1 ESSENTIAL CLINICAL SKILLS

Staff Section Editors

Helly Goez MD FRCPC
Division of Pediatrics Neurology
Assistant Dean, Associate Professor, Diversity, Faculty of Medicine and Dentistry
Stollery Children Hospital, Glenrose Rehabilitation Hospital, Edmonton Clinic Healthy Academy

INTRODUCTION

In this section, you will find some of the basic skills required to be a clinical clerk or intern. Your role as a medical student will transition from preclinical to clinical years. As a student, your primary goal is to gain an understanding of normal human physiology and pathology, while your clerkship experience will require you to act as an information gatherer (in the form of a patient history and physical examination) and then as an interpreter, using your evolving knowledge of disease and treatment. At this juncture, a refresher on the basic skills will be useful to ensure your performance and value in the context of your clinical team.

In an OSCE or clinical scenario, you may be asked to interpret basic clinical laboratory tests or radiographic images to demonstrate your skill as a diagnostician. The following pages discuss those skills in further detail. Regardless of the test, do not forget the clinical context in which these tests are being interpreted. Carefully read the clinical vignette provided with the case and use it to corroborate your interpretation of the test. That is, do not let the pressure of limited time in the OSCE allow you to forget that there is an individual behind the numbers reported.

This section also contains information on the basic written clinical communication skills: prescriptions, progress notes, and orders. These written forms of medical communication are essential to the work that physicians do. On the wards, a clearly written and concise progress note will give your colleagues, residents, staff, consultants, and interdisciplinary team members the ability to follow a patient's progress during a hospital stay or over the course of multiple clinic visits. It will also give you the ability to track and quickly recall the details of a patient you are following. Having a systematic approach to constructing clearly written prescriptions and orders will ensure that the care of your patients is carried out and medication errors are reduced.

On the ward, in clinic, and in examination settings, medical students often are asked to provide an oral summary of a patient interaction. When a resident or staff physician asks you to present a case, organization is key to delivering a useful synopsis. Before beginning the presentation, take a few seconds to think about what it is that you want to convey. Consider your message. Implicitly painting a picture of the pertinent positives and negatives will help your audience understand the patient's presentation and help narrow the differential diagnosis. It also will demonstrate that you have paid careful attention to the possible differential. A clinical presentation does not need to include every last detail about the patient—it should simply provide sufficient information to make a decision on the current clinical presentation. As an analogy, think of your clinical presentation as an iceberg, of which only 30 percent is above the water's surface with the vast majority hidden below. Similarly, you should present only the key findings, while keeping in your own mental reserve the remaining 70 percent of detail in the event you are asked. Practicing your own oral presentation style and taking into account the type of feedback and questions that are asked are essential to building your confidence and skill. At the end of your presentation, be ready to discuss your differential diagnosis and steps that can be taken in the investigation, management, and disposition of the patient.

We hope that you find these essential clinical skills useful as you begin your clinical placements and prepare for OSCEs.

Sincerely,
Edmonton Manual Editorial Team

ADMISSION & DAILY ORDERS

Authors: Shawna Pandya MD, Brian Yong MD, Darren Nichols MD CCFP

Orders (DAD-DAVID)

- The traditional DAD-DAVID format, with some examples of common orders, is included below:

D-A-D

- Date/Time
- Admit to (ward/service)/Allergies
- Diagnosis

D

- Diet
  - Diet as tolerated (DAT)
  - NPO ± ice chips (e.g., pre-op, aspiration risk), clear fluids, full fluids, thickened fluids (dysphagia diet)
  - Advancing diet (NPO → sips → CF → FF → DAT)
  - Canadian Diabetes Association: diabetic diet (small-1600kcal, med-1800kcal, lrg-2000kcal)
  - Cardiac/heart healthy diet, Mediterranean diet
  - Fluid restrictions, salt restrictions
  - Consider Speech Language Pathologist consult for dysphagia, aspiration risk, swallowing study
  - Consider Dietary consult for content, consistency, and quality

A

- Activity
  - Activity as tolerated (AAT)
  - Ambulation orders; if necessary consult OT/PT
  - Up in chair for meals
  - Non-weight bearing (NWB), weight-bearing as tolerated (WBAT), full weight-bearing (FWB)
  - Bed rest (BR), bed rest with bathroom privileges (BRwBP)
  - Bed/chair alarms
  - Elevate head of bed (HOB) to 30 degrees
  - Precautions: fall, seizure, spinal, etc.
  - DVT prophylaxis, compression stockings

V

- Vital Signs
  - Clarify what routine VS are for each service, add on as necessary (i.e., HR, BP, RR, Temp, SaO2)
  - Include on-call orders: set parameters (e.g., call MD if sBP ≥180 mm Hg)
  - Specify frequency, e.g., q1h, q4h, close observation (q15min), nurse in room, constant security (psychiatry, geriatrics), routine/regular observation (pediatrics), when awake
  - Neurovitals (GCS and pupils)
  - Peripheral pulses/doppler (vascular patient)
  - Capillary glucose

I

- IV
  - Fluid, route, rate
  - Replace NG losses 1:1 with 1/2 NS + KCl 10 mEq/L
  - Pediatrics: consider Wt, age, deficit, maintenance, and ongoing losses
  - Parameters for locking IV when patient drinking well
  - Saline or heplock
  - Emphasize oral route when possible
- Ins/Outs
  - Ins/Outs with shift change (surgery, volume status patients, heart failure)
  - Foley catheter
  - Daily weights
- Infection Control
  - Droplet
  - Contact
  - Droplet and contact
  - Airborne
  - Significant organism

- Investigations
  - Blood – Hematology: CBC-D, reticulocyte count, PT/INR + PTT, crossmatch, type & screen
  - Blood – Biochemistry: liver function tests, liver enzymes (AST, ALT, ALP), urea, Cr, electrolytes (Na^+, K+, Cl-, HCO3-), glucose, Ca^{2+}, Mg^{2+}, PO4, TSH, Vit B12, folate, CK, CKMB
  - Blood – Culture: viral serology, culture
  - Urine: R&M, C&S
  - Imaging: X-ray, MRI, CT, bone scan
  - Other: ABGs, ECG, echo
  - Consults: Pharmacy, OT/PT, Dietician, Social Work, Pastoral Care, other specialties

D

- Drugs
  - Check allergies (include latex, iodine, and tape allergies)
  - Include drug name, dosage, route, frequency and stop date if applicable (i.e., ATBx, narcotics)
- 6Ps:
  - Pain (analgesia) – always include a Tylenol order unless contraindicated (maximum 4 g from all sources per day; max 2 g if hepatic dysfunction)
  - Puke (antiemetics) – always include an antiemetic order, especially if patient on narcotics
  - Poop (bowel care) – especially important if patient is on narcotics
  - Pillow (sedation) – sleeping pills
  - PE (DVT prophylaxis)
  - Previous home medications
- Drains
  - Foley (to urometer)
  - NG to low wall suction
  - G-tube or T-tube to straight drainage
  - Reprime JP/hemovac qshift and PRN
- Dressings
  - Change dressing QD, PRN; remove staples day x post-op

Signature

- Signature, printed name, training (e.g., SI-3, PGY-4, etc.), pager number

Note: Be sure to flag the patient's chart once orders are written. Any stat, important, or nuanced orders should be communicated to nursing colleagues.

APPROACH TO ACUTE & CHRONIC PAIN

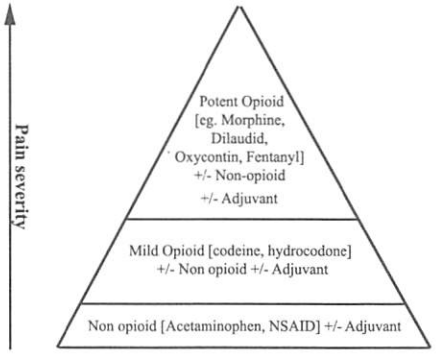
Current Editor: Hana Yu MSc MD

Background

- Pain is the most common reason for emergency department visits
- Pain is both a physical and psychological experience
- Chronic pain is a common and complex disorder often requiring multidisciplinary treatment
- Nociceptive pain: the activation of nociceptive pain pathways via tissue damage. Differentiate between somatic pain (sharp, stabbing, well localized, etc.) and visceral pain (dull, achy, poorly localized)
- Neuropathic pain: pain resulting from an abdominal neural activity. Patients may experience burning, tingling, shooting, or electric pain

History

- OPQRST pain history: Onset, Provoking or Palliating factors, Quality, Radiation, Severity, Treatment
- Are there associated symptoms such as fever/chills, cough, decreased range of motion in joints, paresthesias, etc.
- Assess how pain is affecting day to day function (work, activities of daily living, personal life, sleep)
- Past medical history and medication use (history of cancer, musculoskeletal injury, psychiatric diagnoses, peptic ulcer disease, chronic kidney disease, cardiovascular disease, anticoagulation)
- Social history (occupation, recreational drug use, social support system)
- The Visual Analog Scale (VAS) is a standardized means of assessing pain severity. Clinically, patients can also rate pain severity between 1-10 (Verbal Numeric Rating Scale) or use the Wong-Baker FACES pain scale, which is suitable for patients of a variety of ages, all cultures, and those with cognitive impairment.
- Address chronic pain and its management in the acute pain setting (e.g., post-operatively). It is recommended that patients continue their baseline analgesics even in the setting of new acute pain.
- Continually reassess the etiology of the pain in the setting of failing analgesia as medical comorbidities and surgical complications may be confounding variables.



Adapted from WHO Pain Ladder

Analgesic Ladder: initially developed by WHO to address cancer pain. It has now been adapted as a stepwise approach to the management of pain from any etiology
Multimodal Analgesia: involves the use of medications synergestic analgesic effects while minimizing the side effects of approaching the dose ceiling of individual

Acute Pain

- Definition: "the normal, predicted, physiological response to an adverse chemical, thermal, or mechanical stimulus" (Carr and Goudas, 1999
- Generally resolves within one month.
- Treatment – Effective pain control is achieved via multimodal analgesia and the analgesic ladder
- Multimodal Analgesia:
  - Involves the use of medications from different pharmacological categories for synergistic analgesic effects while minimizing the side effects of approaching the dose ceiling of individual medications
- Analgesic Ladder:
  - Initially developed by the World Health Organization to address cancer pain. Has now been adapted as a stepwise approach to the management of pain from any etiology
  - Start with non-opioid medications and then sequentially add increasingly potent medications until pain is relieved. Adjuvant therapy may be added at any point of treatment

Drug	Equi-analgesic dose (mg)	
	Parenteral	Oral
Morphine	10	30
Codeine	130	200
Fentanyl	0.1	-
Hydromorphone	1.5 - 2	6 - 7.5
Methadone	10	20

From Miller, Eriksson, Fleisher, Wiener-Kronish & Young, 2010, p. 2774

Breakthrough Pain (BTP)

- Definition: a transient exacerbation of pain that occurs in the context of otherwise stable baseline pain
  - When treating BTP pharmacologically, one must first ensure that baseline pain is being treated effectively with appropriate doses of around-the-clock (ATC) analgesics. This results in smoother pain control and decreased need to "play catch up" with PRN medications.
  - Regular reassessment of baseline pain is recommended. Should persistent BTP be occurring before scheduled doses of ATC analgesic, consider either increasing the total daily dose of ATC medication by 25-50% or shortening the dosing interval if the patient is already at the maximum tolerable daily dose (i.e., divide the same dose of daily medication such that it is administered at shorter intervals throughout the day).
  - If the patient is utilizing several PRN doses in addition to ATC medication, adjust the total daily ATC dose. This is done by taking the daily sum of the PRN doses, converting it into the dose equivalency of the ATC medication, and then dividing it equally to be added to the ATC medication. A new PRN dose can be added, which is usually 5-10% of the new total daily ATC dose.

Chronic Pain: pain persisting for 3 months or longer

- Treatment:
  - Multidisciplinary approach is key
  - Treatment options in chronic pain generally include pharmacological, behavioral medicine, physical medicine, neuromodulation, and surgical approaches
  - Generally, the initial pharmacological therapy is targeted depending on whether the pain is nociceptive or neuropathic

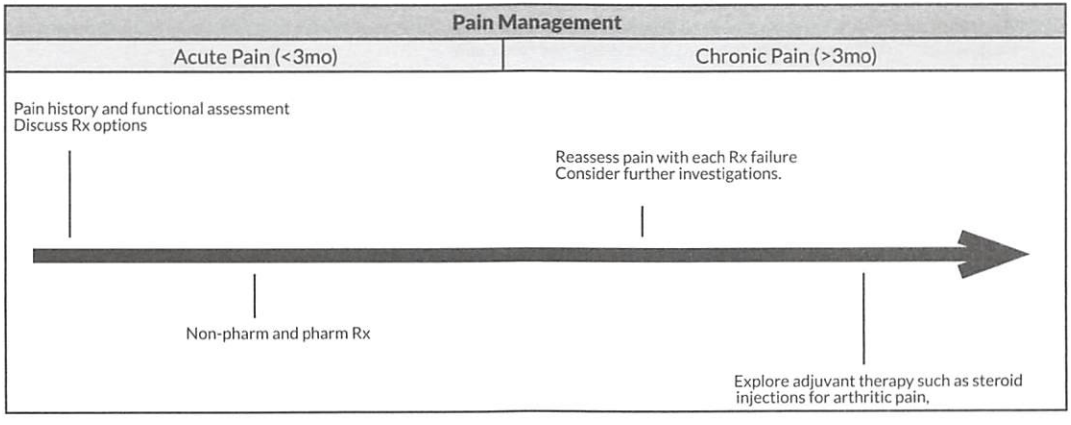
Nociceptive Pain

- Medication:
  - Similar medications as neuropathic pain; however ,first line therapy includes both non-opioid analgesics (e.g., acetaminophen, NSAIDs) and, in some cases, extended release opioids in addition to attempts to relieve source of pain (see pain ladder)
- Non-pharmacological:
  - Cognitive Behavioral Therapy (CBT), relaxation therapy, meditation, aerobic exercise, acupuncture
  - Physical Therapy, Occupational Therapy
  - Thermal compresses

Neuropathic Pain

- Initially try to establish etiology of chronic pain and target therapy to the cause
- Medication:
  - First line therapy includes: TCA Antidepressants, Gabapentin, and Pregabalin
  - SSRIs
  - Topical therapy (e.g., topical lidocaine, capsaicin cream) may be used in conjunction
  - Opioids are not considered first line therapy as they are not as efficacious in neuropathic pain yet carry many side effects. May sometimes be used for BTP.
  - Surgical intervention in the form of spinal cord stimulators or transcutaneous electrical nerve stimulation (TENS) may be an option for certain patients with refractory, severe neuropathic pain

Many patients will benefit from referral to chronic pain or palliative care clinics to ensure continuity and close monitoring.
*Always remember to manage the side effects of the medications being used (i.e., oversedation, nausea, and constipation with opiates and the need for gut protection in the form of Proton Pump Inhibitors when using NSAIDs).



ESSENTIAL DERMATOLOGY

Current Editor: Yan Fei Chen

Common Conditions

- Acne vulgaris, atopic eczema, psoriasis vulgaris, actinic keratosis, seborrheic keratosis, basal cell carcinoma, androgenic alopecia, vitiligo, melasma

High Mortality/Morbidity

- Melanoma, Stevens-Johnson syndrome, toxic epidermal necrolysis, necrotizing fasciitis, pemphigus vulgaris

HISTORY

ID	• Patient's name, age, gender, ethnicity, occupation (occupational exposures)
HPI	• Seven key questions: when, where (site of onset), symptoms (e.g., pruritic, painful, asymptomatic, systemic symptoms), how has it spread, have the lesions changed, provocative factors (heat, cold, sun, exercise, travel history, drug ingestion, pregnancy, season), which treatment(s) have been tried and which have worked
RED FLAGS	• Constitutional symptoms (malaise, unexplained fevers, weight loss, anorexia, night sweats, etc.) • Signs of Stevens-Johnson syndrome/toxic epidermal necrolysis: positive Nikolsky's sign (light lateral pressure causes the skin to form a bullae or slough off), painful rash with mucosal involvement • Pain out of proportion to physical exam findings (necrotizing fasciitis)
PMHX	• Previous skin cancers, shingles, psoriasis, thyroid disorders, DM, atopy (atopic dermatitis, allergies, allergic rhinitis)
PSHX	• Previous operations
PO&GHX	• STIs, pregnancy, sexual activity, HIV risk factors
MEDS	• Present and past medications (both topical and systemic), supplements
ALLERGIES	• Food, medication, environmental
FHX	• Psoriasis, atopy, skin cancer, genodermatoses (e.g., tuberous sclerosis, neurofibromatosis)
SOCIAL	• Sun/chemical exposure, smoking, EtOH, IVDU, pets, travel history, hobbies, sick contacts
ROS	• Done as indicated by the clinical situation with particular attention paid to possible connections between signs and disease of other organ systems (e.g., a patient with a lesion suspicious for melanoma would require a complete ROS looking for any sign of organ dysfunction suggesting metastases) • Myalgia, arthralgia, fever, oral ulcers, Raynaud's phenomenon • Remember to ask about hair and nail changes as many conditions that affect the skin also affect hair and nails (e.g., psoriasis)

PHYSICAL

Define lesions using SCALD

- Size of lesion
- Color: erythematous, violaceous, hyperpigmentation, hypopigmentation
- Arrangement: grouped (clustered lesions), serpiginous (wavy or serpent-like appearance), reticular (net-like arrangement), target (looks like a bull's eye – central erythema surrounded by pale edema and peripheral erythema), discoid (resembles a disc)
- Lesion morphology: primary morphology (see Box 2) and, if applicable, the secondary morphology (see Box 3)
- Distribution: diffuse (e.g., viral rashes and drug reactions), extensor (e.g., psoriasis), flexural (e.g., atopic dermatitis), dermatomal (e.g., shingles), symmetric, photodistributed (areas exposed to the sun)
- In addition
  - Assess suspicious lesions for malignancy (Box 1)
  - Examine the hair, scalp, and nails (Box 3)
  - Look for other systemic features common in autoimmune and infectious disorders: arthralgias (psoriatic arthritis, Reiter's syndrome or Lupus), fever (viral), oral ulcers, sores on palms and feet (syphilis), malar rash (SLE), Gottron's papules and heliotrope rash around eyes (dermatomyositis)

BOX 1: Signs of Melanoma
Asymmetrical skin lesion
Border irregularity
Color variation
Diameter (new lesion >6mm)
Evolution (changes in size, color or bleeding)

BOX 2: Primary Lesions

- **Macule**: flat, non-palpable lesion, <1 cm
- **Patch**: flat, non-palpable lesion, >1 cm (e.g., vitiligo, café au lait spot)
- **Papule**: palpable lesion, elevated above the skin, <1 cm (e.g., molluscum contagiosum, acne vulgaris)
- **Plaque**: palpable lesion, elevated above the skin, >1 cm (e.g., psoriasis)
- **Vesicle**: <1 cm blister (e.g., varicella, contact dermatitis)
- **Bulla**: >1 cm blister (e.g., bullous pemphigoid)
- **Pustule**: superficial cavity of the skin that contains a purulent exudate (e.g., folliculitis)
- **Nodule**: <1 cm deep palpable solid lesion within the skin; depth and size differentiate a nodule from a papule; often better felt than seen (e.g., lipoma)
- **Tumor**: >1 cm nodule
- **Cyst**: cavity containing fluid or semi-solid (e.g., pilar cyst)
- **Wheal**: rounded or flat-topped papule or plaque that is evanescent due to edema of the dermis (e.g., hives, angioedema)

BOX 3: Secondary Lesions

- **Scales**: excess keratin (e.g., psoriasis)
- **Crusts**: dried serum, scab (e.g., impetigo)
- **Erosion**: loss of epidermis, heal without scarring (e.g., dermatophyte infection)
- **Ulcer**: loss of epidermis and dermis, heal with scarring (e.g., stasis ulcer)
- **Fissure**: linear loss of epidermis and dermis
- **Atrophy**: thinning of epidermis or dermis causing depression (e.g., morphea)
- **Scarring**: abnormal formation of connective tissue after dermal injury (e.g., keloid)
- **Special Lesions:**
  - **Excoriation**: scratch mark; if lesions occur at site of scratching it is called Koebner's phenomenon
  - **Comedone**: hair follicle plugged with sebaceous and keratinous material (e.g., acne)
  - **Petechiae**: <0.5 cm deposits of extravasated red blood cells (RBC) suggestive of vasculitis
  - **Purpura**: >0.5 cm petechiae (e.g., senile traumatic purpura)
  - **Telangiectasias**: dilated superficial blood vessels (e.g., rosacea, basal cell carcinoma, CREST syndrome)

INVESTIGATIONS (BASED ON CLINICAL SUSPICION)

Investigations

- CBC, ESR, CRP; ANA if suspicious of connective tissue disease
- TSH, fasting glucose, blood culture; LFTs and organ specific labs if suspicious of other systemic disease
- Imaging as relevant for systemic disease or melanoma staging
- CXR (heart or lung pathology)
- Skin scraping for fungal KOH test
- Wood's lamp for depigmented lesions
- Surgical/Diagnostic Interventions
- Biopsy: shave (epidermal), punch (extends into dermis and subcutaneous tissue), or excisional
- Biopsy is indicated in lesions suspected of neoplastic, bullous disorders or unclear diagnosis with clincial exam alone

