases of invasive Meningococcal disease (IMD, caused by Neisseria meningitidis) is aggressive contact tracing to identify people at increased risk of disease (i.e., close contacts) (Public Health Agency of Canada).
- Management of close contacts of cases with conjunctivitis or pneumonia is the same as for close contacts of IMD.
- Chemoprophylaxis should be offered to all persons having close contact with an IMD case during the infectious period (the 7 days before onset of symptoms in the case to 24 hours after onset of effective treatment).
- Appropriate agents for chemoprophylaxis include ciprofloxacin, rifampin, and ceftriaxone (Public Health Agency of Canada — Recommendations for Chemoprophylaxis (2)).

Aseptic meningitis
- Aseptic meningitis refers to the clinical presentation of meningitis, but with negative bacterial cultures (either blood or CSF).
- Most common causes are the entroviruses (A. Tunkel).
- Typically self-limiting disease course (often requires supportive treatment).
- HSV-caused “aseptic” meningitis may be treated with acyclovir (unclear evidence).
REFERENCES


### 26. Periodic Health Assessment Screening

#### KEY FEATURES

1. Do a periodic health assessment in a proactive or opportunistic manner (i.e., address health maintenance even when patients present with unrelated concerns).

2. In any given patient, selectively adapt the periodic health examination to that patient’s specific circumstances (i.e., adhere to inclusion and exclusion criteria of each manoeuvre/intervention, such as the criteria for mammography and prostate-specific antigen [PSA] testing).

3. In a patient requesting a test (e.g., PSA testing, mammography) that may or may not be recommended:
   - Inform the patient about limitations of the screening test (i.e., sensitivity and specificity).
   - Counsel the patient about the implications of proceeding with the test.

4. Keep up to date with new recommendations for the periodic health examination, and critically evaluate their usefulness and application to your practice.

#### When should we screen?

- When the disease often has a significant detrimental impact on quality of life or longevity.
- When treatment (before symptoms occur) is more effective than treatment that is delayed until symptoms appear.
- When the prevalence of disease in the population to be tested is high enough to outweigh the risk and costs associated with false positive results.
- When we have a “good” screening test available:
  - Inexpensive to administer (including procurement and processing costs).
  - Acceptable to the population being screened (i.e., minimal discomfort, unpleasantness, or inconvenience).
  - *FIT seems to out-perform the older FOBT testing (in that more people in the target population complete the test) due to the "less disgusting" nature (Chambers et al.)*
  - Reliable (repeated testing should yield the same result for the same individual).
  - Valid (able to distinguish between disease and non-disease states).

#### Preventative health guidelines

The following organizations develop primary care-focused preventative health screening guidelines that are relevant for the Canadian population:

- **Canadian Task Force on Preventative Health Care (CTFPHC)**
- **U.S. Preventative Services Task Force (USPSTF)**
- **Canadian Agency for Drugs and Technologies in Health (CADTH)**
- Various membership organizations in North America (i.e., Canadian Cardiovascular Society, American Academy of Physicians).

The interactive tool at [objectivehealth.ca](http://objectivehealth.ca) summarizes some of the content above, and may be useful to you in practice (disclosure: developed by the 99 Topics for the CCFP team)! A recent CFP article ([Shimizu et al.](http://example.com)) does an excellent job of summarizing screening guidelines as well.

#### LIFESTYLE

- **Discuss:**
  - Smoking cessation.
  - EtOH intake limited to ≤10 drinks/wk for women, ≤15 drinks/wk for men.
  - Aim for 150 min/wk of moderate intensity exercise.
  - Balanced diet: fruit, vegetables, whole grains, healthy fat, <2000mg/d salt intake (800mg sodium).
  - Limit sun exposure: sunscreen, protective clothing.
  - Safe sex, STI counselling, and contraception.
- **Supplement:**
  - Vitamin D: 400-2000 IU/d
  - Calcium: at least 1000 mg/d from diet (increase to 1500-2000 mg/d if pregnant or lactating).
  - New! *Folic acid: 0.4-1mg/d for all women of childbearing age.*
AAA

- **USPSTF 2005**, CTFPHC (in development):
  - The USPSTF recommends one-time screening for abdominal aortic aneurysm (AAA) by ultrasonography in men 65-75yo who have ever smoked.

BREAST CANCER

- **CTFPHC 2011**:
  - For women 50-74yo we weakly recommend routinely screening with mammography every 2 to 3 years.
  - We recommend not routinely performing clinical breast exam alone or in conjunction with mammography to screen for breast cancer.
  - We recommend not advising women to routinely practice breast self exam.
  - Breast Cancer Risk Assessment Tool may be helpful for quantifying risk to patients.

