1. What if my patient becomes unresponsive?

Even though there may not be one apparent, things happen to patients for a reason. It's usually a matter of figuring out the context – this can be hard for a newbie who's still trying to figure out which way to turn the stopcock on an a-line. The thing to try to focus on however is pretty simple - keep in mind the basic question: "What's wrong with my patient?" This sounds stupid but actually isn't, since the poor newbie is still struggling to remember how to read the CVP – and it's definitely a fact that equipment of any kind has a genuine hatred for new staff. I remember flushing a toilet once in a patient bathroom during my first week at a job, and watching horrified as the plumbing came apart, off the wall, in front of my eyes, convinced I'd somehow flushed it wrong…

The goal for the new ICU nurse (for any ICU nurse) is to try to figure out what the patient is doing – but when your patient does something unexpected, there is just no substitute for experience. So go get some: go get the resource nurse, and go get the team. This points up the most basic principle of ICU nursing: it is a group process. Tattoo that backwards on your forehead so you can read it in the mirror. Several heads are always better than one. Don't get isolated in your room.

Anyhow. Patients can become unresponsive for lots of reasons – your clues will probably lie in the reasons why they were admitted in the first place. There's a neat maneuver that they do in the ER when a patient comes in unresponsive: they give a quick cocktail of meds that might reverse whatever is causing the problem: an amp of D50 for low sugar, an amp of narcan to reverse opiate OD, a dose of thiamine for (is it the Wernicke's alcoholic thing?) – There might be some others. What's a banana bag?

Unless the situation is really emergent (brand new seizures in a patient with a broken foot), you usually have some sort of diagnostic context to help you puzzle things out – is there an underlying neuro problem? Is their blood sugar too low? Blood pressure low? - have they flipped into some unpleasant cardiac rhythm? Rather than trying to think of every possible cause, my point is that you will almost always have something to go on.

2: Has a change in mental status?

This is so common in the unit (and I'm not just talking about the patients), and can have so many causes. All sorts of meds will do this for example, and often you'll discover that the Ativan you've been giving at bedtime makes Mr.Yakowitz confused, every time, but that he does fine with benadryl to help him sleep, or the other way around. What's the patient's ammonia? Is his calcium really high? Is he "sundowning", or "sun-upping"? Is it "ICU psychosis"? Lots of things to think about – try to think about reasons having to do with his admitting diagnosis.

3: What if I have a change in mental status?

Otherwise known as “Alteration in Reality, Potential vs. Actual”. I’ve been a night nurse for 20 years – I know about this one. Lots of reasons for your own mentation to change: not enough sleep, not enough caffeine, too much caffeine, low blood sugar…one time I had to do the "dad thing" to a young woman who insisted, as she was sliding down towards the floor, that she was just fine, she needed to go turn her patient right now… I had to speak to her firmly, stuck her in a chair, and someone got her some orange juice. How is it that some people "forget to eat"? Not to be antifeminist or anything, but this really is totally a chick thing. Guys never "forget to eat" – forget to eat? I carry power bars in my bag to eat standing, a bite at a time along with some Gatorade if I can’t get out to the back room. What good are you to your patient if you’ve fainted on the floor?

The point is: this really is one of the hardest jobs there is. Not kidding. Nurses have such an ingrained sense of how little they matter that they have trouble perceiving their own value, much less the real impact of the anxieties and burdens that come with working in the ICU. Take care of yourself – you really took on the big one when you came to the MICU. You’re in the majors now.

One more thought: is there another profession that sees death so often? In this job we may spend 25 years treating patients who are trying to die…give yourself credit, and wear the invisible golden badge (the one only your co-workers can see) with pride.

4: Climbs out of bed?

Happens all the time. Your responsibilities are simple: keep your patients safe. If your patient is competent, oriented, can get up and wants to, then you should help her. (If she’s still intubated that might be a problem.) Keep the bed in low position. Know the hospital’s restraint policies. Read more on this topic in "The House of God", by Samuel Shem. Kind of a dirty book, but hilarious. Not very accurate on nurses, though.

5: Climbs out of bed naked, and runs around the unit?

Well, this one does actually happen once in a while. He’s still your patient though, isn’t he? Call security, call the team, and try to keep him safe until you can get him back into the bed. Something that came up recently in the "Med Tips" article might be useful to keep in mind – a patient who’s pulled out all his IV lines can still be given safe sedation by nebulizing a dose of, say, 5 mg of morphine through a neb mask. Surprising but true – a year or so ago we had a similar situation, and the attending pulled that idea out like a rabbit out of a hat. Worked really well.

6: What is the APS?

This stands for the Acute Psych Service – this is an in-house resident psychiatry service available for emergency consults 24/7. Good to know.

7: What if my patient starts refusing treatment in an emergency?

Obviously this depends on the situation. An elderly patient with a terminal illness may be completely rational (and I believe in the right) if she decides to refuse being intubated for the fourteenth time.

A patient we had some months ago demonstrated a different scenario: a man in his fifties, I think with some degree of COPD at baseline, but with a clearly treatable pneumonia that was pushing him over the edge towards the snorkel. He began to refuse everything – nebs, meds, and began climbing out of the bed to go home. This is the kind of situation that legally requires a stat assessment of competency – we called the APS.

8: What if my patient seizes?

For the first time ever? For the third time in an hour? Intubated? Not? The basics are clear – get help, get the team, treat acutely with things like benzos (we usually use Ativan for acute seizures), try to keep the patient's airway clear and prevent aspiration. If he's intubated, try to get a bite block in place – we've seen patients bite through the pilot line, deflating the ETT cuff. Also, biting the tube closed is not usually a healthy thing – I've seen agitated patients arrest doing this once or twice over the years. The jaws are very strong. Put seizure padding on the bed rails, and check their dilantin level, or valproate, or whatever.

