Not a drug reference in any official way – there's certainly lots of those around – but personal experiences and perspectives, some stories. **Take all the material here with many grains of salt,** and always check with your own pharmacy staff and references if you have questions. Which you should.

Remember Rule # 1 "There are no stupid questions."

Rule # 2: "Refer to Rule # 1".

What put the idea into my head: a new person was asking "Is it true that tylenol can really make a patient's blood pressure drop?". (It can, but may be a long time before you see it.) Another one: "I hate adenosine – it's ten seconds of pure terror." You get the idea. The meds are organized by system, and broken down in an extremely efficient way, which is to say the way they occurred to us. Please feel free to send in thoughts that you'd like to add!

1- CNS Meds:

1-1- Anticonvulsants / Anti-edema

- Dilantin/phenytoin
- fosphenytoin
- Valproate
- Tegretol/carbamazepine
- Phenobarbital
- Mannitol
- Keppra/levetiracetam

1-2- Psychiatric Meds:

Acute Confusion/Delirium

- Cimetidine (strange story)
- Haldol/haloperidol

Psychosis

- Risperidone
- Zyprexa/olanzapine
- Clozaril/clozapine
- Seroquel/quetiapine

1-3- Encephalopathy:

- Lactulose
- Thiamine

1-4- Depression:

- SSRIs: Prozac/fluoxetine, Zoloft/sertraline, Celexa/citalopram, Paxil/paroxetine
- MAOIs: Parnate/tranylcypromine, Nardil/phenelzine
- Tricyclics: Amytriptyline, Imipramine, Desipramine
- Wellbutrin/buproprion
- Lithium
- Dextroamphetamine

1-5- Sedatives/ Pain Meds:

- Propofol
- Opiates: Fentanyl, MS04, Methadone, Dilaudid/hydromorphone
- Narcan
- Benzos: Valium/diazepam, Ativan/lorazepam, Versed/midazolam
- Benzo drips: versed
- Flumazenil

1-6- Withdrawal:

- ETOH, opiates
- Serax/oxazepam, Librium/chlordiazepoxide
- Clonidine

1-7- Paralytics:

- Nimbex/cisatracurium
- Vecuronium, Pavulon/pancuronium, curare

2- Cardiac Meds:

2-1- Repleting the Elements: K^{+}, Mag^{+2}, Ca^{+2}, $P04^{+2}$

2-2- Hemodynamics: Controlling the rate:

- Digoxin
- Calcium channel blockers: diltiazem, nifedipine, verapimil
- Beta blockers: propranolol, metoprolol, esmolol, nadolol
- Alpha/ Beta blocker: Labetolol

The volume:

- Diuretics: lasix/furosemide, diuril/chlorothiazide, bumex/bumetanide, edecrine/ethacrynic acid, mannitol, diamox/acetazolamide

The squeeze:

- Nipride/nitroprusside, nitroglycerine
- Oral

2-3- Antiarrhythmics:

- Amiodarone
- Lidocaine
- Procainamide
- Adenosine

3- Pressors and Vasoactives (drips):

3-1: "Up" meds:

- Neosynephrine/phenylephrine
- Levophed/norephinephrine
- Dopamine
- Vasopressin
- Dobutamine
- Zebras: epinephrine, amrinone, milrinone, isuprel

3-2: "Down" meds:

- nitrates
- nipride
- labetolol
- regitine/phentolamine (in case of local infiltration trouble)

4- Pulmonary Meds:

- Beta agonists: albuterol, ipratroprium
- Mucolytics: mucomyst/acetylcysteine, DNAse
- Theophylline
- Glycopyrrolate

5- Gut Meds:

5-1: Too much acid:

- H2 Blockers: Cimetidine, Ranitidine, Famotidine
- Proton Pump Inhibitors: Prilosec/omeprazole, Nexium/esomeprazole
- Antacids

5-2: Coating the Stomach: carafate

5-3: Moving it Along: colace, senna, mag citrate, go-lytely, enemas, reglan/metoclopramide

5-4: Stop the Bleeding: octreotide, vasopressin/ NTG

5-5: Clearing the Ammonia: Lactulose

6- Endocrine Meds:

- Insulin: drips, sliding scales, regular, NPH, humulin, glargine
- Oral hypoglycemics: glucophage/metformin, glitazones
- Thyroid meds: Synthroid/levothyroxine
- Cort stim tests: cosyntropin
- DDAVP/desmopressin
- Steroids: hydrocortisone, methylprednisolone, pulse steroids, stress steroids

7- Renal Meds:

- renal dopamine
- phosphate binders: calcium acetate
- epogen/epoetin alfa

8- Anti-infectives:

- gram positive infections
- gram negative infections
- fungal infections: amphotericin B, ambisome, fluconazole
- TB meds: rifampin, INH/isoniazid, ethambutol
- Drug resistance: Linezolid/Synercid/quinupristine
- Antivirals: acyclovir, protease inhibitors

9- Antipyretics:

- Tylenol/acetaminophen
- Motrin/ibuprofen
- Aspirin

10- Anticoagulants:

- Heparin
- Coumadin/warfarin
- Low Molecular Weight Heparins: fragmin/dalteparin, lepirudin
- platelet drugs: plavix/clopidogrel, integrilin/eptifibatide, reopro/abciximab, aspirin
- Clot busters: TPA, streptokinase

11- Anti-Anti-coagulants:

- Protamine
- Vitamin K/

12- Miscellaneous Meds and Weird Reactions:

- Cancer chemotherapy in the MICU
- Code drugs
- Malignant hyperthermia and NMS: Dantrolene, bromocriptine
- Mucomyst for preventing renal dye damage
- Glucagon for beta-blocker OD
- THAM/tromethamine
- Reglan – strange extrapyramidal reactions
- Oral meds for low BP: midodrine, pseudoephedrine

Drug Fevers, Rashes

1- CNS Meds:

Remember, meds are always given in a context: there are things you're trying to accomplish, or prevent, or improve, or get more of, or get rid of. Goals you're trying to reach. Basic goals in neuro-med: preserve what you've got, closely monitor for changes, control seizures, minimize brain swelling. (Neuro is really not my thing – I floated once to a neuro ICU, and the charge nurse says, "So, uh, what do you know about neuro nursing?" I said: "Um, the brain is in the head, don't let it swell up." She said, "You're going to do just fine.")

Preserve What You've Got: to preserve what the patient's got, you have to be able to assess what's going on. This means that you may have to lighten a sedated patient sometimes, which can be problematic in some situations - status asthmaticus requiring paralysis and sedation is a good example. One way to observe neuro function might be to lighten sedation - a little - and see if they start responding vital-sign-wise to changes in position, voices nearby, etc. Rapid lightening may mean using short-acting meds like propofol – for situations requiring barbiturates and the like you'll probably have to check with the neuro folks for advice on how long it may take for your patient to wake up. Frequent pupil checks are good too.

More recently – of course, it's not recent to the newer nurses, but it seems like last week to me – we've started using BIS monitors to follow depth-of-sedation on our paralyzed patients. I understand that the guidelines from On High suggest completely lightening paralyzed or sedated patients once a day – mm. Not sure how practical that really is. BIS monitoring makes for a nice indicator, and we generally believe what they say.

1-1: Anticonvulsants:

Seizures are no fun. (I said I wasn't a neuro nurse, right? Do uncontrolled seizures really damage brain tissue? "Uh oh, he's going to be fried now…")

Dilantin: Dilantin works pretty well – I don't know what the stats are, but lots of seizure situations that I've seen have come under control with a dilantin load followed by regular dosing. Dilantin will definitely drop BP – it has to be given at a rate of 25mg/minute, so loading doses and reloads have to be given on a pump. There also used to be a story that you couldn't give dilantin po if the patient had a stomach full of tube feeds – is that still true? What if it takes four hours for the stomach to empty? Do you shut them off and wait? That long? And what was that old business about the gums growing?

Ok, new ICU nurses: what's the therapeutic range for this drug?

There's a new version of dilantin/ phenytoin, called **fosphenytoin** – apparently it's a lot safer to give intravenously, creates less hypotension.

Valproate: We see this one sometimes, given both po and IV. No big impression one way or the other. They do follow levels on this drug I think, like they do with Dilantin.

Tegretol: Don't see this one used too much lately. Can cause drops in white counts?

Phenobarb: Once in a while people will come in getting phenobarb as a maintenance med for seizure control, but rarely. I'm not even sure if they're using phenobarb comas anymore for status epilepticus – obviously I haven't had a patient like this in quite a while. Somebody check this out?