CARDIOVASCULAR DISEASE

- **USPSTF 2015**:
  - The USPSTF recommends daily low-dose aspirin use for the prevention of cardiovascular disease and colon cancer in adults 50-59yo who have a 10% or greater 10 year CVD risk, are not at increased risk for bleeding, have a life expectancy of at least 10 years, and are willing to take low-dose aspirin daily for at least 10 years.

CERVICAL CANCER

- **CTFPHC 2013**:
  - For women 25 to 69yo we recommend routine screening for cervical cancer every 3 years.
  - For women ≥70yo who have been adequately screened (i.e. 3 successive negative Pap tests in the last 10 years), we recommend that routine screening may cease. For women >70yo who have not been adequately screened we recommend continued screening until 3 negative test results have been obtained.

COLON CANCER

- **CTFPHC 2016**:
  - We recommend screening adults aged 50 to 74 with FOBT (either gFOBT or FIT) every two years or flexible sigmoidoscopy every 10 years.
  - We recommend not screening adults aged 75 years and over.
  - We recommend not using colonoscopy as a screening test.

DIABETES

- **USPSTF 2015**:
  - The USPSTF recommends screening for abnormal blood glucose as part of cardiovascular risk assessment in adults aged 40 to 70 years who are overweight or obese.
  - However, screening for diabetes did not improve mortality rates after 10 years of follow-up (Selph et al.)
- **CTFPHC 2012**:
  - Ask about symptoms of diabetes: unusual thirst, frequent urination, weight change (gain or loss), extreme fatigue or lack of energy, blurred vision, frequent and recurring infections, cuts and bruises that are slow to heal, and/or tingling or numbness in the hands or feet.
  - FINDRISC Diabetes Risk Calculator to distinguish low-, moderate- and high-risk patients:
    - Recommendation to not screen in low-risk, 3-5 yearly in moderate-risk and annually in high-risk patients.
    - Major risk factors include: WC > 102cm, BMI > 30, age > 55, gestational diabetes, first- or second-degree relative.
HYPERLIPIDEMIA

- CCS 2012:
  - Screen 3-5 yearly, or annually if previous (Framingham Risk Score) FRS >5%.
  - CVD Risk Equivalents that imply FRS >20% and require treatment:
    - Coronary Artery Disease.
    - Peripheral Vascular Disease.
    - AAA.
    - Diabetes and age ≥40.
    - CKD (eGFR ≤45 or ACR ≥30 or (eGFR ≤60 and ACR ≥3)).

HYPERTENSION

- CTFPHC 2012:
  - We recommend blood pressure measurement at all appropriate primary care visits.
  - Also see: CHEP 2014.

OBESITY

- Measure BMI and/or WC: if obese (30 ≤ BMI < 40), offer to refer to structured behavioural interventions aimed at weight loss.

OSTEOPOROSIS

- Osteoporosis Canada 2010:
  - From age 50, assess 10-year risk with: FRAX WHO Fracture Risk Assessment Tool for Canada +/− BMD measurement.
  - Indications for BMD measurement:
    - Age ≥65.
    - Clinical risk factors for fracture (menopausal women, men 50–64yo).
    - Fragility fracture >40yo.
    - Prolonged use of glucocorticoids (>3 months of prednisone 7.5mg daily equivalent).
    - Use of other high-risk medications.
    - Parental hip fracture.
    - Vertebral fracture or osteopenia identified on radiography.
    - Current smoking.
    - High alcohol intake.
    - Low body weight (<60kg) or major weight loss (>10% of body weight at 25yo).
    - Rheumatoid arthritis.
    - Treat those with a high 10-year risk (>20%), consider treatment for those who are moderate risk (>10%).
    - Let fracture risk (not bone density) be the trigger for considering treatment.

LUNG CANCER

- CTFPHC 2016:
  - For adults aged 55–74 years with at least a 30 pack-year smoking history who currently smoke or quit less than 15 years ago, we recommend annual screening with low-dose CT (LDCT) up to three consecutive times.

PROSTATE CANCER

- CTFPHC 2014:
  - Recommends against screening with PSA in men aged 55+. DRE is not recommended.