9: Repeatedly?

Hopefully you've got the neuro service on hand (or their assessment and treatment plan). Is this a sudden change, increasing in frequency? Time for more benzos, more dilantin? Phenobarb? Time for neuro to come back and have another look? Time for another (don't say it!) CT scan? Ack!

10: Does repeated seizure activity really injure the brain?

I ran this one past Jayne, and her opinion is that it's the hypoxia that goes along with repeated seizures (if the patient isn't intubated) that causes the damage. We got a patient in last week who'd been in "status epilepticus" for 90 minutes without being intubated – she woke up and wanted to know what all the fuss was about. (How did she breathe that whole time?)

11: What is a "blown pupil"?

This refers to a pupil that's suddenly gone big, independently of the other one. This usually something acutely bad is happening on one side (the opposite side?) of the head. Jayne: is this because rising ICP compresses the area that holds one of the optic nerves? Pretty clear that I'm no neuro nurse.

12: What if they're both blown?

Make sure it's not just dark in the room. Has the patient's level of consciousness has changed? Everybody knows about "fixed and dilated" pupils – this is a true BBIT (big bad ICU thing), indicating a prolonged hypoxic injury to the brain.

A couple of exceptions: sometimes a patient will have a dilated eye exam done by opthalmology, and they may not remember to tell you that your patient's pupils are going to be sort of massively enlarged for a while. The other thing is that a patient who is post-code may have dilated pupils from atropine, rather than hypoxia – these should go back to normal after several hours.

13: What if my pupils are blown?

Don't forget to bring sunglasses after your eye exam. Otherwise, is the room candle-lit? You know what to do.

14: Acute CVA?

The single enlarged pupil may be your major clue if you have a patient who is otherwise sedated or chemically paralyzed. The critical piece of the puzzle is: is this an embolic or a hemorrhagic event? Quick trip to the CT scanner.

We had a really nice example of "brain attack" treatment a few weeks ago. Gentleman about 80 years old came into the ER, suddenly unable to move his left side, unable to speak, previously completely functional. Apparently the window for assessment and treatment is really short for this kind of event – two hours? – Jayne says it's three hours for an embolic stroke, as against the 4-6 hours that an MI patient has to get into the ER for lytic treatment. Anyhow, I guess the window was still open, and the man flew right through it, got his lysis, and by the time he was finishing up the dose, back in CT scan, he was able to speak and move freely. By the time he got to us, and when I was doing my resourcely nurse duties running around checking things out, he looked up at me.

"How are you doing, sir?" says I.

"I'm just fine, sir." says he, "How are you?" (Yeah, they call me "sir" now. Getting grey around the edges. Okay, more than just the edges.)

I guess I looked a little funny with this enormous goofy grin on my weary old-gome-nurse face, as I stood there in the doorway – he looked as if he was a little worried about me. "Saves" like that are so incredibly gratifying – we rescued this guy from being paralyzed and speechless, hopefully for the rest of his life. So cool.

15: Sudden rising ICP?

What year is it?

Not too long ago, a patient couldn't tell me who the President was, but he looked pretty much with it, so I asked: "Well then, who's Monica Lewinsky?" That got an enormous grin – I think he was pretty well oriented.

Everybody remembers the triad (is it "Cushing's" triad?) of symptoms: falling heart rate, depressed respiratory rate, and widening pulse pressure: systolic heads north, diastolic heads south. The "critical element" however, as they say, is much easier than that: mentation goes first. A patient who was previously arousable and oriented will abruptly become too "sleepy" to respond to questions. Intra-cranial bolts are nice I'm sure, and once or twice a year we get them, but the first real clue to rising ICP is the patient's decreased level of consciousness. (Whenever we get a bolt I always ask the neuro ICU nurses to come down and tell me if it's working right. Anything that I only see once a year makes me nervous.)

These changes can be really abrupt - here's a story by way of example: years ago, I think during the Crimean War, I was working in a medical CCU and, being the owner of the only open ICU bed in the hospital, I was sent the gift of a fresh post-op craniotomy patient for recovery. As my grandpa would say: "This, I know from nothing!" But, a nurse is a nurse is a nurse, right? Anyhow, I got explicit instructions on postop assessment from the neurosurgeons, and I just documented the crap out of that entire situation – the patient's answers to questions, strengths of extremities, pupil exams, tongue stuck out at midline (didn't Mark Green get into trouble with his tongue that way?), severity of postop pain – I'm sure there were others which now, 700 years later I don't remember. Anyhow, I sent the patient to the floor after the prescribed number of hours postop with a sheaf of documentation, after a surgical postop check to clear the transfer.

I got a call about 20 minutes later – had the patient been unconscious when he left the CCU? No way, I told them – check the assessment sheets. Between the unit and the floor, the patient had become suddenly unresponsive. Zapped to CT scan – she'd re-bled. Unbelievable. Be very careful!

Sedation /Paralysis:

16: What if my patient is under sedated?

This is a complex subject, and there's more than you probably ever wanted to know about it in the "Sedation and Paralysis" FAQ. Apparently the studies consistently show that nurses always think that their patients are under sedated, and doctors always think the opposite (what else is new?) The essential point: keep the patient safe, and as free of pain and distress as possible. Make sure that you communicate carefully with the team, and document your assessments.

17: Over sedated?

