

Labs

I must be out of my mind. Am I confused? Would I know? Who in their right mind sits down and writes things like this for the fun of it? Did I get all my meds for today? Can I at least have one hand loose?

This one took a while to put together, as it required a lot of looking-up on the part of the preceptor, something he often doesn't have time to do at work. Got to love the web! As usual, please remember that this article is not meant to be the final word on anything, or even comprehensive in any way. Nurses at the bedside have to work on the fly, and the things that they need to keep in their heads have to be practical and brief – not that this article is very brief, but hopefully the items are. This information is supposed to reflect what a preceptor might teach a new orientee, or maybe to answer some of the questions that the orientee might come up with. Each item in this article is backed up by (apparently) an average of not less than eight thousand pages of reference material in 37 different languages – I just tell what I know! **Please make sure that you check your own references to verify lab/drug and toxic ranges!**

Let us know when you find errors, and we'll fix them up right away. Thanks!

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1- What are some of the labs that we follow on our patients in the MICU?

There are a lot of labs out there, and they come in a wide variety of flavors. If you never got comfortable with frequently looking up lab results on the floors, you're probably going to have to get over that one quickly, since watching trends of one kind or another is about 90% of what we do in the ICU.

The basic idea is often really easy: if some lab value is way out of line, then something having to do with the patient probably is too. Doh! You don't want to be wrong about this, which is why the team will sometimes ask you to re-send a spec. Which of course is frustrating when you think that your GI-bleed patient isn't losing his blood pressure because he forgot to drink his Gatorade this morning or something...

Remember that basic physiology thing about how the body is made up of subsystems? That sort of basic sort of thing? The labs reflect those systems and how they're doing (or not doing) at whatever it is that they're meant to do. Simple example that comes to mind: if the kidneys aren't clearing nitrogenous wastes from the blood, then the levels of those wastes will rise – makes sense to interpret that as kidney failure, right?

But nothing is ever as simple as you'd like it to be. My son and I just bought an elderly motorcycle...(what do you mean, "don't talk about the motorcycle"?...what do you mean, "it has nothing to do with the topic"?...it's got plenty to do with the topic...you're just jealous, 'cause...what do you mean, you "wouldn't get on that thing even if..."...so we had two quads in the unit last month, so what?!)

Anyway, for the ICU newbie there's lots to learn, as usual, and also as usual the best thing is just to try to get some idea of what you're looking for, and then to accumulate mileage and experience – then the things that you learn by reading will make lots more sense. Don't try to memorize it all at once – come back and re-read this article a year from now. This is especially true when it comes to motorcycles. See, the float bowl in the carburetor...ow!

2- Chemistries:

There's lots of chemistries out there, but the basic ones are always easy to get, and can give you lots of clues about what you're looking for. Maybe I can get one of the kids to draw the little diagram thingy.

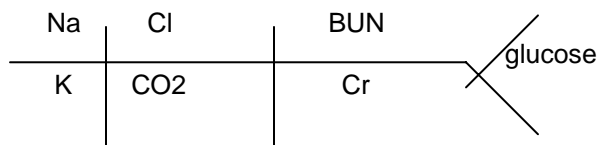
Now here's the thing – every day these kids come home from school:

"Hey kids, whad' ya learn at school today?"

"Nothing." And man, you can sit there and ask them about school until your jaw just drops right off, but they just won't tell you a thing. Then later, daughter # 2 wanders by where I'm struggling to do some (probably) really easy thing with the word processor, and she says: "Dad!! Use a text box!"

The preceptor: "What's a text box?"

D # 2: "Here, just get up and let me show you." Eight lightning moves follow, a nice box or line drawing (as below) appears, and I'm still in the dark. Nice drawing, but still in the dark. I never did that to my parents, not once. Ever. Well...once maybe with the cable box.



Right – this is the little electrolyte drawing gatsby, which makes it easy to remember the values that you want to write down someplace quickly, like on your scrub pants. This is one of those doctor-ish things that nurses hate, but actually (like lots of other things) it isn't hard to learn at all – seven items? And you use them all the time anyhow, or most of them anyway – and it makes things easy to write down.

Let's take these guys one at a time, and please remember that all this info is strictly "from the hip" - I mean, you can keep on going and going with this stuff, and pretty soon you're an endocrine fellow or something. So all this stuff is "with a lot of lies thrown in", as they say.

2-1: "Basic Chems"

2-2-1: Sodium / Na+ (135 - 145 meq/l):

Sodium is confusing – like lots of things in the physiology world, it doesn't always do what you think it's going to do, or what you want it to do. I guess lots of things are like that. In fact, the motorcycle ...ow!

The basic idea is that sodium is a solute, floating around in the serum solvent. If Mr. Shmulewitz has a TIA and lies on his bed for three days before his bum of a son-in-law comes to check on him, he's going to get very – what? Very dry – dehydrated, mostly from "insensible loss" - I think that you lose something like a liter and a half every day this way, mostly through breathing and sweating. And that's when things are normal – imagine what happens to marathon runners. No wonder they don't look so good at the end. "Pruned."

Anyway, if some of the solvent goes away, that leaves more solute in what's left, correct? So if you measured Mr. S's sodium before his TIA, it might've been something like 138. After three days of not drinking anything, it might be in the 150's. Too high! All sorts of unpleasant things can happen – seizures, drain damage, renal failure (why?), and so on.

Here's a formula for figuring out exactly how dry they are (the water they should have, but don't have, is the "Free Water Deficit". Although if you're using Poland Springs, it ain't free – what then, huh, smart guy? Some people think they know everything...)

Jayne: "You realize that stuff isn't even slightly funny, right?" - Self: don't destroy a man's dreams dear, they're all we have.

$$\text{Free Water Deficit} = (0.6 \times \text{pt's weight in kg}) \times [(\text{pt's sodium} / 140) - 1.0]$$

So let's try it: say the patient weighs 70 kg, and his sodium is 160 (oof – he's dry!)

First part: $0.6 \times 70\text{kg} = 42$

Second part: His sodium is 160, divide that by 140, that gives 1.14. Subtract 1.0 from that, you get .14

Last part: $42 \times .14 = 5.9$ liters. Call it six liters. That's about 13 pounds.

That's a lot of liters, in case anybody's counting. Try it sometime.

What about other way? What if Shmulewitz turns out to be one of those people who insists on drinking eight glasses of water a day? (Like my dad.) And what if his doctor puts him on a diuretic, say bid for his swollen ankles, because he won't stop drinking them ("gotta flush the kidneys!")? (This next part is probably mostly lies, but it was explained to me this way once) – it turns out that the loop diuretics make you dump not just potassium, but all the other cations that float around dissolved in the serum : sodium and hydrogen come to mind. In fact whenever you hear the word "diuretic", you should immediately respond in your head with "K⁺!". (Make sure their creatinine is okay before you give any.)

Apparently people dump enough sodium in urine in response to diuretics to cause a significant drop – actually, I was told that you pee half-normal saline. What if you now replace the lost volume with pure water – tap water, or bottled? No electrolytes in it at all. You can see what's coming, right? - having dumped lots of sodium, Mr. S. now takes in lots of solute; both of which maneuvers make his sodium drop a whole lot. If the solute levels get too low, water may start moving into the third space ("Head for the third space, Mr. Spock." – One eyebrow goes up: "Um, captain, can I pee first?"). Gatorade! (Who's that Picard guy, anyhow?)

(Losing a lot of hydrogen can produce bad things too – several days of diuresis will usually produce an alkalosis, because it leaves a lot of bicarb floating around with no hydrogen dancing partners – they all got peed out. Because the patient's fluid volume has "contracted", they call this a "contraction alkalosis".)

"Third - spacing" of fluid into the brain tissue in response to hyponatremia can result in a rising intracranial pressure. I hate it when that happens – all sorts of unpleasant things can result, right? Including, possibly, herniation. Ack! Quick now – what's the first sign of rising intracranial pressure?

Back to the patient. So – what to do? Hypernatremia usually means that a lot of circulating volume has been lost – give some back! Hyponatremia? - got too much volume going around? - Restrict fluid intake for a few days and the patient should straighten out. Might want to give some hypertonic saline, usually as 3% saline, in case overdiuresis or something has caused too much sodium loss.

Now comes an extremely important thing. Try very hard to remember this. Can anybody pronounce the following?: "**Central Pontine Myelinolysis**". This is a truly awful result of **too rapidly correcting hyponatremia**, in which crucial parts of the pons (in the midbrain, is it?) become de-myelinated. Stripped. Leaving the patient possibly quadriplegic, possibly comatose, possibly (shudder) "locked-in". Oh yeah, and maybe dead.

Central pontine myelinolysis happens when hyponatremia is corrected too quickly.

Apparently it is **entirely avoidable**. The way we do it nowadays is: treat the patient cautiously with IV fluids (the team will have all sorts of groovy calculations to do here involving weight, renal function, age, and probably the color of the socks the patient was wearing on admission), but the main thing is that the **sodium must not be corrected faster than one mEq per hour**. Which means sending electrolytes every hour. Make out those slips!

2-1-2: Potassium/ K (3.5- 5.0):

Critically important, especially for the heart. Take a look at the section below on “cardiac labs” for more on the subject of electrolyte repletion.