SEXUALLY TRANSMITTED INFECTIONS

- Screen annually until 25yo for chlamydia and gonorrhea (syphilis, Hep B and HIV if high-risk), and thereafter if ongoing high-risk behaviours.
Explaining risk

- Decisions to screen or not to screen for disease require you to have an understanding of the benefits and risks so that you can have an informed discussion with your patients.
- Whenever possible, use natural frequencies to convey benefits and risks to patients, and use visual aids where available (see sample dialogue and graphic below) (Petrisor and Tornetta).
“Mr. Johnson, as you can see in the graphic below, if we were to test 1000 men similar to yourself, of about your age and of average risk for prostate cancer, 102 of them would ultimately be diagnosed with prostate cancer. Of those 102 men however, only 5 of them will die from prostate cancer despite being screened, and only 1 man will escape death because they were screened with the PSA test. On the other hand, of those same 1000 men, 178 of them will have a positive PSA despite actually not having prostate cancer, and the next investigation that is performed to rule out cancer in these men is a prostate biopsy. Of those 178 who go on to have a biopsy, 4 of them will have complications (such as infection or bleeding) that requires hospitalization.”

---

**Benefits and Harms of PSA Screening**

The Canadian Task Force on Preventive Health Care recommends against screening for prostate cancer with the PSA test

- The CTFPHC found that the potential small benefit from PSA screening is outweighed by the potential significant harms of the screening and associated follow-up treatment.
- Men should understand that PSA screening may result in additional testing if the PSA level is raised.
- To save one life we would need to diagnose 27 men with prostate cancer

**RESULTS OF SCREENING 1,000 MEN WITH THE PSA TEST**

- Among men who **are screened** with the PSA test, the risk of dying from prostate cancer is 5 in 1,000
- Among men who **are not screened** with the PSA test, the risk of dying from prostate cancer is 6 in 1,000

**What are my risks if I don’t get screened?**

- Among men who **are screened** with the PSA test, the risk of dying from prostate cancer is 5 in 1,000
- Among men who **are not screened** with the PSA test, the risk of dying from prostate cancer is 6 in 1,000

**Complications of treatment for prostate cancer**

For every 1,000 men who receive treatment for prostate cancer:
- 114–214 will have short-term complications such as infections, additional surgeries, and blood transfusions
- 127–442 will experience long-term erectile dysfunction
- up to 178 will experience urinary incontinence
- 4–5 will die from complications of prostate cancer treatment

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Infographic reproduced in full with permission.
RECOMMENDED #FOAMED RESOURCES

REFERENCES
Petrisor, B. A., & Tornetta, P. Communicating study results to our patients: which way is best? *Indian journal of orthopaedics*, 42(2), 140–3. doi:10.4103/0019-5413.40249
## 27. Personality Disorder

### KEY FEATURES

1. Clearly establish and maintain limits in dealing with patients with identified personality disorders. For example, set limits for:
   - appointment length.
   - drug prescribing.
   - accessibility.
2. In a patient with a personality disorder, look for medical and psychiatric diagnoses when the patient presents for assessment of new or changed symptoms. (Patients with personality disorders develop medical and psychiatric conditions, too.)
3. Look for and attempt to limit the impact of your personal feelings (e.g., anger, frustration) when dealing with patients with personality disorders (e.g., stay focused, do not ignore the patient’s complaint).
4. In a patient with a personality disorder, limit the use of benzodiazepines but use them judiciously when necessary.
5. When seeing a patient whom others have previously identified as having a personality disorder, evaluate the person yourself because the diagnosis may be wrong and the label has significant repercussions.

*Always ask about suicidal ideation in any mental health presentation!*

### Diagnosis

- **DSM-V**: impairments in personality (self and interpersonal) functioning and the presence of pathological personality traits. To diagnose a personality disorder, the following criteria must be met:
  - Significant impairments in **self** (identity or self-direction) and **interpersonal** (empathy or intimacy) functioning.
  - One or more pathological personality trait domains or trait facets:
    - Negative affectivity.
    - Detachment.
    - Antagonism.
    - Disinhibition vs compulsivity.
    - Psychoticism.
  - Impairments in personality functioning and the individual’s personality trait expression are **relatively stable across time** and consistent across situations.
  - Impairments are **not better understood as normative** for the individual’s developmental stage or socio-cultural environment.
  - Impairments in personality functioning and expression are **not solely due to the direct physiological effects of a substance** (e.g., drug of abuse, medication) or a general medical condition (e.g., severe head trauma).

- Patients may **frequently present with a range of complex issues** including relationship problems, mood issues or features of dissociation such as isolation.

- **When should a diagnosis be made?**
  - Distinct, **long-term pattern of maladaptive responses** to events, difficulties in emotional regulation, impulse management and interpersonal functioning.
  - Diagnosis should **not** be made during acute events, transient psychological issues or substance intoxication. Chronic patterns of drug use can mimic personality problems.