They do have to wake up sometime. Use your judgment, keep the patient safe. Jayne points out that new practice guideline from the Society for Critical Care Medicine say that sedated patients need to be awakened every two hours to make sure that everything is working, neuro-wise. This seems kind of impractical to me, but I guess they know what they're talking about. I always try to document my sedated/paralyzed patients' neuro status carefully: a chemically paralyzed patient will still have pupillary reflexes, right? So if one pupil suddenly gets big – well, what you have there is sort of your basic clue.

18: When should I use Narcan? Romazicon?

Narcan is the drug that pushes opiates off of their little cell receptor sites, so it's used for opiate overdose situations, and sometimes for patients who aren't able to tolerate their prescribed pain meds too well. Romazicon is the same thing except different – it works on benzo receptors. You have to be careful with flumazenil – it can provoke seizures in chronic-benzo-using patients. Be careful with narcan too – a patient can become frighteningly agitated after a dose of narcan. I usually put soft restraints on the patient ahead of time. And maybe pad the ceiling.

19: How do I know if my patient is withdrawing from something?

Usually the picture is pretty clear: agitation, tachycardia, hypertension - and you'll have some idea of what to expect if your patient is admitted as an OD of one kind or another. If your patient is admitted intubated, maybe after being found down, maybe with an big aspiration pneumonia, maybe brewing ARDS, sedated with propofol, and two days along they start to become tachy, hypertensive…if the ER was doing it's job, they'll have sent a tox screen on admission, so you'll have that to work with. And the timetable does vary for withdrawal, but the thing I try to think about is DT's – usually the symptoms will start between 48 and 72 hours after the person's last drink.

20: What if paralysis won't take effect?

Some patients just don't paralyze. I'm sure there are very good, and horribly complex physiological reasons why they don't paralyze, but all I care about is whether or not my patient is ventilating, so would you all stop the intellectual discussion and give me a suggestion as to how we should control this guy before he codes? This is similar to the situation where the anesthesia resident stands there teaching the intern the fine points of intubation while the patient's sat is falling (which is being watched mainly by the nurse while this intellectual discourse goes on). And falling. While we remind them. Again.

Surgical intern says to me once, not very happy: "No one ever listens to me!" I suggested: "Try being a nurse." She didn't like that answer…

Where were we? Before starting paralysis, if possible, it's good to document a baseline "twitch", or train-of-four response, using a peripheral nerve stimulator, if only to document that they do or don't respond to it. This gets a bit into the voodoo realm sometimes – some patients just don't seem to paralyze, or twitch, or both. Twitch response may have to do with peripheral edema over the nerve that you're trying to stimulate – but remember that your first goal is not the twitch number – it's the patient's condition. You can twitch them every whichy-way, but the point is to get the patient into some sort of safer condition than the one they were in before you started. There's lots more about this topic in the "Sedation and Paralysis" FAQ.

21: Won't wear off?

Progress has definitely been made on this one, and without going into too much detail, suffice to say that titrating to the train of four has given us a way to keep from giving too much paralytic drug. In the old days, a patient was either "paralyzed" or "not-paralyzed" – and apparently they sometimes soaked up too much med over the time they spent on the drug. Titrating to one-out-of-four on the TOF let's us minimize the dose, so that they won't have to cook off large amounts of drug after their lungs get better.

The other thing: paralytics and steroids seem not to mix. Certainly "pulse dose" steroids of something like a gram (!) of methylprednisolone seem to make the effects of paralysis linger on and on – and "stress" doses of 60mg may do the same. Something about "steroid myopathy" – as we say in Boston: "Alls I know is, don't give 'em togedda!"

CV: (Pump, Volume and Squeeze)

Pump

22: What if my patient suddenly becomes bradycardic?

Scary one. Two main possibilities for this: first, has the patient acutely obstructed her airway? Acute hypoxia produces bradycardia. Has she plugged her ET tube? With her thumb? Tootsie Roll? Anybody suctioned her lately?

A word about suctioning goes here. Somewhere along the line the word got out that using saline lavage while suctioning is not the right thing to do. This is simply wrong. As I read somewhere (on a different subject): "All the studies demonstrating this point are wrong, and should be burned." Just last week we had a vented patient whose respiratory rate had been rising all evening – it was currently in the 60's. I was the resource RN, and probably the senior nurse to the next by about 13 years. Actually, it might've been 20. (And that's another whole story too, isn't it?) After some discussion I went into the room with the respiratory therapist – we lavaged and suctioned her ET tube with ten cc's of saline a couple of times and produced a large, dryish plug. Her respiratory rate went to the 20's, her heart rate dropped forty points, her sat went up – use that saline!

Jayne: "You are just totally wrong on this one. I have a whole bunch of studies at work that show that what you're doing is opening up a sterile, closed system, and introducing something foreign into the system. Sending the saline down the tube will break up the mucus that's trapping the bacteria, and then if you ambu them, you're just pushing the bacteria down further into their lungs, and making them sicker!"

Myself: "Phooey. And we use the inline suction thing anyway. But I'm putting in your opinion, all right?"

J: "Yeah, well, I'm right, and you're wrong."

She has a tough job. We've been together for 25 years this August. I used to sit behind her in nursing school…

Sleep apnea people are at sometimes at risk for bradycardic events because they're obstructing – which of course is their problem, right? They obstruct every four minutes, wake up with a snort, and go back to sleep for another 90 seconds, all night long. Suppose they have COPD as well, and someone gets nervous and applies too much oxygen when the patient comes in with a flare – total setup for respiratory suppression, right? These patients easily become C02 "narced" (pronounced "narked"), which is to say "suffers an episode of hypercarbic narcosis" – or even better! – has an "alteration in gas exchange secondary to Pickwickian body habitus and history of toxic tobacco exposure resulting in chronic obstructive breathing pattern resulting in an alteration of the human spirit, potential versus actual…" – right. Sorry Aunt Nanda… (!)