BOX 4: Hair, Scalp, and Nail Examination

- **Hair and Scalp:** texture, scars, thinning, absence (alopecia) or excess (hypertrichosis), infestations (lice), masses (on scalp), plaques, crusting
- **Nails:** clubbing, thickness, pitting (psoriasis), separation from nail bed (onycholysis), yellow discoloration of nail bed (oil drop sign for psoriasis), other discoloration, periungual erythema, splinter hemorrhages (endocarditis)

TREATMENT

Emergent

- Stop offending agent if drug reaction (e.g., Stevens-Johnson syndrome/toxic epidermal necrolysis)
- Start antimicrobials for infection (e.g., cellulitis) or immunosuppressive agent for immune mediated disease

Treatment Options

- Topical: steroids, antibiotics, antifungals, emollients, retinoids, etc.
  - steroid strength depends on dosage and vehicle
  - steroid examples: weak (hydrocortisone acetate 0.1% cream), moderate (mometasone furoate 0.1% cream), strong (clobetasol 0.05% ointment)
- Systemic medications: immunosuppressives (oral/IV steroids, methotrexate), retinoids, ATBx, antimalarials
- Light: narrow band ultraviolet B, ultraviolet A (PUVA), laser therapy
- Surgical: curettage, cryotherapy, electrotherapy, scalpel

Follow-up

- Monitor skin findings over time to evaluate progression, monitor for medication side effects

Referral as indicated

FLUID RESUSCITATION

Authors: Malgorzata Ejsmont MD, Adam Dryden MD, Timothy Yeh MD FRCPC

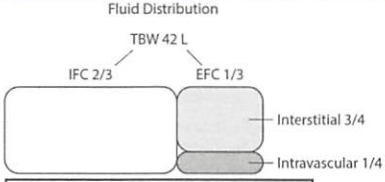
FLUID BALANCE

Basics

- Fluid makes up 50-60% total body weight (TBW)
- Water movement: mainly osmotic forces (Na, K)

Distribution (see figure)

- Note: Intravascular volume → ECF (plasma 3-3.5 L) + ICF (RBC 2-2.5 L) in 70kg male



Fluid Requirements

- Total: maintenance + replacement (existing and ongoing deficits)
- Maintenance requirements: ~2.5 L/day (4-2-1 Rule) (see table)

Maintenance Fluids	
Wt	mL/kg/hr
first 10 kg	4
next 10 kg	2
each kg > 20 kg	1

Compensation in Hypovolemic States

- Initially: sympathetic outflow → vasoconstriction, tachycardia
- Over hours: extravascular → intravascular shift

FLUID THERAPY (IV)

CRYSTALLOID	• Aqueous solution of salts ± glucose (see table below: Common Crystalloid Solutions) • Equilibrate in entire ECF: given as 3-4 x volume deficit • Initial resuscitation in hemorrhagic/septic shock, burns → maintain cerebral perfusion › Isotonic (replacement): if H2O and electrolyte deficit (distribute ECF) › Hypotonic: if free H2O deficit only (distribute ICF/ECF) › Hypertonic: in severe, symptomatic hyponatremia (shift ICF → ECF)
COLLOID	• Large molecules maintain plasma oncotic pressure • Mostly intravascular (altered if ↑ vascular permeability): given as 1:1 ratio of volume deficit • Use: severe intravascular deficits prior to arrival PRBC, large protein loss (e.g., burns) • Caution: prepped in NS (hyperchloremic acidosis), allergic reactions, antiplatelet effects, possible renal injury from excessive colloid use › Blood derived: albumin (5% and 25%), plasma protein fraction (5%) › Synthetic: Dextran™ (40 and 70), Hetastarch™ (6%), Pentaspan™ (pentastarch), Voluven™ (hydroxyethyl starch)
BLOOD	• PRBC: compensate for anemia, not volume deficit • Give when anemia risks > risks of transfusion (Hgb 70-80 g/L) • 1 unit: Hgb ↑ 10 g/L, warm to >37° if need >1-2 units

COMMON CRYSTALLOID SOLUTIONS

Solution	Tonicity (mOsm/L)	pH	Na	Cl	K	HCO3	Ca	Mg	Glucose (g/L)	Caution
			(mEq/L)							
Intravascular	290	7.4	140	105	4	24	2.4	2	0.7-1.1	
NS (0.9%)	308	5.5	154	154	-	-	-	-	-	hyperchloremic acidosis
Ringer's	273	6.5	130	109	4	28*	3	-	-	lactic acidosis
Plasmalyte	294	7.4	140	98	5	-	-	3	-	
D5W	253	5.0	-	-	-	-	-	-	50	hyperglycemia
1/2 NS	154	5.5	77	77	-	-	-	-	-	hyponatremia
3% Saline	1026	5.0	513	513	-	-	-	-	-	hypernatremia, hemolysis

*converted from lactate

Crystalloid vs. Colloid

- Extensive meta-analysis found no difference in survival between resuscitation with colloid vs. crystalloid
- Cost differential guides recommendations in favor of crystalloids (controversial in some cases)

FLUID THERAPY (ORAL)

- Preferred in pediatric patients with mild-moderate dehydration (equivalent to IV therapy)
- Maintenance solutions (Pedialyte™, Ricelyte™): Na 45-50 mEq/L, glucose 2-3 %
- Rehydration solutions (WHO formulation, Rehydralite): ↑ Na (60-90 mEq/L) → improve water absorption
- Administer slowly to decrease emesis: mild (50 ml/kg over 4 hrs), moderate (100 ml/kg over 4 hrs)

ASSESSMENT AND TREATMENT

Initial Assessment: ABCs, VS (BP, HR, RR, Temp, SaO2), IV, monitor, SAMPLE Hx

- Assessment of severity (see table)
  - › Consider DDx (see *Trauma* [in Surgery] and *Shock* [in Internal Medicine])
- Fluid resuscitation goals: restore vital organ perfusion, maintain adequate oxygen delivery, limit ongoing loss of RBC

HEMORRHAGIC SHOCK CLASSIFICATION							
Class	Blood Loss (mL)	Blood Loss (% blood vol)	HR	BP	RR	U/O (mL/h)	Mental Status
I	<750	<15%	N	N	N	N	slightly anxious
II	750-1500	15-30%	>100	N	20-30	20-30	mildly anxious
III	1500-2000	30-40%	>120	↓	30-40	5-15	confused, anxious
IV	>2000	>40%	>140	↓	>35	Anuric	confused, lethargic

IV Access

- 2 large bore IVs (16/18) in peripheral veins → investigations, type & cross match (if necessary)
- If severe shock or peripheral IV not achieved → large catheter introducer (8-9 Fr) in internal jugular/femoral vein
- If major vascular injury in abdomen/pelvis → establish vascular access above diaphragm (subclavian/jugular)

Hemodynamic Assessment

- Continuous monitoring of VS (BP, HR, RR, Temp, SaO2), ECG
- Frequent reassessment for signs of decreased perfusion: mental status, capillary refill, temperature of extremities
- Other: urine catheter (end organ perfusion), arterial line (serial ABGs, normalization of lactate), central venous pressure, central venous oxygen saturation (ScVO2 – O2 extraction, global perfusion)

IV Therapy (also see *Pediatric Emergency* [in Pediatrics])

- Isotonic crystalloids: (RL or NS) x 2-3 L wide open (adult)
  - › Hemodynamically unstable: suggests >15-20% blood volume lost or significant ongoing loss →transfusion
  - › Adequate response → continue crystalloid, monitor hemodilution
- Blood transfusion
  - › If not typed/cross-matched → transfuse O+ (male), O- (female)
  - › Severe hemorrhagic shock → transfuse initially
  - › Bleed controlled → target Hgb >70 g/L
- Hemorrhage, large volume resuscitated: dilutional coagulopathy
  - › Massive transfusion protocol: 6u PRBC, 6u FFP, 1 pool PLTs

Referrals

- Surgery (control bleed), ICU if need for vasoactive drugs to support arterial pressure or cardiac output, circulatory instability unresponsive to volume replacement, decreased LOC, need for invasive monitoring, etc.

INTERPRETATION OF ABDOMINAL RADIOGRAPH

Authors: Babak Maghdoori MD, Christopher Fung MD, Ed Wiebe MD FRCPC

HISTORY

Determine the following prior to acquiring/reading the radiograph:

- Patient's name, age, gender, and date of image
- HPI: acute/chronic, level of progression of the disease
- Availability of previous imaging
- Determine the type of the image (supine/prone/decubitus)
- Ensure completeness of the abdominal film series (i.e., "the 3 views": supine AXR, erect AXR, and a CXR)
  - Note: if patient is unable to stand for an upright abdominal radiograph, acquire a left lateral decubitus AXR

INTERPRETATION

Basic approach: ABCD or GreatBigFART

- Air (free air/gas pattern)
- Bones (fractures, metastatic disease)
- Calcifications (GU stones, gallstones, lymph nodes, calcified AAA wall)
- Density (soft tissue structures)

Great – gas pattern
Big – bowel wall air
F – free air
A – air fluid levels
R – air in rectum
T – thickened bowel wall

Technical Quality

- Level of penetration
  - Normal penetration: vertebral columns (lumbar and thoracic) can be seen clearly
  - Under-penetration: AXR is too white
  - Over-penetration: AXR is too dark
- Inclusion: ensure that the entire abdomen (diaphragm to the proximal femoral head) is included in the study

Foreign/Therapeutic Objects

- Comment on any lines, iatrogenic devices, tubes present (e.g., foley, NG tube, ECG leads), and/or swallowed/inserted items

Gas Patterns

- Normal: gas present in the stomach and a few loops of transverse colon/sigmoid, colon/rectum
  - In a healthy, ambulatory adult the small bowel usually has very little or no air unless the patient has recently eaten
- Abnormal: multiple loops of small bowel and/or large bowel filled with gas (supine); multiple air fluid levels and/or a paucity of gas in the sigmoid colon/rectum (upright AXR); bubbles of air in bowel wall (pneumatosis)
  - DDx for abnormal bowel gas pattern: ileus or mechanical obstruction
    - Ileus: typically seen in post-operative patients (gas is present in sigmoid colon/rectum)
    - Mechanical obstruction: sick patients (no gas in the sigmoid colon/rectum, unless distal obstruction)
  - DDx for small bowel obstruction: adhesions, hernia, tumor
  - DDx for large bowel obstruction: tumor, diverticulitis, volvulus (sigmoid or cecum)
- Extraluminal air:
  - Rigler/double wall sign: sign of pneumoperitoneum with air outlining both sides of the bowel wall
  - Evaluate underneath the hemidiaphragms for possible free air
  - Pathologically significant unless recent intra-abdominal surgery (up to 5-7 days post op)
  - DDx: perforated ulcer, diverticulitis, perforated ischemic bowel, etc.

Bowel

- Carefully look for bowel wall thickening and narrowing of the lumen, as well as air in bowel wall
  - Small bowel diameter <3.5 cm; large bowel diameter <6 cm (variable); cecal diameter <9 cm

Bones

- Begin with the spine, then study the ribs, followed by the pelvis, and finally the upper femurs
- Determine the proper alignment of the vertebral bodies, pedicles, and spinal/transverse processes
- Evaluate for signs of osteoarthritis, scoliosis, and other degenerative disease in the vertebral column
- Overall, carefully examine the bones for any fractures, lytic/blastic lesions, and/or metastatic disease
  - Note: bowel gas patterns, in particular around the pelvis, may closely resemble lytic patterns

Calcifications and Abnormal Densities

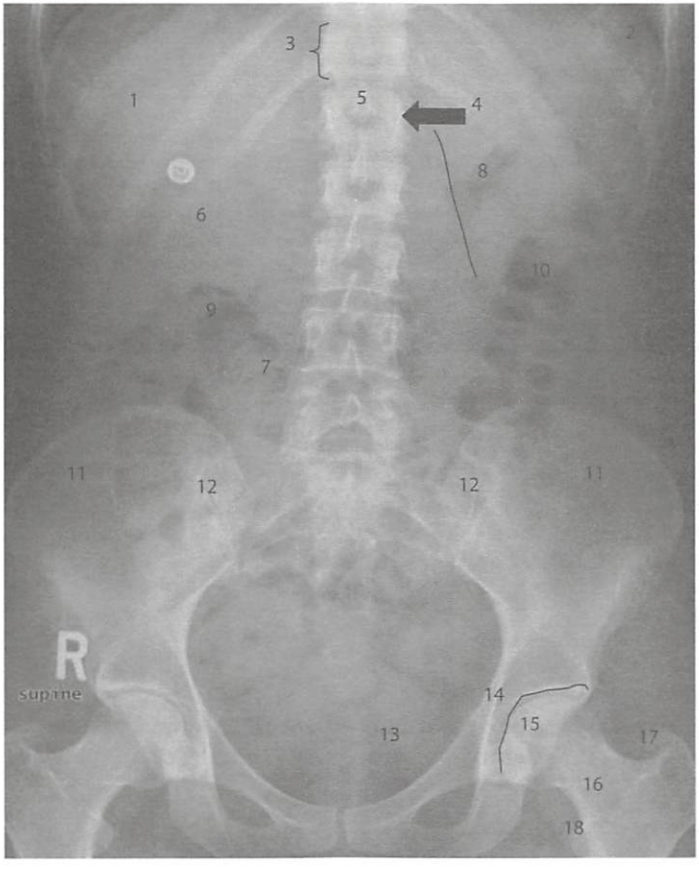
- DDx: GU stones, gallstones, chronic pancreatitis, calcified lymph nodes, fibroid tumors, calcified arteries, calcified wall of aortic aneurysm, etc. Note that phleboliths are commonly seen and generally not clinically significant.

Free Fluids

- Free fluid may be due to an exudative, transudative, or bleeding process
- The distance between flank fat pads and the colon may be used as a diagnostic measure
  - If the distance >1cm then increased possibility of free fluid in the paracolic gutters

Organs/Soft Tissues

- Try to visualize liver, stomach, spleen, kidneys, bladder, psoas margins
- Plain X-ray films are generally not used to examine subtle changes in organs. For instance, the liver's radiological shadow is very misleading as an index of its size, unless there is extreme hepatomegaly
- Useful for obvious changes such as masses, presence/absence of organs, calcifications/calculi, etc.



Normal Supine Abdominal X-ray Legend	
1. Liver	10. Gas in splenic flexure
2. Spleen	11. Ilium
3. T12 vertebral body	12. Sacroiliac joint
4. Pedicle	13. Bladder
5. Spinous process	14. Acetabulum
6. Right kidney	15. Femoral head
7. Right psoas margin	16. Femoral neck
8. Gas in stomach	17. Greater trochanter
9. Gas in hepatic flexure	18. Lesser trochanter

INTERPRETATION OF ABG

Current Editor: Hana Yu MSc MD

BASICS

- Patient ID/date of test
- Patient demographics: age, gender, ethnicity
- Pre-test data: determine if the test was performed with patient on room air or O2 flow
- Compare: previous ABGs if available

DIFFERENTIAL DIAGNOSIS

SELECTED CAUSES OF ACID-BASE DISORDERS				
Respiratory Acidosis	**Respiratory Alkalosis**	**Wide Anion Gap Metabolic Acidosis**	**Normal Anion Gap Metabolic Acidosis**	**Metabolic Alkalosis**
• Respiratory center depression • Neuromuscular disorders • Airway obstruction • Lung parenchymal disease • Mechanical hypoventilation	• Acute/chronic hypoxemia • Respiratory center stimulation • Mechanical hyperventilation	• **M** – methanol • **U** – uremia • **D** – DKA • **P** – paraldehyde • **I** – isopropyl alcohol • **L** – lactic acidosis • **E** – ethylene glycol • **S** – salicylates	• **H** – hyperalimentation • **A** – acetazolamide • **R** – renal tubular acidosis • **D** – diarrhea • **U** – ureteric shunt • **P** – post-hypocapnea • **S** – spironolactone	• Exogenous alkalis • Diuretics • Post-hypercapnea • Mineralocorticoid effect • Hypercalcemia • Vomiting • Volume contraction

BASICS OF INTERPRETATION

What is the clinical context?

- Past medical history: CHF, COPD, renal disease, acute overdose, etc.
- Stable or unstable condition
- Is this patient being ventilated? If so, how is O2 being delivered?

How is the patient ventilating? (PaCO2)

- PaCO2 <35 mmHg then suggests hyperventilation
- PaCO2 >45 mmHg then suggests hypoventilation

What is the patient's arterial oxygenation? (PaO2)

- Normal PaO2 is 80-100 mmHg depending on age (PaO2 ↓ with age)
- Causes of low PaO2 include
  - › High alveolar PaCO2 from hypoventilation
  - › ↓ atmospheric O2 content and/or atmospheric pressure
  - › Ventilation/perfusion mismatch

Are the lungs working as normal oxygenators? (A-aDO2 or PaO2/PAO2)

- A-aDO2 determined by PAO2 – PaO2 (normal <15 mmHg)
  - Normal Aa gradient is age dependent. Expect Aa gradient = Age/4 + 4
- PAO2 is calculated, PaO2 is measured
  - › Alveolar air equation: PAO2 = [FIO2(PB – 47)] – [PaCO2/0.8]
    - FIO2 = fraction of inspired O2
    - PB = barometric pressure (~760 mmHg at sea level)
- Consider A-aDO2 when patient is on room air
- Consider PaO2/PAO2 when patient is on supplemental O2
  - › Hypoxemia with normal A-aDO2 → suggests hypoventilation or high altitude (↓ atmospheric pO2)
  - › Hypoxemia with ↑ A-aDO2 or ↓ PaO2/PAO2 → suggests ventilation-perfusion mismatch, shunt, or low DLCO

CLINICAL APPROACH TO INTERPRETATION

Does the patient have acidosis or alkalosis?

- pH <7.35 = acidosis
- pH >7.45 = alkalosis

Is it a primary respiratory (PaCO2) or metabolic (HCO3) problem?

Is it acute, partially compensated, or fully compensated? (Base-excess and HCO3)

Summary of ABG Findings in Acid-Base Disorders		Respiratory Acidosis	Respiratory Alkalosis	Metabolic Acidosis	Metabolic Alkalosis	Respiratory Acidosis + Metabolic Acidosis	Respiratory Alkalosis + Metabolic Alkalosis
Acute	PaCO2	↑↑	↓↓	Normal	Normal	↑↑	↓↓
	pH	↓	↑↑	↓↓	↑↑	↓↓	↑↑
	Base Excess	0	0	Negative	Positive	Negative	Positive
Partially Compensated	PaCO2	↑↑	↓↓	↓	↑	-	-
	pH	↓	↑	↓	↑	-	-
	Base Excess	Positive	Negative	Negative	Positive	-	-
Fully Compensated	PaCO2	↑↑	↓↓	↓↓	↑↑	-	-
	pH	Normal	Normal	Normal	Normal	-	-
	Base Excess	Positive	Negative	Negative	Positive	-	-

COMPENSATION RULES

Acid/Base Disorder	Compensation Rule
Acute respiratory acidosis	↑PCO2: ↑HCO3 = 10:1
Chronic respiratory acidosis	↑PCO2: ↑HCO3 = 10:4
Acute respiratory alkalosis	↓PCO2: ↓HCO3 = 10:2
Chronic respiratory alkalosis	↓PCO2: ↓HCO3 = 10:5
Metabolic acidosis	Expected PCO2 = 1.5(HCO3) + 8 ±2
Metabolic alkalosis	Expected PCO2 = 0.7(HCO3) + 20 ±5

INVESTIGATIONS

Blood Work (if not already obtained from ABG)

- Hgb, electrolytes, glucose, urea

Anion Gap

- Anion gap = ([Na+]) – ([HCO3-] + [Cl-])
- Normal anion gap is 10-14 mEq/L
- Anion gap is dependent on albumin and serum phosphate
  - Normal Anion Gap = 0.2 x (albumin (g/L)) + 1.5 x (phosphate (mmol/L))
  - Most of the time we can estimate expected anion gap as ~Albumin (g/L)/4, ignoring phosphate
- Increased anion gap suggests an increased number of unmeasured anions

Osmolar Gap

- Osmolar gap = measured osmolality – calculated osmolality
- Calculated osmolality = 2[Na+] + [urea] + [glucose]
- Normal osmolar gap <10 mmol/L
- Osmolar gap >10 mmol/L suggests potential toxic osmole ingestion

Note: The approach to interpretation presented here focuses on a single acid-base disorder. Real-world clinical scenarios often involve multiple acid-base disorders and should be interpreted in the context of clinical information.