Remember a couple of things:

- Potassium can be tricky to give. It's very irritating to the stomach – nauseated patients will not be able to take it po. A conversation I heard once involving a young doc:
 - Nurse: "I'm not sure you want to give this patient potassium by mouth y'know, he's got an empty stomach and doesn't feel too well."
 - Doc: "How else could we give it?"
 - RN: "Well, he's only got peripheral IV access, so we can only give him 10 meq per hour in a dilute mix of eighty in a liter – that's 125cc/hour. You could put in a central line to give less volume."
 - Doc: (appalled) "But we have to diurese him! He's in CHF, and the fluid overload is causing increased hydrostatic pressure to progress retrograde from the LV into the pulmonary circulation of the whangbang kabam and the elang badoodang doodah day!"
 - Outcome: the patient vomited his potassium. Got a bed bath and a central line.
- IV potassium can only be given at fixed rates. Peripherally (try to avoid this, since you can really injure someone's arm if this stuff infiltrates) we can give it as described above. Centrally we can give 20meq per hour.
- IV potassium must be delivered on a pump. No exceptions. If it goes in too quickly, as with many drugs, disaster may result.
- Keep an eye on the patient's creatinine – a failing kidney will not excrete potassium at a normal rate, and your patient may end up with a K way higher than you wanted. And if it looks like your patient is heading into renal failure, you might not even want to replete a low K at all.

2-1-3: Chloride (95 – 105 meq/l):

Reader, sadly here your preceptor fails you. I don't know hardly nothing about chloride, except that if a patient is fluid resuscitated with many liters of normal saline, (and each of those Na's carries a little Cl along with it), the patient can develop "hyperchloremic acidosis". Of course there's a zillion and a half other things that I'm sure you ought to know about this ion, including the fact that hyperchloremia seems to show up in most metabolic acidosis's, but your preceptor has totally dropped the ball on this one. (Hangs head in shame. Then - remembers motorcycle. Happy again!)

2-1-4: Bicarb (22 – 29 meq/l):

This is a confusing one for those of us who only remember biochemistry as a bad memory. And I have a really bad memory in general. Bicarb is also described as carbon dioxide, probably because they associate with each other in the carbonic acid reaction – I seem to remember

arrows pointing both ways, indicating that the reaction could go forwards and backwards. (At the same time? I had a car like that in nursing school.)

The important thing is that this number, whether expressed as serum bicarb or as serum CO₂ (not pCO₂, which is something else), indicates the amount of bicarb present in the blood, available as "buffer".

This gets into acid-base balance, which I think is going to require an article of its own! Meantime, take a look at the section below on "Anion Gap" – yeah, like I understand that stuff – under "Respiratory Labs".

2-1-5: BUN (10- 26 mg/dl):

"Blood Urea Nitrogen" represents the amount of nitrogenous waste in the blood, which is supposed to be cleared by the kidneys. The BUN number always travels accompanied by its partner **2-1-5-1: creatinine (0.6 – 1.3 mg/dl)**, but it's the creatinine number that is actually telling you directly how well the kidneys are working, since a rising BUN by itself can just indicate dehydration, just like a high sodium can. (A high admission hematocrit can be a clue too. Also the prunelike appearance. Some of us just look that way at baseline. Sigh.)

The thing to remember is that it's the creatinine that indicates if the kidneys are in trouble or not. High is bad. Someone told me once that if the creatinine increases by one whole number, it represents the loss of a third of the patient's kidney function, which means you can't do that very often!

So look at the ratio: a high BUN with a normal creatinine means a dry patient whose kidneys are still okay – if she gets hypotensively dry, her kidneys may become unhappy as a result of being under-perfused. Something like BUN of 70 with a creatinine of 1. High ratio. If the creatinine starts to rise, then real trouble is coming, because the kidneys are getting into trouble at the tissue level, maybe in the form of acute tubular necrosis, never a picnic. Might look like 70 / 3.0 – higher numbers, lower ratio. Comparison is everything, so take a look at a couple days' worth of chems and see if the creatinine has been going up, down or sideways.

Something we've noticed over the years: it seems as though alcoholic patients come in with very low BUN numbers, like 4 or 6. Somebody's opinion was that alcoholics will often drink instead of eating, so their muscle mass isn't very good, and they don't make as much BUN as the rest of us.

2-1-6: Blood glucose (70 – 115 mg/dl):

This is where I hope I don't get into trouble – I was diagnosed with Type 2 DM a couple of years ago, and I'd really like to keep my kidneys, if it's all the same to everyone else. This has given my kids an excuse for pulling various kinds of food right out of my hands with the look of a mean teacher catching a kid eating, uh, candy? I wonder where they learned that? Tight glucose management has gotten a whole lot of recognition lately as critically important in managing really sick people. It turns out from the studies that all sorts of things happen for the better if a patient's glucose is kept under tight control – as a result we've started to use insulin drips a whole lot in our MICU. Apparently everything is affected, from wound healing to recovery from septic infection, to length of stay, etc. We run insulin drips at rates of something like one to ten units an hour, checking glucose with either chems to the lab or glucometers every two hours, with a goal range of 80 to 140.

DKA patients obviously come under the frequent-glucose-check category. These people often require changes in IV fluid treatment every couple of hours, and we check their electrolytes every two hours the way we do with any other patient on an insulin drip. We don't use the same

protocol though – DKA people get NPH somewhere along the line, while the others may or may not.

2-1-6-1: Acetone (positive or negative):

The patient's need for insulin does not go away once their blood glucose comes down. Ketoacids can hang around for a long time afterwards – maybe another 12-24 hours, it seems to me. Continued insulin treatment is what will help the patient cook off the ketoacids, so once the glucose gets down to a reasonable level, the IV hydration fluid usually changes from something like normal saline with some K to something like 5% glucose with K – the glucose keeps the blood sugar up now, while the ketoacids get fixed by the insulin drip which continues to run. Lately they've started using NPH when the glucose numbers get down towards normal, but as with all innovation, I disapprove. Getting old. Change is bad. I give the NPH when they tell me to.

We check the acetone level every four hours until it goes negative. You'll know that the situation is improving anyway, since the serum **pH (7.35 – 7.45)** will improve. We had a patient come in a day or so ago with an initial pH of 6.90 – yow!

The serum CO₂ / bicarb will improve, too – these patients come in sometimes with bicarb numbers less than ten.

2-1-6-2: HbA1C (3-6%):

This is a nice test to know about – otherwise known as glycosylated hemoglobin, it actually indicates the overall trend of a person's glucose over the three month period before the spec gets drawn. They can also work out a mean glucose value for that period – with a value of 6.0%, my mean glucose came out as something like 108 – not too bad. Nice to know that the pills are working...we'll see what my kidneys have to say ten years from now. We don't use these much for acute management in the ICU, but it's good to know how the patient has been doing.

2-1-7: Serum Calcium (8.5 – 10.5mg/dl):

Let's see if I can get this right. Calcium binds tightly to proteins that are floating around in the serum, so the serum calcium number reflects that, and varies as the protein level (measured as albumin) goes up and down. There are formulas to figure out the "corrected" calcium that factors in the numbers that your patient may show if she comes in, say, malnourished. A person's serum albumin can drop drastically in the first couple of days after admission to an ICU, and since proteins are what holds "water" in the vasculature, peripheral edema and third-spacing will start to develop. Start those tube feeds early.

2-1-7-1: Ionized Calcium: (1.0 – 1.3) This is the calcium that isn't bound to protein – it floats about in the serum. We follow these a lot with patients on CVVH, which rapidly sucks electrolytes out of the blood – these patients usually have a calcium drip running.

2-1-7-2: Corrected Calcium: Since serum calcium measurements are affected by the patient's albumin level, you have to figure in a correction if the albumin is off. So – for every drop of 1 gm/dl of albumin, you need to add 0.8 to the serum calcium number that comes back from the lab. The point is that your patient's calcium may higher than the lab number would make you think. This is the kind of thing that gets very important when you have a patient whose calcium comes back high in the first place, like people with bone mets.

2-1-7-3: Calcium changes in citrate toxicity:

This one comes into the picture with CVVH. Let's see if I can get my head around this one. It turns out that calcium – in this case we're talking about free, ionized, unbound calcium, is critical to several steps in the coagulation cascade. Who knew? Think I remember that stuff from nursing school, back in the early Cretaceous Era? No way! Dinosaurs – now those, I remember! One of them taught OB.

Second point: Citrate in solution chelates free ionized calcium – soaks it up, binds it up, removes it from activity – you get the idea. Citrate mix is used more and more often nowadays to keep the CVVH machine from clotting itself up – they call this “regional anticoagulation”.

Three: Citrate is normally rapidly cooked off by the liver, also the kidneys. The end product is apparently bicarbonate.

Four: If the liver and/or kidneys don't, or can't metabolize the citrate, then it hangs around, binding up the free, loose calcium. The ionized calcium number goes down. Hypocalcemia. Citrate toxicity. Bummer. Apparently the serum calcium number rises in this situation – anybody know why?

What to do? The point here is that patients in liver failure sometimes can't tolerate the citrate, and the clue will be the rapidly falling ionized calcium number. At this point we would probably change to a different replacement fluid, usually bicarb based. Bicarb-replaced systems are sort of infamous for clotting up, but if your patient is in enough liver failure to produce citrate toxicity, she's probably auto-anticoagulated enough that she'll anticoagulate the machine as well! Otherwise we use a low-dose heparin drip into the machine circuit.

2-1-8: Phosphorus (2.6 – 4.5 mg/dl):

Pretty important stuff, phosphorus – remember ATP, ADP, those guys? Renal failure patients often get very high phos numbers, since they can't clear it, and they take meds like calcium acetate (“Phos-lo”), or Renagel to bring it down.