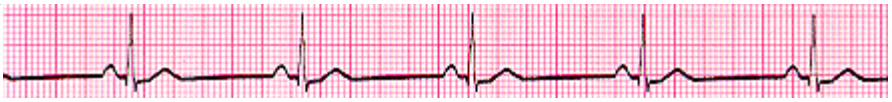
Anyhow – that patient may get narced, stop breathing, obstruct his airway, and brady down. So be alert, and think ahead: what are you going to want to have on hand? Atropine? Sure, but maybe not if the reason for the bradycardia is a closed airway, which you then open with a jaw lift, or an oral airway, or both.

The heart rate ought to pick up once oxygen starts getting into the blood again. If you've given atropine, the heart rate may go up to a zillion – now you have a whole new set of problems. So: keep atropine nearby, sure, but take 30 seconds if you can to see if opening the airway and restoring some oxygen delivery fixes the problem. If not, and the BP is dropping significantly, then go push that atropine!

What else are you going to have on hand if you think this might happen? Oral airway? Ambu-bag, all hooked up? Suction at the bedside working? Plus (big plus here) – did you set your alarm limits nice and tight when you started your shift? If I have a patient who's unstable for any reason at all, I set my limits less than ten points above and below where the patient is at for heart rate and MAP – hey, if I waste printer paper on a bunch of artifact alarms, what does it matter? You can loosen the limits later if you think it's safe.

The other main reason for bradycardia of course is that some unpleasant cardiac thing is happening, usually in the form of some kind of inferior ischemia or MI. These folks will often show you what they're doing by vomiting, or having hiccups along with, or instead of - their chest pain. (Why?) You may know what's wrong just by looking.

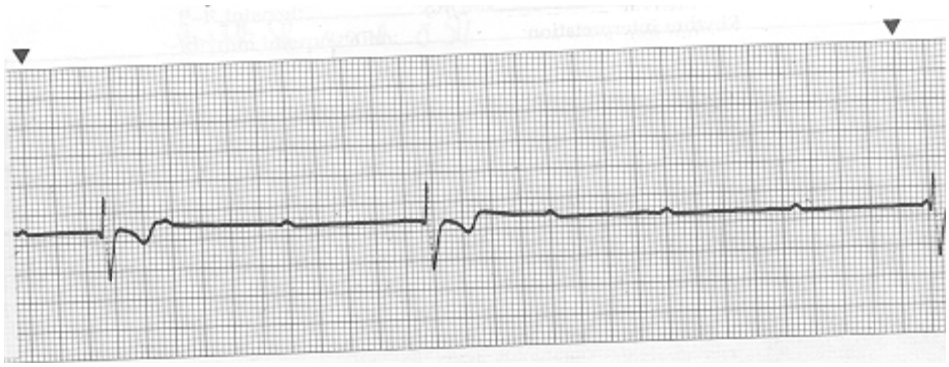
Let's take a minute to look at a couple of the main bradycardic possibilites. Suppose you see this:



Everybody recognize sinus bradycardia? What's the rate – about 55? Does this patient need atropine? No? Remember that atropine is only for symptomatic bradycardia, meaning, "with a low blood pressure". Maybe he's getting lopressor loaded today. But what if the same patient's heart rate had been at about 100 for the whole day before this? And he was vomiting when he did this? And broken out in a sweat, with chest pain and a dropping O2 sat? Blood pressure dropping in this setting might

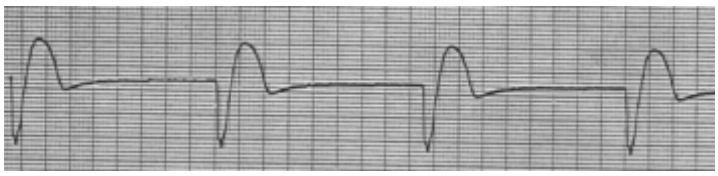
mean some sort of acute inferior-territory problem – it all depends on the context. If this had been the patient's rate all day, with a good BP – probably no problem.

How about this one?:



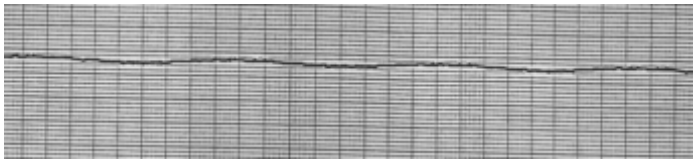
Yikes! Everybody recognize third degree heart block? Everybody know how to use the external pacemaker? Atropine may not help much here…

Here's another:



Ack! Even worse! "Idioventricular" rhythm, otherwise known as "physiology of death". Probably the next-to-last rhythm the poor guy will ever have.

And another:



That’s real bradycardia! (Are the leads on the patient?)

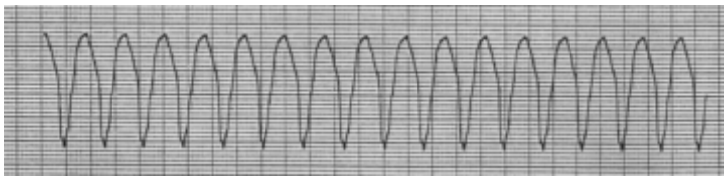
23: When do I give atropine? Do I need an order?