INTERPRETATION OF CHEST RADIOGRAPH

Current Editor: Edwin Cheng MD

HISTORY AND CONTEXT

Determine the following prior to acquiring/reading the radiograph

- Patient's name, age, gender, date of image, and previous imaging available for comparison
- HPI: acute/chronic, reason for chest x-ray, clinical question
- Determine the projection and patient positioning for the image (e.g., supine/upright/PA/AP/lordotic/decubitus)
  - Normally, chest x-rays are done in PA and lateral views
  - AP view for bedridden patients (portable X-ray), lordotic view to image lung apices, decubitus to assess loculation of effusion

INTERPRETATION

Technical quality

- Level of penetration and exposure
  - Normal penetration: intervertebral discs and vascular lung markings behind L heart are clearly visible
  - Under-penetration (CXR: too white): overaccentuation of lung vascular markings; may be mistaken for pulomonary edema
  - Over-penetration (CXR : too dark): difficult to discern vascular markings; false detection of pneumothorax or emphysema
- Adequacy
  - Inclusion: ensure lung fields are complete – the apices to the costophrenic angles should be imaged
  - Symmetry/Rotation: space between medial aspect of the clavicle and midline spinous process should be equal
  - Inspiration: in adult patients, 9-10 posterior ribs or 6 anterior ribs visible above diaphragm

Inspection (systematically work from inside to outside)

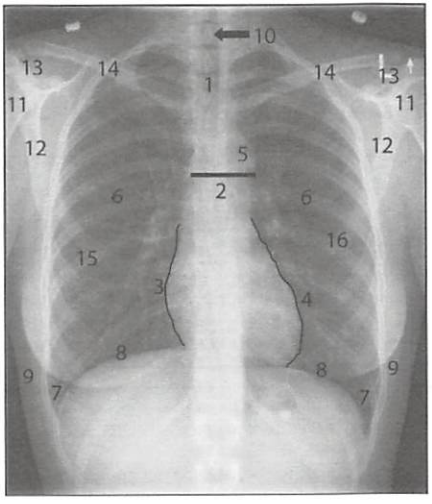
- Comment on any lines, iatrogenic devices, and/or tubes present (central line, chest tube, pacemaker, NG tube, ECG leads, etc.)
- Trachea
  - Check position: if shifted from midline, consider tension pneumothorax or mediastinal mass
- Mediastinum
  - Width >8 cm PA CXR → "widened mediastinum" DDx: aortic dissection, aortic aneurysm, mediastinal mass
- Hila/Lymph nodes
  - Assess for lymphadenopathy
  - If thickness of superior vessels > inferior vessels → vascular redistribution (DDx: edema, effusion, CHF, etc.)
- Heart
  - Determine cardiothoracic ratio: lateral width of heart relative to thoracic cage in PA view <50% normal
  - If enlarged (>50%), possible DDx: cardiomegaly, pericardial effusion
  - Visualization of right and left heart border (see "Pneumonia" on following page)
- Great vessels
  - Assess pulmonary trunk, aortic arch, and descending thoracic aorta for any enlargement and/or calcification
- Lungs
  - Ensure examination of the entire lung field, side-to-side comparison
  - Airspace disease: DDx: pneumonia (pus), pulmonary edema (fluid), hemorrhage (blood), tumor
  - Interstitial disease (lung markings thicker/more prominent): DDx: pulmonary edema, viral pneumonia, inflammation
  - Obvious masses: consider lung cancer, tuberculosis, pulmonary nodules
- Pleura
  - Blunting of costophrenic angles, suspect pleural effusion
  - Look for any abnormalities in the diaphragm (e.g., excessive elevation)
  - Pleural calcifications/thickening, pleural-based mass and calcified plural plaques often 2° to asbestos exposure
- Diaphragm
  - Right hemidiaphragm often slightly higher than the left 2° to the liver
  - Check for free air under the diaphragm on upright projection for pneumoperitoneum
- Bones and soft tissues
  - Examine bones: look for fractures, lytic/blastic bone processes, or any distortion of normal bone contour
  - Cervical and thoracic spine: look for the contour and height of the spinous processes and pedicles
  - Examine soft tissues: breast tissues, axillae, subclavicular areas, etc. Check for subcutaneous emphysema, axillary masses, surgical staples.
  - Bilateral breast shadows: axillary staples ± ipsilateral loss of a breast shadow

LATERAL VIEW

- Examine anatomy as described above, focusing on spine: assess the contour and height of the vertebral bodies
- Follow peripheral parenchyma along spine superior to inferior: lung fields should darken inferiorly. Whitening of lung fields inferiorly (the "spine sign") suggests pathology (e.g., lower lobe pneumonia).
- Assess the hemidiaphragms for evidence of hyperinflation (flattening)
- Confirm pathology seen on PA, view and determine posterior-anterior location

COMMON RADIOGRAPHIC FINDINGS ON A CXR

- Pulmonary edema
  - Vascular redistribution to the upper lobes, interstitial edema (thickening of interstitial markings → Kerley B lines, peribronchial cuffing, visualization of fissures), alveolar edema, pleural effusions, cardiomegaly
  - DDx: CHF, renal failure
- Pneumonia (silhouette sign)
  - R hemidiaphragm not visualized = RLL pathology; R heart border not visualized = RML pathology
  - L hemidiaphragm not visualized = LLL pathology; L heart border not visualized = Lingular pathology
- COPD
  - Frontal: hyperinflated lungs (>10 posterior ribs visualized), flattened hemidiaphragms, bullae
  - Lateral: increased retrosternal air space and flattened hemidiaphragms
  - In COPD, increased risk of apical pneumothorax



Normal Chest X-Ray Legend
1. Trachea
2. Mediastinum
3. Right heart border
4. Left heart border
5. Aortic arch
6. Lung fields
7. Costophrenic angles
8. Hemidiaphragms
9. Breast shadows
10. Spinous process
11. Humeral head
12. Scapula
13. Acromioclavicular joint
14. Clavicle
15. 8th posterior rib
16. 4th anterior rib

INTERPRETATION OF CBC-D

Current Editor: Bradley Brochu MD

HISTORY

- Infection (fever, chills, cough, diarrhea, dysuria, headache, skin infection) & inflammation (joint redness/swelling and rash)
- Malignancy (constitutional symptoms: unintended wt loss, unexplained fevers and night sweats)
- Anemia (SOB, presyncope, sources of blood loss)
- Easy bruising or bleeding
- Identify those patients in need of urgent care [e.g., shock (i.e., tachycardia, hypotension, poor peripheral vascular perfusion), respiratory distress, cardiac ischemia/infarction, and decreased LOC]

PHYSICAL

- Inspection: pallor, jaundice, petechiae, purpura, ecchymosis, joint redness/swelling, skin rash, local signs of infection
- Palpation: lymph nodes, liver, spleen; digital rectal exam is contraindicated if neutrophil count <1.0, inspect the area only
- Percussion: Castell's sign/Traube's space (splenomegaly), liver margin
- Auscultation: flow murmur (high cardiac output in anemia, fever)

Red Flags
• Combined abnormalities including pancytopenia (anemia, thrombocytopenia, and neutropenia) • Fatigue, recurrent infections, abnormal bleeding, Wt loss, night sweats

RED BLOOD CELL INDICES

- Red Blood Cell Count (RBC): number of RBCs per volume of blood; normal values: 4.4-5.7 X 10^{12}/L (♂), 4.0-5.2 X 10^{12}/L (♀)
- Hemoglobin (Hgb): amount of oxygen carrying protein in blood; normal values: 130–175 g/L (♂), 120-160 g/L (♀)
- Hematocrit (Hct): % of whole blood volume occupied by packed RBCs, Hct = RBC x MCV; normal values: 41-52% (♂), 36-46% (♀)
- Mean Corpuscular Volume (MCV): average size of RBCs; normal values: 80-100 fL
- Mean Corpuscular Hemoglobin Concentration (MCHC): average concentration of Hgb inside RBCs, MCHC = Hgb/(MCV x RBC), normal values: 32-36%. Increased: spherocytosis, autoagglutination, lipemia
- Red Blood Cell Distribution Width (RDW): normal RDW is <15%; increased indicates red cell size and/or shape variability and is most commonly related to iron deficiency

POLYCYTHEMIA	
Primary	• Polycythemia vera
Secondary	• Hemoconcentration (dehydration, burns, V/D), response to hypoxia (sleep apnea, COPD and other cardiopulmonary disease, smoking, CO poisoning, renal artery stenosis, high oxygen-affinity hemoglobinopathy), and autonomous EPO secreting conditions and tumors

ANEMIA	
MICROCYTIC (MCV <80 FL)	• **TAILS:** Thalassemia, Anemia of chronic disease, Iron deficiency, Lead intoxication, Sideroblastic anemia • Note: elevated RDW suggests iron deficiency anemia
NORMOCYTIC (MCV 80-100 FL)	
• ↑ **reticulocytes (>2% of RBC)**	• Bone marrow response to the 4Hs (Hemorrhage, Hypoxia, Hematinics, and Hemolysis)
• ↓ **reticulocytes (<1% of RBC)**	• Bone marrow suppression or infiltration (severe nutritional deficiency, ACD, aplastic anemia, bone marrow failure from primary hematologic or metastatic malignancy)
MACROCYTIC (MCV >100 FL)	• Megaloblastic anemia (B12/folate deficiency), liver disease, EtOH abuse, myelodysplasia, thyroid dysfunction, reticulocytosis, anti-DNA and folate drugs, some cases of aplastic anemia

WHITE BLOOD CELL (WBC) COUNT

- Actual number of WBCs per volume of blood; normal values: 4-11 x 10^9/L
- Increased: infection, inflammation, tissue ischemic/infarction, corticosteroids, general physiologic stress response, hematologic disorders (e.g., leukemia, lymphoma)
- Decreased: production (aplastic anemia, folate or B12 deficiency, drugs); increased breakdown/consumption (hypersplenism, sepsis, drugs)

NEUTROPHILS

Role in innate immunity, ANC; normal values: 1.8-7.5 x 10^9/L, ANC < 1.0 increases general infection risk

NEUTROPHILIA	• Infection (primarily bacterial), inflammatory disorders (gout, autoimmune, collagen vascular), drugs (steroids, lithium, catecholamines, G-CSF and GM-CSF), tissue ischemia and infarction (MI, stroke), smoking, pregnancy, post-splenectomy, metabolic (uremia, ketoacidosis), general physiologic stress response, leukemia and myeloproliferative disease, and hereditary (extremely rare)
NEUTROPENIA	
Increased Destruction	• Infection/sepsis, inflammation, autoimmune, drugs
Decreased Production	• Drugs, chemotherapy, bone marrow failure and/or infiltration (including aplastic anemia), cyclic, hereditary • Immunodeficiency
Sequestration	• Hypersplenism

LYMPHOCYTES

Role in adaptive immunity; normal values: 1-4.5 x 10^9/L; reactive lymphocytes in EBV infection

LYMPHOCYTOSIS	• Viral infection (infectious mononucleosis), pertussis, drugs, endocrine disorders (thyrotoxicosis, adrenal insufficiency), allergic reactions, autoimmune disease, lymphoproliferative disorders (CLL=classic), smoking, and transient stress lymphocytosis
LYMPHOPENIA	• Immune deficiency syndromes (including HIV), acute/chronic illness, immunosuppressive therapies (chemotherapeutic agents, radiation), bone marrow failure and malignancy, idiopathic

MONOCYTES

Role in innate immunity; normal values: 0-1.1 x 10^9/L

MONOCYTOSIS	• Chronic infection (such as TB, fungal, bacterial, viral, etc.), inflammatory (sarcoidosis, IBD, collagen vascular disease), neoplasms (hematologic and non-hematologic), post-splenectomy

EOSINOPHILS

Role in response to parasites (especially helminths) and allergic response; normal values: 0-0.7 x 10^9/L

EOSINOPHILIA	• Allergic/hypersensitivity reactions, drug reactions, parasite infestation, autoimmune and collagen vascular disease, cutaneous (pemphigus, eczema, atopic dermatitis), pulmonary (sarcoidosis, bronchiectasis, pneumonia, cystic fibrosis) and GI disorders (celiac disease, IBD), neoplasms (Hodgkin's lymphoma, T cell lymphomas, carcinomas), and certain drugs (cytokine and interleukin therapy)

BASOPHILS

- Role mostly unknown, likely in allergic response; normal values: 0-0.3 x 10^9/L
- Basophilia: allergic/hypersensitivity, inflammatory, endocrinopathy (DM, hypothyroidism), chronic renal disease, and myeloproliferative disorders (especially CML)

PLATELET COUNT

Actual number of platelets per volume of blood; normal values: 150-400 x 10^9/L

THROMBOCYTOSIS	
Primary	• Essential thrombocytosis/thrombocythemia
Secondary	• Hemorrhage, iron deficiency, surgery, splenectomy/hyposplenism, malignancy, acute or chronic inflammatory disease, general physiologic stress response
THROMBOCYTOPENIA	
Decreased PLT Production	• Severe nutritional deficiency, infection and inflammation, chemo and radiation therapy, bone marrow failure (including aplastic anemia) or infiltration (primary hematologic or metastatic disease), hereditary
Increased PLT Destruction	• Immune (ITP, EBV, SLE) and non-immune mechanical (MAHA: TTP, HUS, DIC, HELLP) and mechanical (cardiopulmonary bypass, IABP and ECMO)
Sequestration	• Splenomegaly, massive transfusion

INTERPRETATION OF CREATININE

Authors: Kimberley Krueger MD, Jason Kiser MD, Mark Joffe MD FRCPC

DIFFERENTIAL DIAGNOSIS

Serum Cr increase

- ARF: pre-renal (decreased ECFV, renal artery vasoconstriction); renal (ATN, acute interstitial nephritis, GN, small vessel disease); post-renal (BPH, neurogenic bladder, anticholinergic medications, malignancy, bilateral nephrolithiasis)
- CRF: DM, HTN, PCKD, GN, drug-induced, multiple myeloma, prolonged ARF
- Falsely high in drug interference with assay (cefoxitin, flucytosine)

Serum Cr decrease

- Decreased muscle mass, pregnancy

BASICS OF INTERPRETATION

- Normal values for serum Cr: 50-110 µmol/L. Note: normal value depends on individual patient (e.g., body mass, age; previous creatinine values are needed to interpret value).
- Cr produced from skeletal muscle metabolism, concentration proportional to body mass
- Serum Cr can be used to estimate GFR (endogenous Cr is freely filtered through the glomerulus with minimal tubular secretion and excreted from muscle at a relatively constant rate): GFR is ~ inversely proportional to Cr
- Limitations in ability to estimate GFR exist because:
  - Cr values are influenced by age, muscle mass, and dietary intake
  - Contribution of tubular Cr secretion increase with increasing renal impairment
  - GFR must fall considerably before serum Cr has a notable increase (50% fall in GFR → doubling of serum Cr)
  - Patient must be in steady state for Cr values to be used

CALCULATIONS TO ESTIMATE GFR

Cr Clearance (CrCL)

- CrCL (mL/min) = (urine Cr x urine volume over 24 hrs)/(serum Cr x 140)
- Has the potential to overestimate GFR in patients with advanced kidney impairment

Cockcroft-Gault

- CrCL (mL/min) = (140 – age)(Wt in kg)/(serum Cr in µmol/L) [Note: multiply by 1.2 for males]
- Accounts for age, body weight, and gender influences on GFR

Modification of Diet in Renal Disease (MDRD)

- Complex formula to estimate GFR (reported as mL/min/1.73m^2 body surface area)
- Uses the following indices: age, sex, serum Cr, African descent

CKD EPI

- Formula used to estimate GFR in Alberta labs since 2012
- Uses the following indices: age, sex, serum Cr, African descent

INVESTIGATIONS

Volume Status (pre-renal failure)

Blood Work

- Electrolytes, osmolality, CBC, urea (to differentiate between causes of ARF)

Radiology/Imaging

- Renal U/S (assess kidney size, rule out post renal causes, rule out renal artery stenosis with Doppler)

Special Tests

- Urinalysis (hematuria, proteinuria, casts), U/O, urine electrolytes/osmolality/creatinine to calculate fractional excretion of sodium (to differentiate between causes of ARF), foley catheter (prostatic obstruction)

Surgical/Diagnostic Interventions

- Renal Bx

Fractional Excretion of Sodium
FENa = (UrineNa x SerumCr) / (SerumNa x UrineCr)

TREATMENT

Treat underlying cause (see ARF and CRF [in Internal Medicine – Renal Failure])

Emergent:

- Follow Cr and watch for development of uremic symptoms or other indications for urgent dialysis (acidemia, hyperkalemia, volume overload) in those patients with renal failure
- Uremic symptoms tend to develop once serum Cr >530-710mmol/L or CrCL <10 mL/min

INTERPRETATION OF C-SPINE IMAGING

Authors: Brendan Diederichs MD, Katherine Leung MD, Tom Yeo MD FRCPC

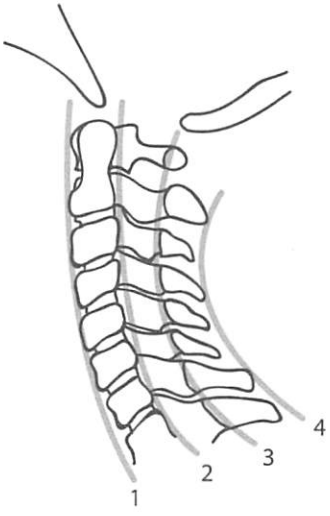
BASICS

Name the study and orientation (e.g., lateral, AP, odontoid)
Identify the patient and date
Comment on technical quality
Ask if previous studies are available for comparison

LATERAL

Mnemonic: All Able-Bodied Premeds Do Anatomy

- Adequacy: assessed by counting the vertebral bodies
  - If you cannot see the top of T1, order a swimmer's view
- Alignment should then be commented on in the lateral view; disruption of the normal contour of these lines may indicate fracture/dislocation
  - 1: anterior vertebral line (in image)
  - 2: posterior vertebral line
  - 3: spinolaminar line
  - 4: spinous process line
- Bones: Each vertebra should be examined for fracture
  - Look for wedge shaped defects indicating compression fracture
  - Examine each spinous process for fracture line
- Prevertebral space
  - 7 at 3, 21 at 7 rule: normal width of the prevertebral line at C3 (7 mm) and C7 (21 mm); widening may suggest pathology
- Intervertebral disc spaces
  - Narrowing may be suggestive of degenerative disc disease
- Atlanto-axial joint
  - Look at the space between the atlas (C1) and the odontoid process of C2 to ensure there is no ligamentous damage resulting in instability
  - This space (atlantodental distance) should be ≤3 mm in width for adults



AP (FRONTAL)

Focus on the vertebral bodies

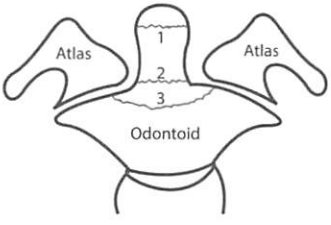
- Look for fractures of the pedicles and facets, as any asymmetry may be pathologic and should be noted
- Examine the spinous processes for alignment and spacing

Examine the soft tissue for any masses or calcifications

ODONTOID

This figure provides an AP view of the odontoid

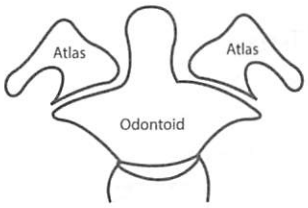
- Alignment
  - Look for the lateral sides of the atlas to align with the edges of the axis
  - Look for equal spacing bilaterally between the axis and the atlas
- Fracture lines through the dens
  - Type 1: through the cephalic dens
  - Type 2: evenly through the base of the dens (most unstable)
  - Type 3: descending into the base of the dens



OBLIQUE VIEWS

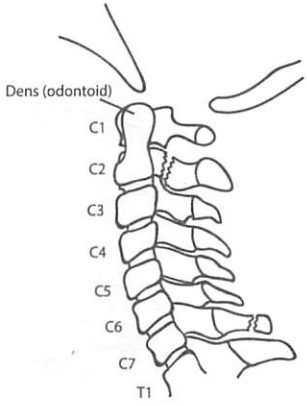
Oblique views may not be available depending on local imaging protocols (not typically performed in acute trauma setting)

- Examine the foraminae
  - Each vertebral body should have a patent foramina; otherwise, suspect fracture with angular distortion, poor positioning, or degenerative uncovertebral joint and/or facet joint changes
- Examine the alignment of facet joints
  - Commonly described as "shingles on a roof" in this view
  - Disruptions of the "shingles" may indicate dislocations of the facet joints
- Examine the pars interarticularis (region of vertebra between superior and inferior facet joints)
  - Scotty dog sign: pars interarticularis fracture in spondylolysis → vertebral body slips anteriorly and compresses the vertebral canal



COMMON EPONYMOUS FRACTURES

- Jefferson's fracture (unstable)
  - Classically seen in the odontoid view as the lateral edges of the atlas extend beyond the lateral edges of the axis (C1 ring has broken open)
- Hangman's fracture (unstable)
  - Due to hyperextension of the neck resulting in bilateral C2 pedicle fracture
  - Often seen in motor vehicle accidents (MVAs)
- Clay-Shoveler's fracture (stable)
  - Avulsion fracture of the lower C-spine (C6 or C7) spinous processes
  - Caused by sudden flexion force of neck and back muscles



CANADIAN C-SPINE RULE

Used for alert (GCS 15) and stable trauma patients in whom C-spine injury is suspected: 99.4% sensitive and 45.1% specific for C-spine injury (P <0.001)