Sometimes patients with poor nutrition will come in with really low phos's, maybe less than 1.0, which we replace with 10 – 30 millimoles of either sodium or potassium phosphate IV, which has to be given slowly on a pump over the better part of a day. There's an oral form too (“Nutra-Phos”). Maybe we could come up with a new product: “Nutra-Phos-Lo” – that would either replace or remove itself, depending.

(What do you mean, “Husbands should be like that!”?)

2-1-9: Lactate (0.6 – 2.2 mmol/l):

Okay – everybody remember the definition of a shock state? I mean besides how you feel after work. Three parts to a blood pressure: pump, volume and squeeze - three shock states: cardiogenic, hypovolemic and septic. Right? All three produce low blood pressure. Low perfusion to the peripheral tissues, which switch from aerobic to anaerobic respiration. The byproduct (the “exhaust”) is what? Lactic acid. More is bad. This lab helps you figure out why your hypotensive patient is so acidotic, although you should probably be able to guess. High is bad.

Another point about pH – a high potassium and a low pH can be a clue that something has died inside your patient. Bowel-infarct patients do this – it's classic, and a critical early sign. (Why does the K rise when this happens?)

2-1-10: Osmolality (280 – 295 mOsm/kgH₂O):

Cute units, huh? (“Yo Einstein! Nice units!”) Almost as good as “dynes/sec/cm⁻⁵”, which is what measures SVR and PVR and the like. This lab becomes very important in the case of increased intracranial pressure – the whole point is to try to keep the brain from swelling up, and treatment is with mannitol, which pulls fluid out of the vascular, fluidy brain by osmosis. Remember, making the blood hyperosmotic means that water will move out of the cells, right? – and into the bloodstream, from where it gets diuresed out. The goal for mannitol treatment is usually to keep the serum osm above something like 310. Dry.

Used to be we'd mannitolize them, and sit them up in a high Fowler's position, which we called “keeping them high and dry” – nowadays I don't think they do that any more. (Oh dear, once again, left behind by progress. Poor pitiful me...wait a minute. Motorcycle! Wahoo! “Look out Edna, here comes the old guy on the bike again! Why does he wear that nurse's cap?”)

2-1-11: What does "hemolyzed" mean?

When you draw a blood spec, it's important to try and remember that you're actually sending off a bunch of red cells that are swimming around in serum. Often we get our lab specs from arterial lines - if you were to manually pull really hard on a blood gas syringe, pulling the red cells through the stopcock, lots of the red cells would break, or burst. Hemolysis. Poor little red cells. Anybody remember the phrase "chief intracellular cation"? Everyone still asleep? This is actually important – the most prevalent positive ion inside the cell is what – anybody remember? Potassium.

When you send off a blood spec for chems, the result you get is actually from the serum (which is why they call them, um, serum chemistries. Doh!) Not from within the red cells. A normal serum potassium level will be something like 3.5 to 5, right? What if all the little red cells get busted – hemolyzed - as the blood spec is drawn? All their intracellular potassium gets to come out and mix with the K that was already in the serum, maybe doubling the result that you get. I'm not sure how they know it, but the chem lab will often mark the results as "hemolyzed" so that you don't jump out of your skin when you see a K of 8.3... Sometimes I think a spec gets "sort of" hemolyzed, although it won't say so on the results – maybe the stat chem result will be 5, and the one from a blood gas will be 3.2 . I think this is probably because people draw the gas specs more gently (the only way, right?) than the suction does in the vacuum tubes.

2-1-12 : Amylase (23 – 85 units/l):

Units of what? Amylase usually rises with it's cousin **2-1-13: lipase (0 – 160 u/l)** when patients develop pancreatitis. Painful. It seems that in recent years the numbers of these patients going to the OR has really dropped – I guess many of them do better if left alone for a period of time. There's a whole staging process for pancreatitis that I don't know much about.

2-1-14: Ammonia (11 – 35 mcmol/l):

(All the experienced ICU nurses give a big sigh when they see this one.) Ammonia is one of the nasty substances that accumulates in the blood when the liver doesn't work – makes people encephalopathic. Some of us are like that anyway. These folks often have a level in the 200's or higher, and treatment involves inducing lots of diarrhea with lactulose. Make sure that you warm

up the duoderm on the rectal bag before you put it on, and it'll stick much better – I put it under my arm while prepping the patient's, um, "area". Use a razor if you need to. Remember that benzoin really hurts on sore skin.

2-1-15: Albumin (3.7 – 5.0 gm/dl):

Very important for a couple of reasons – first, albumin is a main indicator of your patient's nutritional state: low is bad, normal is good. An elderly person can get into albumin trouble in a couple of days without sufficient nutrition, so get the tube feeds going as soon as possible.

Second: albumin is a main constituent of blood protein, which as we all know is what maintains oncotic pressure in the blood vessels – if this drops, then the patient will start third-spacing all that nice IV fluid you've been giving her to keep her pressure up. And if all that fluid leaks out of the vessels, will it help her pressure?

2-2: Renal Labs

2-2-1: Creatinine Clearance (90 - 130ml/min):

This is another name for the glomerular filtration rate – the normal rate at which blood is filtered through the kidneys. Low is bad – the kidneys are unhappy. Creatinine clearance can also be calculated and predicted when patients are on hemodialysis or CVVH. Higher is better, although the numbers are probably different for machine filtration.

2-2-2: Uric Acid (4.1 – 8.8 mg/dl):

Too much of this gives you gout, and also accounts for some kidney stones. Thanks a lot – mine wasn't much fun last year, although I did get my first-ever IV morphine. Wow – worked really well. They tried IV ketorolac first, which did squat.

2-2-3: Myoglobin:

You don't want to see this show up – it's an indicator for muscle damage, much the way CPK is, except that it's extremely nephrotoxic, and is what damages the kidneys in rhabdomyolysis. Think about a bicarb drip, which helps protect them. Anybody know how that works?

2-2-4: Urinalysis:

We send off tons of these – and they give back a lot of information. Some of the main points:

- Color: "straw colored" is always nice. Blood – not so nice, but we have fun with the descriptions: "Oh, it's a nice rose today, but it was definitely merlot yesterday." Drugs like rifampin and pyridium can produce a really nice orange Gatorade color. Methylene blue can make urine a nice teal green. ("Ya tink dat's teal? Nah, you dope, dat's like, aqua! Totally! Ha! Hey Ralpie, dis guy tinks dis heah color is teal, ha ha!")
- Turbidity: is there stuff floating around in it? Casts maybe? Fungal clumps? – time to ask about an Ampho-B irrigant even before the culture comes back. (You sent both UA and C&S, didn't you?)

- pH: very important sometimes, as in rhabdomyolysis, where large-scale muscle destruction releases lots and lots of myoglobin, which will show up in the urine, assuming the patient is making any, since the stuff is so nephrotoxic. The pH of the urine is kept above 7 with a bicarb drip.
- Specific Gravity: higher means more concentrated, lower means more dilute.
- Sediment: any there? Any idea why?
- Blood: shouldn't be any.
- Bacteria: 0 - 1/ml
- WBCs: 0 – 3/ml
- Glucose: none. (Yes, I take my glucophage, and no, I don't check my blood sugars often enough. Grrr.)
- Ketones: also none. (I know, I know!)
- Nitrite (indicates that bacteria are present): shouldn't be any.

2-3: Drug Levels

These definitely come under the heading of “chems”, and we follow a lot of levels in the unit. We do a lot of dose adjustment for renal failure – digoxin and vancomycin are good examples. Some other drug levels that we follow, with therapeutic ranges:

- **2-3-1: Dilantin (Total: 10 – 20mcg/ml):**

You should definitely know this one. Dilantin turns out to be one of the drugs that floats around in two forms like calcium does: free and albumin bound, the free drug being the active part. In general, it seems that following the total number is usually okay, but changes in the serum albumin will change the bound levels of the drug, making more or less of the free stuff, um, free, or actively available, as in renal or liver disease states. Nurses tend to let the physicians worry about calculating corrected levels – it's seems strange though to come across some dosage that they calculated, that comes out to something like 27.32 mg IV q 41 hours. Or something like that. **Free dilantin** is supposed to run around **1.0 – 2.0 mcg/ml**.

- **2-3-2: Valproic acid (50-100mg/l)** We don't do these too often.
- **2-3-3: Tegretol** Also not too often.
- **2-3-4: Lithium** Rarely, we'll see an OD.
- **2-3-5: Theophylline** Hardly ever any more, but this was a real big mover “back in the day”.
- **2-3-6: Thiocyanate (goal: < 30 mcg/ml)** This is a cyanide byproduct of nipride – anyone on a drip for more than a day should probably have these levels followed. Nasty.

Source: <http://www.toxassociates.com/refer2.htm>

- **2-3-7: Vancomycin (30 – 40mcg/ml)** Watch the BUN and creatinine.
- **2-3-8: Gentamicin (4 – 10mcg/ml)** Also watch the BUN and creatinine.
- **2-3-9: Digoxin (0.8 – 2.0ng/ml)** A range you should know. That's nanograms/ml. Got to hurt to do that one in the lab...a digitalized patient with acute-onset renal failure may show up with a level up around 5 or 6 - dangerously high. Go look up "Digibind".

2-3-10: Peaks, Troughs, and Randoms:

There seems to be some confusion about peak and trough levels. Here's what I do: the trough gets drawn first, just before a scheduled dose, when the level should be lowest (the trough of the drug-level graph.)