1-1 also: Drugs for Rising ICP:

Rising intra-cranial pressure is your basic Real Big Bad ICU Thing. Everybody immediately starts thinking about the triad – heart rate down, respiratory rate down, widening pulse pressure, right? Cushing's triad, I think it is. The thing to remember: **mentation goes first**. In other words – the first sign of rising ICP is a change in mentation for the worse. (What do you mean, "How am I supposed to tell with you?") Someone who was oriented becomes disoriented, or progressively unresponsive. **You do not want to wait for the triad to show up before you start treating the patient for rising ICP**. The way I was taught: at the first sign of a problem, get help – and give **mannitol**. It shouldn't hurt, hopefully will help – is that still true? Mannitol always has to be filtered, and the goal is to get the patient's brain "dry": all other things being equal, the serum osm is supposed to get to between 310 and 320 (?)That's pretty dry.

Keppra/levetiracetam – newer anti-seizure med. Not much experience with it, anyone want to contribute?

1-2- Psychiatric Meds:

Lots of psych situations come up in the ICU, and they can take some sorting out, but as I see it at the basic bedside level, you're trying to do two things: keep the patient safe, and try to figure out if something bad is happening. This is often not something that you're going to be able to figure out by yourself, so get help. For really acute situations and questions of competence (what do you mean, "Yours?") there's an Acute Psych Services person on call at all times.

Acute confusion/delirium:

Some situations develop quickly, and you need to have a plan. Sometimes (it seems like less often lately) patients will develop "ICU psychosis" – I remember any number of patients, usually elderly – but not always, who become acutely confused when suddenly taken out of their own environment and stuck in a hospital bed. Managing this can be a tough problem – and sometimes calls for radical measures: intra-aortic balloon patients are famous for getting acutely confused, for example. A ballooned patient can not be allowed to sit up, because the balloon (think of a hot dog at the end of a straightened-out wire hanger) – can be pushed inwards, and poke up through the arch of the aorta.

The result may be that a series of meds get tried on the patient, some of which may work, or not work, or make things worse… the end point however is that sometimes these patients have to be intubated, so that they can be safely sedated. It's always good to have a plan! Of course, a medical intern will look at you like a gunshot buffalo when you suggest that your mildly agitated, ballooned patient may need this at some point… so be ready to climb the chain of command quickly!

• Some patients have weird responses to meds – once in a while you'll see a patient who has a "paradoxical response" to a sedative: in other words, it'll make them more agitated instead of less. Ativan is famous for this, and rarely Versed.

• Another episode I'll never forget: a nice man, who I forget exactly what was wrong with him, who went from oriented to absolutely wild, really screaming crazy, over the period of a couple of hours. Psych came up, took a look, and decided that it was the **cimetidine** he'd been started on. We all looked at each other – this, we'd never heard of. And they were right!

Haldol (haloperidol): Haldol works well sometimes – sometimes very well, sometimes not at all well. The good thing about haldol is that it inhibits respiration the least of all the sedatives we use, which makes it a good choice for patients who are having breathing problems. The last thing you want to do with a patient who's already hypercarbic is to sedate him with something like **Valium**, which will probably double his pC02 and push him over the edge of intubation.

Haldol can also drop a patient's blood pressure, but I think that has more to do with the general effects of sedation, since lots of sedatives will do this. A classic blood pressure scenario involves intubation – you have a patient who's been developing respiratory distress over, say, a week or so. Very dehydrated, hasn't taken in much fluid over that whole time. Comes into the unit in increasing distress – tachycardic, maybe hypertensive, but actually (being so dry), maybe not. Huffing and puffing, agitated, they get a dose of **etomidate** for intubation, or maybe propofol, and boom – blood pressure drops to 60 systolic. And the team is <u>always</u> surprised. Don't you be – make sure that you have good IV access, a running gravity line of normal saline that you can give fluid through, and maybe a dilute mix of **neosynephrine** ready to go on a pump nearby. We use 10mg of neo in 250cc for situations requiring peripheral administration, but only for very short use. Take a look at the "Intubation" FAQ for more on this.

I saw an anesthesiologist give a dose of **ephedrine** in this situation once, and didn't like want happened at all! Heart rate doubled, blood pressure doubled, nothing titratable or controllable about the situation at all! Made me very unhappy, and I don't think the patient liked it either! I like using neosynephrine MUCH better for this situation.

Psychosis

(Just don't say it, okay? What do you mean, "Who are you talking to?") Some people in the unit get treated with antipsychotic meds, usually the ones they were on before they came in. More acute problems obviously take precedence, but I think the idea is to try to keep the treatment going unless it's causing other problems.

Risperidone: I don't know much about this one except that I've seen it work really well a couple of times, for patients who have been severely disoriented and agitated. Sometimes patient will be treated with a variety of meds – say haldol round the clock, maybe along with some antihypertensive to control elevated blood pressure, and maybe a beta blocker to control tachycardia. I seem to remember risperidone working so well that it replaced all of those. You may see this kind of thing sometimes: an agitated patient may be treated with cardiovascular meds, and a more appropriate treatment may not come until psychiatry gets a look at the patient and figures out the problem.

Olanzapine, **clozapine**: these are **zyprexa**, and **clozaril**, respectively. I don't know much about either of them, but I read somewhere that after a patient who'd been in uncontrolled schizophrenia – for many years (maybe 20?, 30?) - was started on one of these, his brother burst into tears, because his brother had finally "come back". If they really work that well, I hope they save some for me….

Something we saw recently about clozaril: we had a patient come in with really high, almost uncontrollable temperatures up to around 106 F, and the team was worried about **neuroleptic malignant syndrome**, because apparently clozaril is on that list somewhere. The fevers actually turned out to be related to her sepsis (pneumonia), and eventually came down, although they treated her anyhow with **bromocriptine** for her possible NMS for the whole ICU admission. Held the clozaril too.

Seroquel – yup, listed as an antipsychotic. A lot of people seem to get this to help them sleep. Works pretty well!

1-3- Encephalopathy

(What do you mean, "You!"? So I like to lie on the couch – so what!?)

Lactulose: The nurse's favorite. Works, though. One thing we've tried that actually turned out to be useful: if a patient has a really sore perianal area, adding lactulose to their tube feed diet a couple of times a day may make their stool liquid enough to be managed with a rectal tube, allowing the skin to heal around it.

Thiamine: Someone explain this one to me: what exactly is Wernicke's thing, and how does thiamine fix it? Something to do with alcoholism. And what is Korsakoff's thing? Rimsky-Korsakoff – he was the russian composer who discovered thiamine?

Banana Bags: 1 amp of mvi, 2 grams of magnesium, 1mg of folate, 100mg of thiamine, in D5 NS (probably), shaken, not stirred. We don't use these, but apparently they're useful in alcohol situations. (Do they keep them behind the bar at Ralph's?)

1-4- Depression

(We're always talking about putting Prozac in the coffee in the nurses' lounge…)

If our patients were the ones you went by - overdoses is what I'm thinking of here - you'd have to say that antidepressants just don't work at all. Of course, that would be like saying that all of us are going to die of ARDS in our 30's. Our patients are sometimes on antidepressants when they come in, and they're usually kept on them while they're with us.

SSRI's: **Prozac**, **Zoloft**, **Celexa**, **Paxil** – Once in a while we'll get an overdose patient in who's taken all their prozac, or zoloft, or whatever SSRI they're on. These drugs are apparently so non-toxic that they're safe to hand over to horribly depressed, possibly suicidal patients, unlike **tricyclics** and **MAOIs**. (Weren't the MAOIs the native people in

Fiji or someplace?) There was an interesting story some years back about Prozac and yawning… try Googling that one ☺

MAOIs: Let's see if I can remember them without looking – **Parnate** is one, and **Nardil**. Sounds like a couple of the characters in the Lord of the Rings. I guess these are really rare now, hardly used any more. One really critical thing to try to remember is that these patients **MUST NOT GET DEMEROL.** (Is that for all of the MAOIs or only some? – I wouldn't want to get it wrong.) I don't know why exactly – but apparently it produces a potentially **fatal reaction**. Important. Another thing with these drugs is that if the patient makes a mistake and eats something wrong containing tyramine (?)– cheese, or wine of one kind or another, or I think maybe snow pea pods or whatever, they can have a truly severe hypertensive reaction, requiring rapid treatment with **nifedipine** – or maybe something like **nipride**? Never seen it happen. I don't think I've ever given anyone any of these meds.