- High risk? (if yes → radiographic imaging indicated)
  - Age ≥65
  - Dangerous mechanism
    - Fall ≥3 feet/5 stairs, axial load to head, MVA high speed (≥100 km/hr) or rollover or ejection, motorized recreational vehicle, bicycle collision
  - Paresthesia in extremities
- Factors allowing ROM assessment? (if no → radiographic imaging is indicated)
  - Simple rear-end MVA
    - Exceptions: pushed into traffic, rollover, hit by bus/large truck/high speed vehicle
  - Sitting in emergency department
  - Ambulatory at any time
  - Delayed onset of neck pain
  - Absence of midline C-spine tenderness
- Able to actively rotate neck 45° left and right? (if no → radiographic imaging is indicated)
- Note: Rule not applicable if:
  - Non-trauma cases
  - GCS <15
  - Unstable vital signs
  - Age <16
  - Acute paralysis
  - Known vertebral disease
  - Previous C-spine surgery

INTERPRETATION OF CT

Current Editor: Edwin Cheng MD

HISTORY AND CONTEXT

Name the study and orientation (e.g., axial images from non-contrast CT head)

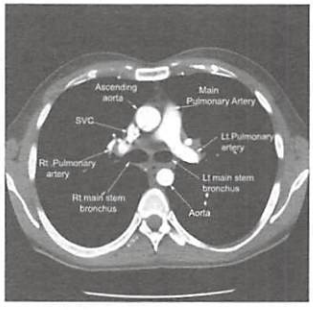
Identify the patient and date along with clinical question and relevant history

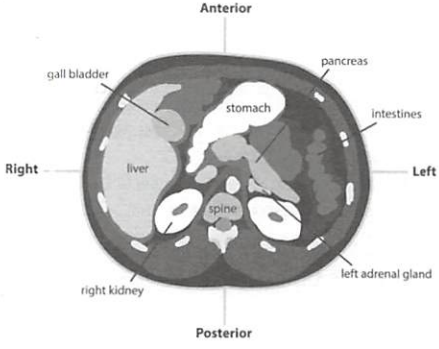
Determine if previous studies are available for comparison

CT BASICS

CT scans are generally viewed in axial sections as viewed from the feet (see figures below)

- Coronal (similar in orientation to a PA CXR) and sagittal reformats are common
- IV contrast studies have vessels appearing white (approaching the attenuation of bone)
  - › In the arterial phase, the arteries are brighter than the veins
  - › In the portal venous phase, the arteries and veins are both bright





- Oral contrast may also be given to enhance the bowel
- Common contraindications for IV contrast include allergy and renal disease (check creatinine before administering contrast)
- Non-contrast studies have darker vessels (approaching the attenuation of soft tissue)
  - › Especially important for identifying renal calculi
- CT images should be windowed according to what you are examining
  - › Windows are often preset and described as "Lung", "Bone", "Soft tissue", etc.
- Beam attenuation is measured in Hounsfield Units (HU)
  - › Air (-1000 HU) < Fat (-100 HU) < Water (0 HU) < Muscle (40 HU) < Bone (1000 HU)

CT ABDOMEN

- CT Abdomen studies typically include slices from just above the diaphragm to the iliac crests
- Be systematic (system or organ based approach) and scroll through image set several times

Abdominal Wall and Intra-abdominal Fluid

- Check for defects in the abdominal wall (e.g., hernias), thickening/irregularities of the peritoneum, and fluid collections . (indicates ascites, hemorrhage, abscess). Also assess for lymphadenopathy.

Bone

- In bone window, assess for fractures, degenerative changes, lytic/blastic lesions, decreased mineralization, symmetry.

Free Air

- In lung window, assess for free air under the anterior wall of the abdomen (if scan was obtained with patient supine).

Stomach, and Small and Large Intestines

- Follow the GI tract from distal esophagus to mid small bowel. Then start at the rectum and work your way up to the ileocecal valve.
- Assess for masses, polyps, wall thickening and other irregularities.
- Assess for distension, obstruction, inflammation, and air in the lumen wall (pneumoperitoneum).
- Look for diverticulae and the appendix (blind ending pouch) by finding the ileocecal valve and scrolling a few slices lower.

Liver, Biliary Tract, Spleen, and Pancreas

- If looking for hepatic lesions, you will often need an arterial phase and portal venous phase.
- Assess the overall density of the liver to look for fatty infiltration.
- Look for masses, cysts, scarring, and enlargement.

- Assess the hepatic duct and common bile duct for dilation, the gallbladder for stones/inflammation/masses.
- Assess the splenic volume and contours.
- Assess for normal pancreatic duct. Look for masses, abscesses or signs of pancreatitis including peripancreatic fluid.

Kidneys and Adrenals

- Look at both adrenals and assess for masses.
- Look at both kidneys for size and shape. Look for masses and cysts. Look for hydronephrosis and hydroureter. Follow the ureter to the bladder, looking for ureteric calculi or a bladder mass

Vasculature

- Assess the aorta and its vessels for aneurysms, dissections, and calcifications

CT CHEST

- The CT chest series will include images from the lower neck to the diaphragm.
- A systematic approach is key to successful interpretation. Review medical history to attune focus.
- The use of contrast enhancement will allow better assessment of the vessels, heart, thyroid, and lymph nodes.
- The commonly used "CT PE Protocol" uses IV contrast timed when the contrast is in the pulmonary arteries and allows for visualization of pulmonary emboli.

Soft Tissues

- In soft tissue window, look at the thyroid gland, thymus, and breasts. Assess for any masses or lymphadenopathy.

Bone

- In bone window, assess for any fractures, lytic or blastic lesions, and masses.
- Look at the ribs, humerus, scapula, and clavicles. Assess the vertebrae and sternum.

Mediastinum

- Look at the different heart chambers, valves, and pericardium. Assess for any lymph node enlargement. Look for masses.

Vessels

- Assessed best with IV contrast. Look at the aorta for dissection, aneurysm, or calcification.
- Look at the branches of the aortic arch. Assess the vena cava.
- Look at the pulmonary arteries, and follow individual arteries in the CT PE Protocol to find emboli.

Pleura

- Look for pleural plaques or masses. Look for pneumothoraces and pleural effusions.

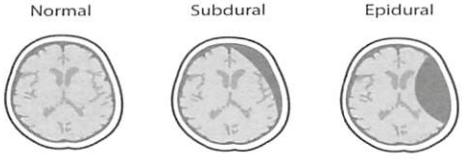
Airways and Lung Parenchyma

- Assess for obstructions, masses, consolidation, atelectasis, nodules, bullae, bronchiectasis, ground glass opacity, emphysema, honeycombing, etc.

CT HEAD

- CT head consists of axial views from the vertex to the skull base
- Medical history can help focus search (trauma, new neurological deficits, query stroke, etc.)
- Start from the bottom and work up to the top (or vice versa)

Examine the **CSF spaces**



- Is there loss of space (effacement/edema) or excess space (atrophy, hydrocephalus)?
- Is the ventricular system normal? Any dilatation, shift, compression, or asymmetry?
- Are the basal cisterns patent? Is there blood (bright if recent) in the cisterns?

Examine the **brain parenchyma**

- Move from central structures outward, ensuring each major area of the brain is symmetrical and without lesions (any soft tissue asymmetry may indicate mass effect).
- Examine for midline shift/mass effect, masses, bleeds, or infarcts.
- Examine grey-white matter differentiation, look for changes suggesting edema.

Examine the **contours of the calvarium**

- Are there any areas of attenuation in keeping with a subdural (lens shaped, crosses sutures) or an epidural (crescentic, within sutures) hematoma?

Examine the **bones (using the bone window)**

- Also look at the sinuses (air/fluid levels, fractures), the orbits, and mastoid air cells.

INTERPRETATION OF ECG

Current Editor: Derek Chan MD MBA CHE

GENERAL

Ensure correct patient ID

Compare with previous ECGs if available

RATE

Each thick line (5 mm box) represents 0.2 secs. Find a QRS that lands on a thick line and use the count off method for successive thick lines (300-150-100-75-60-50)

- If there is an irregular rate, count the number of QRS complexes in the entire strip and multiple by 5 (each strip is 12 secs)
- Bradycardia = <60 bpm
- Normal = 60-100 bpm
- Tachycardia = >100 bpm

RHYTHM

Regularity

- Sinus rhythm: P wave before every QRS, QRS after every P wave, P waves upright in leads II and aVF
- Regular or irregular?
- If irregular, is it regularly irregular, or irregularly irregular?
  - Regularly irregular rhythms include 2nd degree heart block and ventricular bi/trigeminy
  - Irregularly irregular rhythms include atrial fibrillation, multifocal atrial tachycardia, or wandering atrial pacemaker

Important Differentiating Questions

- P-waves (PQRS mnemonic)
  - P: Are P waves Present? (e.g., atrial fibrillation)
  - Q: What is the relationship between the P waves and the QRS? (e.g., sinus rhythm)
  - R: Is the PR interval constant or different? Does the PR interval get progressively prolonged? (e.g., 2nd degree Mobitz I)
  - S: Are the P waves the same Shape? (e.g., multifocal atrial tachycardia or wandering pacemaker)

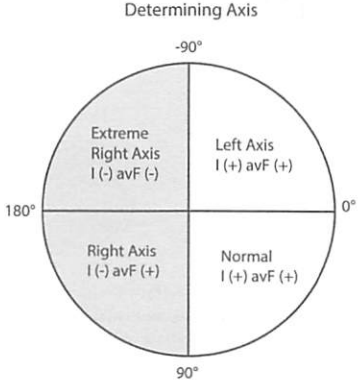
AXIS

Vectors Method

- Lead aVF for vertical vector
  - If QRS is positive, axis points down toward +90°
  - If QRS is negative, axis points up toward -90°
  - The more positive the QRS, the more the overall vector points toward +90°
- Lead I for horizontal vector
  - If QRS is positive, axis points right toward 0°
  - If QRS is negative, axis points left toward 180°
  - The more positive the QRS, the more the overall vector points toward 0°
- Combine the vertical and horizontal elements to estimate the axis

Isoelectric Lead Method

- Determine the quadrant for the axis (see figure)
  - Determine if QRS in lead I and lead aVF is positive or negative
- Look at the 6 limb leads and decide which one is closest to being the isoelectric lead (positive and negative components of the QRS are equal)
  - Draw a line perpendicular to the isoelectric lead vector toward the appropriate quadrant – this estimates the axis to the nearest 30°



Interpretation

- Normal axis is between -30° and +90°
- Note: if QRS is positive in lead I and lead II, then axis is within normal range

DDx of Right Axis Deviation	DDx of Left Axis Deviation
Right ventricular hypertrophy	Left ventricular hypertrophy
RBBB	LBBB
Left posterior hemiblock	Left anterior hemiblock
Lateral infarct	Inferior infarct

INTERVALS

PR Interval—Normal: 0.12-0.20s

- Shortened PR interval
  - Fast conduction along an accessory pathway (pre-excitation, Wolf-Parkinson-White)
  - Junctional rhythms with retrograde P waves (P waves occur within the QRS complex)
- Lengthened PR interval indicates delayed AV conduction
  - Regular rhythm: 1° heart block
  - Irregular rhythm: 2° or 3° heart block, wandering atrial pacemaker, multifocal atrial tachycardia

QRS Interval—Normal: <0.12s

- Many causes of wide QRS complexes are life-threatening, always consider VT
  - Consider LBBB, RBBB, hyperkalemia, interventricular conduction delay
  - New LBBB and dynamic ST changes (MI), RBBB and S1Q3T3 (PE), third degree heart block (MI), fascicular block

QT Interval—Normal: ~ < ½ of preceding R-R interval, QTC < 460ms (male) or 480ms (female)

- Calculate QTc as the QT/√RR interval (0.38-0.42 s)
- Shortened QT interval
  - Hypercalcemia, digitalis
- Lengthened QT interval (mnemonic: DIE, because the most serious complication is torsades de pointes)
  - Drugs: amiodarone, quinidine, procainamide, sotalol, TCA antidepressants, antihistamines, macrolides, cocaine
  - Injury: myocardial infarction, myocarditis, head injury, hypothermia, HIV, anorexia nervosa
  - Electrolytes: decreased K^+, Mg^+ or Ca^{2+}
  - A lengthened QT interval may also be congenital (common)

HYPERTROPHY

Atrial Enlargement

- Left atrial enlargement
  - P-mitrale is a "m-shaped" P wave >0.12s wide with >0.04s between peaks (most common in leads I and II)
  - Negative component of P wave in V1 is greater than or equal to 1x1 small boxes
- Right atrial enlargement
  - P-pulmonale are peaked P waves >2.5 mm tall (most common in leads II and III)

Ventricular Enlargement

- Left ventricular hypertrophy
  - There are multiple criteria with varying sensitivity and sepcificity for chamber hypertrophy
  - Deepest S wave of V1 or V2 + tallest R wave of V5 or V6 > 35 mm (Sokolow Lyon criteria)
  - S wave of V3 + R wave of aVL > 28 mm in men or 20 mm in women (Cornell voltage criteria)
- Right ventricular hypertrophy
  - R:S ratio is >1 in V1 or V2
  - May see evidence of posterior MI or RBBB, RAD (axis >90 degrees), R wave in V1 > 7 mm (not R' of RBBB)

PATHOLOGICAL FEATURES

T waves

- T waves should be positive in leads I, II, and V3-V6, and negative in aVR
- Abnormal T waves: inverted, symmetrical, peaked, or tall (>2/3 of R wave)

ST segment

- Elevation >1 mm in 2 contiguous leads from baseline (defined as the TP segment) indicates acute transmural ischemia
- Depression >1 mm in 2 contiguous leads from baseline indicates subendocardial ischemia
- Concave segments are classic for strain pattern; ST elevation with tombstone segments are classic for acute infarction

Q waves

- Signify necrosis: significant Q waves are 0.04s (1 small square) wide or >1/3 amplitude of QRS

Special Circumstances were an ECG is Difficult to Further Interpret

- **LBBB**: a new LBBB shown on an ECG is alway pathological and may be an indication of MI, but this is not diagnostic. In a LBBB ST segments and T waves are shifted in a discordant direction and can either mimic or hide a MI. LV hypertrophy produces an ECG pattern similar to LBBB with a widened QRS and ST depression or T wave inversion on lateral leads. Therefore, any diagnosis of LBBB may also include a differential diagnosis of MI and LV hypertrophy.
- **Right Ventricular Paced Rhythm**: a right ventricular paced rhythm results in a morphology similiar to LBBB on ECG. This results in similar difficulty diagnosing an acute MI to a new LBBB on ECG with ST segments and T waves shifted in discordant directions.

Location of Infarct	Leads Showing Ischemic Changes	Coronary Arteries
Septal	V1 and V2	LAD
Anterior Wall	V3 and V4	LAD
Lateral Wall	I, aVL, V5, V6	LAD, LCx
Inferior Wall	II, III, aVF	RCA, LCx
Posterior Wall	V1-V3 (ST-depression), V7-V9	RCA
Right Ventricle	V4R, II, III, aVF	RCA

INTERPRETATION OF ELECTROLYTES: CALCIUM

Current Editor: Maryam Soleimani MD

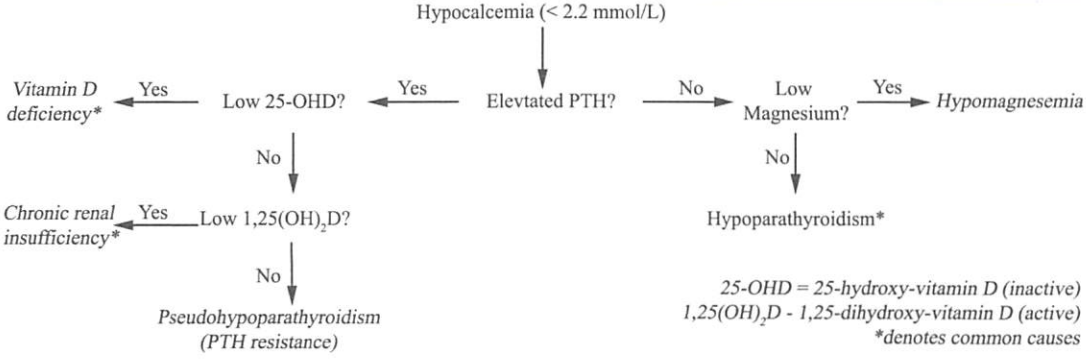
CORRECTED CALCIUM AND PARATHYROID HORMONE

Measured serum calcium must be corrected because serum albumin influences ionized calcium to bound calcium ratio

Corrected calcium (mmol/L) = measured serum calcium + 0.02*(40 – serum albumin)

- If in doubt whether serum calcium reflects ionized calcium, can measure ionized calcium directly
- Ionized calcium normal range is usually ~50% of serum calcium

HYPOCALCEMIA – DIAGNOSTIC APPROACH



HYPOCALCEMIA – HISTORY

HPI: diet and medications (calcium and Vit D deficiency)

PMHx: renal disease, autoimmune disorders, GI absorption disorders

SHx: head and neck surgeries (parathyroid injury)

FHx: familial hypocalcemia, polyglandular autoimmune syndromes

HYPOCALCEMIA – SIGNS AND SYMPTOMS

CNS: confusion, depression, psychosis, seizures, personality changes

CVS: hypotension, prolonged QT interval, arrhythmias

MSK/NEURO: tetany, spasms, myopathies, paresthesias, circumoral numbness

Special tests (T&C signs)

- Trousseau's sign: carpopedal spasm (wrist and fingers flex and draw together) when BP cuff inflated over arm for 2-3 minutes
- Chvostek's sign: facial spasm when facial nerve tapped over the parotid

HYPOCALCEMIA – INVESTIGATIONS

Routine labs

- Electrolytes, magnesium, calcium, albumin, phosphate, Vit D, PTH, ALP, creatinine, urea

Special tests

- 24 hr urine calcium: decreased in hypoparathyroidism and Vit D deficiency

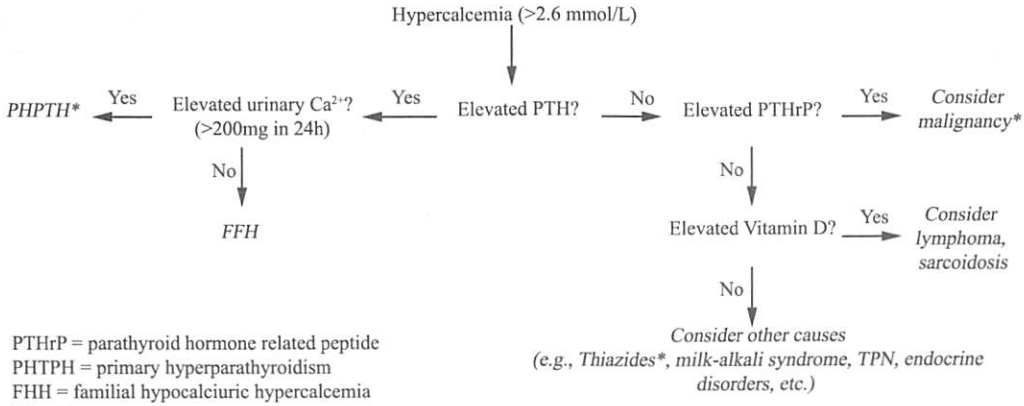
HYPOCALCEMIA – TREATMENT

Emergent (calcium <1.0 mmol/L, acutely symptomatic)

- IV calcium gluconate, correct other electrolyte abnormalities (hypomagnesemia)

Chronic

- Treat underlying disease, diet and lifestyle modification
- Oral calcium and/or Vit D supplementation as appropriate



PTHrP = parathyroid hormone related peptide
PHTPH = primary hyperparathyroidism
FHH = familial hypocalciuric hypercalcemia
*denotes common causes

HYPERCALCEMIA – HISTORY, SYMPTOMS, INVESTIGATIONS, AND TREATMENT

HPI: diet (milk and antacids), drugs (thiazides, lithium, Vit D), bone pain, abdominal pain

PMHx: endocrine disorders, malignancies

FHx: hypercalcemia or MEN syndromes, cancers

HYPERCALCEMIA – SYMPTOMS

"Bones, stones, groans, and psychic overtones"

Note: Symptoms are non-specific and many patients are asymptomatic at time of diagnosis

- CNS: confusion, depression, fatigue
- CVS: hypertension, shortened QT syndrome, arrhythmias
- GI: abdominal pain, N/V, anorexia, constipation, peptic ulcer disease, pancreatitis
- GU: renal calculi, polyuria, polydipsia, renal failure
- MSK: weakness, bone pain, arthritis, osteoporosis, fractures

HYPERCALCEMIA – INVESTIGATIONS

Routine labs

- Electrolytes, magnesium, calcium, albumin, phosphate, Vit D, PTH, ALP, creatinine, urea

Special tests

- 24hr urine calcium: distinguish FHH from primary hyperparathyrodism. FHH is typically associated with low 24 hr urine calcium excretion whereas primary hyperparathyroidism is often associated with elevated calcium excretion.
- Can consider spot urine calcium to creatinine ratio as a surrogate for 24 hr urine calcium excretion
- Serum and urine protein electrophoresis: if multiple myeloma is suspected
- Parathyroid Sestamibi scan: hyperparathyroid lesions

HYPERCALCEMIA – TREATMENT

Emergent (calcium >3.0 mmol/L, acutely symptomatic)