The peak gets drawn about 45 minutes after a scheduled dose ends, when the level should be highest.

Random levels are just that – they're drawn without any relation to the timing of the doses. We follow a lot of random vancomycin levels, because we have a lot of renal patients who can't clear it – we say the dose just keeps going round and round...

2-4: Tox Screen Panel Meds, with toxic ranges:

- **2-4-1: Tylenol (5 – 20 mcg/ml)** Patient have a nice bronzed look?
- **2-4-2: Salicylates (>500mg/l)** Ears ringing?
- **2-4-3: Opiates** (usually represented as "present") Breathing? What's Narcan?
- **2-4-4: Cocaine** ("present", or if you're in the service: "Ho!") Apparently it's important to remember that cocaine hangs around in the urine longer than it does in the blood. One reference we looked at said that the drug reaches peak excretion renally about six hours after a dose.
- **2-4-5: Benzodiazepines** ("present") What's flumazenil?
- **2-4-6: Miscellaneous other things** that show up on a tox screen, among many, (usually reported as "present"): **cannabinoids**, **phencyclidine** ("angel dust"? – I remember a story about a "dusted" patient who pulled his hands right through his handcuffs...), **amphetamines**, **antidepressants** (some of the older ones are very toxic), **oxycodone** (somebody really must've had a lot of time with nothing to do when they figured out about crushing oxycontin and sniffing it. What, did they try everything else in the cabinet before they got to that? Colace? "Hey Ralphie, try one of these!")

2-4-7: A totally cool thing:

A completely off-topic but totally neat maneuver popped into mind at the thought of someone sniffing a colace capsule – Jayne taught me this cool thing if a kid (hopefully a kid) shows up with

some interesting item inserted in the nose: mom (hopefully mom) puts her mouth over the kid's open mouth, she holds the kid's unplugged nostril closed with a finger (hopefully a finger, and not another M&M, or marble, or whatever it is that's plugging the first one), blows with appropriate pressure into the kid's mouth, and with air pressures doing what they do, the item should expel. Ha! Better than calling in the forceps team.

That Jayne – she's a smart one. Twenty-five years now, last August.

2-4-8: Ethanol (toxic > 300 mg/dl, often reported as % of total blood volume, legal limits often 0.08%): I found a conversion: a level of .08% supposedly equals a level of 80mg/dl, and .3% equals 300mg/dl. Can this be right?

Some toxic ingestions can be handled with dialysis – apparently all the alcohols can be removed this way, although we usually treat **ethanol** overdoses with intubation, and then let them “cook it off”.

A short flame: Where is it written that we have to put patients with DT's on benzos to detox them in the ICU? Why in the world don't we just prevent the whole DT thing with an appropriately dosed ethanol drip, then transfer them to detox, and do it the easy way?

2-4-9: Methanol (toxic > 20gm/dl)

Another member of the alcohol family – “wood alcohol”, I think they used to call it. Methanol ingestion patients seem to be so desperate for something alcoholic that they'll reach for anything that even resembles it: antifreeze, paint thinner...you'd have to be pretty thirsty. Methanol is converted to a couple of nasty metabolites: formic acid (“Ralphie! I told you not to eat that whole box of chocolate-covered ants!”), and formaldehyde.

Treatment is way cool: giving the patient ethanol will actually displace the methanol in the metabolic pathway – the bad stuff then cooks off slowly and non-toxically. Once in a while we get a glass bottle of – is it 10% ethanol? – up from pharmacy and hang it at some carefully calculated rate that factors in the weight, age, gender, and probably the renal function of the intern ordering it...

2-4-10: Ethylene Glycol: (“Ethylene! You stay out of that glycol! Don't make me get the hose!”)

Another dangerous substance found around the house, also appearing in antifreeze, brake fluid, etc. Treatment consists of ethanol, dialysis, and buffer in the form of a bicarb drip.

A really important point: ethylene glycol is sweet, and your dog or cat will definitely drink it up if it appears in a puddle under your car. I think that there are alternative antifreezes around.

3 - "Cardiac" labs

The whole point of drawing labs is to get information about what your patient is doing. The first part of cardiac assessment is the patient's rhythm: is he in sinus? Something else? Sinus with ectopy? More ectopy than before? Less? What labs might you think about in this situation? – probably **3-1: electrolytes** first.

3-1-1: Potassium (3.5 – 5.3 meq/l):

Everybody pretty much knows about the importance of K when it comes to issues of cardiac irritability. Actually, it turns out that **3-1-2: Magnesium (1.3 – 2.4 meq/l)** is just as important. (New people, try to remember that the mag thing is still an innovation for us ancient nurses – be patient with us, we'll get it eventually. I still haven't gotten over reaching for lidocaine when my patient has a run of VT. Speaking of which – what should I be reaching for?)

Here's a question – how are you going to give your patient a dose of potassium? Orally? On an empty stomach? To a patient who's been vomiting? (What kind of cardiac event might he be having?) Not orally? How about IV? How dilute does the K have to be – you might have to give 10 meqs per hour through a peripheral vein, in a pretty large volume to keep from burning a hole in the patient's arm. But what if the team doesn't want to give volume – maybe they want to diurese the patient instead? Well, could you mix it with lotion and rub it on his back? How are you going to solve this? (Go back and look at 2-1-2.)

The effect of repleting electrolytes can be really impressive: lots of ectopy may simply go away. The other thing to worry about when giving K is the kidneys – these guys usually excrete potassium at a fairly constant rate; if they're failing, they won't. So if your patient's BUN and creatinine have been rising, and his K is 3.2, what should you do?

Magnesium also turns out to be the treatment for “polymorphic VT”, which is either the same as, or first cousin to Torsades de pointes.

3-2: Cardiac Enzymes

3-2-1: What are cardiac enzymes?

This is a myocardial infarction thing. The idea is that destroyed myocardium releases specific substances into the blood, which can be measured – nowadays they call these the "serum cardiac markers".

3-2-2: Which cardiac enzymes do we follow on our patients?

For a long time we followed creatinine phosphokinase levels "CPK"s, and we still send them, but recently we've started sending troponin levels as well.

3-2-3: Can a patient have elevated enzymes without having an MI?

Well, see, that's the thing. It turns out that almost any situation that causes muscular injury – almost anywhere in skeletal muscle or the myocardium – can cause a CPK bump. Apparently not, however, for troponin, which only shows up in myocardial injury.

3-2-4: Can a patient have an MI without having elevated enzymes?

I don't think so. You read about people having MI's without developing q-waves – maybe because the events are physically very small – but I don't believe that a person can sustain a muscular injury to the heart without releasing some amount of enzymes.

3-2-5: What is CPK again?

CPK is released into the blood whenever there is a muscular injury somewhere. Non-cardiac injuries will release CPK: defibrillation, surgery, trauma or seizures – even IM injections. CPK begins to pop up in the period roughly 4 - 8 hours after an event, and starts dropping 48 hours out.

3-2-6: What is the reference range for CPK?

Our lab uses a reference range of 60 - 400 units/ liter. I know that I've seen patients "rule in" with a peak CPK "inside the range" – say, in the 300's – but the thing is that they show up positive for MB isoenzymes, which indicate specifically that the CPK release is coming from myocardial tissue. They would presumably be troponin-positive as well.

3-2-7: What are isoenzymes?

I thought that question was coming! It turns out that different tissues produce different sub-species of CPK when injured, which can be measured and expressed as a percent number of the total amount of CPK that's been released.

3-2-8: What is the "MB fraction"?

"MB" refers to a sub-species of CPK isoenzyme that gets released from cardiac muscle after an injury occurs - anything higher than 3% of MB isoenzyme means that the CPK is coming from myocardium (the isoenzyme number is reported as ng/ml). It isn't always diagnostic of an MI specifically, since other situations can cause tissue injury to the heart: cardiac surgery for example, or defibrillation, but you get the idea. You might see a rise in CPK/MB in a patient whose chest had struck the steering wheel, producing a "cardiac contusion"...everyone reading this has their seat belt on, right? I wear mine in the shower, but hey, that's just me.

3-2-9: What are MM and BB?

It's been a while since I even saw these used, but I think that MM is the CPK isoenzyme that gets released in skeletal muscle injury, and BB is the one released when brain tissue infarcts. Does that mean that brain tissue is structurally muscular? (Mine isn't. Wish it was.)

3-2-10: Does a higher CPK mean a larger MI?

That's the idea – but you have to make sure that you're looking at the right kind of CPK. If the MB isoenzyme forms more than 3% of the total number, then that points to MI as the source of the release. A person who's been knocked off a bicycle (somebody opened their car door right in front of me once) might have a result in the thousands, but if the MB "iso" wasn't there, then you'd have to say that the CPK "bump" came from skeletal muscle instead of the heart.

During the Crimean War, we used to call a small MI a "subendo": sub-endocardial, meaning small, and not all the way through the muscle wall ("transmural"). This kind of event usually went with a CPK peak of something less than a thousand: 400, 500 maybe. I think these are the MIs that nowadays are often called "non q-wave" events, because the muscular injury isn't big enough to generate the dreaded evil q's.

Big MI's on the other hand are pretty unmistakable – you may see CPK peaks of 3 - 4000 or more, with an MB fraction – well, what would it have to be? 3% (or more) of 3000 – um...well, one percent would be 30, right? So 3% would be 90? So if the MB number came back at something like 300, that would be 10% - definitely a cardiac event. Pretty big one.