### Types of personality disorders

**CLUSTER A: ODD, ECCENTRIC**

- **Paranoid**: Pervasive distrust and suspicion. Interprets others as harmful or deceiving.
- **Schizoid**:
  - Diagnosis:
    - Pervasive pattern of **detachment from social relationships** and **restricted emotional expression in interpersonal settings**, beginning by early adulthood and present in a variety of contexts, as indicated by **four (or more) of the following**:
      1. Neither desires nor enjoys close relationships, including family.
      2. Almost always chooses solitary activities.
3. Little interest in having sexual experiences with others.
4. Takes pleasure in few, if any, activities.
5. Lacks close friends other than first-degree relatives.
6. Indifferent to praise or criticism from others.
7. Emotional coldness, detachment or flat affect.
   • Does not occur exclusively during the course of schizophrenia, a mood disorder with psychotic features, another psychotic disorder or pervasive developmental disorder, or is not due to a direct physiological effect of a general medication.

• **Schizotypal**: Eccentric behavioural patterns with decreased social relatedness.
  - Important to distinguish from schizophrenia.
  - Diagnosis:
    - Impairments in **personality functioning** manifest by:
      - **Identify**: distorted self-concept.
      - **Self-direction**: unrealistic goals.
    - Impairments in **interpersonal functioning**
      - **Empathy**: difficulty understanding impact of their own behaviours on others.
    - Impairments in developing textbf{intimacy}
    - Pathological **personality traits**
      - **Psychoticism**: eccentricity, cognitive dysregulation including unusual thought processes), unusual beliefs and experiences.
      - **Detachment**: reduced emotional response and withdrawal.
      - **Negative affectivity**: suspiciousness and heightened expectations of ill-intent.

**CLUSTER B: DRAMATIC, OVER-EMOTIONAL**

• **Anti-social**: unempathetic, callous, manipulative. Previously known as 'sociopathy'.
• **Borderline**: unstable identity.
  - Substance use is a common co-morbidity.
  - Diagnosis:
    - **Poorly developed self image** and self-direction with excessive self-criticism, chronic feelings of emptiness and instability in goals or values.
    - Impairments in interpersonal functioning – **difficulty maintaining relationships** and recognising feelings with others. Instability with intimacy.
    - Pathological personality traits:
      - **Negative affectivity**: unstable mood, depressivity, anxiousness and separation insecurity.
      - **Disinhibition**: engagement in risky, self-damaging activities.
      - **Antagonism**: hostility in response to minor insults.
    - Frequent self-harm and suicide ideation to prevent real or perceived abandonment.
    - Often due to childhood trauma and attachment issues.
• **Histrionic**: excessive attention seeking behaviour, lacks self-reflection.
• **Narcissistic**: self-centred, grandiose.
  - Seek admiration with perceived self-importance and exaggerates achievements.

**CLUSTER C: ANXIOUS, FEARFUL**

• **Avoidant**: inhibited, sensitive to being negatively perceived by others. Difficulties forming interpersonal relationships and avoid social situations.
• **Dependent**: excessive need to be cared for due to fear of separation.
  - Difficulty with autonomy and taking responsibility.
  - Risk of exploitation by others. Chronic, low self-esteem.
• **Obsessive**: Perfectionistic, rigid, control.
  - Self-imposed high standards leading to impaired function, distress and isolation.

**PERSONALITY DISORDERS**

“Mad, bad, sad” — Mad (cluster A: odd, eccentric), Bad (cluster B: dramatic, emotional, erratic) and Sad (cluster C: anxious, fearful).
Principles of Management

- **Goal:** help the patient reflect and understand their mental experience and behavioural responses to maximise stability, well-being and social functioning.
- **Cloninger’s 4 stages of treatment:** (Cloninger et al.)
  - **Stage 1:** Crisis management.
    - Risk assessment of self-harm, suicide ideation, ability to accept support.
  - **Stage 2:** Stabilization.
    - Engagement, presenting issues and symptoms.
  - **Stage 3:** Personality change.
    - Insight and mentalisation.
  - **Stage 4:** Understanding.
    - Self-awareness of underlying causes of difficulties and dealing with past trauma.
    - Learning psychological strategies to manage stress, dysphoria and negative cognitions.

