

Tool Box

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Assessments; remember your ABCs!

What will your client look / sound like if they are having airway problems?

What will your client look / sound like if they are having breathing problems?

What does skin look like with no circulation?

(Whether the cause is from epinephrine causing vasoconstriction or from a blocked artery)?

What would skin look like if I blocked a vein that drains that part of the skin?

Must know normal parameters for BP, Pulse, Respirations, Temperature, Pulse Oximetry

Lab Values every SN should know!

Fluids / Electrolytes:

What are the electrolytes? Na⁺, K⁺, Ca⁺⁺, Mg⁺⁺

(Abnormal K⁺ causes cardiac problems/abnormal Na⁺ causes CNS problems)

Water likes to follow sodium (Na⁺)

Be able to describe oncotic pressure (plasma proteins pulling water in their direction)!

Be able to describe active transport of Na⁺ and K⁺ and where their concentrations are highest at rest?

❖ We want Na⁺ in ECF normal serum lab 136-146 mEq/L

❖ We want K⁺ in ICF normal serum lab 3.5 – 5.1 mEq/L

❖ We don't want Ca⁺⁺ in ICF normal serum lab 8.6 – 10.1 mg/dL

We want it hiding (outside the cell or in the endoplasmic reticulum) if it is unduly released it will activate a lot of enzymatic activity that can cause the cell to stop function and dissolve!

(Apoptosis = cell death)

❖ We want an optimal level of Mg⁺⁺ in the ICF (inside the cells) normal serum lab 1.3-2.1 mEq/L

Anticoagulant/bleeding times:

❖ PTT (APTT), PT/INR---if these go UP you will bleed EASIER

❖ Platelets - if the PLT level goes down, you will bleed easier

❖ D-Dimer - clot related lab

❖ Norms PT 11-15; PTT 60; INR 1; Plt 150-450

Hematology:

Indicators of oxygen carrying capacity

❖ Hgb normals M= 13.5 - 17.5 F= 12.0 -16.0

❖ Hct normal M= 39 - 49% F= 35 - 45%

❖ RBC counts M= 4.3 - 5.7 million / mm³ F= 3.8 - 5.1 million / mm³

Pancytopenia = bleeding, anemia, infection (mouth sores)

Bone marrow or other RBC replacement function

❖ What is the Platelet level? Thrombocytopenia = too few platelets; affects ability to clot

❖ MCV – Mean Corpuscular Volume normal 80-100 fL

Macrocytic anemia = B12 or Folate deficiency (may be pernicious anemia) – High MCV

Normocytic anemia = bleeding

Microcytic anemia = IDA (iron deficiency anemia) – Low MCV

Indicators of infection

❖ WBCs normal (varies with age) 4,500 – 11,000 / mm³

Leukocytosis = WBC >11,000 (infection or leukemia depending on how high it goes)

Neutropenia = WBC < 1 (1000 in some places) – at BIG risk for infection

Liver Function

- ❖ AST/ALT --- SGOT/SGPT

Enzymes from hepatic cells, *an elevation in these labs means that the liver is in trouble*
Hepatotoxicity manifests as lethargy and jaundice

Renal Function

- ❖ BUN / Creatinine(Creat)

Normally the kidneys can keep most of these leftovers cleaned out of blood.

If these are elevated in the blood that means that the kidneys are sick because they aren't doing their job

- ❖ Proteinuria (urinalysis) **We normally don't have protein in our urine.**

If we do, that may mean that the kidneys are sick!

Cardiac Enzymes

- ❖ CK, CK-MB, Troponin

Enzymes from heart cells, **an elevation in these labs means that heart cells have died!**

Diabetes

- ❖ HgbA1c = glycosylated hemoglobin (lab test) i

Indicates the average blood sugar over the course of 2-4 months

- ❖ **A1c normal = < 6, 6 -7= ideal diabetic controls > 8 requires med/diet review**

- ❖ **Glucose normal = Fasting 70 – 100 (Diabetic > 130)**

90 min post 100 – 140 (Diab > 180)

Nutrition

- ❖ Serum albumin level (an indicator of nutrition status)

If too low, get edema (especially in periphery) because of low serum oncotic pressure

- ❖ Normal level in men and women >3.5 g/dL

CRITICAL REMINDERS;

Anti - coagulant Therapy

Thrombosis- patient is admitted for a clot (brain, leg, lungs, heart) they are started on heparin and coumadin.

- ❖ **Watch the PTT**

- ❖ **Therapeutic(good/desirable) INR** for someone with a clot or history of clot is **2-3**

- ❖ **NORMAL INR** for someone with no clot or no history of clot is **1**

Coumadin / Warfarin - Anticoagulant (PT/INR)

- ❖ K Coumadin takes a few days to get the INR to a therapeutic level (and you still have to monitor)

- ❖ Antidote is vitamin K

- ❖ What safety precautions do these people need to take?