3-2-11: How many CPKs should be drawn, and how far apart?

We usually send three CPK specs, eight hours apart. If a cardiac patient has some sort of complicating event later on – say a spell of a-fib, or maybe recurrent pain, then we'll probably send another three sets to see if there's been another injury. (A small CPK release is called a "leak", and a really small release is called a "leaklet".)

Remember that CPKs are going to go way up in any situation that produces skeletal muscle injury, but the situation that really makes the numbers get scary is **rhabdomyolysis** – which I think got mentioned somewhere earlier. This is a pretty dangerous scenario that shows up sometimes when someone's been lying on the floor, say, for a couple of days without moving – maybe intoxicated, maybe after a stroke, something like that. We've seen CPKs get up into the range of 30,000. Pretty high.

3-2-12: What is troponin?

It turns out that there's an even more sensitive test for myocardial injury, and her name is troponin. There are three types of troponin: CTnI, CTnC, and CTnT, which is the one we use (we call it "troponin-t").

It turns out that troponin is a really sensitive and accurate indicator of myocardial injury, which makes it preferable to CPK, since you can get knocked off your bike three times a week and not release troponin until the frustration makes you have an MI. Troponin is a definitive indicator for non-q-wave MI; there's no confusion about whether your CPK bump is coming from your broken arm or from the chest pain that you got when that idiot opened his door in front of you for the fourth time this month.

Troponin also stays elevated for something like a week after an MI, so someone who comes in four days after his event - when his wife finally convinces him that he doesn't look so good - will still have diagnostic levels to prove what's going on. Just didn't want to miss the post-game show, y'know.

Nothing's perfect though, and other conditions can make troponin rise: renal failure can cause the only "false elevation" of troponin that we've heard about. Other causes of troponin release: an episode of CHF can do it, and obviously any myocardial injury besides an MI will do it too: cardiac contusion, defibrillation, myocarditis, ablation (that's where they burn out the WPW thing in the EP lab, etc.), but the important point is that there's no confusion as to the source of the enzyme release. They say that troponin may replace CPK testing entirely sometime soon.

3-2-13: What is the reference range for troponin?

Our range is 0.00-0.09 for "normal". Anything above 0.10 is considered a "rule-in" – here's a quick example: we had a patient whose CPK came back at 184, with an MB of 5.1. If you calculate it out, the MB turns out to be 2.8% of the total (5.1 turns out to be 2.8% of 184. I realize not everyone is as stupid as I am with numbers, but it always helps me to say things several times.) So the MB fraction is a hair under 3% - maybe not an MI? But the troponin at the same time was 0.14 – strictly speaking, a "rule-in". The next set of enzymes showed a CPK of 440, with an MB fraction of 4.4%, and a troponin of 0.19. Helpful. Definitely a little MI.

3-2-14: How often should troponins be drawn, and how far apart?

We send troponins and CPK/MB's on the same schedule – it's the same red-top spec, every 8 hours times 3.

3-2-15: What is "washout"?

This is a "reperfusion" phenomenon that you see when a patient gets clot-busted. Visualization exercise, okay?: everybody see the little clot that's plugging the coronary artery at the narrow spot? (No Ralphie, the heart. The big red thing. No, the one that's moving.) There isn't much gas exchange in the tissue beyond an arterial plug, or exchange of anything for that matter, and CPK will accumulate downstream in the ischemic tissue which is not quite dead yet, but will be soon, if the clot doesn't get busted. If perfusion is suddenly restored, all that CPK gets blown out into the circulation at once – if this occurs within the 6 hour "window", then the affected area of myocardium will hopefully be saved, and the CPK bump will only indicate the transient injury, instead of tissue death. Close one!

What else might you expect during a reperfusion period that might make you a little nervous?

3-2-16: Can cardiac enzymes go up if a patient is ischemic, but not having an MI?

CPK /CKMB may not rise after an ischemic episode, but troponin does in about a third of patients, raising the theory that "micro-infarcts" are occurring. "Angina producing necrosis." Bad prognostic sign, worse than if there's no troponin release.

3-3: Lipids

A **lipid profile** is made up of a number of tests:

- **3-3-1: total cholesterol (<200mg/dl)**
- **3-3-2: HDL (30-60mg/dl)**
- **3-3-3: LDL (<190mg/dl)**
- **3-3-4: Triglycerides (<180mg/dl)**

We send these as part of the workup on cardiac admissions, and oftentimes our patients get a daily dose of one statin drug or another, but we don't follow them much as a matter of course. One exception: patients on propofol get a lot of lipid from the emulsion that it's made of – sometimes the lipids are removed from their TPN as a result.

An interesting story – we had a patient a while back who was some kind of incredible congenital hyperlipidemic – I forget what the numbers were exactly, but there was a white sediment in his blood spec tubes. Scary.

4- Respiratory Labs:

Obviously the main topic here is blood gases. There's more than you probably ever wanted to know about blood gases in the faq on "Vents and ABG's", so take a look over there for details, but we can take a quick overview:

4-1: ABGs:**- 4-1-1: pO₂ (80 – 100 mm Hg) :**

This is the good stuff – this is what you’re trying to deliver. A rough rule of thumb that’s often helpful if you’re trying to figure out how your patient’s doing: the pO₂ should be something like four or five times the FiO₂.

Example: you’re breathing room air, which has an FiO₂ of 21%, right? And if we stuck you for a blood gas, your pO₂ should be something like 80-100, which is roughly four or five times the FiO₂ number. So if you intubated me, put me on 100%, and stuck me, my pO₂ would be upwards of 400. I hope. The point is, the next time someone tells you how great it is that your patient has a pO₂ of 80, and they’re on 100% and 10 of PEEP, you’ll know that 80 is actually really very low, compared to where they ought to be. Hypoxic. Remember: “oxygenation” means just that: how well the blood is getting oxygen delivered from the alveoli.

- 4-1-2: pCO₂ (35 – 45 mm Hg):

This is the bad stuff, the stuff you’re trying to get rid of. “Exhaust gas” – comes out of the tailpipe. (So if you use Red Man in your pipe, and you exhale blue smoke, does it mean you need a ring job? As for me, just make me a DNR – I had a cracked block last year anyhow. It’s that diabetes, man.) Too much pCO₂ and the carbonic acid reaction goes the wrong way – respiratory acidemia results. Get rid of too much and the reaction goes the other way – respiratory alkalosis. Remember that “ventilation” refers specifically to how well your patient is clearing CO₂. Different conditions often produce specific effects on blood gases: patients in CHF will usually be hypoxic, but won’t have much trouble ventilating. Pneumonia patients are the other way – they may oxygenate fairly well, but they don’t ventilate good. Correctly. Properly. Whatever.

- 4-1-3: pH (7.35 – 7.45):

pH tells you lots of things, and it takes time to learn how to put it in the right context. Just looking at it from the respiratory standpoint, the pH will go up or down as the pCO₂ does, except in the opposite direction: if pCO₂ goes up, pH goes down, and vice versa.

- 4-1-4: bicarb (22-29 mmol/l):

The range for this one is “the age you’d like to be”. (Thanks, Laura!) Bicarb is the buffer that floats about in the serum, and counterbalances the carbonic acid. (Something tells me that there’s a little more to it than that...). The kidneys do the job of either holding onto bicarb or dumping it, as blood pH conditions change – it takes three or four days to come to a new stable state.

4-2: Carboxyhemoglobin (bad effects show up anywhere from 10-30%, serious effects >40%):

This is a scary one. Carbon monoxide just loves hemoglobin so much that it will push oxygen out of the way just to take its place on the Hb molecule – that’s to say, it “binds preferentially”, and is rather a bear to get rid of. All that CO-saturated hemoglobin (that’s the “carboxy” part) is not carrying oxygen around – it would sort of make sense that if 40% of your Hb was saturated with something besides oxygen, then bad things would ensue. Duh. Ever read “Coma”?

Treatment is so cool: serious exposure cases go into the hyperbaric chamber, and sent “down” to a depth of whatever number of atmospheres, as though they were going down in a diving bell,

or being treated for the “bends”. Pressurized. ENT has to come in and puncture the patient’s eardrums to prevent tympanic rupture. Hyperbaric oxygen is apparently really useful in preventing bad things like neurological sequelae. I hate it when those happen!



A hyperbaric chamber, in Scotland I think. Big!

4-3: Methemoglobin (goal: <4%):

This is another substance that likes to bind to hemoglobin and that can interfere with oxygen delivery if too much of it accumulates. This one appears when inhaled nitric oxide therapy is used for pulmonary hypertension. (Not nitrous – ha ha! – why are my feet so big? Hmm, that’s a big drill...) Too much methemoglobin: bad.

4-4: What is an anion gap (10-14 mEq/l), how do I calculate it, and why is it listed here under “respiratory”?

Acid-base involves everything. After thinking about it, and thinking about what to put in this section, I decided to leave it here under the heading of “respiratory” because the part of acid-base that you’re probably going to work with first is the ventilation kind – but remember that the non-respiratory, the metabolic components, are just as important to think about. So maybe this will help you keep them both in mind.

Calculating an anion gap is supposed to help you figure out what’s going on with your patient’s acid-base balance thing. “Why the hell is he so acidotic?” Or alkalotic. There are “gap” acidosis’s, and “non-gap” acidosis’s.