The policy is "Give atropine for symptomatic bradycardia." - lots of people get totally wound up, ready to give atropine when the patient is still making a pressure – and it’s hard not to want to just charge ahead and do it. But try to wait just a little and see what happens. If the patient loses pressure, you are absolutely authorized to go ahead and give the atropine. (Make sure their airway is open. Yours too.)

24: Tachycardic?

This is usually going to be some kind of arrythmia. Sinus tachycardia happens, for sure, but usually it creeps up over the period of some hours at least, and is usually pointing to something happening: the patient is spiking a temp, or getting dry, or agitated, or some combination of the three. Sudden supra-ventricular tachycardia is often something like a burst of rapid PAT, which will likely stop as suddenly as it started, or rapid a-fib, which won’t. The essential point here is: "Is the patient making a pressure or not?" If they are, then you have time to try different things – if they aren’t, you don’t. Take a look at the articles on "Arrhythmia Review" and "Defibrillation" for ideas on how to identify rapid arrhythmias, and how to go about treating them: some rhythms get defibrillated, and some don’t, and it’s a good idea to be ready to tell which is which. We’ll do some basic review here.

25: Sudden VT? Narrow complex? Wide complex?



Ok, ready? VT? SVT? Narrow or wide complex? That’s all good stuff to know, but go back to the essential point: are they making a pressure? Yes? Sometimes you’ll see a patient maintain a pressure in VT, and there are algorithms for that, but remember not to defibrillate someone who’s awake! Stop making a pressure? Think it’s VT? Pretty darn sure it’s VT – nice wide complex? Patient’s "out of it"? Try a

precordial thump. This is something you see the old nurses do: they'll see VT on the central station monitor, and a newbie nurse assigned to the room may be standing there (no offense now, okay?), like a "deer in the headlights", and the old RNs will stand up in a group and yell "Hit him!" as they scramble for the defibrillator, cart, ekg machine, docs, etc. I've precordially thumped several patients back into sinus rhythm in my day – all I can say is that I think it's still useful, even though I think it's not part of the protocols any more.

This is the kind of situation where ACLS comes in handy – if you have the kind of mind that memorizes easily, then you'll have absolutely no trouble remembering what to do when everyone is yelling at everyone else in the middle of a code situation. I don't memorize well at all, but what I can do is to learn from experience – for some reason memories come up in my mind literally from years before, and I'll say "Hey, I've seen this, I know what to do."

ACLS is a wonderful thing, and it's way cool to be ACLS certified, but the basics of BLS still cover most of what you want to do in a code situation: A,B, and C. Let's do these individually for a minute:

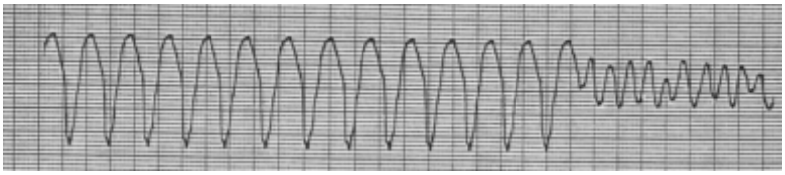
A: Is the airway open? No amount of dramatic maneuvering with defibrillators, wires, or external pacemakers is going to make the least bit of difference unless the patient's airway is open. Some months ago we had a patient who brady'ed down with a low O2 sat, and people were in there doing all sorts of stuff, but having the chance to stand back a little, you could clearly see that the patient's airway wasn't open. We did a jaw thrust and things got better very quickly.

B: Breathing. Once the airway is open, get your ambu bag and mask and get some gas exchange going. Make sure the bag has good 02 flow. An oral airway will usually do a good job of keeping the airway open under the mask. Suction, suction, suction.

C: Circulation. You know this part. "Hut hut hoo!" (Wait – isn't that something else?)

Now's time to think about cardioversion and defibrillation. Take a look at the FAQ on the subject for lots of info and some nice pictures.

26: VF?

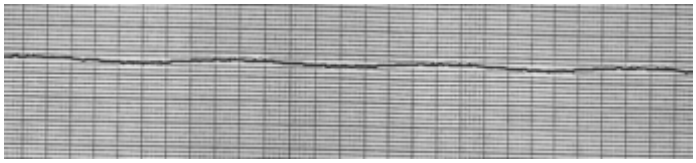


So - what happened here? Just when you were hoping that things couldn't get worse, they did. This is the thing about VT – even if your patient is making a pressure initially, they may lose it – sometimes because they've gone into VF. This situation calls for immediate defibrillation – but try to get the airway open, too…

27: Rapid AF? What is RVR?

RVR stands for Rapid Ventricular Response – the ventricles are responding to so many atrial signals that they haven't got time to fill properly, so the blood pressure may drop impressively. That situation usually calls for synchronized cardioversion right away.

28: Asystole?



Ugly. We saw this one before – everybody remember the first thing to do? Are you going to call a code if the patient is eating dinner in this rhythm? Unresponsive? You or them? Okay – now call a code, get the Zoll, start BLS, bag the patient…what do you mean, you can't do all of that at once?

29: How do I work the Zoll?

External and internal pacing devices both work the same way – you pick a rate that you'd like to pace the patient at, then turn up the milliamperage output until you capture the patient. Obviously it's the

delivery system that's different: in this case the electricity is being delivered through pads on the patient's chest and back. External pacing is tricky, and it makes a lot of sense to spend time looking over the box, the pads, the sensing and output cables – the whole setup, before you have to use it. Take a look at the FAQ article on "Pacemakers" for more on this topic.