Use an electric razor,

Limit the green leafy vegetables-high in Vitamin K,

No contact sports,

Watch injuries closely for excessive bruising / prolonged bleeding

Use a soft bristle tooth brush)

- ❖ Takes longer to clear than heparin (up to one week)

Heparin/Enoxaparin - Anticoagulant (PTT) - No heparin with a PLT < 100

- ❖ Advantage over coumadin; starts to work AND metabolized quickly – stops soon after med is discontinued

- ❖ Antidote is protamine sulfate

- ❖ What safety precautions do these people need to take?

Same as Coumadin

Aspirin - Anticoagulant anti-platelet (even in lo-dose; 81 mg daily preventive)

- ❖ Suspect a heart attack – give an aspirin! (outside of hospital)
- ❖ GI upset (ulcers?)
- ❖ GI bleed - anybody with a GI bleed or **history of GI bleed should not use aspirin or other NSAIDS** (ibuprofen)

SURGERY - if the patient has surgery coming up we need to stop the anticoagulants / anti-platelet meds

COMMON DIAGNOSIS DRUG REMINDERS

CHF – How do we deal with the fluid overload? (Nancy McMahon, RN, BSN, 9-10-03)

- ❖ **Pump it** – digoxin is a positive inotrope to help push it along.(Slow HR – pump more effective)
- ❖ **Park it** – nitroglycerin will cause massive veno-dilation so decreases the amount of fluid the heart has to pump w/each beat
- ❖ **Pee it** – diuretics to get rid of the fluid
- ❖ Dopamine is a potent vasopressor (it makes arteries constrict) So it will increase _____???

BNP (stands for Brain-Type Natriuretic Peptide)- if this is elevated your patient has CHF (congestive heart failure)

ACE inhibitors - the nurse must ask what is the K+? **ACE inhibitor will make the pt hyperkalemic.**

- ❖ Lisinopril, fosinopril, captopril, enalapril
- ❖ This drug inhibits the Renin Angiotensin system which is a major stimulator of Aldosterone
- ❖ **Aldosterone causes us to RETAIN Na+ and lose K+ (hypokalemia)**
- ❖ Lack of aldosterone (which can be produced in response to hyperkalemia) can lead to hyperkalemia

Diuretics

- ❖ **Loop – furosemide - Lasix** (what is the K+?) the patient may become hypokalemic
- ❖ **K+ Sparing diuretics – SPIRONOLACTONE (Aldactone)** is an aldosterone antagonist
Spironolactone is the opposite of furosemide
- ❖ **Watch blood pressure and lytes (Na+ and K+) with any diuretic administration**

Digoxin - **what are the pt's PP? (pulse and potassium)**

- ❖ Digoxin, helps increase the force of contraction of the heart, it is a positive Inotrope
- ❖ **do not give with pulse less than 60 unless MD orders**
- ❖ **do not give with a potassium less than 3 without MD order**

Beta Blockers –

- ❖ Atenolol, Propranolol, Labetalol, Metoprolol
- ❖ **What is the pulse?(do not give with pulse less than 60 unless MD orders)**
- ❖ **Watch use of beta-blockers in diabetics.**
Part of the response to hypoglycemia (because we gave them too much insulin) is the SNS response mostly attributed to epinephrine.
SO If we use beta blockers in these people, **they may have very very very low blood sugars before they experience a hypoglycemic warning** (tremors, tachycardia, diaphoresis, etc.)
- ❖ **Watch use of beta-blockers in asthma.**
If we block beta adrenergic stimulation in a person with asthma, **we will inhibit bronchodilation!**
- ❖ **Albuterol (COPD) will make your patient tachycardic (Sympathetic drug)**
It is a B2 agonist (it stimulates the sympathetics on the lungs and heart)

- ❖ **Ipratropium will make your patient tachycardic (Parasympathetic drug)**
It is an anticholinergic (it blocks the parasympathetics on the lungs and other places)
- ❖ **Atropine (t-tachy) is the opposite of adenosine (d- down) ---- pulse**
- ❖ We put small amounts of epinephrine in SC lidocaine that we use to numb a localized area.
The reason for this is that the epi causes local vasoconstriction and therefore the lidocaine is not washed away by blood flow and we get the local anesthesia that we want.
Epi should not be used in pinna, fingers, toes - WHY?

CANCER

CA Chemotherapy acts on rapidly dividing cells –

- ❖ these include hair, mucosa (oral, GI), WBC, PLT, RBCs.