- IV fluids with goal urine output being roughly 100-200 cc/hr , bisphosphonates, calcitonin, correct other electrolyte abnormalities
- Loop diuretics to promote excretion of calcium should only be used after dehydration is corrected and only if patient appears fluid overloaded (most patients are in fact dehydrated)

Chronic

- Treat underlying disease, stop offending medications, diet and lifestyle modification, consider surgery if applicable

INTERPRETATION OF ELECTROLYTES: POTASSIUM

Current Editor: Hana Yu MSc MD

HYPOKALEMIA – DIFFERENTIAL DIAGNOSIS AND INVESTIGATIONS

Decreased intake (uncommon): starvation or clay ingestion

- Redistribution into cells: acid-base (alkalosis), hormonal (insulin, β2 adrenergic action), anabolic (B12 or folate administration, TPN, granulocyte-macrophage colony-stimulating factor), other (hypokalemic periodic paralysis)

Extrarenal losses (urine K^+ <20mmol/d)

- GI losses: vomiting, diarrhea, tube drainage, laxative abuse
- Skin loss: excessive sweating

Renal losses (urine K^+ >20mmol/d)

- Hypertensive (mineralocorticoid excess): primary or secondary hyperaldosteronism, Cushing's, CAH
- Hypotensive or normotensive:
  - Acidemic: DKA/renal tubular acidosis (Types 1 & 2)
  - Alkalemic: diuretics
  - Variable: vomiting/hypomagnesium/drugs (amphotericin)

HYPOKALEMIA – TREATMENT

Recognize signs/symptoms of hypokalemia (see box)

Calculate transtubular potassium gradient (TTKG):

- TTKG = (K^+urine)(Osmserum)/(K^+serum)(Osmurine)
  = >4 (renal loss), <2 (extrarenal loss)

Treat underlying cause and/or remove offending medication

Replace K^+ slowly: oral preferred over IV KCl (max dose 20-40 mEq oral, max infusion 20mmol/h IV); never bolus K^+

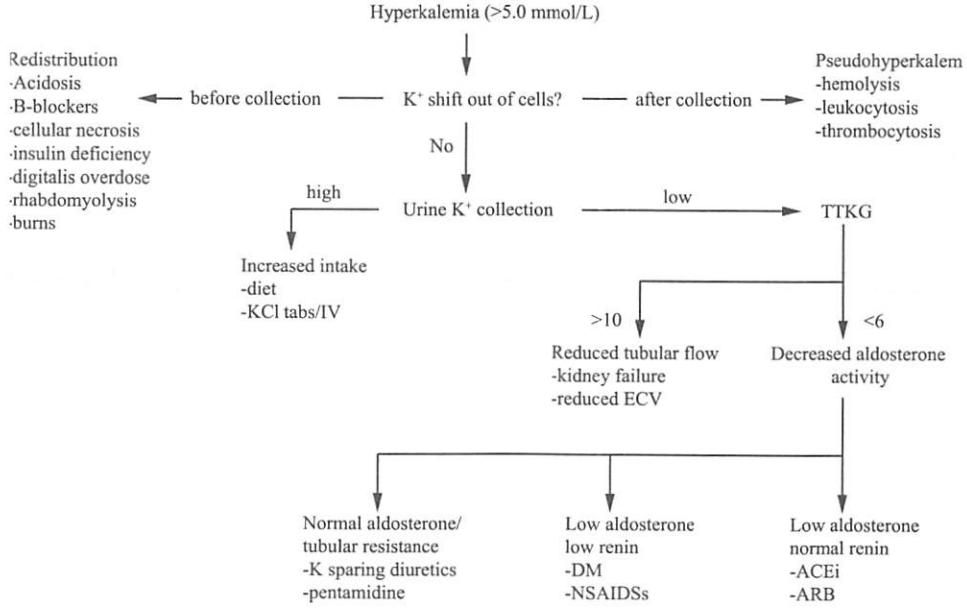
Check serum K^+ frequently to avoid over-correcting

Consider role of K^+-sparing diuretics in treatment plan

1 mEq/L decrease reflects ~200 meq total body loss

Signs/Symptoms of Hypokalemia

- Nausea, vomiting
- Muscle weakness (if severe: ascending paralysis, ileus, respiratory failure, rhabdomyolysis, arrhythmias)
- ECG changes: flattened T wave, ST segment depression, high U wave

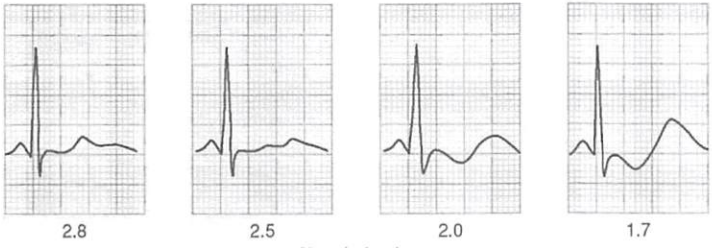


HYPERKALEMIA – DIFFERENTIAL DIAGNOSIS AND TREATMENT

HYPERKALEMIA DIFFERENTIAL DIAGNOSIS	
Cellular Shift	• Acidosis, insulin deficiency, beta-blockers, tumor lysis, burns, hemolysis
Decreased Excretion	• Renal failure, hyperaldosteronism
Drugs	• TMP-SMX, succinylcholine, K-sparing diuretics (e.g., spironolactone)

Note: Need to rule out pseudohyperkalemia via repeat serum K level before treatment if value does not match clinical scenario

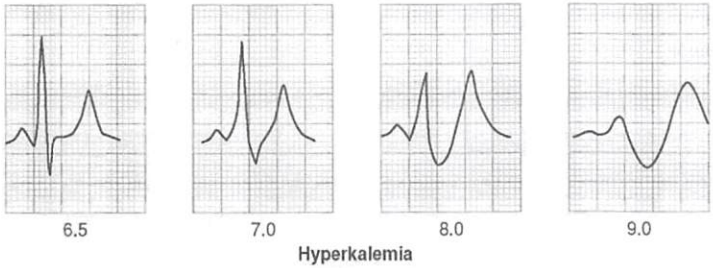
HYPERKALEMIA TREATMENT	
K^+ <6.0 and normal ECG	• Treat underlying cause • Stop K^+ intake and meds that increase K^+ • Promote K^+ loss (see "Remove K^+" below)
K^+ >6.0 and/or ECG changes* A MEDICAL EMERGENCY: TREAT URGENTLY	• Antagonize effects of K^+ on myocardium (10 mLs of 10% calcium gluconate IV over 2-3 mins) • Shift K^+ into cells: › Insulin + glucose (e.g., 10 units of rapid acting insulin + 50 mL of 50% glucose solution IV as a bolus followed by an infusion of glucose-containing solution) – Always give the glucose before the insulin!! › If patient is significantly hyperglycemic, insulin alone can be provided • β-agonist (Ventolin) › Bicarbonate • Remove K^+ › Loop diuretics (furosemide) › Cation exchange resin (e.g., Na or calcium polystyrene sulfonate = Kayexylate®); give with lactulose to promote osmotic diarrhea and prevent sticking › Hemodialysis



Hypokalemia

ECG Changes - Hypokalemia

PR interval lengthens, ST segment depresses, T wave inverts, and U wave is appears and becomes more pronounced.



Hyperkalemia

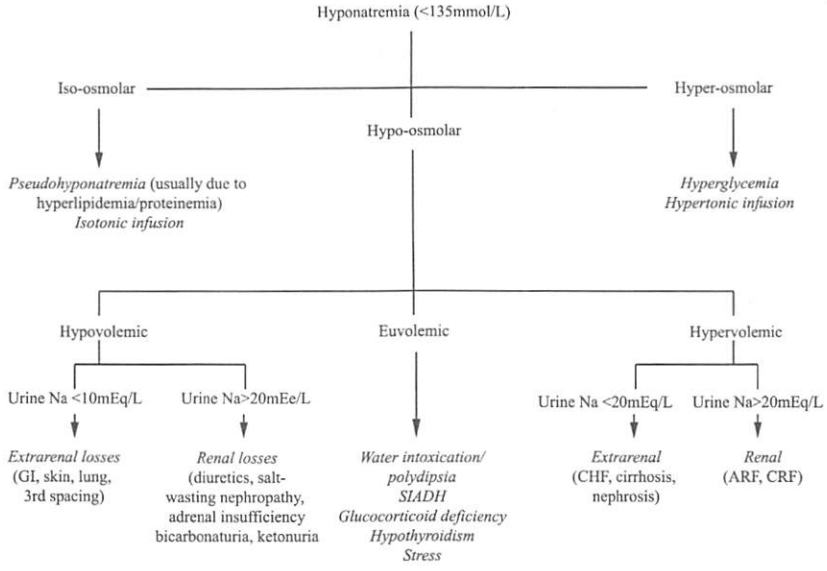
ECG Changes - Hyperkalemia

Progression from tall peaked T waves (5.5-6.5mM), to a loss of a P wave (6.5-7.5mM), to a widened QRS complex (7.0-8.0mM), then a sine wave pattern (>8.0)

INTERPRETATION OF ELECTROLYTES: SODIUM

Authors: Kimberley Krueger MD, Jason Kiser MD, Mark Joffe MD FRCPC

HYPONATREMIA – DIFFERENTIAL DIAGNOSIS AND INVESTIGATIONS



MANAGEMENT OF HYPONATREMIA

Recognize signs/symptoms of hyponatremia

- Headache
- N/V
- Anorexia
- Lethargy
- Seizures
- Decreased LOC
- Coma

Complications of Hyponatremia

- Seizures
- Coma
- Respiratory arrest
- Brain damage
- Brainstem herniation
- Death
- If correction too rapid: osmotic demyelination of neurons

Order basic investigations

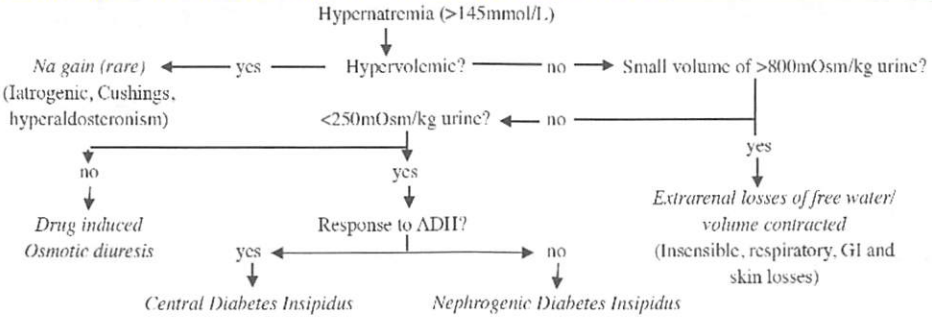
- Serum electrolytes, glucose, Cr, BUN, osmolality
- Urine sodium, osmolality
- Consider ordering: TSH, free T4, cortisol level

General treatment guidelines

- Fluid restrict to 1L/day (if hypovolemic give NS)
- Treat underlying cause
- Monitor both serum sodium and urine output to ensure appropriate correction rate

GENERAL MANAGEMENT	
Acute hyponatremia (<24 hrs)	· Rapid correction in symptomatic patients or those with large drop in sodium
Chronic hyponatremia (>24 hrs)	· Slow correction (≤8mM/24 hours correction) as rapid correction can cause osmotic demyelination syndrome
GENERAL CHOICE OF IV FLUID (IN HYPOOSMOLAR HYPONATREMIA)	
Asymptomatic	· Normal saline
Symptomatic/seizing	· Hypertonic 3% saline; consider demeclocyline if patient refractory to treatment

HYPERNATREMIA – DIFFERENTIAL DIAGNOSIS AND INVESTIGATIONS



HYPERNATREMIA – SIGNS, SYMPTOMS AND TREATMENT

Recognize signs/symptoms of hypernatremia

- Thirst
- Weakness
- Altered mental status
- Convulsions
- Coma

H_2O Deficit
H2O deficit = [TBW x (serum Na^+ – 140)]/140
TBW_{male} = 0.6(Wt) TBW_{female}=0.5(Wt)

General treatment guidelines

- Give free water (PO or IV)
- Restrict salt
- Treat underlying cause
- Monitor the serum sodium regularly to ensure <0.5 mmol/L/hr of correction
- H2O deficit = [TBW x (serum Na? – 140)]/140
- TBW_{male} = 0.6(Wt); TBW_{female}=0.5(Wt)

Complications of Hypernatremia
• Increased risk of vascular rupture (can lead to intracranial hemorrhage) • If correction too rapid: cerebral edema

If **hypovolemic**: follow general guidelines

If **hypervolemic**: remove excess Na^+ with diuresis/dialysis and replace water deficit with free water

INTERPRETATION OF LFTS & ENZYMES

Authors: Mackenzie Lees MD, Simon Turner MD, Gordon Lees MD FRCSC

INTRODUCTION

Liver enzyme abnormalities are common

1:10 Canadians will have at least one elevated liver enzyme during routine screening

Normal LFTs do not exclude the possibility of chronic liver disease

TYPES OF LIVER FUNCTION TESTS (* = FIRST LINE)

Hepatocyte damage/inflammation (increased transaminases = cell death)

- *ALT: sensitive and specific marker of liver injury/inflammation
  - Normal range: <50 U/L
  - Nonhepatic increases: severe rhabdomyolysis, systemic myopathies
  - Can remain elevated weeks to months following liver injury
  - Can be normal in presence of liver disease (EtOH, chronic HCV or inactive cirrhosis)

AST, ALT >1000	· Ischemic injury, toxic injury, acute viral hepatitis, acute biliary obstruction, autoimmune hepatitis (less common)
AST >500	· Unlikely to be EtOH-related liver disease

- *AST: less sensitive and specific marker, useful as a screening test to determine AST/ALT ratio
  - Normal range: <40 U/L
  - Nonhepatic increases: MI, extensive surgery, PE, rhabdomyolysis
  - AST/ALT <1: viral hepatitis, autoimmune liver disease
  - AST/ALT >2: EtOH liver disease, biliary obstruction, NAFLD
  - Decreasing AST/ALT ratio over time: can indicate progression to cirrhosis
  - AST/ALT ratio is not useful in fulminant liver failure

Biliary function

- Bilirubin: usually increased in bile duct injury, cholestasis, and severe hemolysis
  - Normal range: 1-20 mmol/L
  - Normal bilirubin is 70% unconjugated and 30% conjugated; hyperbilirubinemia is considered unconjugated if >85% of bilirubin is unconjugated.
  - Test for indirect/unconjugated (RBC lysis) vs. direct/conjugated (biliary); determine cause of high total bilirubin
  - An increased bilirubin due to hepatic cause usually indicates advanced liver disease
- *ALP: Elevated in all forms of cholestatic liver disease, mild elevation in cirrhosis
  - Normal range: 25-110 U/L
  - Can also be high in pregnancy (placental ALP), bone disease, renal disease
- GGT: useful to confirm that high ALP reflects hepatobiliary disease (order GGT when elevated ALP)
  - Normal range: <65 U/L
  - Specific, with poor sensitivity: can be high following recent EtOH ingestion, secondary to antiseizure medications, renal disease, pancreatitis, DM, CAD, prostate cancer

Liver synthetic function

- Coagulation factors (INR): PT-INR, measures extrinsic pathway, Factor VII dependent (synthesized by the liver)
  - Normal range: 0.8-1.2
  - Can be high with Vit. K deficiency
- Albumin: major plasma protein (synthesized by the liver)
  - Normal range: 33-49 g/L
  - Can be low in liver disease, malnutrition, diarrhea, iron deficiency, infection

QUICK LFT REFERENCE FOR JAUNDICE			
	Pre-hepatic (i.e., hemolysis)	Hepatic	Post-hepatic (i.e., biliary)
AST, ALT	Normal/unchanged	↑↑	↑
ALP	Normal/unchanged	↑	↑↑
BILI	↑	↑ or ↑↑	↑↑

WHEN TO USE LFTS

Routine annual evaluation of patients with risk factors for liver disease

- Risk factors: obesity, DM, dyslipidemia, Hx of autoimmune disease, Hx of nephrotic syndrome, family Hx of liver disease, high-risk behaviors (IVDU, EtOH, sexual promiscuity, tattoos, non-sterile ear or body piercing, residence or travel to developing countries, occupational exposures)
- Hx of unexplained RUQ pain, dark urine, acholic stools, jaundice
- Findings on physical exam: unintentional Wt loss, stigmata of liver disease, impaired cognitive function

APPROACH TO ORDERING LFTS

Initial screening tests: ALT, AST, ALP, bilirubin, INR, +/- GGT, albumin

Use pattern of liver enzyme/LFT abnormality along with patient history and clinical context to guide further investigations and treatment (see table below)

PATTERN OF LFT ABNORMALITIES

Hepatocellular cause

- Very high ALT (e.g., 200-2000 + U/L)
- Moderately high ALP, unlikely >2x normal
  - Elevated AST and ALT >2x in ↑↑ ALP → likely hepatocellular cause
- Variable bilirubin, with degree of elevation proportional to severity of damage
- Prolonged INR

Cholestatic cause

- Very high ALP and bilirubin (direct)
- Normal ALT, unless severe
  - Similar increase in ALP and AST (e.g., 4x normal) → likely cholestatic cause

Test Result	Possible Cause	Initial Action	Management
↑ AST/ALT <1.0	Liver damage • Viral • EtOH • Autoimmune • Heritable disorders	• Perform additional blood tests (2nd line LFTs, HCV/HBV serology, autoimmune panels, iron saturation, ceruloplasmin, a1-anti-trypsin) • Abdominal U/S • Liver Bx	• Treat underlying cause • Specialist referral
↑ AST/ALT >2.0	• EtOH • Obesity • Hepatotoxic drugs	• Recheck LFTs q3-6 mos	• Reduce EtOH ingestion • Change hepatotoxic meds • Lose weight if BMI >25 • Specialist referral if LFT >1.5x normal 2x, 6 mos apart
↑ ALP AND GGT ± ↑ BILIRUBIN	Cholestatic • Biliary obstruction • Primary biliary cirrhosis • Primary sclerosing cholangitis • Autoimmune hepatitis	• Additional testing: autoimmune panels, viral serology, abdominal U/S, liver Bx	• Treat underlying cause • Specialist referral
↑ BILIRUBIN ONLY	• Hemolysis • Biliary obstruction	• Check direct/indirect bilirubin	Indirect • Look for cause of hemolysis Direct • Likely Gilbert's syndrome • No further treatment necessary
↑ GGT ONLY	• EtOH ingestion • Antiseizure medications • Renal disease • Pancreatitis • DM • CAD • Prostate cancer	• Review patient's PMHx • Identify risk factors	• Treat underlying cause

INTERPRETATION OF LIPIDS

Authors: Adarsh Rao MD, Carol Chung MD, Arnold Voth MD FRCP

BASICS

The lipid profile includes: total cholesterol (TC), high-density lipoprotein (HDL), low-density lipoprotein (LDL), and triglycerides (TG).

1. Decide who to screen with lipid panel
2. Estimate risk for major adverse cardiac events based on risk factors
3. Classify patients into low, intermediate, or high risk
4. Treat according to risk category

Recommendations made are based on the 2016 Canadian Cardiovascular Society guidelines.

RISK FACTORS FOR CAD

Male >45 years old or female >55 years old

Cigarette smoking

DM

High cholesterol (TC, LDL-C, or apoB)

Low HDL-C

HTN

Family Hx of premature CAD
(male <55 years old, female <65 years old)

Elevated inflammatory biomarkers
(especially hs-CRP)

Overweight/obese

Physical inactivity

LIPID SCREENING GUIDELINES	
Men >40 years of age; women >40 years of age (or postmenopausal) Consider earlier screening in ethnic groups at increased risk such as South Asian and First Nations individuals	• All patients with the following conditions regardlessof age: • Clinical evidence of atherosclerosis • Abdominal aortic aneurysm • Diabetes • Arterial hypertension • Current cigarette smoking • Stigmata of dyslipidemia (arcus cornea, xanthelasma or xanthoma) • FHx of premature CVD or dyslipidemia • Chronic kidney disease • Obesity (BMI >30 kg/m^2) • Inflammatory bowel disease • HIV infection • Erectile dysfunction • Chronic obstrucive pulmonary disease • Hypertensive diseases of pregnancy

INVESTIGATIONS

History and Physical

- PMHx: angina, DM, HTN, chronic kidney disease, RA, SLE
- Lifestyle: obesity, sedentary lifestyle, EtOH, smoking
- Family Hx of premature CAD
- Signs of hyperlipidemia include atheromata, xanthoma, tendinous xanthoma, corneal arcus

Blood Work

- Screen patients with lipid profile (TC, LDL, HDL, and TG) as part of global CVD risk estimation
- Non fasting lipid profile is considered reliable and acceptable as long as triglycerides are less than 4.5 mmol/L
- Research does not support use of biomarkers in risk assessment at this time
- Screen for secondary causes if indicated by clinical scenario, e.g., hypothyroidism, chronic kidney disease, drugs (tamoxifen, glucocorticoids, ß-blockers), nephrotic syndrome, DM, liver disease
  - › hs-CRP, TSH, fasting glucose, Cr, electrolytes, urea, GGT (if EtOH suspected), HbA1C, apoB

Risk Estimation

- Use a risk calculator every time lipid testing is performed, e.g., Framingham, QRISK2
- Sum of scores for multiple risk factors: age, sex, total cholesterol, tobacco use, HDL, BP (both sBP and dBP), presence of diabetes used to estimate 10-yr risk of CAD and stratify risk for treatment decisions
- Presence of some conditions put a patient in the high risk category irrespective of measured LDL, arguing for pharmacotherapy
- In general, match the intensity of preventative effort to the patient's absolute risk estimate

INTERPRETATION OF LIPID PANEL

If TC >6 mM or LDL >5 mM, suspect familial hypercholesterolemia

If TG >5 mM, suspect familial hypertriglyceridemia

If fasting TG are >5 mM – TG will be increased in non-fasting states

TREATMENT

THERAPY BASED ON CVD RISK	
10-Year CVD Risk	Recommendation
≥20%	• Discuss and strongly encourage statin (preferably high intensity)
≥10-19%	• Discuss and offer statins (preferably moderate intensity)
<10%	• Re-test lipids in five years with risk estimation. Repeat sooner if other CVD risk factors develop.