- **Drug treatment**
  - Often patients will already be taking a variety of psychotropic medications.
  - **Non-pharmacological treatment** should be emphasised.
    - Minimal evidence for long term treatment with anti-psychotic drugs.
    - **Limit** use of benzodiazepines and opioids (due to ↑risk of dependence).
  - **Prescribe with care:** single pharmacy, clearly identified outcomes, avoid polypharmacy and frequent dispensing.
    - Short-term psychotropics may be required for stabilization:
      - SSRI's: treat symptoms of depressed mood and irritability.
      - Psychotic-like symptoms: low-dose quetiapine, clozapine.
      - Aggression: mood stabilizers, carbamazepine.

Challenges with treatment

- **Negative counter-transference**
  - Perceived as "challenging" patients due to complex, chronic nature of the condition.
  - Recognize treatment frustrations as **manifestations of the patient’s own distress**.
  - Share management responsibility to ensure consistent, adequate support for ideas and treatment approaches.

- **Risk assessment**
  - Suicidal or self-harming thoughts and behaviours are common!
  - Include a history of suicidal behaviour and impulses, current level of distress, past response to stress and capacity to accept support.
  - Maintain patient engagement, document of risk assessment and consider involving patient’s family.

- **Developing a care plan**
  - **Acute crisis plan** for the physician and patient to identify potential triggers, self-management strategies and crisis services.
  - **Long term goal** to decrease crisis presentations, promoting stability.

- **Maintaining boundaries**
  - Show interest and empathy while maintaining clear boundaries.
  - Schedule regular, structured appointments.
  - Prescribe carefully with clearly defined outcomes and regular reviews.

When to refer

- Care in personality disorder diagnoses requires a **team approach**: patients should be co-managed with a psychologist, psychiatrist and/or mental health service. Particularly in cases of:
  - Multiple co-morbidities.
  - Uncertainties in regards to pharmacological treatment or risk assessment.
RECOMMENDED #FOAMED RESOURCES


REFERENCES


28. Urinary Tract Infection

See Dysuria.
29. Sepsis

Causes

**SEPTIC ORIGIN SITES**

LUCCASSS — Lung, Urine, CNS, Cardiac, Abdomen, Skin, Spine, Septic Arthritis

Lung and urine are the most common: always get a CXR and U/A!

- **Lung** infection is most common (pneumonia). Older or immunocompromised patients may not have many signs: lack of infiltrate early on, can have a normal WBC/CRP.
- **Urine** infection is the second most common source of sepsis. Be mindful that asymptomatic bacteruria is common in the elderly (as much as 10%), if you find a urine infection that may be your source but you should keep looking to rule out other sites.
- Think of **CNS** infections if there is altered LOC, new headache, or meningeal signs.
- **Cardiac**: think of endocarditis in every septic patient: listen for the new murmur, and look for peripheral signs (Janeway lesions and Osler nodes). Consider this diagnosis upfront in IVDUs.
- **Consider an abdominal source** if there is abdominal pain, constipation, nausea, vomiting, or diarrhea. Have a very low threshold in the elderly and immunocompromised to consider and investigated an abdominal cause of sepsis (mesenteric ischemia, bacterial translocation in obstruction, etc.).
- **Every septic patient should receive a full skin exam** to look for cellulitis, necrotizing fasciitis (which is difficult to differentiate from cellulitis early on), or invasive devices that can be a source of infection (urinary or intravenous catheters).
- When you have a septic patient without an identifiable source, next consider **spinal pathology**: epidural abscess, osteomyelitis (not just of the spine), and discitis. These patients can present with subtle neurological signs in conjunction with the typical septic signs and symptoms. Consider this diagnosis upfront in IVDUs.
- **Septic arthritis** can affect any joint, but the knee is most common, and the most common pathogens are *S. aureus* and *N. gonorrhea*. Consider this diagnosis in patients with prosthetic joints, RA, HIV (immuno-compromised), and recent surgery. If you have a concerning joint, you should perform arthrocentesis. ESR or CRP are not clinically useful.

Treatment of SIRS/sepsis

There is lots of new evidence and ideas in the management of sepsis. Early Goal Directed Therapy (EGDT) has given way to evidence from newer trials such as **PROMISE** and **ARISE**. Regardless, most healthcare facilities should have a sepsis protocol to get things started.