Immunosuppression - means that the patient is at risk for opportunistic infections - fungal, herpes, tuberculosis

- ❖ **Steroids** (prednisone, given IV as hydrocortisone and methylprednisone) anti-inflammatory
“**Solu-medrol,**” “**Asthmacort**”
Prednisone - will send blood sugar up (why the hyperglycemia?)
Increased appetite, weight
ANTI-inflammation and ANTI-Immune
Used to prevent rejection of a donated organ
Used to stop an inflammatory response (asthma, Systemic Lupus Erythematosus)
- ❖ **Cyclosporine, ALG** (anti rejection in transplant)
- ❖ **Chemotherapy** (cancer treatment)
"cortisol" Stress hormone is (our drug that looks like cortisol is prednisone/also causes hyperglycemia)

PAIN

Any narcotic (sedating)

- ❖ Think safety (will they trip and fall while on the drug?) and constipation.
- ❖ Why do people with chronic pain need to be on a high fiber diet or stool softeners?

INFECTION

- ❖ **Antibiotics** - kill even the good germs/bugs in your gut so you get diarrhea
Killing these good germs/bugs allow for fungal infections to happen because the normal bacterial flora competes with fungi for food. Competition is gone? - fungal infections go nuts.

THYROID

- ❖ Synthroid/levothyroxine for hypothyroidism.
- ❖ **TSH is High - we need more T3/T4.**
- ❖ **TSH is low - we need less T3/T4**
Common lab to check is TSH level. Will check a thyroid level if needed

DIABETES

- ❖ Even non-diabetics may run higher than normal BG when traumatized – may receive insulin (see – cortisol)
- ❖ **Insulin:**
Basal - NPH – long acting
Insulin glargine - a CLEAR LONGACTING (weird) insulin that can NOT be mixed with anything.
lasts for 24 hours and has NO peak (all other insulins have a peak)
Prandial (pre) – Aspart (Novolog) - 0-15 min before eating
Regular (R, Novolin) – 30 min before eating
- **Clear to Cloudy** - when insulins can be mixed (only regular and NPH) always draw up the regular (clear) insulin first. Make sure to check your drug guide before you mix any insulin.

- ❖ The counter-regulatory hormones (to insulin) are (Cortisol, Epi, Glucagon) **So** what would the symptoms of hypoglycemia be? Hmmmmm..... (hint - adrenergic)

HYPOGLYCEMIC reaction - give IV (50 gm) or PO dextrose and/or subcutaneous glucagon

HYPERGLYCEMIC reaction - In diabetic ketoacidosis, why do we have to give IV fluids, potassium and insulin? (Remember how we can reduce serum potassium????)

PARKINSON'S

- ❖ L - Dopa to replace this potent neurotransmitter
- ❖ Parkinson's (too little dopamine) is the opposite of Huntington's (too little acetylcholine)
- ❖ Dopamine - Low dose has RENAL action / High dose cardiac action
- ❖ Dopamine is a potent vasopressor (it makes arteries constrict)

Antiparkinsonian agents are divided into cholinergics and dopamine agonists. Cholinergics work by the blocking or competing at central acetylcholine receptors; dopamine agonists work by decarboxylation to dopamine or by activation of dopamine receptors; monoamine oxidase type B inhibitors increase dopamine activity by inhibiting MAO type B activity.

(Skidmore-Roth, Linda. *Mosby's Drug Guide for Nurses*, Elsevier)

OCULAR Drugs (Glaucoma)

- ❖ Cholinergics – Parasympathetic promoters; work on cholinergic/nicotinic/muscarinic receptors. (The opposite of drugs like bronchodilators.)
- ❖ Beta-2 Adrenergic agonists – “Alphagan” - DO NOT USE w/ MAOI
- ❖ If you overdose on a MAOI (monoamine oxidase inhibitor) you could have a hypertensive crisis

Acetylcholinesterase is to ACh just like Monoamine oxidase is to Norepinephrine and dopamine (2 hormones)

Norepi could stay around longer and cause prolonged sympathetic stimulation

This decrease in Dopamine and Norepi **DEGRADATION** means that their action will be increased!

Why are sympathomimetics called sympathomimetics and sympatholytics called sympatholytics?

GI – Primarily antacids

- ❖ H2 blocker - **ranitidine** decreases gastric acid production by blocking the histamine 2 receptor in the gut. can be used for a long time
- ❖ Proton pump inhibitor
Pantoprazole / omeprazole
inhibits the gut from pumping protons into the stomach (a proton is H⁺ = acid)
only to be used for one month
- ❖ Motility – prokinetic (For a sluggish stomach / bowel – move food out faster)
metoclopramide
encourages upper GI motility (given to diabetics with autonomic dysfunction of the gut)
suppresses emesis - given for N/V

Any GI upset, ulcer - need to be thinking of NSAID cause (ASA, Ibuprofen) (APAP or Acetaminophen does not fall into this category) - why not?