Recommend lifestyle changes for all patients: Mediterranean or DASH diet, exercise, Wt loss, smoking cessation, moderation of EtOH intake

Manage additional medical risk factors to ↓ CAD risk (treat HTN and DM)

Pharmacologic therapies

- Treat to target if pharmacotherpay is started: LDL <2 mmol/L or >50% LDL reduction. Repeat cholesterol panel 3 months after therapy change.
- Statins
  - First line of treatment for all patients when drug therapy is warranted
  - Statin can be expected to lower relative risk of CVD by 25-35%
  - HMG-CoA reductase inhibitors slow cholesterol formation by inhibiting rate-limiting enzyme
  - Major side effects include myalgia, elevated ALT, CK elevation, myopathy, rhabdomyolysis (rare), liver failure (very rare)
  - Monitor CK and ALT in symptomatic patients and those at high risk of adverse events
  - E.g., atorvastatin 10-80 mg po daily, rosuvastatin 2.5-40 mg po daily, simvastatin 5-40 mg po daily
- Non-statin lipid lowering agents
  - Additional agents are considered in patients who are not at target despite statins
  - Ezetemibe is second line to reach target LDL. Niacin, fibrates, and bile acid sequestrants can be used if LDL is NOT at target. Addition of second line agents not useful if LDL is at target.
- ASA
  - Consider after statin therapy in high risk individuals with low risk of bleeding
  - Relative benefit approximately 10%
  - Annual risk of major bleed approximately 0.5%

THERAPY		ESTIMATED BENEFIT (RELATIVE RISK REDUCTION)	EXAMPLE IF BASELINE RISK ESTIMATED AT 20% OVER 10 YEARS		
			Absolute Risk Reduction	Number Needed to Treat (NNT)	New Risk Estimate
Smoking Cessation		Recalculate without smoking	9%	12	11%
Mediterranean Diet		30%	6%	17	14%
Exercise		30%	6%	17	14%
Statin Intensity	Low	25%	5%	20	15%
	Moderate	30%	6%	17	14%
	High	35%	7%	15	13%
ASA		12%	2%	50	18%

- **Note:** Example used a 53 year old male smoker with total cholesterol 5, HDL 1.2 and systolic BP 128, estimated risk from Framingham (from http://cvrisk.mvm.ed.ac.uk/calculator/calc.asp andhttp://chd.bestsciencemedicine.com/calc2html#basic) to attain a 20% risk over 10 years.

INTERPRETATION OF PFT

Current Editor: Bradley Brochu MD

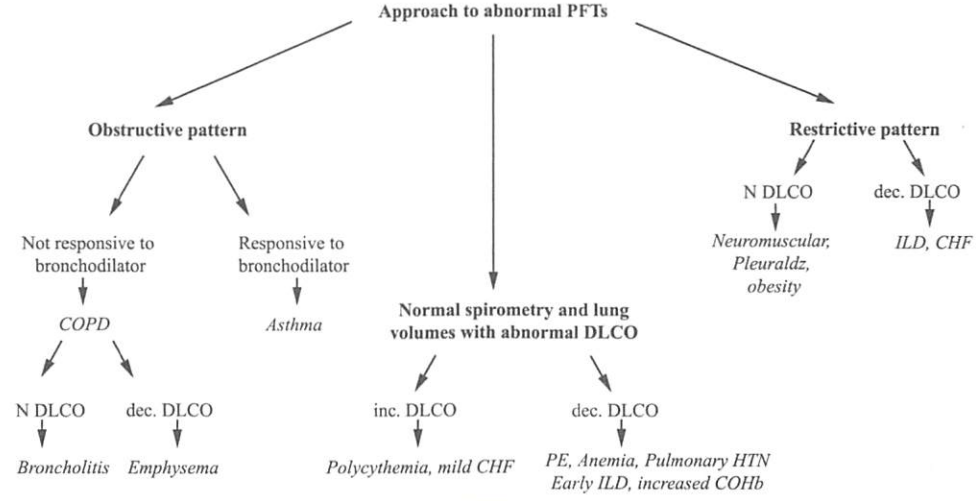
BASICS

Patient ID, age, gender, ethnicity, Ht, Wt, BMI (used to calculate reference values)

Test information

- Date of test
- Pre-test data
  - Pre-test medications taken
  - Smoking and past medical history
- Compare with patient's previous PFTs (if available)
- Contraindications
  - Respiratory distress, angina, pneumothorax, ongoing hemoptysis, active TB

DIFFERENTIAL DIAGNOSIS



SPIROMETRY

Quality

- Acceptability: requires 3 artifact-free maneuvers (e.g., no cough within 1 sec, no glottis closure)
  - Note: a minimum of 6 secs of sustained expiration on each maneuver is required
- Repeatability: requires 3 maneuvers where the two largest values of FEV1 are within 0.150L of each other and the two largest values of FVC are within 0.150L of each other

Flow-volume loop

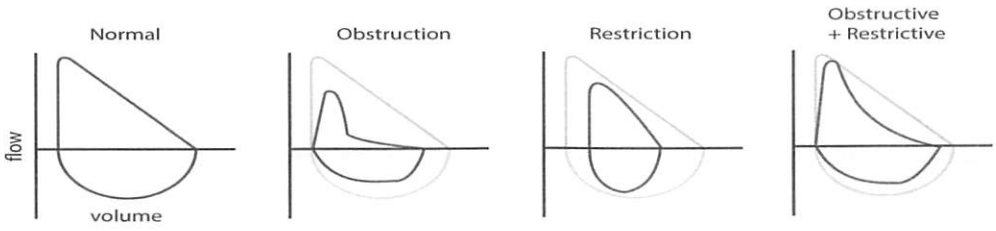
- Right displacement of tidal flow loop and tidal flow curve approaching maximum expiratory flow curve is suggestive of airflow limitation
- Flattening of the inspiratory or expiratory flow curve is suggestive of a thoracic airway obstruction. This could be either variable or fixed and either intra or extra thoracic in origin (e.g., tumor, thyroid goiter).

Diagnostic criteria for airflow obstruction and significant reversibility

- Is there obstruction?
  - FEV1/FVC <0.7 – yes
  - FEV1/FVC >0.7 – no
- How severe is the obstruction?
  - Mild: FEV1 >70%,
  - Moderate: FEV1 60-69%,
  - Severe: FEV1 35-49%,
  - Very Severe: FEV1 <35%

- Is there bronchodilator response?
  - ≥12% and ≥200 mL improvement in FEV1 post-bronchodilator indicates a significant bronchodilator response, i.e., reversible airflow obstruction
- COPD
  - Post-bronchodilator FEV1/FVC <0.7 AND FEV1 <80% predicted. Airflow obstruction is not fully reversible.
- Asthma
  - FEV1/FVC ratio less than lower limit of normal based on age, sex, height, etc. AND increase in FEV1 post bronchodilator ≥12%

Selected Examples of Flow-Volume Loops



A) normal flow-volume loop

B) typical obstructive defect with FEV1 < lower limit of normal

C) typical restrictive defect with TLC < lower limit of normal

D) typical mixed obstructive and restrictive defect

LUNG VOLUMES

Quality vital capacity measured by lung volumes (slow expiration) should be > FVC measured by spirometry (forced expiration), especially in obstructive disease

- TLC measured by lung volumes should be > VA measured by diffusion capacity

Total Lung Capacity: may be measured using either N2 dilution method or body plethysmography

- >120% predicted suggests possible hyperinflation (emphysema)
- <80% predicted suggests possible restrictive ventilatory defect
- TLC may be underestimated if using the N2 dilution method (measures only ventilated lung volume); using body plethysmography is the best method as it measures total lung volume
  - If TLC measured by body plethysmography is greater than N2 dilution, it suggests the presence of bullous lung disease

Residual Volume (RV)

- >120% predicted suggests gas trapping due to poor patient effort or obstruction

Expiratory Reserve Volume (ERV) and Functional Residual Capacity (FRC)

- In obese patients, both ERV and FRC are decreased

Lung Volume Guide	
TIDAL VOLUME (TV)	• Volume of air moved into or out of lungs during normal quiet breathing
FORCED VITAL CAPACITY (FVC)	• Maximum volume of air forcibly expired after maximal inspiration
FUNCTIONAL RESIDUAL CAPACITY (FRC)	• Volume remaining in lungs after normal breath
TOTAL LUNG CAPACITY (TLC)	• Volume of air at maximal inflation
VITAL CAPACITY (VC)	• Maximum volume of air exhaled after maximal inspiration
RESIDUAL VOLUME (RV)	• Volume of air remaining in the lungs after maximal exhalation

DIFFUSION CAPACITY

Check for DLCO correction for Hgb or VA (alveolar volume)

Decreased DLCO adjusted for VA suggests reduced diffusion capacity due to parenchymal abnormalities, abnormal concentrations of hemoglobin or carboxyhemoglobin, or the presence of pulmonary vascular disease

AIRWAY RESISTANCE

Increased airway resistance suggests turbulent airflow (airway constriction, edema, etc.)

INTERPRETATION OF URINALYSIS

Authors: Aamir Bharmal, Cathy Lu MD, Valerie Luyckx MBBS

INDICATIONS

UTI, pyelonephritis
Renal calculi
DM
Acute or chronic renal failure
Pregnancy
Hematuria
Undifferentiated abdominal or flank pain

APPEARANCE

CLEAR, LIGHT TO DARK YELLOW	• Normal variation with different physiological states of hydration
COLORLESS	• Diabetes insipidus, diuretics, excess fluid intake
DARK	• Acute intermittent porphyria, advanced malignant melanoma, cholestasis
CLOUDY	• UTI, amorphous phosphate salts, blood, mucus, bile
PINK/RED	• Blood, Hgb, sepsis, dialysis, myoglobin, food coloring, beets, sulfa drugs, nitrofurantoin, salicylates, laxatives (phenolphthalein)
ORANGE/YELLOW	• Dehydration, phenazopyridine (pyridium), bile pigments, drugs (rifampin)
BROWN/BLACK	• Myoglobin, bile pigments, melanin, cascara, iron, nitrofurantoin, alkaptonuria, metronidazole
GREEN/BLUE	• Urinary bile pigments, indigo carmine, methylene blue
FOAMY	• Proteinuria, bile salts

HEMATURIA

Type	↑ Free Hgb, myoglobin	↑ Whole cell (erythrocyte)
• Differentiate free Hgb from RBCs by centrifuging urine • (supernatant is colored if pigment is free)	• Transfusion reaction, intravascular hemolysis, burns, crush injury, tissue ischemia	• Neoplasm, coagulopathy, menses (contamination), glomerulonephritis, foreign body (especially Foley catheter), stones, renal infarct

*To further delineate the nature of hematuria, analyze the nature of the sediment microscopically

Glucosuria

ENDOCRINE	• DM, pheochromocytoma, Cushing's disease, hyperthyroidism, pancreatitis
MEDICATIONS, BURNS	• Steroids
RENAL	• Defects in tubular reabsorption mechanisms (e.g., Proximal Renal Tubular Acidosis)
IATROGENIC	• SGLT-2 inhibitors; false positive with large doses of aspirin, ascorbic acid, cephalosporins

Ketonuria

ENDOCRINE	• DKA, hyperthyroidism
METABOLIC	• N/V/D, starvation
MEDICATIONS	• Parkinsonian medications, stimulant laxatives

*Used primarily to detect acetone and acetoacetic acid, does not detect β-hydroxybutyric acid

LEUKOCYTE ESTERASE

Dependent on the presence of esterase from granulocytic leukocytes, used to detect 5 WBCs/HPF or lysed WBCs

May not be reliable in children with UTI, generally detects pyuria, not bacteriuria

When combined with the nitrite test, leukocyte esterase has a PPV of 74% for UTI if both tests are +ve and a NPV of >97% if both tests are –ve, (therefore, seen as surrogate markers for bacteria)

NITRITE

Coagulase-splitting bacteria convert nitrates to nitrite, urine must be in the bladder for >4 hrs prior to voiding for conversion, nitrates must be present

Some bacteria do not produce nitrite (*E. faecalis*)

Morning sample is preferred (stasis)

False negatives ➔ dilute urine, non-nitrate reducing bacteria, insufficient bacterial counts

PH

Range 4.6-8.0 (utility is often limited to select clinical situations)

Acidic	Basic
• High-protein (meat) diet	• UTI
• Acidosis (secondary to ketoacidosis in starvation, diabetes)	• Metabolic alkalosis • Renal tubular acidosis
• Uric acid stones	• Diet (high vegetable diet, milk, immediately after meals) • NaHCO3 therapy, vomiting

PROTEIN

Normal protein excretion is <150 mg/24 hrs (10 mg/100 mL in a spot specimen); proteinuria on dipstick requires quantification with 24hr urine studies

Dipstick protein detection is limited to albumin primarily and will not detect immunoglobulins (urine protein electrophoresis must be utilized to detect immunoglobulins)

DDx includes benign and pathological causes (see *Proteinuria* [in Internal Medicine])

SPECIFIC GRAVITY

<1.016	1.016–1.022	>1.022
• Diabetes insipidus (central or nephrogenic), pyelonephritis, glomerulonephritis, water load with normal renal function	• Average with normal fluid intake, • isosthenuria: SG fixed at 1.010, regardless of intake • suggests renal tubular dysfunction	• Volume depletion, CHF, adrenal insufficiency, DM, SIADH, ↑ proteins (nephrosis), newborn state • if markedly ↑ (1.040–1.050), artifact or recent administration of plasma expanders • radiographic contrast media

Note: Interpret in light of relative fluid status

UROBILINOGEN AND BILIRUBIN

Limited utility (dipstick analysis), bacteria in gut utilize conjugated bile to form urobilinogen ➔ cleared by the liver

Normal, low level of urobilinogen excreted in the urine daily, but hemolytic processes or hepatocellular disease can lead to increased urinary levels

Complete biliary obstruction ➔ absence of urobilinogen in the urine

1% of conjugated bile is filtered and usually undetectable, ↑ levels associated with states of conjugated hyperbilirubinemia

MICROSCOPIC EXAMINATION OF THE URINARY SEDIMENT

BACTERIA OR LEUKOCYTES	• >5 WBCs/HPF indicates significant pyuria (DDx: infection, neoplasm, calculus disease, etc.)
ERYTHROCYTES	• >5 RBCs/HPF on one occasion or >3/HPF on multiple warrants further investigation (DDx: glomerular disease, neoplasm, trauma)
CRYSTALS	• Normal patients and stone-formers will both form uric acid, phosphate and oxalate crystals; preferential formation of stones based on the acidity or alkalinity of the urine
CASTS	• Hyaline (commonly benign), hemegranular (ATN), WBC (AIN, pyelonephritis, glomerulonephritis), RBC (glomerulonephritis), fatty casts (nephrotic syndrome, DM, damaged renal tubular epithelial cells), epithelial (tubular damage, nephrotoxin, virus), waxy (severe CRF, amyloidosis)

INTUBATION & LUMBAR PUNCTURE

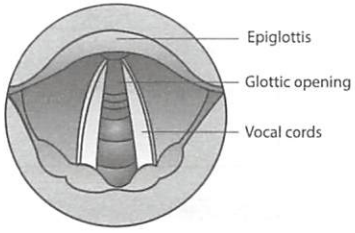
Authors: Maleka Ramji MD, Keir Peterson MD CCFP

INTUBATION

Indications

- Depressed level of consciousness such as in head trauma or after administration of general anesthetic
- Actual or impending airway obstruction
- Clinical signs of respiratory failure or fatigue; Hypoxemia
- Failure to achieve adequate ventilation with non-invasive means

Preparation



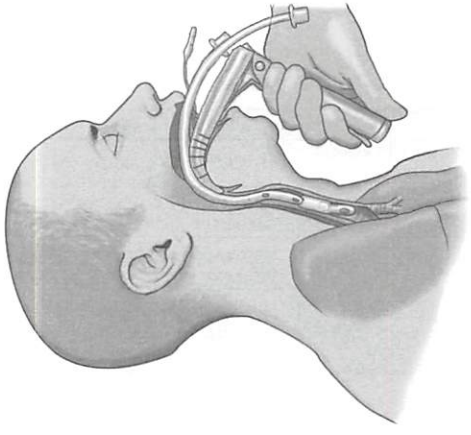
- Sedation: midazolam, propofol, ketamine
- Neuromuscular blockade: succinylcholine
- Opioid: fentanyl
- Equipment
  - Cuffed ETT; usually 8.0-8.5 mm for adult male; 7.5-8 mm for adult female
  - Laryngoscope (usually Macintosh blade size 3 or 4)
  - Suction
  - Adjuvants, such as bougies, glidescopes, or lightwands

Evaluation - LEMON

- Look – foreign object, neck size, teeth, jaw
- Evaluate using 3:3:2 rule – 3 fingers in mouth (TMJ mobility), 3 fingers from mentum to hyoid bone (mandible length), 2 fingers from hyoid to thyroid (neck length)
- Mallampati classification
- Obstruction – soft tissue swelling, foreign body, excessive soft tissue (obesity), burn/inhalation injury
- Neck mobility

Procedure

- Pre-oxygenate the patient with 100% O2, unless patient is at high risk of aspiration with bag mask ventilation
- Position the patient
  - Ensure the patient's head is level to the umbilicus of the intubator
  - Take appropriate precautions to minimize neck movement when intubating a patient with a potential C-spine injury
  - Elevate the head to the "sniffing position", so that the tragus of the ear is in the same horizontal plane as the sternal notch. This will maximize view of glottis opening.
- Hold the laryngoscope in the left hand and insert into the right side of the oropharynx; the tongue will shift to the left and up into the floor of the pharynx
- Advance the blade into the vallecula and apply force upward and forward toward opposite junction of wall and ceiling to elevate epiglottis. Do not rotate the laryngoscope using your wrist as this increases the likelihood of dental damage.
- Hold the endotracheal tube with the right hand and pass the tip through the abducted vocal cords, into the upper trachea and past the larynx. The tube should be at approximately 23 cm at the teeth for a male and 21 cm at the teeth for a female.
- Withdraw the laryngoscope and inflate the cuff
- Secure the endotracheal tube



Post Intubation

- Confirm proper ETT placement
  - Exam for symmetrical chest rise and tube condensation
  - Listen for equal breath sounds and absence of epigastric sounds.
    If breath sounds are louder on the right side than the left, this could indicate right mainstem bronchial intubation.
  - Check return of ETCO2

Chest X-ray should be performed after emergency intubation

Complications of intubation

- Recurrent laryngeal nerve injury
- Hemorrhage
- Aspiration secondary to esophageal intubation
- Tracheal stenosis
- Dental fractures
- Dislocation of arytenoid cartilage
- Vocal cord injury

LUMBAR PUNCTURE

Indications

- Suspected CNS infection (e.g., meningitis)
- Suspected subarachnoid hemorrhage
- Suspected CNS disease (e.g., Guillain-Barré syndrome, carcinomatous meningitis)
- Therapeutic relief of pseudotumor cerebri
- Administration of intrathecal therapy

CT Head prior to LP recommended if:

- age >60
- immunocompromised
- history of CNS disease
- seizures within the past 1 week
- focal neurologic abnormalities
- papilledema
- obtunded or unconscious

Contraindications

- Absolute
  - Infected skin at intended site of needle entry
  - Unequal pressures between the supratentorial and infratentorial compartments (identified through CT head)
- Relative
  - Signs of increased intracranial pressure
  - Coagulopathy (anticoagulation therapy or disorder, bleeding diathesis)
  - Brain abscess