There's a new version of one of these meds out now in patch form – Emsam, I think it's called.

Tricyclics: We do see tricyclic overdoses sometimes. Unlike the SSRIs, these meds can kill, and I guess pretty quickly – they interfere with cardiac conduction. I don't remember – do these patients ever buy temporary pacing wires? I think they also have to get a **bicarb** drip, something about protecting the kidneys by alkalinizing the urine.

Wellbutrin: I think I've given this a couple of times, but it's not much of an ICU med. I think it's also on the "relatively safe" list.

Lithium: for bipolar disease, right? Famous line: "I don't miss my lows, but I sure do miss my highs." Lithium can definitely be toxic – not sure what the treatments are, but I know that they do draw levels. Come to think of it, it seems as though we ought to develop a "Tox and Poisoning" FAQ. I don't seem to recall giving lithium to patients while in the MICU. My colleague Cathie just leaned over and mentioned that a renal attending told her that chronic lithium use for bipolar disease seems to be associated with eventually developing renal failure. (So should I stop taking it now, or can I still go on for a while? Where's the men's room?)

Dextroamphetamine: Once in a while this one still pops up. I think the idea is that the drug will supply some kind of energizing pop in the morning to patients who can't seem to mobilize themselves. Doesn't seem to do much, and I seem to recall that it effectively stops people from sleeping. Add a sleeping med in the pm, and you wind up with a seriously messed up situation.

1-5- Sedatives / Pain Meds

Propofol: Ah, the "Milk of Amnesia". ICU nurses love propofol. Works well, works quickly, wears off almost as fast as it goes to work. Two main cautions: propofol will drop your patient's blood pressure, sometimes more, sometimes less, so be ready. Second: **propofol will make your patient stop breathing.** So be ready for that too. There's more on all this in the "Sedation and Paralysis" FAQ.

A nice thing about propofol is that it has no effect that I know of on the gut, so your patient can be started on tube feeds without having to worry about opiate ileus

problems. Another thing to remember is that it comes as a lipid mix, so your TPN patient may need to have lipids removed from her formula if she stays on the drug for a while.

Opiates: We use a lot of opiates in the unit, sometimes in combination with benzos or propofol. Most people have a pretty good idea of what opiates are all about by the time they get to the unit, but they probably haven't seen them given in the doses that we use for long-term sedation. It's good to keep in mind that their strengths are different: **morphine** is what everything else is compared to – so for example one milligram of **dilaudid** is equivalent to 4 or 6 mg of morphine. I think 50mcg of **fentanyl** equals 2-4 milligrams of morphine – there are equivalency charts around that you can look at.

Fentanyl: Fentanyl is the propofol of the opiates – works quickly, wears off quickly. Used to be, we ran people on morphine drips for the long term, but nowadays we're using fentanyl because apparently it causes less of a histamine release, which is better for patients who have an inflammatory component, like asthmatics. Sometimes we use a lot - we're running a woman now on a fentanyl drip at 2200mcg/ hour, when the usual range is something like 100-300. That high of a dose usually means that they've been on it for a while – at least a couple of weeks. My impression is that people habituate pretty quickly to fentanyl, and may need a higher dose every few days to keep a steady level.

Patients like that will definitely have to be weaned carefully to avoid withdrawal. (You may not avoid withdrawal completely as you wean the med, but you should understand the idea.) The usual method is to try to wean the drip 25% per day – which does not mean 4 days – it means 25% each day of what's running. So the lady on 2200mcg would go down by 25% the first day, to 1650. Second day, you'd go down by 25% of the 1650, or 420-odd, to about 1235. And so on. Even then you may have to pop them back up sometimes.

How would you know if your patient was withdrawing? What could you do to tell?

MSO4: I got my first - ever personal exposure to morphine a couple of months ago, when my rapidly advancing age introduced me to the happy concept of kidney stones. I got a total of nine mg in two doses, and I can't say it made me euphoric, but I sure was happy the when the pain went away. Took a nice nap too. (Apparently I was quite a spectacle. Cried like a baby. My wife points to her uterus and says: "See?")

We use little bits of morphine in the unit for pain management, apart from narcotic drips, because it's easily reversed. A patient with respiratory problems after surgery may only get small doses of morphine on a careful prn basis – there are always lots of things to think about. Ten milligrams of morphine is equivalent to 10mg of morphine…wait a minute…..Doh!

Here's a neat morphine trick: the next time a patient manages to do a Houdini and yanks out everything including ET tube, IVs, and all, and you have no access to try to give something to calm him, try nebulizing five milligrams of morphine through a mask…one of our attendings came up with this on the fly one night, and it worked really well.

A word about PCA pumps in the MICU – "Patient Controlled Analgesia". I'm all for patient-controlled analgesia – I think it's a great idea. Just not in the ICU. A postop patient in the ICU is there for serious problems – blood pressure maybe, breathing more likely. I think the nurse at the bedside should be the one in control of pain management

– he needs to be the one giving the doses and watching what happens. Not the patient. Just my opinion.

Methadone: Now and again we have addicted folks in the unit, and they are usually started on methadone right away to prevent withdrawal. (Of course they sometimes come in as Mr. or Ms. Who Knows Who They Are – unresponsive, sometimes no ID, sometimes no clothes…so it can be hard to tell. Tox screens help sort things out.)

Dilaudid: Dilaudid (hydromorphone) is strong stuff. The chart says that 1mg of dilaudid (hydromorphone) is equivalent to 4mg of morphine. We use dilaudid drips occasionally – I'm never really clear on why, since usually we go for agents that wear off more quickly.

A couple more things from the chart: **Demerol** 75mg, and **oxycodone** 30mg are each equivalent to ten of morphine…

Opiate Antagonist: Narcan ("Narcotic Antagonist")

We used narcan last week – we got a patient with respiratory depression, apparently from her patient-controlled-analgesia (she'd had a fractured hip repaired). I guess she got too much. (What did we just say about PCA pumps…?)

I've learned to be careful when giving narcan – if your patient is intubated and lined (or even if they're not), you may want to think about applying restraints before giving the dose. As I understand it, Narcan pushes the opiate molecules off their receptor sites, and if the patient was in pain before, it may be worse afterwards.

In other words, they may levitate, scream, shout, swear, and otherwise demonstrate an alteration in comfort level secondary to an alteration in opiate receptor status mediated by pharmacologic alteration of pain management as evidenced by, uh, screaming. (Got to love nursing diagnosis.) That's to say, they become distressed. So - narcan isn't always a nice thing to do. The hip lady got some, which was before I came on, and I wonder how she felt – would it have been kinder to intubate her and let her "cook off" her opiates to a safe level? Risk? Benefit? Judgment call.

The other thing about narcan is that it doesn't last very long – it doesn't "cure" an opiate overdose. Your patient may have to be intubated for respiratory support until the overdose is metabolized off.

Benzos

Valium (diazepam): seems like we don't use Valium so much these days; it seems like we use **Ativan** (lorazepam) instead. Patients at risk for alcohol withdrawal get put on Ativan doses round the clock, with extra doses prn. Alcohol withdrawal can require really impressive amounts of benzos – back in the Valium days we used to see patients get thirty to forty milligrams IV. Every hour. Or more.

Versed (midazolam): Used mostly for procedures on un-intubated patients, such as those undergoing endoscopy. It seems to work well, and it's very short-acting. I'm supposed to have an upper endoscopy myself at some point, and I'll have a report from the field to add at some point.

Ok, here's my report. So I turned 50 – it happens. Went off for my endoscopy – both ends, such a lovely birthday present. Man, that versed is the greatest! I didn't remember a THING!

Benzo drips – versed drips are the one we use. The other benzos tend to hang around for a long time, which is why we try to use the shorter-acting agents. The idea is to try to shorten ICU stays, rather than lengthen them.

Benzo Antagonist: **Flumazenil**. This is Narcan for benzodiazepines. Needs to be used carefully, since a patient that takes benzos chronically can be pushed into seizures by this drug. For overdoses, we usually let the patients cook off the drug while they're intubated (since their main lethal effect is that they make people stop breathing). Then once awake, out comes the tube, they get evaluated by Psych, and off they go.