30: How do I work a temporary pacing box?

This is the controller for an internal wire, as opposed to the external box we were talking about above. Same basic idea – the patient is not making enough intrinsic signals to generate a heart rate fast enough to make a decent blood pressure; something like ischemia, or an acute infarct involving the SA or AV nodes is disrupting the process. A wire is placed – almost always in the cath lab, although at really rare intervals you may see one "floated in" at the bedside – and is connected to a generator box. Same idea: set the "rate" control at some rate that you think will make a blood pressure, then increase the milliamp output until the heart is captured 100%.

Once the box is set you'll probably have to worry more about the wire being dislodged than working the box itself, although you need to have the basics in mind.

There is a third knob besides "rate" and "milliamps" on an internal controller, labeled "sensitivity" (doesn't always work on males). If the patient's heart rate does come up, you probably want to let it capture, because intrinsic rhythms are usually the best ones – setting the sensitivity lets the box sense the patient's intrinsic rate. Or not. In emergent situations with the patient's rate at 22, (or zero!), you usually want to set the box to just pace – not sense. In that situation, the knob is set to "least sensitive", or "asynchronous".

31: What if my patient's K is 1.9?

Well, how the heck did that happen? These things don't come out of the blue, y'know! This is sometimes the patient who got overdiuresed – too much lasix? I guess! This sort of underlines my fear about some of these drugs that we give patients to take home: here's a patient with heart trouble, probably CHF, probably prone to some arrhythmias anyhow, who gets sent home with a diuretic that makes him "dump" potassium. And, sure, he gets a scrip for potassium too – but what if he doesn't like the taste of it? Ack!

This person is going to need replacement treatment right away. Our rules are: no more than 20 meq of potassium through a central IV per hour, max. You can give a dose orally at the same time – but make sure the patient's creatinine is okay! (Why?) And make sure the patient stays on a monitor until you know he's not going to keel over in VT!

It really is amazing how, in some patients, replacing electrolytes can make arrythmias go away. Some people are very predictable this way: "Oh, did he have a run of ten beats? Yeah, he has one every night – did he get his Mag dose yet?"

32: 6.9?

Then again, there can be too much of a good thing! Is this result for real? Could it be a hemolyzed spec? (Quiz question for later – what does hemolysis have to do with it? Clue: draw specimens gently from arterial lines.)

If it is real, then the danger is severe – the patient may go into an arrhythmia just as she would if her K were low, or maybe brady down to about nothing. Couple of maneuvers to make here: first we might give a dose of regular insulin – 10 units IV push, followed by an amp of D50. (Jayne says to give the D50 first – makes sense to me. In the blood sugar world, sort of too high is much better than way too low.) The insulin will push the potassium from the plasma into the red cells circulating in the blood, so the plasma level will drop. That same insulin dose will of course drop the patient's blood sugar too, so that's why they get the D50. The problem is that the potassium will leak back out after a short while, so this only buys you some time. Giving calcium chloride is supposed to help protect the myocardium from irritability in this situation. I'd be pretty irritable myself.

The second maneuver works better, but takes longer: kayexelate. This stuff is an "exchange resin", which sits in the gut (it has to get into the intestinal tract to work), and swaps ions – one reference I looked at said that one gram of kayexelate will bind one meq of potassium – good to know. This stuff works pretty well, but of course you need to be thinking about what the problem is (isn't that just always the way?) – is the patient in acute renal failure? Everything always depends on the context.

You noticed that little key phrase up there "it has to get into the intestinal tract to work"? Apparently kayexelate doesn't work if it just sits in the stomach, and if your patient has some sort of ileus, then you can give doses through an NG tube all day, but they'll just bounce off the pylorus and come back up the next time you check an aspirate. We see a lot of opiate ileus's – the only thing to do in this situation is to give the med as a retention enema through a rectal tube.

Important things to remember about rectal tubes. First – they don't work very well. You can't give a large volume through one and expect the patient to retain it – what we do is to mix the kayexelate with some normal saline to make it dilute - it's very thick - and then give small amounts every half hour or so through the tube, maybe 100cc at a time. Clamp and unclamp things as necessary to let the dose dwell, then drain, then repeat. Works pretty well. Better than trying to give 500cc, having it leak everywhere, and then having to tell the team that it couldn't be done. This trick works with lactulose too. Don't forget to let the balloon down every four hours.

33: What if my patient is having an acute episode of ischemia, or an MI?

Why don't you ask an easy question, huh? Lots of stuff to think about in this situation, and you know, there's a reason why God created cardiologists…

Some basic thoughts:

Is the primary process MI or ischemia?

- If an MI, should the patient get "clot-busted"? (Are they liable to start bleeding from someplace if they do? Have they recently had surgery? Maybe lysis isn't such a good idea.)

- Should she get an aspirin?

- Should she go to the cath lab? If the goal is to reopen a plugged coronary artery – probably. Is the patient 26 years old? 126 years old?

- Is the patient having specific symptoms that need to be treated right away? The symptoms can vary a great deal, depending on where the MI is territory-wise. Inferior MI people may vomit and go bradycardic (atropine!), while anterior or lateral MI people may become horribly short of breath ("flashing" - although that can happen with an ischemic episode or an MI, and it's important to figure out which is which.) Lots of other arrhythmic possibilities exist too.

Either way, ischemic event or MI, some basic maneuvers usually apply:

- Morphine. (This is all, as always, with MD orders, right?) Make sure the patient has a blood pressure before (and after!) you give it.