- **Septic/SIRS** patients are often hypovolemic: you need to ensure adequate preload with initial intravenous resuscitation of 1-4L of NS or Ringer’s Lactate in adults, or 30ml/kg in pediatrics.
  - Avoid the use of colloids such as pentastarch or hydroxethyl starch.
- After each 1L bolus, **assess for volume responsiveness and fluid overload** using IVC US or straight leg raise test, or (with appropriate training) bedside echo. If the patient continues to be volume responsive, continue fluids. If the patient has signs of fluid overload or is volume unresponsive, stop bolusing.
- If the patient has not responded sufficiently with your initial fluid bolus, **start a peripheral vasopressor** as the next step. Norepinephrine has the greatest evidence and is safe to run while resuscitating in a peripheral IV (cite). Start at 2-4mcg/min and increase as clinically indicated. Typical required dose range is 8-12mcg/min.
- **Start empiric antibiotics early**! Every hour of delay can increase mortality by 7.6%. Use broad spectrum antibiotics initially, keeping in mind the likely source, local resistance patterns.

**THERAPY TARGETS**

As mentioned above, evidence regarding routine management of EGDT targets is conflicting. Until further studies are done, it is probably reasonable to measure and attempt to achieve one or more of the EGDT targets:

- Mean arterial pressure (MAP) ≥ 65 mmHg.
- Urine output ≥ 0.5 ml/kg/hr.
- Central venous pressure (CVP) of between 8 and 12 mmHg, when central venous access is available.
• Central venous hemoglobin saturation ($S_{cv}O_2$) $\geq$ 70%, when central venous access is available.

**RECOMMENDED #FOAMED RESOURCES**


**REFERENCES**

Weingart, S. Peripheral Vasopressor Infusions and Vasopressor Extravasation. [http://emcrit.org/podcasts/peripheral-vasopressors-extravasation/](http://emcrit.org/podcasts/peripheral-vasopressors-extravasation/)

30. SOOs

From the podcast: practicing for the SOOs

Notes from an interview with: Dr. Sonali Srivastava, FMR2, and Dr. Mark Karanofksy — Family physician at Jewish General Hospital (McGill University training site). Listen to the podcast at the 99T website.

How do you think residents should prepare for the SOO? What specifically can they do to practice?
- Practice with staff, and practice within a study group — each prepare a different case and practice with each other.
- Be careful not to read all the cases beforehand.

How many SOO’s do you think they should do before the actual exam?
- No real number, until you are comfortable.
- Once you master the process of the exam, practice only if you need more confidence.

How much before the exam should they begin practicing?
- It is hard to do an interview with two problems and a social context in R1. Usually we do 1-2 sessions in R1.
- In R2 we do 4 sessions through the residency programme, but then the residents will do a few on the side with each other. A few residents seek out staff to do other SOOs with them in the last few months. I wouldn't worry or spend too much time until 3-4 months into R2. Before then you’re likely not ready.

What are the common mistakes people make?
- Cheating on the review of systems.
- Time management.
- Not listening to the answers the patient gives.
- Not listening for cues.
- Not asking questions for all reasonable differential diagnoses (you'll never know what counts for marks and what doesn't).
- Forget to ask for old records.
- Forget to say you would perform physical exam.
- Forget to FIFE: but don’t do it obviously / on auto-pilot!
- Not telling the patient what you think the diagnosis / differential is.
- Missing the context integration statement.

Where do people often lose marks?
- All over the place!
- Usually I have seen candidates miss on the differential, review of systems, premature closure, and forgetting to ask about the social context.

If someone gets stuck mid-SOO, what should they do?
- Summarize! "Do I understand you correctly?"

In your experience, what makes someone really great at doing the SOOs?
- Smooth, patient-centred interviewing; not "machine gunning" yes/no questions, calm approach, interested in making the patient better and negotiating a plan – not dictating one.

Do you remember any candidates that stood out for either being really great or losing steam? What did they do to get themselves in that position?
- Don't challenge the examiner or question the ethics of the exam!
- Don't try to get the examiner to tell you the second problem directly.
- Don't listen to the answers then just move on. Don't ask questions just to ask, the responses should be heard and addressed if needed.
Chapter 30. SOOs

- Abrupt, arrogant, or condescending approach does not go over well in this exam.

**Let’s say it all goes wrong during the SOOs and the resident feels the actor was not being true to their part, what systems are in place for reporting this?**

- Forget about it and move on to the next one. You’ll have time after the last case is done to think about it.
- Address your concerns to the staff on site – there is a site coordinator. Also write it in your feedback after the exam. Speak to the on-site staff they will help direct you if you feel there was an issue.
- Don’t forget, other candidates may get different information based on the questions they ask. Examiners don’t volunteer information at times that easily if it says in the script to only answer if candidate asks specifically. You may not get information because you didn’t ask the right question.