I remember when they were doing the double-blind trials on this drug – apparently the research team was notified whenever a benzo overdose showed up in the ER, so up they came, and there was my intubated patient in the bed, very sleepy, not really rousable, but otherwise stable. The nurse working on the study told me "You know, we never know if we're giving the drug, or just a dose of normal saline." They pushed the dose, the woman's eyes opened wide, she sat straight up in the bed (in her four-point leathers), and almost levitated. The nurse: "I guess that was the drug, huh?" Ya think?!

1-6- Withdrawal

Patients can withdraw from all kinds of substances: alcohol and opiates are the ones we see the most. Lately we've gotten a little better about putting **nicotine** patches on patients who smoke, but I bet it gets missed a lot more than we think.

As we looked at a minute ago, people withdrawing from alcohol usually get treated with benzos – sometimes a whole lot of benzos. It can be scary, and I can remember at least several times being given orders for 30mg of Valium an hour. Or twice an hour. More recently, it turns out that propofol "covers" alcohol withdrawal in pretty much the same way that benzos do. GABA receptors, is it?

So for example, let's think of a scenario: gentleman found "down" in the park, bottles of scotch nearby, vomitus nearby, probable aspiration, intubated at the scene. Comes to the ER, sent to the MICU, nasty looking x-ray (right – you guys who read the X-ray FAQ: any quick and easy clues on a chest film to make you think of aspiration?). The ER nurse gives you report: "He's really been agitated down here – we've given him ten of Ativan over two hours, which is more than I've ever given before, and he's better now, but they had to push a dose of vecuronium to get him through his head and neck CT – he's still got his collar on, and they haven't cleared his C-spines. He's tachy at about 120, sinus, and his pressure is 160 systolic – he only put out about a hundred cc when we put his Foley in…".

This is actually going to be a little bit of a puzzler, because now he's got at least two reasons to be tachycardic. (What are they?) So he comes up from the scanner (thank you, thank you ER, for scanning him before sending him to us), and his vec dose is wearing off, and he's trying to yank out anything out he can reach – his ET tube, his IV's, his Foley, the nurse's fingers, the stretcher rails – at this point the nurse will usually turn to the resident (who hopefully is still in the room), and ask for a propofol order. Our

range is 0 – 300mg/hour ("Okay, so I got him on zero of propofol, and he's been really well - sedated with that, so I haven't gone up at all". I mean, why do they write "zero to 300"?) We'll give a small bolus dose of maybe 20 mg to start, and then depending on size and degree of agitation I might start the drip at 100 /hour, see how it worked, and then go up if necessary, or if the patient was absolutely enormous and totally wild, I might start at 300, and work backwards. The nice thing is that you know that not only is their short-term agitation covered, but withdrawal should be too. (Except that it's probably a little early for DT's, unless he'd been lying in the park for about 3 days.)

So the strategy is something like: propofol for the short term, and round-the-clock benzos until it seems clear that an effective "level" has been reached. "Level" is in quotes because we don't actually measure one, the way we might with digoxin or dilantin, but you get the idea – we're trying to figure out what regimen will keep the patient stable so that they can be weaned off the propofol and the vent. Although that can often take a little longer than anticipated…and remember – no vent, no propofol. It's worth repeating: **Propofol will make your patient stop breathing**.

Other drugs used for DT prophylaxis are also benzos, eventually oral, like **serax** and **librium**, although we hardly ever see them used any more – ativan is all the fashion now. It's interesting to see things come, and go, and come back again, and maybe go away again as the years go by, as (presumably) the studies come in showing that one thing works and the other doesn't.

Clonidine – This is an interesting use for a drug – it turns out that clonidine, the antihypertensive also known as catapres, works to block the adrenergic release of endogenous catecholamines during withdrawal episodes. (Whoa! That sounded good! Bet I can't say that again!) Or so it has been explained to me. Anyhow, patients withdrawing from all sorts of things: alcohol, opiates, cocaine, can be started on clonidine, usually in the form of a patch. Neat! Remember that this is not the same as **klonopin** (clonazepam), which is one of the benzos… which reminds me of the time years ago, hunting around in the med closet, we found that vials of **Pavulon** (pancuronium) looked almost exactly the same as vials of something else – **gentamicin** maybe… yikes!

1-7- Paralytics

There's a whole article devoted to "Sedation and Paralysis", so I won't get into all the details here, but there seems to be some basic difficulty that people have in telling the difference between the two. Every now and then I still hear someone say "Okay, so he's sedated with **Nimbex**…". No. No, no, no. Let's recap the basics here:

1. Sedation is sedation.
2. Paralysis is paralysis.
3. They are not the same. As in: **"NOT THE SAME !**".

Here's a story: patient is being transferred to us from an OSH (Outside Hospital). I'm the charge nurse. Our RN calls their ICU for report:

"Well, we paralyzed him, but he's gotten really tachycardic and hypertensive, so we started him on IV **nitro**, and we've given him a couple of doses of **lopressor**."

At this point our house officers (first-year interns, second-year junior) start looking worried. I can almost read their thought process: "Okay, so the guy's having an MI, maybe he's having an aortic dissection, oh this could be really bad, we better line up the scanner now, maybe we should anticoagulate him, has he gotten his aspirin, what's his troponin, does he have ekg changes, who's the cath fellow on call tonight…". And so on.

I look at my nurse, she looks at me. "What's he sedated with?", we ask, as one.

"**Ketamine**." Oh, this is a weird one. I know that they use ketamine at the animal hospital where I take my greyhound to get her teeth cleaned every couple of years, and I know that there's some street traffic in it as some sort of hallucinogen, but I've never in my – what is it now? – 23 years in the units, seen ketamine used on people. "Who's managing the patient?" we ask.

"Anesthesia". "Well, is the patient getting enough of the drug?" "I don't know", says the RN on the other end, "I've never given it before."

Uh-oh. This is a patient who's chemically paralyzed but apparently hardly sedated, and he's got to be going bonkers with terror on the inside. We turn to the team, who are still rapidly doing the differential diagnosis thing, and say: "Well, we know why he's so tachycardic." Skeptical looks from the team: "Why?" "He's hardly sedated under his paralysis." We explain about the ketamine. More doubtful looks.

The patient arrives. Report from the paramedics: "He's still paralyzed, really tachycardic and hypertensive, so we kept the nitro going in transit." Apparently the patient got no more sedation on his trip.

In our ICU, starting propofol is pretty much standard practice in situations like these. A patient who is intubated, but who may or may not be adequately sedated, or paralyzed, or both – the immediate goal is to produce safe sedation, and to evaluate the whole picture. Let's say this is a fairly large male patient - we might start him with a 20mg propofol bolus, and start a drip at 100mg/ hour – not an enormous dose. Bingo – heart rate drops from 130's to 74, BP from 230 to 120 systolic. The team looks at us in disbelief, as though we had pulled a moose out of a rabbit-sized hat. The patient rules out for an MI.

This is actually a good illustration of an important point, which is that over the course of some three years, a general medical resident spends three months in the ICU. This means that over the course of one year, a brand-new ICU nurse gets four times as much exposure to ICU patients than the residents do. The result is that some things begin to become very obvious to the nurse, that remain intellectual puzzles for the MDs.

Another quick story about this situation: my wife the CNS (she's a real CNS, the kind that gets into the blood and the poo and the everything else, right there in the bed with her orientees and their patients), goes off to one of these conferences of the Inter-Galactic Association of Critical Care Things and Stuff, having to do with intensive care trends, modalities, interventions, and all like that. Physician is standing up, giving a lecture on vent-weaning strategies. She's a pulmonologist someplace, and says something like: "Now, we did an informal survey in our unit, just keeping numbers for a while, and we found that our staff nurses could correctly predict whether our patients would successfully extubate 86% of the time. Would you believe it?" To which the nurses all responded (on the inside): "Well <u>duh</u>!"

And this actually raises another really important point: ICU nurses develop skills that the physicians don't even know exist. This leads to many of the arguments that nurses have with physicians about management of one situation or another – an attending who takes the time to get to know her nursing staff, who takes the time to get familiar with their expertise, will have an incomparably easier time than one who blows them off when we voice our concerns.

Nimbex: (Cis-atracurium) Back in the days when we used to weave in between the mammoths on the highway to get to work, we used to use **Pavulon** (pancuronium) pushes of 2-4 mg every 2-4 hours to keep our patients paralyzed. Then we started using **curare** drips (the stuff that they shoot monkeys with in the rainforest – what happens when they eat the monkey? They must get very relaxed.) Apparently Nimbex causes less of a histamine release than the older drugs, and so is better for situations like asthma, that have an inflammatory component. Same for the change from morphine to fentanyl drips.