- Nitrates – sublingual nitroglycerine is usually the first thing to try here, but if the patient is having an MI, this may actually not be what they need. Watch their blood pressure!

- Oxygen. This is the problem, right? - some part of the cardiac musculature isn't getting 02 – so apply some. Try to remember if the patient has COPD or not. (Why?)

- Is the patient short of breath? Sit her up, way up in a high Fowler's position with pillows supporting her arms. Watch her blood pressure. Does she need diuresis?

- Get lots of EKG's – in fact, leave her hooked up. If the pain comes and goes, try to get EKG's with the pain and afterwards, to see if things are changing. Take a look at the FAQ on infarct localization for help on interpreting these guys. It's not as hard as you think…really!

Volume

34: What if my patient is dry? How do I know?

Arguments can actually break out at the bedside on this one, and not for trivial reasons either. It can be really hard to sort out what a patient is doing if they show up short of breath, looking bad, getting worse, with a diffusely horrible chest x-ray that looks like "wetness". We've seen patients come in who were actually developing something unusual like ARDS after some precipitant like a car crash, or maybe BOOP (discovered by the eminent pulmonologist Betty, back in the 40's, at the Warner Bros. Med School. Didn't she do something else as well?) Or Wegener's, or whatever…

Anyway: is he making urine? Sodium up? BUN up? What's the BUN/ creatinine ratio? Here's a normal one: BUN/ creatinine of 10 / 0.7 .

Now look at this one: 60 / 0 .7 - look different? Clearly a higher ratio than the one before. Which one means "dry"?

Let's take a second to remember what the numbers actually mean. The BUN tells you how much nitrogen waste is floating around in the blood, while the creatinine tells you if the kidneys are actually working or not. If the creatinine is high, the kidneys are in real trouble – maybe "taking a hit". Then the BUN will go up because the kidneys can't get rid of it.

If however the creatinine is normal, then the kidneys are working properly. So if the BUN is high, it means that the patient is dry – their BUN is high because the patient has lost water. Dry. Less water means that everything floating around in the blood becomes more concentrated – red cells, electrolytes, BUN – see? So the hematocrit will go up, the sodium will go up, the BUN will go up…see? Right.

Here's a scenario: Mr.Yakowitz comes into the ER. He's 64 years old, and he's been feeling rotten for about ten days. Hasn't had much to eat or drink in that time. Getting a little short of breath. Chest x-ray is clear, EKG is normal (he's not having chest pain.)

He used to smoke for many years, but he "quit last week". He does wear two liters of 02 at home for COPD. Not making much urine. (Jayne the CNS : "Wouldn't he have RV strain pattern because of the COPD?" – Yeah, okay, smartypants!)

Quick look at the labs: Sodium is 147. Hematocrit is 52. (What reason does this guy have to walk around with a high hematocrit besides being dry?) BUN is 64, creatinine is 0.8. What do you think?

Yup, dry - real dry. I wonder if he's making any urine – these people can get so dry that they can get pushed into renal failure. (Is he pre-renal? Post-renal? Intra-renal? Renal-renal?) This is a classic situation: a COPD patient who gets pushed over the edge by a URI or community-acquired pneumonia. They come in "dry as a dog-biscuit", and their x-ray is clear because their infiltrates won't "flower" until they're hydrated. Then they may get into more trouble handling secretions….

35: What if he's wet?

Opposite problem. Of course there's wet, and there's wet, depending. "Wet" usually means that the patient is fluid overloaded to the point of hypoxia – pushed into a little CHF. If you give enough fluid, almost any patient can get into trouble (also depending) – but it gets a little complex if you have a patient whose blood pressure is low. Volume resuscitation in sepsis can add up to a lot of liters in a very short time – keep careful track! Respiratory "wetness" will usually show up as increasing hypoxia, shortness of breath, bilateral rales to one level or another – you know that stuff.

Another aspect: the patient may look "wet" because she's having myocardial ischemia. Someone with left-sided CAD who has an ischemic episode may develop a sudden problem with her mitral valve. (That's the one on the left side.) Remember the chordae tendonae? – the stretchy things that support the valve leaflets? Ischemia can make them suddenly go all floppy, and then the valve doesn't valve – blood starts leaking backwards with every contraction, back towards the lungs, which get congested, and leaky, and then the little alveoli start filling with water that transudes from the capillaries because of the backup pressure…and it can happen <u>really</u> fast. "Uh-oh. I think he's flashing".

So for sure this ischemic person is "wet" – but should you remove fluid? Probably, but you need to treat the underlying problem, which is the ischemia. So you do the little memory thing: LMNOP.

L: Lasix – most of the time they'll give some. But again, volume overload may not be the real problem.

M: Morphine for the pain, also helps lower BP ("afterload reduction") – which in English means "dilating the arterial system so that the heart doesn't have to work so hard to pump blood into it".

N: Nitrates. You know this stuff – sublinguals, nitropaste, IV nitroglycerine. If you can "fix" the ischemic situation, the valve may start valving again, and you may save your patient an intubation.

O: Oxygen is what the myocardium wants, right?

P: Positioning helps – sit her up straight with pillows under the arms.

36: When should my patient get a central line?

Not too hard to tell – any time your patient needs a pressor, for example. Some patients have terrible veins, and they come in with complex problems, and they start needing all kinds of good stuff like fluids, antibiotics, blood, drips of all description – access is everything in these situations.

37: Where should it go?

Depends. In a real emergency like a code, the team will go for a femoral site – you won't need an x-ray to confirm the position. (Although you can tease the team and ask if they want a KUB.) Not the cleanest insertion prep, but once the patient is stabilized you can go after a line in the neck or the subclavians.