After talking about the gap, I’m going to confess that I don’t use it myself. Never having learned a lot of the horrible chemistry, etc. that lies behind the ways the physicians analyze these situations, I tend to rely on experience, and I can usually come up with something helpful that way. There are a few main reasons why you’re going to see your patient become acidotic or alkalotic, and they’ll become very clear to you inside of your first year’s time in the unit:

4-4-1: Acidoses:

- He’s gained some acid:
 - 1- Did his pCO₂ go up? (How does that make acid?)
 - 2- Is he “shocky”? (How does that make acid? Send a lactate.)
 - 3- Is he in renal failure? There are a couple of kinds of “renal tubular acidosis”.
 - 4- Is he in DKA? (How does that make acid? Send the ketones.)
 - 5- Has he gotten 12 liters of normal saline today? (Can someone explain hyperchloremic acidosis to me?)

- He's lost some bicarb:
 - 1- Does he have an ileal loop? (They can dump bicarb like mad. Sodium, too.)
 - 2- Has he been climbing mountains and taking too much Diamox? (Dumps bicarb in the urine. Hey, it could happen! And monkeys could...never mind.)
 - 3- Has he been poisoned? Ethylene glycol, aspirin overdose? Methanol?

4-4-2: Alkaloses:

- She's lost some acid:
 - 1- Has her NG tube been putting out liters and liters a day? (Lots of HCL loss there.)
 - 2- Has she been over-ventilated? (How does that make alkalosis?)
 - 3- Has she been diuresed for several days running? (Loop diuretics make the kidneys dump H⁺ ions as well as potassium. Acid loss, right?)

- She's gained some bicarb:
 - 1- Has she been getting into the family-sized antacids again? I never go to the warehouse stores any more.

Let's take an example of the thought process (assuming there is a thought process): patient comes in intubated, history of COPD, they put him on a vent at 100% FiO₂, rate of 12, tidal volume of 600, 5 of PEEP to hold things open. You send a gas, and here it is:

(pO₂ / pCO₂ / pH / bicarb): 325 / 40 / 7.56 / 39.

Well – you can turn the oxygen down, that's for sure. PCO₂ looks good – right on the ideal number. But what's up with the pH? Wicked alkalotic, as we say in Boston.

Hmm – look at the pCO₂ again. That's okay. So this isn't a respiratory thing. So what does it have to be? Got to be a metabolic thing. Look at the bicarb – way high. Definitely a metabolic thing.

Actually, this is a pretty common scenario for COPD people: they walk around with a pCO₂ around 50 most of the time anyhow, right? – hence the saying that they belong to the “50/50 club” – meaning their pO₂ and pCO₂ are both usually around that number.

If you sedated me, say with 0.5 of morphine (I'm a very sensitive individual...wiping tear away), and made my pCO₂ go up to 50, what would my pH normally do? Anybody remember the rule? If the pCO₂ changes 10 points, the pH changes .08 – so with a pCO₂ of 50, my pH would be 7.32.

But this guy walks around with a high pCO₂ all the time. His baseline gas would be something like 50 /50/ 7.40 – how come is that? It's on account of he's compensating – remember compensating? And how does he do that? By holding onto bicarb with his kidneys – look: his bicarb is wicked high. Compensated.

So what happens when he comes in, and you tube him? He's probably been in trouble for a couple of days – his pCO₂ has been even higher than usual, and his kidneys are just a-hangin' on to every little bicarb molecule that goes by, and he's doin' okay, keeping his pH fairly normal, but his pCO₂ goes up a little more, and a little more, and then bam! Narced. Stops breathing, almost. Gets tubed.

So okay – we tube him, and we ventilate him, and we “blow him down” to a normal pCO₂. But by this time he’s saved up so much bicarb over the past couple of days that he’s got this enormous reserve of it floating around in his blood – so if we blow him down from his pCO₂ of about 75 that he’s been compensating for all this time, what happens to his pH? Wicked alkalotic.

So – what to do? Well, he’ll re-equilibrate in a few days – it takes three or four days for the kidneys to straighten things out again (assuming that they work). Or we could let his pCO₂ rise some, which would normalize his pH some. Or we could give him diamox for a day or two, to make him dump out some of that bicarb. But somebody better figure out what pushed him into trouble – pneumonia, COPD flare, CHF, whatever - and treat that at the same time, yo!

4-4-3: Calculating the Gap:

The preceptor had to go look this one up. (Hanging head in shame – the preceptor can’t calculate an anion gap?)

Here’s a formula: the Anion Gap = (serum Na⁺) - (serum chloride + serum bicarb).

In English, this turns out to be the difference between the main serum cation, minus the sum of the main serum anions.

- So: the calculation itself turns out to be easy: take the level of the main cation, sodium - let’s say it’s 135.
- Now the sum of the two main anions: chloride and bicarb, let’s say 100, and 25. Add those up: 125.
- So what’s the difference between the sodium and the total of the negatives? 10?
- That’s the gap.

As usual, my explanations will not be the same as those you get from your medical teams – and they probably shouldn’t be. Current book-knowledge is exactly what they’re supposed to have, and bedside experience is what you and I are supposed to have, and we’re supposed to put those together – it often works out very well!

5- Liver Function Tests

5-1: A story.

A story goes here – just skip this part if you don’t want to go off on a sidetrack for a while. I’m not sure how to tell this – if there aren’t any numbered questions I get disoriented. It was one of those rare experiences that have made a permanent impression in my memory (and there’s not much room in there.) It’s lots easier to describe VT – “just the facts, ma’am”. (Young people: who said that?) This is harder.

Some years ago – maybe ten?, a gentleman came into the unit with this rare liver thing: “sclerosing cholangitis”, which is apparently one of these conditions that you can manage but never cure, and which kill you in the end. The bile ducts become spontaneously inflamed, sometimes they close and have to be stented open. Transplant didn’t seem to be an option in his

case, and I think that he was in the unit that first time because he had an acute obstructive jaundice that they were trying to treat with stenting procedures.

I have a famously bad memory for some things. Maybe that's just a trick my mind plays on me, since I can apparently remember "escape - capture bigeminy" without much trouble, and how arcane is that? Very. House officers will come back as juniors into the unit a year after their intern month – I'll remember the face, usually not the name, and embarrassedly sneak over to the photo list to quickly check – then act like I never forgot it...but I won't forget Charles Mifune (not his name): name, face, case, or family.

I usually ask the same set of simple questions by way of getting to know a new patient – entering a room under the assumption that I may have to do something unpleasant to, or with, someone that I've never met before, I usually will give a nod, and say "Mr. Yakowitz?". You have to be careful to watch how far to extend yourself in this situation – a patient may show you in the first seven seconds that he wants you to stay in his room for the next three hours, and the job being what it is, you have to evaluate time limits right away. Or not. There's a lot of variation.

I was quickly impressed. Mr. Mifune had a surprising natural dignity, which is something you read about sometimes – here it was for real. I could never call him Charles – there was something in me that had too much respect for the man, which only grew as I got to know him, to allow for familiarity, no matter how many times I cleaned him up in the bed. But there was no lack of mutual affection either – he liked me, I liked him, maybe because we saw similarities of personality – which means he probably never got a really good look at me, anyhow! He was of Japanese descent, and seemed to embody some qualities that I think of as Japanese, mixed with some American ones: he was reserved, but friendly – there was no social barrier – his whole life had been lived in an American context. He seemed quietly disciplined. Apparently from a poor working background in Hawaii (he used to take his wife on walking dates to an ice-cream stand before they married), he couldn't afford law school, and so became a postal worker until retirement. I think his wife had worked on a pineapple plantation. They'd had something like six children – all of whom seemed to have absorbed his work ethic. I found out later that they had all worked and scholarshiped their way through college – they would gently tease their dad, copying things he'd sternly said to them in the past about homework...and they laughed about the cheap ice-cream dates. I thought they were some of the best dates I'd ever heard of.

The discipline with which he'd raised his family seemed to be of a piece with his personality – who really knows? But it seemed that way. The man carried himself through a really arduous set of tests and procedures with patience, quiet good humor, and simple fortitude – and go watch a few ERCP's if you think this stuff is easy, although I hear that they go more smoothly now than they used to.

His wife seemed to share the same kind of personality. She would sit quietly in the room, exchanging comments with her husband while I would draw labs, or start an IV, or hang meds, always present, always very concerned. They seemed to be best friends, used to helping each other. They seemed to be grounded in reality, probably from all that walking, back in Hawaii. She took him home.

Eight years later. We'd moved to a new location – our shiny new ICU, which was pretty for sure, but I always thought we did a pretty good job in the old one...and back came Mr. Mifune. The dusty, ancient Rolodex that still holds the primary nurse assignments reliably coughed up my name, and I came in to find myself assigned to a patient that I actually remembered for once. They were very happy to see me, and I guess that they associated me with the time that he'd gotten better. Now things were obviously worse - he looked terrible: jaundiced, thin, big belly, tired. And calm, and sad, and brave. I guess he was back in the unit to see what might be done.

It took a couple of weeks, as I remember it. There was always some family member in the room – usually Mrs. Mifune herself – she hadn't changed: still showing that same sense of restraint,

mixed with a totally American sense of humor. And the same feeling of grounding in reality – as if there had never been a TV in their house. They knew what was coming, and they were quite prepared for it, thanks. No hysteria, no screaming, no throwing one's self to the floor, no shouting or threatening of hospital staff. They were ready.