**Any words of wisdom would you like to impart?**

- 15 minutes: 2 problems, 1 social context. 1 context integration statement, and manage both. Check with the patient: make sure they agree with each plan.
- Don’t leave the room early. You are allowed to, but don’t. Ask more questions.
- Don’t cheat on the review of systems or the differential diagnoses questions.
- With the three-minutes-left prompt, say something like “So I see you have problem A and also Problem B all in the social context C. That must be difficult and I want to help you through it”.
- In management, remember to offer pharma and non-pharma options for treatment if applicable and to involve a support network in plan.
31. SAMPs

SAMP Basics

- You must do the practice exams online from the CCFP — they helpfully use the same computer system as the actual examination!
- The SAMPs are patient-centered cases: all questions start with a patient case stem, with questions related directly to it.
- Questions are based on the basics of family medicine: two half-days, ~20-25 cases in the morning, ~20-25 in the afternoon.
- Tested content is consensus practice guidelines and general knowledge.
- Practice setting will be identified in the question: office, emergency room, inpatient setting. Answer the question with setting in mind!
- Use commonly-used abbreviations only; if in doubt, write it out (“blood culture” instead of “BC”).
- Generic or trade names are both acceptable.
- There are often more right answers than responses required.
- Only one answer per line will be marked.
- CBC is not an answer on this exam! (Be specific: hemoglobin, white cell count). Similarly, don’t write: LFTs, lipid panel, “lytes”, Celiac panel, ABGs.
  - (Only two exceptions to this rule: Urinalysis, and “WBC and differential”).
- No negative marks for incorrect answers. Don’t finish the exam without writing an answer (however unsure you may be) for each question!
- Exam committee is made up of Family Physicians from across the country, not specialists.
- Questions are meant reflect family MD experience in typical practice (rural or urban).
- CCFP Self Learning modules: avoid MCQs (not representative), example SAMPs may be helpful.
- PBSG learning modules (“McMaster PBLs”) are usually guideline-based — a good resource for study!

Most common family medicine diagnoses

**OUTPATIENT:**
- Hypertension.
- Hypercholesterolemia.
- Diabetes mellitus.
- Post-menopausal bleeding.
- Pregnancy.
- Urinary tract infection.
- Depression.
- “Routine” medical examination / preventative health screening.
- Abdominal pain.
- Sinusitis, pharyngitis.
- Well baby check (i.e. milestones).
- Parkinson’s.
- Rabies.
- PMR/Temporal arteritis.
- Lithium surveillance/toxicity.
- Hemochromatosis.
- Celiac disease.
- Back pain.
- Smoking cessation.
- Migraine.
- STIs (know treatment as well).
- Thyroid disease.
- Basic palliative care.
- Basic infectious diseases (i.e. HIV, Hep C, TB, PID).
Chapter 31. SAMPs

INPATIENT:

- Chest pain.
- Pneumonia.
- Congestive heart failure.
- Asthma.
- Cellulitis.
- Fluid/electrolytes/acid/base disorders.
- Stroke.
- Cardiac dysrhythmias.
- Diabetes mellitus.
- Coronary artery disease.

DOSES TO KNOW:

- Ibuprofen, acetaminophen (pediatric).
- Amoxicillin (pediatric and adult).
- Anaphylaxis in adults and kids.
- STI treatment.
- *H. pylori* eradication (choose one option).
- Smoking cessation drugs.
- ACLS drugs.
- General rule is to learn one first-line, and one second-line pharmacological option from a different class for common conditions:
  - Pick 1 drug from two different classes for hypertension (i.e. an ACE-i, diuretic, CCB, etc.).
  - Pick 1 drug from two different classes for depression (i.e. citalopram 10mg PO OD, amitriptyline 10mg QHS).
- Learn common side-effects / drug interactions:
  - Statins (myalgia).
  - NSAIDs (gastroitis; cardiac risk factors).
  - ACEIs (cough, hyper-K, ↑Cr).
  - HIV meds (fatigue, nausea, diarrhea).
  - Oral contraceptive pills.
  - Antidepressants.

Marking

- The passing score is determined by a group of reference candidates: graduates of Canadian residency programs in family medicine who are sitting the examination for the first time — all candidates are compared to this group.
- Most CCFP exam failures are due to clinical exam (SOOs, OSCE) (for IMGs).
- Re-write exam at next sitting: if only fail SAMPs, only re-write SAMPs.