Vecuronium: "Vec" – also hardly used anymore, but once in a while the ER will give a dose – 10mg? to a patient who's severely agitated (**along with sedation!**) for a scan before coming up to us. Wears off in an hour or two. While the OR people do it, I can't say that we ever reverse paralysis nowadays – we just keep the patient sedate and wait for it to wear off.

2- Cardiac Meds:

Well, here's a topic that could (and does) take up whole textbooks. But the goal here is not to create a reference, but just to yak about meds and how well they work or how weird they are, so maybe it won't go on forever. (It'll just feel that way.)

2-1- Repleting the Elements: Very important – but you guys know about this.

K^+: You always need to know what your patient's potassium is. If you're diuresing him, plan ahead – do you need to remind the team to order K along with the **lasix**? And by the way, what's the BUN and creatinine? (And why am I asking?) K generally wants to be above 4.0 if the patient is on digoxin…

Mag^+2: Mag turns out to be right up there with K^+ in terms of keeping the heart happy. If your patient is having ectopy, know both values, and be aggressive in treating them.

Calcium: Lots of argument about replacing calcium, but in practice, we do it. Certainly patients on CVVH need it, because the machine sucks it (along with K^+) right out of them, so they usually get replaced continuously. We tend to replace calcium when it's low in our unit.

Phosphorus: We replace P04 with either sodium phos or potassium phos, usually something like 10 or 20 or 30 millimoles. Has to be given slowly – last night I gave the last of a 30 millimole dose, which took up a central line port for the whole shift.

2-2- Hemodynamics: Controlling the Rate

Many of the treatment goals in the MICU have to do with controlling one or the other of these, because too much of either one increases cardiac work, either from the effort of a rapid rhythm or the work of pumping into a really tight arterial system. This is described in typically roundabout fashion as "myocardial oxygen consumption" – which is like measuring a car's horsepower by measuring gasoline consumption. Makes sense I guess…anyhow, the point is that a heart that's hurting doesn't want to beat too quickly, or work too hard at pumping blood into the arterial system. A rate nearer that of NSR is generally better, and a relatively dilated (rather than constricted) arterial bed is easier to pump into. (This last is what "afterload" is all about. "Preload" has to do with the amount of volume arriving in the LV – it's all supposed to get pumped out, right? If not, then things begin to back up – CHF ensues.)

Digoxin: Dig ("Didge") is one of the real oldies – apparently people have been using digitalis leaf to control heart rate for at least a couple of hundred years. I can still just about remember the days before dig levels – the joke was "Load 'em till they start vomiting, then back off." We do it a little better now. Know your patient's BUN and creatinine. Dig is also just about the only oral inotrope around – there used to be a little blue pill in trials called **enoxamone**, which appeared to work very well – at least some of the time. Any new ones coming along?

Calcium Channel Blockers: **Diltiazem, Nifedipine, Verapamil** - these were the original three, and they formed a range of effect: Verapamil slows rate the most, nifedipine drops blood pressure the most, and diltiazem is in the middle somewhere. I know that most places use a lot of dilt drips – for whatever reason, I've only seen it done once or twice.

Beta Blockers: inderal/propranolol, metoprolol, esmolol, nadolol, etc.

These are the meds most commonly used for controlling heart rate. A basic concept to grasp is the adrenergic receptor thing – there's lots about this in the "Pressors and Vasoactives" file, but basically there are three receptor groups (this is with a lot of lies thrown in – I understand that there are more, but keeping it simple will help here.) Alpha receptors live in the arteries. You have **1** heart, so that's where the beta-**1**s live. You have **2** lungs, that's where the beta-**2**s live.

If you agonize a receptor, then it does more of what it's supposed to. If you antagonize it, then it does less of what it's supposed to. So if you give a beta-agonist, like **albuterol**, then the beta-2s in the lungs do their thing, and the bronchi open up. (And the heart rate goes up). Beta agonist.

So then giving the opposite of albuterol, namely a beta-antagonist, or beta-"blocker" (everybody says "aha!" at this point), will block the betas, lowering the heart rate and, if you're unlucky, provoking an asthma attack in your patient. Which is why asthmatics don't get beta-blockers, they get something like verapamil instead.

We use a lot of lopressor and verapamil for rate control in our unit. Once in a while we'll see an **esmolol** drip, but I have to say that I think it doesn't really work very well.

Of course, there's always the heart rate that's too slow, but that's a whole other article…

Labetolol – this is a neat idea: it's both an alpha and a beta blocker. So it produces arterial dilation, and slower heart rate.

The Volume:

Diuretics: Everybody remembers that the three parts of a blood pressure are "pump", "volume", and "arterial squeeze"? Strategies for lowering blood pressure center around picking one of these. The pump component is treated by lowering the heart rate, with one or the other or even a cocktail of the meds we were talking about above.

The volume aspect is what diuretics are all about. There are a couple of kinds – the most commonly used are the **loop diuretics**: **lasix**, **diuril**, and I think **bumex**, which I understand were invented by the famous medical rock star Don ("Loopy") Henley. Something like that. I think I'm right when I remember being told that lasix and bumex work on one part of the loop, and that diuril works on another part, and that the combination effect makes the drugs work together more powerfully – "synergistic diuresis" – wasn't that on the flip side of "Hotel California"…?

One recent development in the diuretic world is the idea of giving a big wallop of diuretic to patients who are acutely sliding down the slope into renal failure – the patients who have taken a recent kidney hit because of a prolonged hypotensive episode for example. The theory is that doing this will push the failing kidneys into making good amounts of urine, so that the patient will be able to get rid of fluids, if nothing else. At least that way they won't get into CHF. Obviously this doesn't solve the whole problem though.

Edecrine is a diuretic that can be given to patients with a lasix allergy. Turns out that patients with a sulfa allergy can be allergic to lasix as well…

The two last ones are **mannitol** and **diamox**. Mannitol we use in neuro situations when the worry is increasing ICP – "osmotic" diuresis literally shrinks the brain. (Wouldn't work for me – too small already.) Mannitol easily forms crystals, so it gets filtered.

Diamox is interesting – it causes the kidneys to dump bicarb, so it's useful in alkalosis. Something I heard, which may or may not be true: you go climbing up Mount Everest. The air gets thin, and you start breathing rapidly – producing a respiratory alkalosis, right? Breathing rapidly blows off C02, which leaves you with a carbonic acid deficit, and a relative bicarb excess. (An absolute excess would be if you drank both bottles of Maalox you'd brought along.) Now your fingers and toes start tingling, you get shivery – what to do? Take your diamox pills – you'll dump bicarb, and your ph will come back down. Of course, now you have another problem, right? You come back down the mountain, and your breathing straightens out, and wups! Ack! Where'd your bicarb go! Now you have a new problem!

The Squeeze

The third part of a blood pressure is "arterial squeeze". This is what SVR means: systemic vascular resistance, one of the numbers that comes up when you "shoot numbers" with a PA line. High is tight, low is loose. Another word for this resistance faced by the LV – which determines how hard it has to work to empty itself into the arterial bed – is "afterload". High afterload can be a good thing sometimes, usually in some form of hypovolemic or cardiogenic shock state where getting tight is the only thing keeping your patient's pressure up. But a nicotine addict will walk around in a tight state – which puts lots more work onto his heart. Also bad for anyone with any degree of

pump failure – CHF, in other words. Loosening up a tight afterload really helps a failing heart. Flowers and nice dogs work too, maybe through the same mechanism.

Most commonly used in blood pressure situations are **nitrates**, which in the unit usually come in the form of IV nitroglycerine. Nitrates open up the arterial bed – and sure, they do do that - I mean we've all seen patients with angina respond to sublingual nitrate pills. But honestly, IV nitro just doesn't live up to its reputation as an antihypertensive.

For really serious hypertension situations, the drug to use is nipride (nitroprusside). This drug is da bomb. It really is like a bomb, too. This is a really serious, powerful drug, sort of the opposite of norepinephrine. **Nipride must run in a line all by itself.** Anything put to run through a nipride line may kill your patient. (It also isn't compatible with anything.) Don't even think about flushing a heplock with nipride in it – aspirate it instead, then flush with saline. Very serious medicine.

A useful nipride tip: if you use a very concentrated mix, like 250mg in 250cc, then if you decide to doulbe your rate, your rate change may only be a cc or two per hour. This is a bad idea – a much better idea is to use a dilute mix. I like 50mg in 250cc – that way if I want to change the dose, the change in rate will be something like 20 up to 35 cc per hour. The drip is much more titratable that way, and you'll be able to "fine-tune" the drip much more closely.