Procedure

- Position
  - Lateral recumbent position: patient should be requested to adopt the fetal position, with back flexed, in order to widen the gap between the spinous processes
  - Sitting position: alternative position, may be preferred in patients who are obese; patient should be requested to lean forward to open the interlaminar spaces
- Landmarking
  - Palpate the posterior iliac crest and visually draw a line between the superior borders of the posterior iliac crests; this line will intersect the L4 spinous process
  - Using this landmark, identify the L4-L5 interspace for your needle insertion site (L3-4 interspace may also be used)
  - Remember that the spinal cord normally ends at around L1-L2 levels
- Preparation
  - Apply mask, gown, and gloves using sterile technique
  - Cleanse the skin with chlorhexidine three times
  - Drape the area with a sterile cloth
  - Ensure that the needle insertion site is blotted dry with a gauze pad
- Anesthesia
  - Administer local anesthesia with injection of 3-5 mL of 1% lidocaine; retract the plunger to ensure no blood
- Collection
  - Advance needle with stylet in place, aiming towards patient's umbilicus; bevel parallel to spinal column; feel for a "pop" as the dural membrane is pierced
  - Remove the stylet to check for CSF during advancement if unsure
  - Use a manometer to measure opening pressure (N = 7-15 cm H2O)
  - Collect 4-10 mL of CSF; approximately 10 drops per tube
  - Tube 1: cell count; Tube 2: gram stain, C+S, +/- fungal; Tube 3: glucose, protein; Tube 4: cell count; Tube 5: virology, mycology, cytology
- Removal of needle
  - Reinsert the stylet to avoid entrapment of a nerve root in the dura as the needle is removed

Complications

- Post procedure headache
- Infection (meningitis, discitis, vertebral osteomyelitis)
- Bleeding
- Cerebral herniation (most serious complication)
- Radicular symptoms and lower back pain (not uncommon)
- Epidermoid tumor (occurs years after the procedure is performed)
- Abducens palsy
- Trauma to nerve roots or conus medullaris

Post LP Headache

- Most common complication
- Occurs in 10-30% of patients
- More common in women of younger age
- Usually begins within 48 hrs of the procedure
- Headache is often positional: worse when upright, better when supine
- Treat with caffeine and/or analgesics
- In case of failure of above therapies, epidural blood patch may be offered

FAMILY HISTORY & PEDIGREE

Authors: Christina Beach MD, Norma Leonard MD FRCPC

BASICS

Mendelian Inheritance

- Autosomal recessive: M=F; trait expressed with inheritance of two mutated copies of gene
  - › e.g., cystic fibrosis, sickle cell anemia, Tay Sachs disease
- Autosomal dominant: M=F; only one copy required to express phenotype; on average, half the offspring of an affected parent may also be affected; may "skip" a generation if incomplete penetrance
  - › e.g., familial hypercholesterolemia, hereditary colon cancer, polycystic kidney disease, Huntington's disease, neurofibromatosis
- X-linked: M>F; son of carrier female has 50% probability of being affected, daughters of affected males are obligate carriers, daughters of carrier females have 50% chance of being a carrier, carriers usually do not express affected phenotype or have a mild form of disease
  - › e.g., Duchenne muscular dystrophy, hemophilia A

History SCREEN

- **SC:** Is there **S**ome **C**oncern of a familial disease?
- **R:** Are there **R**eproductive issues (i.e., miscarriages, infertility) in the family?
- **E:** **E**arly deaths/disease onset in family members
- **E:** **E**thnicity (e.g., Ashkenazi Jews, French Canadians)
- **N:** **N**on-genetic risk factors

Multifactorial

- Most common form of inheritance: arises from interaction between multiple genes ± environment
  - › Neural tube defects, DM, HTN

Chromosomal disorders

- Number of chromosomes
  - › e.g., Down syndrome (trisomy 21), Klinefelter syndrome (XXY)

Rearrangements: translocations, inversions

- › e.g., CML

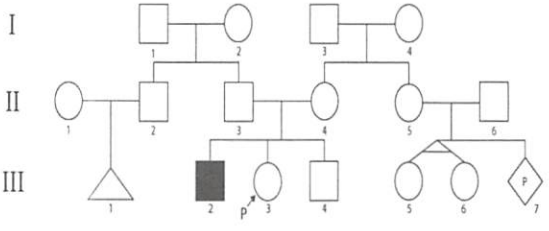
HISTORY

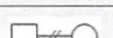
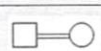
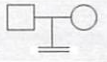
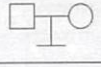
ID	• Patient's name, age, gender, ethnicity
CC	• As per concern prompting clinic visit
HPI	• Age of onset of condition, details of diagnosis, genetic and medical tests previously performed
RED FLAGS	• Physically or socially isolated populations, congenital anomalies, family history of early onset cancer or disease, presence of disease in an unlikely individual (e.g., male family member with breast cancer), unexplained mental retardation or developmental delay, advanced parental age (mother >35 y/o, father >50 y/o)
PMHX	• Hx of pregnancy (e.g., prenatal care, teratogen exposures), birth, development history, past illnesses
PO&GHX	• Multiple spontaneous abortions or stillbirths, infertility, maternal age, triple test results (AFP, βHCG, estriol), abnormalities detected on U/S
FHX	• Sibling/offspring with known chromosomal abnormality, consanguinity (i.e., parents related to each other by blood), early onset of disease/death in family members, reproductive concerns in multiple family members
SOCIAL	• Adoptions, non-paternity, EtOH and tobacco use

PEDIGREE CONSTRUCTION

Pedigree Conventions

- Mark the proband (index case) and consultands (individuals seeking genetic counseling)
- Organize family by generations – each generation on a separate line and designated by Roman numerals from top to bottom
- Label individuals with Arabic numerals from left to right in a generation line
- In a subline, arrange individuals from eldest to youngest
- In unions, the male is on the left wherever possible



PEDIGREE SYMBOLS					
	Female by phenotype		Male by phenotype		Sex not specified
	Affected individual		Carrier (obligate)		Asymptomatic/ presymptomatic
	Spontaneous abortion		Spontaneous abortion (affected)		Termination of pregnancy
	Pregnancy		Pregnancy (affected)		Multiple individuals (unknown number)
	Multiple individuals (known number)		Egg/sperm donor		Surrogate
	Deceased		Proband		Consultand
	Relationship		Relationship no longer exists		Consanguinous relationship
	Monozygotic twins		Dizygotic twins		Twins (zygosity unknown)
	Biological parents		Infertility		No children by choice/ reason unknown
	Adopted in		Adopted out		

INVESTIGATIONS

Blood Work

- Newborn screening panel (in Alberta: endocrine, amino acid, organic acid, fatty acid oxidation disorders + biotinidase deficiency, CF). As with any protocols, check with your provincial/national guidelines.

Radiology/Imaging

- Prenatal: U/S (18-20 wks)
- Postnatal: U/S, X-ray

Special Tests

- DNA sequencing, PCR, karyotyping, fluorescence in situ hybridization (FISH)

Diagnostic Interventions

- Prenatal: MSS, amniocentesis, chorionic villus sampling, preimplantation genetic diagnosis (during in vitro fertilization), fetal blood sampling by cordocentesis

TREATMENT

Emergent

- Varies according to condition (e.g., neurosurgical evaluation in patient with osteogenesis imperfecta with neurological changes, dietary interventions for patients with metabolic disorders such as PKU)

Treatment options

- Medical: enzyme replacement, pharmacological
- Surgical: Plastic Surgery (e.g., cleft palate), tumor resection, organ/bone marrow transplant

Follow-up with genetic counselor and multidisciplinary team as needed

Referrals

- Genetic counselor, Dietician, neurodevelopmental clinic, support groups, Psychologist, specialist physician (e.g., Endocrinologist)

PRE-OPERATIVE EXAM

Authors: Imran Raghavji MD, Eric Chou MD, Surita Sidhu MD

BACKGROUND

Anesthesia is required for surgical interventions in order to provide analgesia, anxiolysis, amnesia, areflexia, and hemodynamic stability. The pre-operative assessment should identify the presence of any factors which may increase the risk of perioperative mortality and morbidity, with particular attention paid to cardio-respiratory reserve. The physical exam emphasizes airway anatomy.

A pre-operative Internal Medicine assessment prior to surgery would focus on cardiac risk (would a patient benefit from cardiac investigation +/- cath), perioperative diabetes, anticoagulation, other medication management, etc.

HISTORY

ID	• Patient's name, age, gender
SURGERY INDICATION	• Anatomical region • Emergent or elective
PAST SURGICAL/ ANESTHETIC HX	• Surgical procedure and history of perioperative course (especially related to anesthesia)
PAST MEDICAL HX	
CNS	• Seizure history • Stroke and residual deficits • Increased ICP • Spinal cord disease, especially high cord lesions and autonomic dysreflexia • Neuromuscular disease (myasthenia gravis, dystrophy)
CV	• CAD • Previous MI and any interventions • CHF • Systemic or pulmonary HTN (resting BP) • Valvular disease, especially aortic stenosis • Arrhythmias • Exercise tolerance (ability to climb 1 flights of stairs = 4 METs)
RESP	• Asthma (severity, treatment) • COPD (home O2) • OSA (use of CPAP) • Restrictive lung disease • Recent infections
GI	• GERD • Hepatic disease
RENAL	• Renal disease • Dialysis (type, frequency, last run)
ENDOCRINE	• Diabetes and systemic manifestations • Adrenal insufficiency • Thyroid disease • Exogenous steroid use
HEMATOLOGIC	• Anemia • Coagulopathy • Bleeding diathesis
MSK	• Osteoarthritis (neck and back) • Diseases affecting airway manipulation: rheumatoid arthritis, ankylosing spondylitis, Down syndrome, scleroderma • Cervical tumor/infection

CURRENT MEDICATIONS	• All medications • Peri-operative management › Anti-coagulants/anti-platelets and last dose › Oral hypoglycemic agents › Steroids in last 6 weeks › ß-blockers, ACEi, ARB, diuretics
ALLERGIES	• Medication and specific reaction, latex
SOCIAL HX	• EtOH, smoking (pack years), drugs
FAMILY HX	• Malignant hyperthermia • Cholinesterase disease (pseudocholinesterase deficiency)
OTHER	• Pregnancy, obesity
FASTING	• Last time of ingestion of food or drink

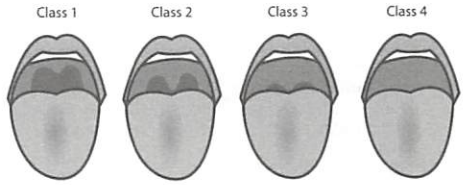
PHYSICAL

General Approach

- VS (BP, HR, RR, Temp, SaO2), Ht, Wt, BMI
- Established IVs and potential sites for invasive monitoring/regional anesthesia

Airway Exam

- Mallampati score (see figure)
- Dentition (prostheses, caps, crowns, chips)
- Mouth opening (>2 finger breadths ideal)
- Thyromental distance: mentum to thyroid notch (>6cm)
- TMJ ROM (ability to protrude lower jaw)
- Neck
  - Length, circumference, cervical anatomy
  - ROM (flexion and extension)



Cardio-Respiratory

- Inspect: JVP, WOB (work of breathing)
- Palpate: radial and carotid pulse for rate, rhythm, quality
- Auscultate for breath sounds and adventitious lung sounds, heart sounds, murmurs

Other

- Neurologic exam if indicated through surgery, history, or utilization of regional anesthesia technique

INVESTIGATIONS

Blood Work

- CBC ± type & screen; electrolytes, glucose, Cr; LFTs; coagulation studies; ß-HCG

Imaging

- ECG; chest x-ray; echocardiogram; PFTs, or spirometry

Indications for Investigations	
CBC	• Major surgery; cardiovascular, renal, pulmonary or hepatic disease; malignancy, hematologic disorder
PTT/INR	• Hepatic disease; anticoagulant therapy; bleeding diasthesis
ELECTROLYTES CR	• Hypertension; renal disease; diabetes; adrenal disease; diuretic use
FASTING GLUCOSE	• Diabetes
PREGNANCY TEST	• Women of childbearing age
ECG	• Heart disease; hypertension; diabetes; men >40 y/o, women >50 y/o; cerebral trauma; CVA
CXR	• Cardiac or pulmonary disease; malignancy; age >60 y/o

RISK CLASSIFICATION

American Society of Anesthesiologists

- ASA I – healthy, fit patient
- ASA II – patient with mild systemic disease with no functional limitation (controlled HTN)
- ASA III – patient with severe systemic disease causing functional limitation (stable CAD)
- ASA IV – patient with severe systemic disease causing constant threat to life (unstable angina)
- ASA V – moribund patient not expected to survive >24 hrs with or without surgery (ruptured AAA with shock)
- ASA VI – organ donor
- Add the E classification for patients undergoing emergency procedures

PRESCRIPTIONS & PROGRESS NOTES

Authors: Shawna Pandya MD, Brian Yong MD, Darren Nichols MD CCFP

GENERAL

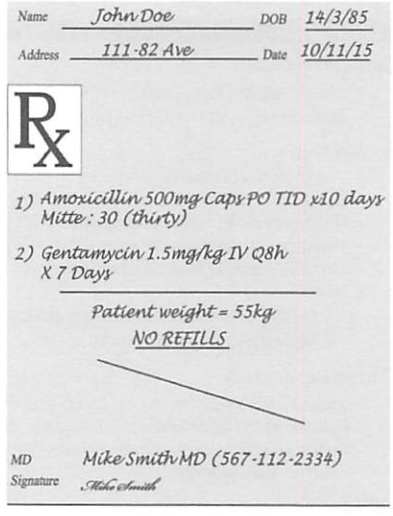
To minimize medical error, avoid using abbreviations that can be misinterpreted

- Use "daily" instead of "qd"
- Use "alternate daily" instead of "qod"
- Write "2" instead of "2.0," and "0.1" instead of ".1"
- Use "mcg" instead of "ug"

PRESCRIPTIONS

The following information should be on a prescription

1. Date
   - Time must be included if in hospital
2. Patient name and address
   - Consider writing out to prevent patient removing stickers to sell Rx'n
3. Medication Information
   - Name of drug or ingredient(s)
     - › Generic name unless using combination product
     - › Spell the drug name correctly and write legibly
   - Strength
     - › Include patient Wt for Wt-based dosages
   - Dosage form
     - › Caps, tabs, gtt (drops), crm (cream), etc.
     - › Consider putting parentheses around dosages and writing out numbers so they cannot be easily altered (e.g., consider "(30) (thirty) tabs" rather than "30 tabs")
     - › When writing dosages in mg/kg (especially in pediatrics) also write total dosage (e.g., gentamycin 82.5g (1.5mg/kg) IV q8h x 7d, Pt Wt 55kg
   - Route of administration
     - › PO (oral), IV (intravenous), IM (intramuscular), SC (subcutaneous), PR (per rectum)
   - Directions for use
     - › Daily, bid (twice/day), tid (thrice/day), qid (four times/day)
     - › PC (after meals), AC (before meals), qHS (at bedtime), qAM (daily before noon), qad (every other day)
     - › PRN (as needed)
     - › q#h (e.g., q4h, q6h, etc.)
   - Quantity (especially for PRN dosing)
     - › Mitte (send): number of caps, pills, etc.
4. Number of refills authorized (write out and circle); interval between each refill
5. Prescriber's name and phone number
6. Prescriber's signature
   - Orders written by medical students must be approved/co-signed by a physician before implementation
   - Print out name under the signature
7. Draw a line under last Rx'n to prevent others from adding additional drugs
8. Outside of hospitals – controlled drugs (e.g., narcotics) require triplicate prescriptions



PROGRESS (SOAP) NOTES

Date, time, patient ID, age, gender, admission presentation (diagnosis if known), Hx (significant comorbidities), if surgery – include # of post-operative day and any surgical complications

S – Subjective

- Symptomatic information: how the patient feels, how they are coping
- Historical information: changes since last note, new symptoms (shortness of breath, chest pain)
- Caregiver information: events in the past 24 hrs (from nurse and family members)
- General review: behavior, activity, sleep, appetite (N/V), bowel/bladder routine, pain control

O – Objective

- VS (BP, HR, RR, Temp, SaO2) (on RA or on O2), T-max, daily Wt
- General appearance (acutely ill, appears well, etc.)
- Physical exam findings (FOCUSED: usually include CVS, RESP, ABDO)
- If applicable: incision/wound (clean/dry/intact), dressings
- Ins & Outs including drains (number of/output/character)
- New laboratory results, microbiology (culture report), x-rays, pathology reports
- Allied health updates/consult reports

- Review medications, do not rewrite. Assess whether any need to be added or discontinued.

A – Assessment

- One line summary of patient's status (improving, worsening, same)
- Summary of known medical problems (optionally can add inactive issues)
- Short DDx

P – Plan

- Evaluation: additional laboratory studies and procedures needed for management of each problem
- Therapy: medical treatment, education, etc.
- Disposition: Plan for patient discharge (home, long term care, etc.)
- Note: Assessment and plan are often combined (see example)

Signature

- Sign and print name, include level of training (e.g., SI-3 for third year intern, PGY-4 for fourth-year resident), and pager number

Medicine SI Progress Note *Jan 1, 2015 1400h*

Patient ID: 70 y/o male admitted for delirium secondary to infection (urosepis vs. peri-rectal abscess) ± opioid toxicity.
PMHx – rectal tumor (currently actively being investigated, followed by Dr. Smith), rectal fistulas/abscesses, HTN, GERD, PVD.
Lives independently.

S: Patient's rectal pain still present but has decreased in severity. Mentation improving. Continued delirium (says someone was trying to sell him "dope" yesterday in hospital). No overnight concerns from caregivers and family. Patient had one bowel movement yesterday (no hematochezia or melena), normal urine output, and slept for 8 hours.

O/E: VS – BP = 137/62, SpO2 = 96% 2L, HR = 110, RR = 20, T 36.4. General – Alert. Oriented to person (not time or place). CV – N S1/S2. No S3/S4/murmurs. RESP – AE=AE. No adventitious sounds. GI – BS +, abdo soft, non-tender. No organomegaly.
U/O - 2000mL/24hrs (>30cc/hr)

Labs/Investigations: Blood culture pending. Urine culture – no growth (post antibiotics)/urine culture (Sep 17th pre-ATBx) – E. coli, CFU108/ CT abdo/pelvis (Sep 22nd) – no intra-abdominal hemorrhage. Invasion of rectal tumor into right levator ani muscle. R inguinal LN spread of tumor. R ischiorectal fluid representing possible ischiorectal abscess.
Abnormal labs: Cl-: 109. HCO3: 22. ALT: 53. AST: 86. Hb: 98. Plt: 521. WBC: 23.9. Ca2+:2.02

A/P
Active Issues

- Delirium – Improving. Secondary to urosepsis/rectal abscess/opioid toxicity. Infections being treated with meropenem (see below). Dilaudid decreased to hydromorph contin 3mg po bid and hydromorphone 2-4mg po q4h PRN.
- Urosepsis – ESBL positive. Has had ESBL positive urine for mos. Being actively treated and followed by Dr. Johnson in community. ID consulted, being treated with meropenem 500mg IV q6h.
- Rectal abscess – Elevated white count and CT suggests current active rectal abscess. ID consulted, being treated with meropenem 500mg IV q6h. Multiple rectal fistulas, abscesses and surgeries since 2005. Followed by Dr. Smith.
- Rectal tumor – diagnosed July 2013. Being followed by Dr. Anderson (Oncology) and Dr. Smith (Surgery). Currently the patient is in the process of being appropriately staged. Treatment plan is still under discussion.
- Anemia – 2 units pRBCs transfusion on admission. Cause unknown.
- Code Status – Full code
- Disposition – home once delirium has cleared

Inactive Issues

- HTN – stable. On Adalat XL 60mg po daily.
- GERD – stable. On pantoprazole 40mg po daily.
- PVD – treated with femoral-femoral bypass graft in past.

Jane Doe, SI-3
969-9999

PROCEDURE & WARD CALL NOTES

Authors: Ryan Gallagher MD, Carrie Ye MD

PROCEDURE NOTE

Date and Time

Who: who was present for the procedure, including supervising staff if applicable; with pelvic & other sensitive exams, make sure to document other staff present in the room

Consent: comment on whether consent was obtained (was patient alert or unconscious), and if complications/risks explained

Indication: purpose of the procedure

Preparation: materials, positioning, sterility, relevant investigations

- e.g., aseptic technique, freezing application, INR

Description: technique, attempts

Findings

Complications

Outcome

Specimens taken (e.g., Bx for pathology)

Instructions given to patient, family

Signature

Example: Lumbar Puncture

June 5, 2017; 1315h; Lumbar Puncture Procedure Note

- This procedure was supervised by house staff Dr. [Name]
- Written consent obtained and potential complications explained
- Indication for LP: fever and headache
- INR, PTT, and PLTs normal
- Patient in seated position and prepped using sterile technique; landmarking for L4/5 interspace done using iliac crest palpation
- 3cc of 1% lidocaine was used for anesthetic
- 20 gauge spinal needle was introduced successfully into the arachnoid space on 1st attempt
- CSF fluid was light yellow and clear; 5 cc CSF collected in 5 tubes
- Tubes sent for: cell count and differential, gram stain and culture, glucose, protein, HSV PCR
- Patient tolerated procedure well; no complications
- John Doe SI-4

WARD CALL NOTE

Common calls: chest pain, shortness of breath, urinary incontinence/retention, pain medications, drug reaction

- Date, time, service you represent
- When and why you were called
- ID: one line summary of patient age, gender, reason for admission
- Medical Hx: quick list of active medical issues + relevant PMHx
- S: Event details, HPI from patient/family/staff
- O: VS and relevant physical exam
- A: Impression (include DDx)
- P: Plan
  - Actions taken
  - Investigations ordered
  - Medications given
  - Outstanding issues/results for day staff to follow up on
- Note: every good note is followed by a turnover to corresponding staff in A.M.
- Remember to write a follow-up note once the results of the investigations ordered come back and patient has been re-evaluated

What to ask for on the Phone
• ID: name, ID #, age, location • Vital signs • Progression of problem • Patient information (e.g., SAMPLE Hx) • What has been initiated • Who else has been called

While Walking There
• Think of differential for concern • Review management of problem

Example: Chest Pain

June 2nd, 2017; 2115h; GIM Student Intern On-call

Called at 2100h by patient's nurse re: acute onset chest pain

Pt ID: 57y/o M admitted May 28th with confusion, found to have bilateral pneumonia, currently treated with IV levofloxacin

PMHx: DMT2, HTN, BPH, smoker

S: New onset chest pain around 2040h, gradually worsening-left anterior chest, 5/10, no radiation. Was lying in bed at onset of pain. No change with deep inspiration/cough/position. Has had similar pain before but has not sought medical attention. No leg swelling/SOB/palpitations. No metallic taste in mouth.