As jaundice worsens, so does confusion. Bilirubins rise, LFTs rise, ammonia rises, treated with lactulose – we know what that does. Even confused – and I think he was almost unique in this way – Mr. Mifune always remembered who I was. He would gently rouse from his sleepiness and say, a little startled, “Hi Mark!” He never became combative, although he did forget where he was. He would accept our answers to his confused questions, and he would always help turn himself in the bed when he needed cleaning up.

I got to know some more of his family members. One son was angry – not at us, not at the hospital – angry at fate, he was, and he knew it. He would look furious –when I could, I'd bring him a coffee and we'd talk. Mr. Mifune had had relatives and friends who'd been in the 442nd Regimental Combat Team – the Japanese-American unit that lost so many men running uphill into live fire from Monte Cassino in the dead of winter, 1944. I actually knew about this a little, since my son has a jones for the History Channel and we like to sit together sometimes on the couch and watch. Did I ever get a grin from the patient's son when he realized that I had heard of that unit!

Charles died, quietly, sleepily, probably very comfortably, and I was there. Mrs. Mifune, crying, hugging me hard, said “You were here at the beginning, and you were here at the end. We were so glad.” I had no words. What a privilege. I want to be Mr. Mifune when I grow up.

5-1-1:



Deep breath. Okay - back to liver function tests. Anybody else need the box of tissues besides me?

From the point of view of a preceptor, I should say that I don't look at liver function tests as much as I ought to – all I want to know is: are they rising or falling? – since they generally move in a group. (“This way, group!”)

I guess it's clear that humor is my coping mechanism, huh? Better than skydiving, I guess. Cheaper insurance, too.

The notes here are based on some quick reference checks – anyone with a correction to make, please send it along? It turns out that the ratios of one LFT to another can indicate what your liver-failure patient's underlying problem may be: tissue based maybe, or obstructive. These diagnostic problems don't usually turn out to be something that I worry about when my goal is to keep the patient alive overnight – unlike knowing the difference between rapid a-fib and VT. I find myself checking the PT and PTT much more frequently in liver-failure situations, along with ammonia checks to see if the lactulose is working. Another clue is that the patient wakes up – but that's an assessment detail that I leave to the experts. (grin!)

A couple of the main LFTs:

5-2: Bilirubin, direct (0 – 0.3mg/dl) and indirect (< 1.0 mg/dl):

Let's see if I have this right. Bilirubin is metabolized in the liver – conjugation, they call it. (“I am bilirubin, you are bilirubin, he is bilirubin, they was bilirubin...”). Something like that. Bilirubin has to be conjugated so that it can be eliminated (mostly) in the bile, and (a little) in the urine. Direct bili is the part that has been conjugated, and the indirect bili is the part that hasn't. The total **(0.3 – 1.9mg/dl)** bilirubin is both of them added together. Basically what I remember is that the normal numbers are very low – anything greater than 1.0 in either one makes me look twice.

An example of how I might use LFTs at the bedside: we had a patient not too long ago, I think mentioned elsewhere in this article, who had taken a really impressive amount of acetaminophen and then waited to come in until she'd absorbed most of it from her gut. This is your basic bad liver situation – you can almost hear the freight train of liver failure bearing down on your patient, who is stuck on the tracks. I think that the LFT specs aren't sent more often than qd, but certainly this patient's LFTs are going to be high, and going to get higher quickly – just a matter of time until she starts becoming jaundiced. (Where does the patient become jaundiced first? Yes, Hermione?) Call the ELAD team.

Some others:

5-3,4: ALT, AST:

(I think these are the new names for what I used to call SGOT and SGPT). These reflect liver cell damage or death, as opposed to an obstructive problem. ALT is apparently the most specific to the liver.

5-5: Alkaline Phosphatase:

(“Geez, would ya look at this guy's alk phos rising?”) This one seems to be more related to problems in the biliary tree itself.

5-6,7: PT/PTT

For sure the PT and PTT are indicators of liver failure – when the patient is really in trouble, as in maybe thinking pre- ELAD, they're often on continuous FFP infusions to supply the clotting factors that the sick liver can't make.

5-8: SPEP: This test separates out four of the proteins made by the liver to see whether there's not enough, or maybe too much of one kind or another: albumin (hold on to that – you need that), and the alpha, beta and gammaglobulins.

6- Hematology Labs

They still call the basic hematology spec the **Complete Blood Count (CBC)** – we'll take the parts one at a time.

6-1: Hematocrit (36 – 46%)

Hematocrit tells you how many red cells are floating around in the serum. The number is reported as a percentage because of the way it's done – they spin the spec tube in a centrifuge, and the red cells settle to the bottom – if the tube is half-full of red cells, then the "crit" is 50%. If it's a third full of red cells, the crit is 33%. The crit number will definitely go up and down as your patient de- or re-hydrates, and a dry, debilitated person admitted with a normal number may show you that he actually lives really low once he gets "tanked up". Renal failure patients can fool you that way. (Why do renal failure patients run chronically low hematocrits, and how is that treated?)

A crit drop after aggressive hydration is called "dilutional", which is clearly not the same as a "delusional" crit drop, which is where you think the crit has gone down, but it hasn't. That's the kind I have. I think.

The rules for transfusion have really changed over the past few years, and patients often run with numbers that would have made us very nervous in the past: low 20's sometimes. It turns out that transfusions are dangerous – well, um - I think we knew that. But the statistics are clear: more patients die if they're transfused than if they're not. This does not mean that you shouldn't get a ton of blood set up for your big GI bleeder – but the lady in room 92 who's just a slow vent-wean may not need to be kept at 30 the whole time she's in the ICU.

6-2: White count (4.5 – 11 thousand/cc): Defenders against evil. There's a number of different kinds, (determining how many there are of each is the **6-2-1: differential**), and there's a basic breakdown of the types in the faq on "Blood and Transfusion": T-cells, B-cells, helper cells, polymorphonuclears, basophils, eosinophils...a few things come up with some regularity in the unit:

- Total number: higher usually means something bad is going on infection-wise. Watching the number rise and fall from day to day isn't always very meaningful – it can vary a lot from day to day without a real change in the patient's status. Watching the trend over several days is helpful. Steroids will make your patient's white count rise.
- "Bandemia": Bands are immature white cells – if their numbers rise, it means that the marrow is cranking them out rapidly, probably in response to a bacterial infection. Developing bandemia is also sometimes called a "left shift" – having to do with the way the cells sort out, I guess. Sounds like when your kid joins the anarchists and starts living in a tree...like dad did!
- **Eosinophils:** Sometimes you'll see an order for this when the docs are trying to figure out if the patient is having a drug reaction – usually a drug fever or rash.

6-2-2: “It’s a bad sign when the white count is higher than the crit.”

6-3: Platelets: (130 – 450 thousand /microliter): Microliter? One millionth of a liter? Pretty small of a volume for 130 – 450 thousand of anything to swim around in, but I guess they’re right – why did I always think that it was per cc? Definitely important – can’t clot without these - don’t leave home without ‘em. We transfuse plates for low counts if patients are actively bleeding or if their count drops below 20K, although that number seems to change at times. Does anybody know - why do platelets come in six-packs?

The problem with giving repeated platelet transfusions is that they don’t work very well after the first few – it’s an antibody-mediated thing as I recall. I remember giving “HLA-matched” platelets in the past, but we don’t seem to do that nowadays. Are they pre-matched now? I have no idea.

6-3-1: Heparin-Induced Thrombocytopenia (positive or negative)

We definitely see patients come up positive for this one now and again. Usually the lab gets sent twice; if a patient’s count has dropped drastically over a day or so, we change our line flushes to normal saline and start sending the specs. Stop the sq heparin too, and get out the air boots.

H2 blockers can also really hurt your patient’s platelet count, which I understand is why we don’t see cimetidine around anymore – we used to give that stuff like water. We use ranitidine now, apparently much better, although still on the platelet-problem list to some degree.

6-4: Coagulation Studies:

6-4-1: Prothrombin Time/ PT (10-14 seconds)

6-4-2: PTT /partial thromboplastin time(normal 20 – 40 seconds, therapeutic 50 – 70 seconds)

6-4-3: INR /International Normalized Ratio (normally 1.0 – therapeutic 2.0 – 3.5)

We still send the “coags” (“co-aggs”) the way we used to: the PT and the PTT, but in recent years the INR has become a standard part of the coag report, replacing the PT – newer nurses look at me blankly when I ask what their patient’s PT is. In amongst the horrible complexity of the clotting cascade there are two anticoagulation paths that we follow: the PT/INR (coumadin therapy) represents one of them, PTT (heparin therapy) the other.

Sometimes you’ll see a patient come in who may have gotten a little confused about his pills at home, with a PT greater than 50, an INR greater than 20. That’s pretty anticoagulated. These people often show up with GI bleeding of one kind or another, sometimes with spontaneous bleeding in the head. Liver failure patients look similar – the continuous FFP drips are to keep them from losing what blood they’ve got.

6-5: D-Dimer (normal <250 micrograms/liter):

D-dimer turns out to be about having clots: it’s a material released by the degradation of fibrin, which means that there’s a big clot process going on somewhere. Iraq, maybe. D-dimer will pop up if a patient has a DVT, a PE, or in DIC, when supposedly zillions of little micro-clots are being formed. D-dimer is part of the **6-6: DIC screen**, a raft of labs that goes off in two tubes, iced, and includes the coags, a d-dimer, and other measures of clot activity including **6-7: Fibrin Split**

Products (normal is < 10gm/ml) – if the FSP is high, then a lot of clotting (and clot breakdown) is going on, as in DIC.