There are lots of oral antihypertensives: oral nitrates like **isordil**, oral **ace inhibitors** like **captopril**, alpha blockers like **prazosin** – (remember about antagonizing the alphas? – makes the arteries dilate.) The thing about oral meds is that they're not particularly titratable, which is something that you really want in the ICU. The nice thing about IV nitro and nipride is that their effects go away really fast – turn the drips off, and their effects are gone within a minute or two. For the same reason, changing the drip rates produces quick responses in the patient. Titratable. It's a lot harder to go down and suck out an oral med that's halfway through the small intestine than it is to turn down the nipride… I have to say that it seems like the best oral BP med is sustained-release nifedipine. Excellent drug, but too powerful in it's regular form.

Ischemia We all know about this one, right? Tight coronary artery lesions, not enough **oxygen** is getting to the cardiac muscle tissue, and pain ensues. What to do? Anything that increases oxygen delivery, or that decreases the workload of the heart will help. So: first drug is what? Oxygen!

Next: probably nitrates. Sublingual, IV nitro, etc. Then, or maybe even at the same time, rate control. Ask the patient if he has asthma…

2-3- Antiarrhythmics

Obviously dating myself here, but of course the first thing I think of is **lidocaine**. There have been big changes in antiarrhythmic treatments lately though – now **amiodarone** comes first. Great drug – does all sorts of neat things like converting a-fib into sinus rhythm, controls ventricular arrhythmias – in fact, it comes first in the VT/ VF code algorithms now. Two percent of people on amiodarone get nasty fibrotic lung effects. Very unpleasant. Amiodarone gets loaded, usually 150mg over ten minutes (fast!), then

dripped at 1 mg per minute for something like 6 hours, then half a milligram for another 18 hours and then changed to oral.

We really don't see anything like the numbers of VT/ VF codes that we used to back in the Ice Age – the difference is that now we clot-bust most MI patients – it's the MI's that mostly make for the really awful arrhythmic codes.

Lidocaine – yup, once in a while we still use lido. 50 to 100mg load, followed by a drip at 1-2 mg per minute. Lido can make people acutely bonkers, which usually goes away if it's stopped.

Procainamide – second antiarrythmic up until the rise of amiodarone – is it third now? Proc ("Proke") also gets loaded, and then run in mgs per minute. Been a long time since I saw this up and running. Be careful – a proc load can really drop your patient's pressure.

Bretyllium – gone altogether now, I think. I remember being really impressed when they were coding "ET" – before or after he phoned home?, and they called for bretyllium…that was a while ago.

Adenosine – I love and hate adenosine. Very powerful drug, used to break rapid supraventricular rhythms – the problem is, it creates about ten seconds of asystole in both the patient and the nurse giving the drug…

3- Pressors/ Vasoactives

These meds have a FAQ file all to themselves, so that's where to go for the detailed stuff. The idea behind vasoactive drips is that they either raise ("Up" meds), or lower heart rate and/or blood pressure ("Down" meds).

3-1: "Up" Meds

Simple concept – raising blood pressure, which is what the word "pressor" is all about.

This stuff calls for a little memorization. The thing to figure out is: which set of receptors do you want to work on, and why? The answer depends on which of the three parts of the blood pressure is being affected. If it's pump (heart), then it's the beta-1s you want to go after. If it's volume – then you may not want to give a pressor at all if you can avoid it. If it's arterial squeeze (sepsis), then it's the alphas out in the arteries that you want to hit. There's more than you probably ever wanted to know on this subject in the "PA-lines" FAQ, which is where we go into this stuff in more detail. The main point is that "shooting the numbers" with your PA line gives you indices for all three parts: cardiac output/ index tells you about your patient's pump status, the SVR tells you about the arterial squeeze, and the CVP, wedge pressure, and stroke volume tell you about the, uh, volume.

Neosynephrine – (phenylephrine): "Neo" is pure alpha. This is what you want to use in sepsis, when the arteries are dilated. It doesn't have any direct effect on heart rate, but some people worry that it can constrict the coronary arteries too, producing angina, etc.

I can't say that I've ever noticed that to happen, although these patients can certainly get angina from the tachycardic reflex to sepsis – "rate-related angina".

Levophed (norepinephrine) – this is sort of a "kitchen sink" pressor – it kicks both alphas and betas, but much more the first than the second. It gets used in sepsis a lot, and it works well, but if you notice that your septic patient is getting even more tachycardic than before, and maybe having some ectopy too, it might be a good idea to change to neo.

Dopamine – dopa is the only pressor that can be started on the floors, so no matter why the patient is having pressure problems, this is the one that they put up. It helps, too, but it's not always the drug you want to go up on – if your septic patient is tachycardic, using a lot of dopamine can only worsen that problem, and sometimes it can provoke VT or VF. Then again, for a patient with an inferior MI (the kind that makes brady-arrhythmias), it might be just the thing. A wire might be better though.

Vasopressin: I just don't understand this drug at all. We use it in two ranges, one for sepsis, one for GI bleeding, in both cases to tighten up arteries. But the dose for GI bleeding is ten times the rate of the dose for sepsis? How come it doesn't produce ten times the blood pressure? We used to use vasopressin together with IV nitroglycerine for GI bleeds before **octreotide** came along – what I do remember is that it causes bradycardia, or some slowing anyhow, often to heart rates in the 50's - that I would never have believed in septic patients who usually run in the 130's for days at a time. But what the heck – it works, I run it.

Dobutamine – This is the pure beta pressor (the only one with a "b" in it.) This is what you want to use when the pump isn't pumping – cardiogenic shock. Two problems with that: first, it "whips" a heart that's already failing – a balloon pump is much better. Second: it very easily causes lots of arrhythmias – something your cardiac patient doesn't need. Make sure their electrolyes are ok.

Rarely used (Zebras): Epi, Isuprel, Amrinone, Milrinone. We'll use these drips sometimes in special situations – like maybe at the end of a code. Epinephrine – very powerful, very arrhythmogenic. Isuprel – "Prel": a powerful beta drug, also causes lots of arrhythmias, can be hard to control. I haven't seen a Prel drip in many moons. (Jayne: "Isuprel is completely gone – nobody even makes it any more.") Amrinone – "the yellow stuff", and it's cousin milrinone – sort of the same as dobutamine, except different – maybe we see these once a year. Anybody know if they're used more in the CCU?

For extravasations: Regitine. This is a good one to know about, even if you never have to use it. A patient who has to get pressors through a peripheral line is at a real risk for injury around the site if the drip infiltrates into the tissue. Regitine is an alpha blocker, and gets injected ("infiltrated") into the tissues around the IV site to relax the vasoconstriction. Never seen it done, but I saw a dopamine infiltration wound once. Ugly.

3-2: "Down" Meds

Nitrates: nitrates are vasodilators – they open up coronary artery flow, and they lower SVR, so the heart gets 1: a better muscular blood supply and 2: less arterial resistance to push against. Nice.

Nipride – the ultimate down med. This is what you want to use if your patient has malignant hypertension, or something very acutely dangerous, like a leaky aortic aneurysm. Very powerful. Be especially careful with this one. The nice thing is that it both works and wears off so quickly, so that you can titrate it very easily – it doesn't take ten minutes for a change to show up.

4- Lung Meds

Beta Agonists: Albuterol, ipratoprium. Simple enough – these are beta agonists, so they make the bronchi open up. Remember that they can make your patient very tachycardic for the same reason.

Mucolytics: mucomyst – we used to use this a lot to loosen up secretions, but not in recent years. The in-house CNS (that's as in my house, and my spouse) says it's because it can cause hemorrhagic bronchitis. Sounds like a good reason. Lately we've been giving a couple of doses of mucomyst orally before and after IV dye loads required for contrasted scans – apparently it helps prevent renal failure problems afterwards. How the heck did they figure that one out?

DNAse: Are they still using this? We saw it used for a while some years ago, mostly with CF patients who had very thick, tenacious secretions. I have no idea how well it works.

Theophylline – talk about going in and out of fashion. We used to give this stuff by the barrel, seemed like. Now I see it given once very couple of months, maybe. Those poor COPD patients are shaky enough without being made lots more tachycardic by theo…

Glycopyrrolate – another one we used to use a lot, nebulized, mostly to "dry up" patients with lots of secretions. Also used in place of atropine for bradycardia, I think.