O: VS: HR 101 & regular, BP 135/82, SpO2 94% on 2L O2 (unchanged), T 37.2, RR 18, GCS 15/15

- CVS: radial pulses strong/regular bilaterally, JVP 3 cm ASA, normal S1/S2, no murmur/S3/S4, pain not reproduced on palpation
- RESP: breath sounds equal bilaterally. No adventitious sounds.
- ABDO: soft, non-tender, no masses
- Investigations:
  - This AM: Hgb 140, platelets 210, WBC 13, neutrophils 10.9. Lytes normal (K=4.1)
  - CXR (Nov 2): bilateral airspace disease, no pleural effusion/pneumothorax
  - ECG (May 28th): NSR

A: 57 y/o M with multiple cardiac risk factors, admitted with pneumonia, now experiencing 5/10 atypical chest pain.

- DDx: Acute coronary syndrome, esophageal spasm, pleuritis secondary to pneumonia, GERD

P: ECG stat, trops/CK, Nitrospray, CXR, repeat CBC-D, electrolytes

June 2nd, 2017; 2145h; Follow-up Note

S: Patient's pain decreased with nitrospray. Now 1/10.

O: Repeat VS: unchanged except HR now 85

- CXR: unchanged, ECG: NSR, no T wave or ST changes compared with previous, 1st trop (-)
- Rest of blood work pending

A/P: Patient's pain resolving, no evidence of ACS on first ECG/trop

- Repeat ECG and trop in 8 hrs
- Nitrospray PRN for pain
- Consider Cardiology consult in AM for risk stratification (MIBI or EST)

UNDERSTANDING ANTIBIOTICS

Author: Maleka Ramji MD

BASIC PRINCIPLES

ATBx only effective against bacteria

Need effective concentration at the site of infection

Need appropriate duration of therapy

Inappropriate use contributes to the development of resistance

For most infections, the goal of treatment is concentration > minimum inhibitory concentration (MIC) except where host defense is inadequate. In this case, concentration must be > minimum bactericidal concentration (MBC).

CONSIDERATIONS FOR ANTIBIOTIC SELECTION

Site

- E.g., eye/brain/prostate = non-fenestrated capillaries, which impede drug diffusion
- Low pH in abscesses inactivate aminoglycosides and erythromycin
- Ability to reach site (e.g., aminoglycosides not absorbed orally; endocarditis creates huge vegetative growths, therefore longer duration required; foreign objects such as catheters and joint replacements are prone to biofilm production, which provides a mechanism of resistance)

Immune status

- May determine the organism (e.g., *Pneumocystitis carinii* in HIV patients)
- May determine antimicrobial agent and dose (i.e., 'big guns' in immunosuppressed patients)

Hepatic and renal function

- If decreased, may require dose adjustment (drugs either renally or hepatically metabolized)
- Certain ATBx contraindicated (e.g., tetracycline in renal failure, chloramphenicol in babies due to inability to glucuronidate)

Pregnancy and breastfeeding

- Teratogenicity of certain ATBx
- Altered pharmacokinetics because of increased maternal volume of distribution, cardiac output and clearance

Medication history

- Allergies, interactions

Setting of acquisition of infection

- Country/region
- Community vs. hospital acquired, ward vs. ICU
- Contact with animals

CLASS OF ATBX	MOA	EXAMPLES	SPECTRUM OF ACTIVITY	THERAPEUTIC INDICATIONS
Penicillins	• Interfere with the synthesis of cell wall peptidoglycan • Results in formation of defective cell walls that lyse and result in death of the organism • Bactericidal		• Streptococci • some enterococci • N. meningitidis • Many anaerobes (excluding B. fragilis) • Spirochetes (Treponema) • Corynebactium diphtheriae	
		• Pen G (IM/IV) • Pen VK (PO)	• Most active against non ß-lactamase producing Gram+ organisms, anaerobes and selected Gram- cocci	• Acute Pharyngitis (Group A Strep), Group B Strep, • Syphilis, Listeria, Actinomyces
		• Cloxacillin (PO/IV/IM)	• Bulky side chain provides increased activity against ß-lactamase producing staphylococci	• Staphylococci skin infections (excluding MRSA)
		• Amoxicillin (PO) • Ampicillin (IV/IM) • Covers the same organisms as penicillin, but also covers additional gram-negative organisms	• More activity vs. enterococcus, Listeria monocytogenes, increased Gram- (E. coli, H. influenzae, P. mirabilis, Salmomella, Shigella)	• Streptococcus pneumoniae infections (sinusitis, otitis, CAP, AECOPD) (beware that resistance to penicillins is increasing), H.pylori eradication in triple therapy, prevention of infective endocarditis in susceptible patients
		• Ticarcillin-clavulanate (IV)	• Increased spectrum to include P. aeruginosa, P. mirabilis, Enterobacter, some serratia, some B. fragilis • Activity vs. non-ß-lactamase producing E. coli, H. influenzae, N. gonorrhea	• Similar to Pip-Tazo
		• Piperacillin • Pip-Tazo (Piperacillin + Tazobactam) • Gives broad coverage	• Excellent activity vs. Nisseria, H. influenzae, enterobacteraciae, pseudomonas • Used in combination with tazobactam for activity against ß-lactamase producing Gram- and Gram+ organisms	• Severe intra-abdominal infections • Severe nosocomial infections • Empiric therapy in high risk febrile neutropenic patients + aminoglycoside
		• Imipenem-cilastin (IV) • Meropenem (IV) • Ertapenem (IV)	• Broadest spectrum • Ertapenem advantage once daily dosing • Most Gram+ (excluding MRSA, enterococci), Gram- (including Pseudomonas), most anaerobes (except C. difficile) • Resistant to most beta-lactamases	• Most Gram+ & Gram- bacteria • Severe intra-abdominal infections • Severe polymicrobial skin and soft tissue infection • Fournier's gangrene • Severe ventilator-associated pneumonia where Pseudomonas and S. aureus coverage is needed

CLASS OF ATBX	MOA	EXAMPLES	SPECTRUM OF ACTIVITY	THERAPEUTIC INDICATIONS
Cephalosporins	• Bind transpeptidases, disrupt cell wall synthesis, activate autolytic enzymes and cause bacterial cell lysis • Bactericidal	First Generation • Cefazolin (IM/IV) • Cephalexin (PO)	• Increased stability to beta-lactamases produced by Staph (not active vs MRSA) • Moderate activity vs Strep • More activity vs. Gram– (E. coli, P. mirabilis, K. pneumonia, Moraxella sp.) • NOT active vs enterococci, Chlamydia and Mycoplasma, or Haemophilus species	• Surgical prophylaxis, soft tissue and skin infections (impetigo, cellulitis erysipelas caused by MSSA) and bony infections like osteomyelitis and septic arthritis
		Second Generation • Cefuroxime (PO/IV) • Cefprozil (PO) • Cefoxitin (IM/IV) • Cefotetan (IM/IV)	• Slightly decreased activity vs. Gram+ cocci, increased vs. Gram- rods, 2 subgroups: › Respiratory: increased activity vs. H. influenzae & M. catarrhalis › GI/GU: increased activity vs. B. fragilis	• CAP, otitis, sinusitis • Abd/pelvic infections, surgical prophylaxis
		Third Generation • Cefotaxime (IM/IV) • Ceftriaxone (IM/IV) • Ceftazidime (IM/IV) • Cefixime (PO)	• Enhanced activity against aerobic Gram- organisms such as Enterobacteriaceae; while retaining good activity against streptococci (except enterococci), it is less active against staphylococci • Cefazidime active vs. Pseudomonas but not good for Strep pneumoniae	• Sepsis, meningitis (penetrate the blood brain barrier), hospital acquired pneumonia • Ceftriaxone DOC for Lyme disease • Cefixime DOC for uncomplicated gonorrhea
		Fourth Generation • Cefepime (IV/IM)	• Good Gram+ & Gram- coverage • Increased resistance to beta-lactamases (inclusive of Staph and Enterobacter) • Lacks coverage of B. fragilis	• Similar to 3rd gen. • MonoRx for nonlocalizing febrile neutropenia
Aminoglycosides	• Inhibit protein biosynthesis by binding to 30S ribosome; Bactericidal	• Gentamicin (IV/IM/OTIC/OPTH) • Tobramycin (IV/IM/OPTH)	• Gram- • Pseudomonas • No activity vs. anaerobes	• UTI • Synergistic with wall active agents
Macrolides	• Inhibits protein synthesis by binding reversibly to the 50S ribosomal subunits of susceptible microorganisms	• Erythromycin (PO/IV/TOP/OPTH) • Clarithromycin(PO) • Azithromycin (IV/PO)	• Staph, Strep, anaerobes (excluding B. fragilis), • N. gonorrhea, atypical (legionella, mycoplasma, chlamydia), Bordetella pertussis	• Alternative in acute pharyngitis • 3rd line for otitis media, AECOPD, sinusitis • Alternative for superficial S. aureus infection, acne vulgaris, CAP, whooping cough, Chlamydia trachomatis & prophylaxis of ophthalmia neonatorum due to N. gonorrhea or C. trachomatis

CLASS OF ATBX	MOA	EXAMPLES	SPECTRUM OF ACTIVITY	THERAPEUTIC INDICATIONS
Tetracycline	• Inhibits bacterial protein synthesis by binding to 30S and to some extent the 50S ribosomal subunits • Alters the cytoplasmic membrane causing leakage	• Tetracycline (PO, OPTH) • Doxycycline (PO) • Minocycline (PO)	• Gram+, Gram-, anaerobes, Chlamydia, Mycoplasma, Nocardia, Rickettsiae, Plasmodium, Vibrio Cholera, Brucella	• CAP, AECOPD, acne, chlamydia trachomatis, rocky mountain spotted fever • brucellosis, Lyme disease • malaria prophylaxis
Lincosamide	• Binds to 50S ribosomal subunit of susceptible bacteria, reducing the rate of nucleic acid synthesis and ceases protein synthesis	• Clindamycin (PO/IV/IM/TOP)	• Gram+, anaerobes including B. fragilis, clostridium (excluding C. difficile), Gardnerella vaginalis	• Mixed skin infections, • 3rd line acute pharyngitis, Group A Strep necrotizing faciitis (in combo with penicillin), bacterial vaginosis
Metronidazole	• Reduced substrate, activated by nitroreductase, affects anoxic or hypoxic cells causing loss of the DNA helix and form and impairment of cellular function	• Metronidazole (PO/IV/TOP)	• Anaerobes including B. fragilis, C. difficile • Some H. pylori, amoeba and parasites (Trichomonas vaginalis, Entamoeba histolytica, Giardia lambia)	• Anaerobic infections • mixed infections, C. difficile diarrhea or pseudomembranous colitis, bacterial vaginosis, Trichomonas, • PUD triple therapy
Sulfonamides/ Trimethoprim	• Inhibits folic acid synthesis by competitively inhibiting dihydrofolate reductase and other steps in the pathway	• TMP/SMX (PO/IV)	• Gram+, Gram-, plasmodia, Chlamydia, toxoplasma, actinomyces, mycoplasma, Pneumocystic jiroveci (carinii) pneumonia (PJP) • No enterococci or anaerobes	• UTI, prostatitis, traveler's diarrhea, PJP prophylaxis
Nitrofurantoin	• Reactive intermediates alter bacterial ribosomal proteins and macromolecules	• Nitrofurantoin (PO)	• Enterococci and E. coli	• UTI
Quinolones	• Inhibits bacterial DNA synthesis by targeting types II and IV topoisomerases • Causes arrest of DNA replication and subsequent cell death	• Ciprofloxacin (IV/PO/OTIC/OPTH) • Norfloxacin (PO) • Ofloxacin (PO/OPTH) • Levofloxacin (IV/PO) • Moxifloxacin (IV/PO/OPTH) • Gatifloxacin (OPTH) • Gemifloxacin (PO) D/C 2009	• Enteric Gram- and atypical (Cipro has activity vs. pseudomonas) • 3rd and 4th gen have increased activity vs Gram+	• UTI, refractory prostatitis, bacterial diarrhea, respiratory tract infection with penicillin/ macrolide resistant S. pneumonia, AECOPD
Vancomycin	• Inhibit peptidoglycan wall synthesis by binding to precursor	• Vancomycin (PO/IV)	• ONLY Gram+ • MRSA, penicillin highly resistant S. pyogenes/S. pneumonia, E. faecalis, E. faecium, anaerobes (excluding B. fragilis)	• Severe or life-threatening staphylococcal infections when MRSA is suspected (cloxacillin is the drug of choice for Staph aureus) • Vancomycin PO ONLY for the treatment of antibiotic associated pseudomembranous colitis produced by C. difficile

PRE- & POST-OPERATIVE ORDERS

Authors: Patricia Lee MD, Sarah Lai MD, Kamran Fathimani MD FRCSC

PRE-OPERATIVE ORDERS: ABCDEFGHI MNEMONIC

Date, time, planned procedure, diagnosis

- Antibiotics: Ancef (Cefazolin) ± Flagyl (Metronidazole), Clindamycin ± Ciprofloxacin (on call to OR vs. STAT)
- Blood Work: CBC-D, electrolytes, BUN, Cr, INR, PTT, type & screen or cross-match
  - Bowel prep: laxatives, enemas, suppositories
  - Booking the case: call OR
- Consent (informed): discuss potential benefits, risks, and alternatives of surgery with patient and family
  - Consults (if indicated): Anesthesia, Internal Medicine, ICU, Cardiology
- Drugs: previous medications, cardiac medications (continue ß-blockers and statins; hold diuretics and ACE inhibitors), anticoagulation (hold Coumadin™, ASA, Plavix™, therapeutic IV heparin/SC LMWH)
- Eating/drinking: NPO after midnight
- Fluids: IV NS or RL maintenance (4:2:1 rule; approximately 100-125 cc/hr)
- Glucocorticoids: If patient on long-term steroids give stress dose steroids
- Heparin: DVT prophylaxis (Heparin 5000 units SC 2h before OR); hold if neuraxial anesthesia is expected
- Imaging
  - CXR: >50 y/o or previous abnormal within 6 mos
  - ECG: >50 y/o or as indicated by history

POST-OPERATIVE ORDERS: AD-DAVID MNEMONIC

Date, time, admit to surgery, diagnosis

- Admit to surgery under Dr. _____
- Diagnosis
- Diet: NPO, ice chips/popsicles, TPN, PPN, CF, FF, DAT, tube feeds, diabetic diet, cardiac diet, renal diet
- Activity: AAT, NWB, WBAT, bed rest, full spine precautions, HOB >30°
- VS: q2h, q4h, routine
- IV: NS, RL, etc.
  - I&O: q2h, q4h, qshift
  - Investigations: (depending on surgical procedure)
    - CBC-D, electrolytes, BUN, Cr
    - LFTs: AST, ALT, ALP, bilirubin, lipase
    - INR, PTT
    - CXR or necessary imaging
- Drugs:

CNS	• Antiemetics, analgesics
CVS	• Anti-hypertension, cardiac meds
RESP	• Inhalers, O_2, incentive spirometry
GI	• Bowel routine, PPI
ID	• ATBx
DVT	• Prophylaxis: heparin or LMWH, pneumatic stockings

- Dressings:
  - Closed wound: dry dressing PRN
  - Open wound: saline soaked gauze bid/tid
- Drains: JP to bulb suction, NG to low intermittent suction, Foley to urometer, chest tube – 20 cm H2O suction

ECG (EXAMPLES)

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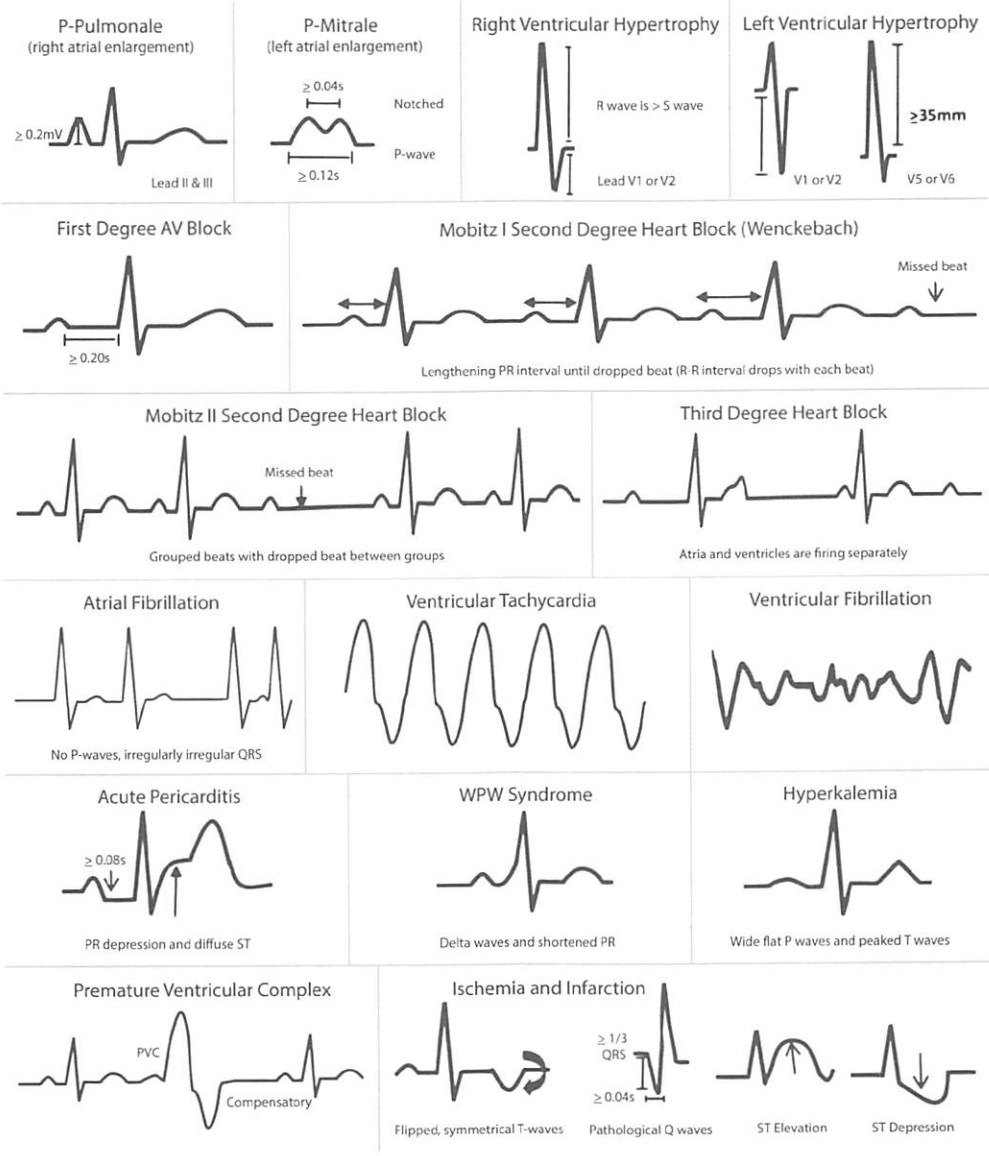

STATION CONTRIBUTORS

Approach to Acute and Chronic Pain Kirles Bishay MD, Kirsten Biefer MD Maryam Soleimani MD

Essential Dermatology Nathan Hoy MD, Isaiah Day MD, Alain Brassard MD FRCPC

Intrepretation of ABG Charles Lim MD, Edward James PLT, Janjeevan Deol MD, Dilini Vethanayagam MD FRCPCC

Intrepretation of Chest Radiograph Babak Maghdoori MD, Christopher Fung MD, Ed Wiebe MD FRCPC

Interpretation of CBC-D Kimberely Krueger MD, Jason Kiser MD, Mark Joffe MD FRCPC

Interpretation of CT Brendan Diederichs MD, Katherine Leung MD, Ed Wiebe MD FRCPC

Interpretation of Electrolytes: Calcium Charles Lim, Craig Domke MD, Lucille Lalonde MD FRCPC

Interpretation of Electrolytes: Potassium Kimberely Krueger MD, Jason Kiser MD, Mark Joffe MD FRCPC

Interpretation of ECG Charles Lim, Craig Domke MD, Lucille Lalonde MD FRCPC

Interpretation of PFT Maleka Ramji MD, Mohit Bhutani MD FRCPC

ECG Examples Odell Pui MD, Sabira Suleman MD, James Huang MD

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