6-8: Fibrinogen (200 – 400mg/dl): Another part of the DIC screen. This is one of the proteins in the clotting cascade – if a patient is in DIC, then fibrinogen gets used up rapidly by the disseminated clotting process – which is to say, its titer goes down. Helpful in making the diagnosis.

6-9: Erythrocyte Sedimentation Rate (0 – 20mm/hour)

I don't see as many orders for these specs as we used to, but they still crop up. This involves measuring how rapidly the red cells in a spec tube settle towards the bottom in the space of an hour. Apparently it isn't diagnostic by itself, but it's used as an indicator for the presence of lots of different kinds of inflammatory processes, MI, or tumor activity. Used often in monitoring arthritis, maybe? I've never gone looking up a sed rate on one of my patients and gotten upset on getting the result. If the cells fall faster, the inflammation is worse – as a patient responds to treatment, the cells fall more slowly. Anybody know why?

6-10: Coombs Test – Direct and Indirect:

Direct Coombs testing is an auto-antibody-vs-RBC test. Indirect Coombs tests are used by the blood bank in determining possible reaction to transfusion. Is the one pre-transfusion, and the other for detecting reactions? Pretty obvious I don't know much about these, and while we do send them off, we don't make much use of them at the bedside. Make sure you check blood products properly! (What would you do if you thought your patient was reacting?)

7- ID Labs:

7-1: Cultures: blood, urine, sputum, CSF (Did I miss any?)

We send a lot of cultures – maybe too many, they tell us. We get routine culture reports back in one day, finals in three, and the results include **7-2: sensitivity reports** as to which antibiotics the bug is sensitive to. These are starting to get scary of late – we had a patient recently whose decub wound was colonized by acinetobacter (say that three times quickly) – the report came back something like this:

Vancomycin: RESISTANT	Methicillin: RESISTANT	Metronidazole: RESISTANT
Gentamicin: RESISTANT	Cefuroxime: RESISTANT	Penicillins: RESISTANT
Cefazolin: RESISTANT	Amikacin: PARTIALLY RESISTANT	
Linezolid: BETTER NOT BE!		

You get the idea. Wash your hands.

7-3: A couple of specialty tests:

- **7-3-1: TB:** we send three sputum specs for **AFB** (Acid-Fast Bacillus). Smear results come back within 24-48 hours, and the culture specs are usually held for something like 8 weeks.

- **7-3-2: CD4 (T-cell count) (500 – 1500/ml):** trends are the key – lower counts correlate with progression of HIV.
- **7-3-3: Viral Load:** Another part of the HIV test panel: usually reported as high, intermediate or low - depending on the test method used the numbers are variable. Jayne says that with the protease inhibitor cocktail some patients' levels come back "undetectable". Cool.
- **7-3-4: Branch-chain DNA and PCR, "polymerase chain reaction":** These are two methods used to "provide more evidence" when the cultures aren't definitive. I understand that PCR is used to replicate cells in cultures nowadays to help identify organisms that might otherwise not show up in the dish. Very cool.
- **7-3-5: Kary Mullis:** Another interesting thing about PCR is its inventor: the scientist who won the Nobel Prize for it named Kary Mullis. He's apparently fond of surfing, and thought up the PCR idea while driving on the California highway in his Honda Civic. (With or without his boards?) He won the prize, but his parent company apparently got the rights to PCR, which they sold for some \$30 million. Oh well – back to the surf for the next idea.

7-4: CSF: It seems like most of these specs are drawn to rule out one kind of infection or another, so ID isn't a bad place to put them, although there are other reasons for doing LP's as well.

7-4-1 Which kind of infection?: One of the main things that you're trying to figure out here is whether an infection is present at all; and if it is, whether it's viral or bacterial. A clue seems to have to do with how many white cells show up in the CSF, and what kind they are:

- Viral infections: mostly lymphocytes, glucose higher than 3.0 mmol/liter. Herpes simplex turns out to cause many cases of viral meningitis, and they use the PCR method to verify it.
- Bacterial infections: mostly neutrophils, glucose less than 2.0 mmol/liter - bacteria eat up the csf glucose and, virii don't.

7-4-2 Some normal values for CSF: here's another case where some of the tests should go somewhere else maybe, like chemistry, but they're relevant here. The point is that things overlap for important reasons.

- glucose: 40-85 mg/dl
- opening pressure: 50-180 mm H2O
- white cells: not many, i.e. less than 5 per cc – lots of whites may also point to tumor activity.
- red cells: none, unless the LP is traumatic
- color: should be crystal clear – a yellow tint ("xanthochromia", which apparently means "yellow tint") means either red cells have been in the csf, or jaundice, or maybe eating too many carrots. Cloudy csf often points to bacterial meningitis.

- Protein: not much compared to the serum, maybe 35mg/dl. Increased amounts of protein in the csf might mean a breakdown in the blood-brain barrier, pointing to infection, inflammation, or even tumor activity.
- (I don't think I have a blood-brain barrier. What were we talking about? Hey - where's my bag of carrots?)

8- Endocrine Labs: We send these often enough to mention them:

8-1: Thyroid studies:

We usually send the “panel”: T3, T4, and TSH are the ones that come to mind. Got to have enough TSH coming from the pituitary, otherwise the thyroid won't make the other ones. We give synthroid to patients who need it, usually because they've been diagnosed on the outside and come in with it listed in their history. Recently we had a patient in myxedema coma, which got the resident teams very excited – severe hypothyroid. She got intravenous thyroid treatment, something I don't think I've ever seen before. Cool.

8-2: “Cort-stim” tests:

These are to figure out if your patient's adrenals are working. A baseline cortisol level gets sent – why always in the morning? Some circadian thing there. (After working nights for 20 years they'd have a hard time with mine.) Then the patient is given a dose of IV ACTH, also known as cosyntropin – then after an hour the cortisol level is remeasured to see how well the adrenals responded. Sometimes you'll see a seriously hypoadrenal patient wean off pressors after being started on IV hydrocortisone. Impressive.

8-3: Testosterone: (“Yo, I'm a donor for that stuff, man!”) Couldn't resist. I don't think I've ever sent one.

9- Immunology:

9-1: A New Discovery - “Anti-RN” Antibodies:

Immune problems in general involve testing for problems in “self-recognition” - a condition which afflicts many nurses. I believe that this may be caused by “Anti-RN Antibodies”. I propose a research study: “Healing Our Pain: Exploring Levels of Anti-RN Antibodies in a Hospital Population of Nurses, Physicians, and Assorted Family Members: Do We Immune-Oppress Ourselves?”

Hmm. Maybe the Therapeutic Touch people could help us out here – “Feel the aura healing the antibodies...” Yo, keep your hands off my aura, man! Chi-square regress this! (Arrggh... spouse speaking soothing words into left ear while I get dragged once again into the ambulance...but I took my pill today!)

9-2: ANA:

(No, not that one.) “Anti-nuclear antibodies”. These can show up in patients with a number of disease states: lupus, rheumatoid arthritis, scleroderma, and also after certain chemical exposures. The result is reported as a titer: the patient’s serum is diluted, and the last dilution that still shows the antibody is the result – i.e. 1:20, or 1:60. Higher would presumably be worse – the antibody is still detectable at higher levels of dilution.

9-3: ANCA:

“Anti-Neutrophilic Cytoplasmic Antibody” – also auto-immune-produced. Titer results. This tests for a variety of unpleasant rheumatologic conditions: Wegener’s granulomatosis, polyarteritis nodosa, glomerulonephritis - rheumatologic things.

9-4: Rheumatoid Factor:

Also a titer-based test that indicates the presence of an antibody, this time for rheumatoid arthritis, positive in 70-80% of cases, usually at > 1:80.

9-5: Scleroderma Antibody:

Apparently not so useful, as it only shows up in 20% of patients with the disease. Phooey. Then again, would I really want to know? I guess so.

9-6: Immunoglobulins:

I wish I knew more about this stuff. Immunoglobulins turn out to be the proteins made by plasma cells in the bone marrow - they attack antigens introduced by specific invaders like bacteria or foreign chemicals. Immunoglobulins get the abbreviation “Ig” (pronounced “eye-gee”), and there are five main kinds: IgG (gamma), IgM (mu), IgA (alpha), IgE (epsilon), and IgD (delta). These guys take various forms, and do a number of different things, but basically their job is to hunt down and help kill off invading micro-organisms.

I know that I’ve hung IgG in the past, but it seems to have been pretty rare, and I forget what for.

10- Odd Labs: A few of these that don’t seem to fit easily into other categories:

10-1: Beta hCG:

Once in a while we’ll send one of these, often to make sure that a patient isn’t pregnant before we give some drug or treatment that might be dangerous to the baby.

10-2: Haptoglobin:

This one’s always been a mystery to me, but it doesn’t turn out to be too hard – it’s an indicator of hemolysis. Turns out that haptoglobin is a protein, made by the liver, that binds to the small fraction of hemoglobin that floats around unbound to any red cells – normally a very small

amount, which would only make sense. This normally small amount increases when red cells start to hemolyze, which also makes sense – more hemoglobin breaks loose and floats around.

Next – after binding up free hemoglobin, the haptoglobin-hemoglobin, um, molecule?, is taken up by the liver again, where things get recycled – and the haptoglobin is destroyed. Apparently this stuff isn't made very rapidly, so the level goes down. Low haptoglobin – hemolysis. Can someone tell me: so does this get sent after suspected transfusion reactions?

11- A nice picture.



Ain't it pretty? Old. Wahoo!