5- Gut Meds

5-1- Too much acid:

H2 Blockers: cimetidine, ranitidine, pepcid. Cimetidine was apparently the wonder of it's day, setting all sorts of world records for drug sales, and it did a lot of good, too. Then apparently we found out that it often makes platelets go away – we give it rarely now, giving zantac a lot of the time instead. My wife's hospital uses a lot of pepcid (famotidine) – we hardly ever do.

Proton pump inhibitors: prilosec, nexium. ("Mom, I hate my cousin Ralph. Do I have to sit nexium?") Apparently the greatest thing since sliced cimetidine for gastric acid. Sure works well for me…

Antacids – another big change over the years. I remember giving 60cc of **Mylanta 2** every two hours through an NG tube. No more. Do we even stock it now?

5-2- Coating the stomach: Carafate (sucralfate)– this is good stuff, but it'll block up an NG tube very easily. I dissolve mine for a while in warm water, and follow it with a warm flush.

5-3- Moving It Along: Colace, senna, mag citrate, go-lytely, enemas: You've got to keep it moving, and the sooner you start your patients on this stuff the better. We see lots of opiate ileus, and lots of our patients get **reglan** (metoclopramide) around the clock when they're on fentanyl or morphine drips. Recently somebody got a dose of **neostigmine** for an opiate ileus – I hear it works well for that, but I haven't seen it done myself. And you know that trick they've been trying with narcan through the NG tube for the past twenty years? Doesn't work.

5-4- Stop the Bleeding: Octreotide: The word is that octreotide tightens up the arterial flow to the splanchnic-perfused areas, and I guess the studies are clear about its effectiveness. I do know that octreotide has almost completely replaced the previous combination of vasopressin and nitroglycerine that we used to use.

5-5- Clearing the Ammonia: Lactulose – it works, but it seems like an awful price to pay for everyone involved. Sometimes patients with varices can't have an NG tube but still need the med – these people get retention enemas through a rectal tube. The trick is not to give too much at once: usually we dilute a dose of lactulose with an equal amount or more of NS, and infuse not more than 200cc or so at a time, letting it stay in place for about 30-45 minutes. Rectal tube balloons have to be deflated every four hours for about half an hour, and the tube itself is supposed to come out of the patient once a shift.

6- Endocrine Meds

Insulin: drips, sliding scales, regular, NPH, humulin, glargine. Lately we've been tearing our hair out (I have less to pull out than most, which may or may not be an advantage) over the decision to put most of our patients on insulin drips to try to keep them in the "tight control" range of 80-120. This means an awful lot of running around with glucometers, a lot of sore patient fingers, a lot of titration between drips and tube feeds, etc., etc. We do a lot of q4-hour checks with sliding scales too. We check patients on insulin drips every two hours, on the idea that regular insulin peaks two hours after a change. **Glargine** insulin, which goes by the trade name of Lantus, is the newest horse out of this particular gate – apparently the dose is given sq once daily, and there are no peaks or drops; just a nice constant delivery. Sign me up!

Oral hypoglycemics glucophage/metformin, glitazones: Lots of patients come in that are managed on the outside with oral meds, but usually they're so stressed during their stay in the unit that they wind up on one of the sliding scale treatments.

Thyroid replacement: Synthroid. We give these when people are on maintenance management.

"Cort stim" tests: the idea here is to check a morning cortisol level (I'm pretty sure the level has to be drawn early in the morning, having something to do with "inner-clock" timing of cortisol secretion). Then a dose of cosyntropin is given, and another cortisol level drawn an hour later. Tells you if your patient's adrenal cortex can respond normally

or not. Sometimes septic, hypotensive patients turn out to be "hypo-adrenal", and giving them steroid replacement is supposed to help. Apparently under debate.

DDAVP/desmopressin – this one's interesting. This turns out to be vasopressin, which turns out to be anti-diuretic hormone, ADH. Two uses for this one: first that comes to mind is your hepato-renal patient who's bleeding from here and there, because he's coagulopathic? And uremic? Uremic platelets don't work so well, apparently DDAVP helps them work more better.

The second one is very cool to see, although you may not see it often. Remember any of that pituitary stuff? Hypo-pituitary, all that? We had a young man in awhile back, he'd had some sort of tumor removed, pituitary I guess, and he couldn't make any of his pituitary hormone things any more, had to take them as supplements.

So let's see – two ways ADH can go. You can make too much, which is SIADH, right? You don't pee, much, because you have too much anti-diuretic hormone… ok. Then there's the other way, which is diabetus insipidus – you don't make enough ADH, and you can't hold onto water at all. Right – that's what this fellow did – drank all the time, peed hugely… I mean, scarily – a liter an hour when he'd come in, out of whack. Like a "siphon", which is what "diabetes" means… so he'd get his dose of DDAVP, and it was as though the faucet had been turned off – all of a sudden his urine output would drop to whatever – 40 an hour? "Anti-lasix". Amazing.

Steroids: Couple of flavors of steroids, given for several reasons – hydrocortisone, which is the generic, sort of "weaker" version, and methyprednisolone, which is as I recall about 7 times more powerful as hydrocortisone, which will put your patient's adrenals right to sleep for a while. Some patients in an acute flare of something bad will get started on "stress" steroids – a COPD flare is probably the most common example. They get methylprednisolone, something like 60mg several times a day.

Now and again you'll see someone get "pulse steroids" – this is usually in some really life-threatening situation like a severe lupus flareup, or I want to say BOOP… this is pretty impressive – the dose is a GRAM of methyprednisolone. When you consider the usual dose is 60mg or so… it's a lot!

Steroids will absolutely play havoc with your patient's blood sugars – especially, obviously, if they're diabetic – or even if they're not. Be aware that you may have to work hard with your insulin management while this is going on.

There's been a lot of debate in recent years about giving steroids to people in sepsis. For the longest time this was considered a really bad idea, supposedly because they inhibit immune response – a bad thing when your patient is infected. Then more recently it became the practice to test septic patients for adrenal suppression – which is what cort-stimming is all about – I guess some people's adrenals go to sleep in sepsis. Then the folks with sleepy adrenals got hydrocortisone.

Now the latest rumor is that the evidence is going the other way. Ask around – not sure what the current word is.

7- Renal Meds

Our patients have a lot of kidney trouble, which only makes sense given what's wrong with so many of them. Kidneys are incredibly sensitive to insults ("You stupid kidney!"), and even a hypotensive episode of 20 minutes can put kidneys into ATN. Patients with hypertension often have kidneys that are used to a high perfusion pressure – sepsis or some other kind of hypotensive shock can have a really unpleasant effect. We also see lots of chronic-renal patients who get hemodialysis outside the hospital and who get into trouble for some reason. (Ask me why I take my **glucophage** every six frickin' hours.)

There aren't a whole lot of meds that immediately come to mind when you think "kidney", besides diuretics. Low dose "renal range" **dopamine** sometimes is still used to encourage kidneys when they don't want to go – once in a while it works, too. **Phosphate binders** like **calcium acetate** are used to, uh, bind up excess calcium that can't be excreted by non-functioning kidneys. And we give the new (anything less than ten years old is new to me) **marrow stimulant factors**: **epogen** for red cells and **neupogen** for whites – I know that epogen replaces endogenous **erythropoietin** ($1.25 for the word "endogenous", please), which is secreted by the kidney – what does neupogen replace?

8- Anti-Infectives

We sure do give a lot of antibiotics in the unit, and it's a good thing too, but I'm pretty sure that we had a patient with a linezolid-resistant bug a while back. (Oh shit…!)

Gram positive/ negative infections: (Why is everyone mad at Gram? What did she ever do to anyone besides make that Christmas fruit bread that everyone hates?) Now I remember why I didn't want to write this part – I am really deficient in understanding antibiotics. Everybody has their thing, and mine is actually balloon pumps. I'm pretty good at blood gases and PA lines too. Daughter number one is doing micro now in nursing school, and she usually quizzes me on things – this time I think it's going to be the other way around.

Ignorance though is no excuse for not knowing the essential points of safety, which in antibiotics usually center around sensitivity reactions, from rashes to anaphylaxis, identifying drugs as the causes of "drug fevers", and knowing which drugs need to be monitored for levels. Vancomycin dosing is level dependent (read: "do your kidneys work?"), and we send peak and trough levels on gentamicin as well. Every drug in the universe has an enormous number of side effects – experience and book time will serve you well when mixed together in fairly large doses. We are always looking things up in our unit, consulting pharmacy, checking calculations with each other. Remember rule # 1: "There are no stupid questions.", to which could be added "Two heads are always better than one."