Some content adapted with permission and thanks from Dr. Laura Bennion MD CCFP Dip Sport Med, Associate Clinical Professor (University of Calgary).
32. Extras

Pearls of wisdom

- If you have a high clinical suspicion, screening tests (a.k.a. those with a high sensitivity) are generally inappropriate — send for the gold standard diagnostic test.
- Similarly, for patients who have a high pre-test probability (a.k.a. you have a high suspicion of disease, and/or for patients with significant risk factors), guideline-based screening intervals may not apply. For example, the Canadian Diabetes Association recommends 3 yearly screening for Diabetes — this recommendation does not apply (and you should test at more frequent intervals) in patients at high risk, or those with symptoms consistent with hyperglycemia.

The ‘Murtagh’ diagnostic model

Professor John Murtagh is the preeminent Australian educator in General and Family Practice. He is the author of John Murtagh’s General Practice, the definitive General Practice reference for Australian GPs.

Ask yourself five questions with every presenting problem:
- What is the probable diagnosis?
- What serious disorder(s) must not be missed? ("red flags")
- What conditions can be commonly missed in this situation? ("common pitfalls")
- Could the patient have one of the "masquerades" commonly encountered?
- Is the patient trying to tell me something? (Look for ‘yellow flags.’)

MURTAGH’S 7 COMMON MASQUERADES

- Depression.
- Diabetes.
- Drugs: iatrogenic, over-the-counter, or self-abuse.
- Anaemia.
- Thyroid and other endocrine disorders (especially Addison's disease).
- Spinal dysfunction (pain syndromes).
- Urinary tract infection.
**An ATLS Cheatsheet**

**ATLS Cheatsheet — 1/2**

<table>
<thead>
<tr>
<th>Section</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Call for help!</td>
<td>“OMIP” : O2 (full open NRB), monitors (ECG leads and pads), IVs (x2 large bore), Pulse check and BP cuff (cycling q30s)</td>
</tr>
<tr>
<td>Airway:</td>
<td>maintain C-spine immobilization - is the airway patent? - evidence of obstruction? - facial trauma? - burns / carbonaceous sputum? - if yes, consider securing an airway early!</td>
</tr>
<tr>
<td>Breathing:</td>
<td>patient talking? - breath sounds equal bilat.? - bilat chest rise? - SpO2?</td>
</tr>
<tr>
<td>Circulation:</td>
<td>peripheral pulses present? - BP, listen for heart sounds - fix large active hemorrhage (life threatening), pelvis, long bones (reduce and splint)</td>
</tr>
<tr>
<td>Disability:</td>
<td>GCS, pupils (unequal? think uncal herniation) - check gross sensation / motor x 4 - lateralizing signs?</td>
</tr>
<tr>
<td>Exposure:</td>
<td>expose patient completely then re-cover (hypothermia) - bleeding? fractures? deformities? - log roll now but only if will change management (i.e. penetrating trauma) - now off the spine board</td>
</tr>
</tbody>
</table>

**ATLS Cheatsheet — 2/2**

<table>
<thead>
<tr>
<th>Section</th>
<th>Details</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adjuncts to Primary Survey:</td>
<td>trauma labs, portable CXR, lateral C-spine, abdo, pelvis</td>
</tr>
<tr>
<td>Secondary Survey:</td>
<td>“AMPLE”: allergies, medications, past medical history, last meal, events leading up to now recheck vitals head to toe physical examination, dermatomal neuro exam if disability was abnormal further imaging as necessary tetanus status / Tdap ensure adequate analgesia pregnancy test in women non-stress test (NST) if pregnant WinRho if pregnant and appropriate</td>
</tr>
<tr>
<td>Finally, consider transfer</td>
<td>to tertiary and definitive care</td>
</tr>
</tbody>
</table>

**Reading suggestions for better clinicians**


**We as physicians are guilty of misusing p-values**

“A common misconception among nonstatisticians is that p-values can tell you the probability that a result occurred by chance. This interpretation is dead wrong, but you see it again and again and again and again and again.” — fivethirtyeight.com

**History & physical examination review**

- Evidence-Based Physical Diagnosis, 3e by Steven McGee is a superb, evidence-based approach to the physical examination skills.
- Excellent free video resource for physical examination skills: The Stanford 25
- UBC Family Medicine Abbotsford — OSCE – General Review

Have an “Extra” that you want to share with residents and physicians early-in-practice? Let us know at brady@drbouchard.ca.

**RECOMMENDED #FOAMED RESOURCES**

33. Contributors

The podcast and these study notes would not have been possible without the contributions of many; for their help we are very grateful. A list of all contributors who have chosen to be identified is included below:

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"The good physician treats the disease; the great physician treats the patient who has the disease."
—Sir William Osler