Fungal infections: Amphotericin B, Ambisome, Fluconazole – Amphotericin B is a toxic drug, and often patients need to be premedicated for it they way you would for a patient sensitive to red cell transfusion: tylenol, benadryl, sometimes steroids. An Ampho-B dose has to go in over something like 6 hours. The liposomal stuff can run a bit faster, and I understand is better tolerated. Fluconazole seems easier in general, but does it cover the same fungal infections? I keep wanting to say "I really should know all this." Which is of course why they publish pharmacy reference books…my wife would look it up on the pharmacy reference that she carries around in her palm pilot at work.

The Epocrates reference is a totally cool thing to get if you're at all gadget-head: you download it free – pretty nice of them, huh? into your PC at home, and then the PC sends it into your handheld through the little cradle. Updates itself automatically, too. I wonder if anybody else just gives their stuff away like that on the web…

TB – **rifampin, ethambutol, INH, maybe clarithromycin** – TB is probably going to earn a FAQ of it's own one of these days – it's a major, serious baddy in the ID universe. Now and then we get a patient who needs to be ruled out, and it becomes a major project: the patient goes into a negative pressure room (it sucks air inwards, to prevent the bug from getting out), and everyone wears N95 masks inside, because it's so easily transmitted. We give the drugs infrequently enough that I always look them up before giving them to make sure I remember what I'm doing. Some years back we had a patient turn positive for something nasty – it was actually Neisseria meningiditis, and all of us who came in contact with him got to take rifampin for two days – makes the urine turn a very pleasant day-glo orange.

Treating drug resistant infections: Linezolid/Synercid - those bugs just don't know when to give up. Of course, we're doing our best to help them mutate, aren't we? This is a battle that is never going to end.

A quick story that goes here: just in case you thought that the superbugs were going to kill us all, and just when you thought that there were no role models left in the world, along comes Dr. Paul Farmer, a physician at one of the big teaching hospitals in Boston. Dr. Farmer has established a field hospital out in the wilds of Haiti someplace, where he treats the local people who have had the misfortune to be infected with multi-drug resistant TB. Scary one. He's achieved cure rates between 95 and 100% by coming up with innovative treatment plans. So cool.

Antivirals: **acyclovir, indinavir**, etc. This part of the treatment spectrum is really starting to get going – the development of the whole HIV treatment process was amazing to me, and I think I've read that the antiviral age isn't quite here yet, but soon ought to be. We give these meds as orals / via NGT for patients who are on them on the outside, usually for HIV. I think there's one for CMV as well.

9- Antipyretics

Tylenol – Yes, it's true, Tylenol can drop a patient's blood pressure. Not often, but it happens enough that we old-gome nurses remember it. Nobody's ever had much of an explanation…

Motrin (ibuprofen) – We use motrin now and again for fevers. Good for cramps too, and I sure hope it really works against Alzheimer's the way they say, because, um…what were we talking about? Some article I saw said that ibuprofen actually breaks down amyloid plaques.

Aspirin – Aspirin used to be the best thing for fevers, and I think we used it on my oldest kid when she was small (she's half-way through her first year of nursing school now). Not any more – that whole business about Reye's syndrome and all, but we do still give it to everyone coming in who's suspected of ruling in for MI. Apparently makes an enormous difference.

10- Anticoagulants

Heparin – we see, or at least rule a lot of people out for the heparin-induced thrombocytopenia thing. It's good to think about it if you see your patient's platelet count dropping. We change our patients' flush lines from heparinized saline to NS for this – it seems as though even just saying "heparin" in the patient's room can make this happen if they have this problem.

Coumadin – Most of our patients are pretty acute, so we don't give coumadin a lot. On the other hand, we do see patients come in who have taken a bit too much for one reason or another, and it's always worth wondering about if your new admission has a really high INR. Make sure they get vitamin K, and don't brush their teeth for a while.

Low-molecular-weight heparins: **Fragmin** – this is one of a whole group of new LMW heparins, which are supposed to anticoagulate as well as heparin does, while doing away with a lot of the PTTs, along with most of the worries about HIT. We'll see, I guess.

Platelet drugs: **plavix, integrilin, reopro.** These are the newer platelet-aggregation inhibitors. Obviously the CCU gets most of the cardiac cath patients, but we still see people from the lab sometimes. I don't know what makes the physicians choose one drip over the other, but they run for a fixed number of hours only, as opposed to heparin running for several days. Plavix is the oral maintenance med in this category – I think there are others, but we rarely give them.

Aspirin: Yup, aspirin.

Clot-busters: TPA, streptokinase – there are some newer ones which the family CNS knows all about, but our patients are hardly ever good candidates for this stuff, since so many of them are coagulopathic already. Once in a blue moon the team will lyse a PE, but as always we'd have to check with pharmacy and our references to run it.

11- Anti-Anticoagulants

Protamine – another med that we give once every year or so. I never have. Remember that it specifically reverses heparin.

Vitamin K – this we give fairly often, at times for those folks we were talking about who get a little too much coumadin. (My wife was doing a community-nursing course years ago, and one of the nurses described an elderly person she had visited at home. She had put all her pills in a candy bowl, and was saying things like "Well, when I'm dizzy I take an extra one of these" – holding up, say, a nifedipine (ack!) – "and when my knees are bothering me I take two of these" – holding up who-knows-what. Good thing she got visited!)

12- Miscellaneous Meds and Weird Reactions

Cancer chemotherapy in the unit

Our hospital requires nurses to go through a certification course to give cancer chemotherapy meds. Now and again we get a patient in who needs them, and the nurses from the oncology floors come down and do it for us. Sometimes we will give a dose of something on the chemo list, but only if it's not at chemo strength – for this we use the special heavy gloves (the coolest purple color), and a special disposal container.

Code Drugs

Atropine, epinephrine, calcium chloride, lido, amio…this is certainly a subject that should take up entire volumes, and does, but here's one thing that immediately comes to mind. Patients will come to the unit after a code sometimes with pupils that appear to be fixed and dilated – atropine will definitely do this. Wears off after some hours. Good thing to remember! Take ACLS. Learn how codes are run. Learn why the meds are given.

Special Situations:

There are some situations that call for specific meds only, and a couple came to mind:

Dantrolene, bromocriptine: Once in a really great while a patient in the OR will develop malignant hyperthermia – a syndrome of very rapid temperature rise, which as I understand it is a reaction to one or the other of the anesthetic agents being given. The fever will go up really fast, and really high – maybe to 108 F. A similar situation is neuroleptic malignant syndrome, where a fever will rise quickly to scary heights, in response to meds like haldol, or zyprexa. I think we had a patient on clozaril who did the same thing. Anyhow, the treatment med to keep in the back of your mind for this situation is dantrolene. Pretty orange color. I think I've seen it given twice, maybe, in the last few years. Supposedly works well – the other med I've seen used for this is bromocriptine.

Mucomyst: lately this old drug has been put to use pre- and post- IV contrast procedures, because there's some evidence that it helps preserve the kidneys from injury. I wonder who noticed that?

Glucagon: for beta-blocker overdose. I've seen this happen once or twice. Another one to keep in the back of your head.

THAM: (Tromethamine) – sort of a "super-bicarb". Powerful stuff, useful for your patient who is horribly acidotic (our patients usually see this drug when they're around 7.0, even with a bicarb drip), and will definitely help your pressors work better as a result. Unfortunately people who get that sick hardly ever get better, so THAM is usually a sign of impending death.

Reglan: This is a strange one: about a year ago we had a patient come in from one of the floors who had developed a sort of "waxy flexibility", which was the term they used to use for catatonic patients who could be put in a position – say, arms above the head – and who would stay that way for hours. The teams finally decided that this poor person was having a very strange reaction to reglan. Never heard of it before or since.

Oral meds for low BP: midodrine, pseudoephedrine. Kind of strange, but it's done. I think this is usually in situations when people have some chronic condition that loosens up their arterial tone – muscular dystrophy comes to mind. Pseudoephedrine is the stuff that dries up your nose in cold pills, and midodrine is actually sort of like oral neosynephrine – it's an alpha agonist agent. Used rarely.

Special thanks to Mark Hammerschmidt and Jayne Mulholland for compiling this resource. Get similiar resources over at http://www.icufaqs.org/