A thing to remember: is your patient on a lot of forward pressure from a ventilator? A lot of PEEP maybe, or a lot of pressure suport combined with PEEP? The patients' lung apices will be pushed up almost to his ears – be careful with subclavians! Everybody know how to set up for a chest tube?

38: Should I give IV fluid?

Depends! Pump, volume, or squeeze? If the patient is "just dry", then the hematocrit will probably be up – most hypotensive situations are usually treated with a bolus or two of normal saline given over a few minutes. For a really rapid fluid bolus you can put the saline in one of the pressure bags that we use for pressurizing arterial and central lines. Remember two things – giving fluid this way through a peripheral vein may blow your only access. Second – (very important, this) - **purge the air from the saline bag before you infuse!** Otherwise the patient will get the air as a bolus. Bad.

39: Should I give blood?

Depends! Do they need it? How would you know?

40: What if my postop patient drops her pressure?

Always scary. The first move is probably to give some volume – it's important to know if your patient got a lot of IV fluid during the case in the OR (and if she made urine during the case). Big postop belly cases will "sequester" (there's a word!) lots of fluid in and around the very vascular tissues everywhere in the abdomen, so they'll act like fluid sponges for at least a day or two. These patients can scare the life out of MICU personnel who don't recognize what's happening.

Another thing that can happen is that the patient simply warms back up. If Mr. Shmulewitz comes back from the OR after a long procedure with his chest or abdomen physically open for several hours, he's going to be very cold indeed when he gets back. Cold makes blood vessels do what now? Constrict, very good. With what effect on the blood pressure? Raises it, correct. Also very good. So as the patient warms up, the vessels will, what?… dilate – excellent. (And after they dilate at about two in the morning, you barium, right?)

And when they dilate, their pressure will do what?

Okay – let's get very ICU here. Ready? Mr. Shmulewitz goes to the OR after it's been found that he's infarcted much of his bowel. He just had to smoke and take birth control pills at the same time, didn't he? Dummy. He's down there for three hours, comes back with a PA line, and the anesthesiologist gives you report. Since the patient has a history of CHF, they tried to run him dry during the case – in other words, they didn't give him a lot of IV fluids, and they used a little neosynephrine to keep his pressure up. He made about 150cc of urine during the case. Blood loss was 500cc, and he got two packed cells intra-op.

Right. You unsnarl the lines, hook everything up, blood pressure is pretty good, say 126 systolic with a MAP of 67. Let's hook up the PA line – core temperature reads 94 degrees.

Let's shoot some numbers: CO /CI /SVR /SV are, respectively: 2.8/ 1.8 /2050 /25 . CVP is 12, PCW is 17.

Vent settings are 60% FiO2, IMV at a rate of 12, tidal volume of 700, PEEP of 15.

Interpretation please? Everyone remember how to interpret cardiac-output/ SVR/ SV numbers?

Something seems to be wrong. Cardiac output is low, but no, he's not cardiogenic. He's tight, that's for sure. Let's check an EKG – no changes. So what's going on? Anybody? Anyone notice the stroke volume? Doesn't look right? Right – that's they key here. Remember the three parts of a blood pressure: pump, volume and squeeze? Which one isn't in good shape here? He's certainly not having any trouble squeezing; look at that SVR. The cardiac output is iffy – is there a pump problem? Only indirectly. If this were cardiogenic shock, which the pattern does look like, it's true, would he be able to empty his LV? No. Not well, anyway. So his wedge pressure would be up, down, or sideways? Up – correct. But this wedge isn't very high. How about the stroke volume? That would be down in cardiogenic shock, but down to 25? That doesn't look right.

In fact, it's very low – the usual SV range is something like 70 –110 cc. Mr. Shmulewitz is dry – they ran him dry during the case, remember? But his abdomen was also open for three hours, right? You think you have insensible loss on a hot day? – just try hanging around for three hours, even in a cold OR, with your abdomen open to the breeze! Enormous fluid loss there. Plus almost all the water component he's got in his whole body is flying to his belly now. No wonder he needs neosynephrine!

So, okay, now we know what's going on. Great – let's give him some IV fluid. But this is the MICU, remember? And the resident is very aware of the history of CHF – once she's persuaded that this isn't cardiogenic shock, she takes her courage in both hands and gives you an order for D5 1/2NS at 75cc an hour for One Liter Only! Maybe you should call the surgeon back.

Now Mr. Y. begins to warm up. Covered up with nice blankets, nice warming circuit running on the vent (still intubated postop) – what happens? He dilates. Are you ready to barium? Not yet! Pressure drops some more. Let's shoot numbers again, in the same order. This time: 2.2 / 1.5 /2400/ 18. CVP is 10, PCW is 16. Ack! Even tighter! Bet he's losing his peripheral pulses at this point, fingers are blue…what to do? (That SV is awful low…)

Anybody catch the ringer in this situation? (Meaning, I threw in something that really does happen, but makes the situation less obvious than it might usually be.) Stroke volume is really low – he's obviously dry. But the CVP and wedge pressures are fine – is he really all that dry?

The ringer is the PEEP. (Strictly speaking, this situation really is too hard for beginners. But this is the kind of thing that you're going to see, and it can't be bad to throw in an example of something complex. Come back and look at it again a year from now.)

PEEP does what exactly? It sets an expiratory pressure limit, which is to say, the patient can exhale, but only to a point. The vent will maintain "x" amount of forward pressure through the ET tube at the end of expiration. Forward pressure. Into the chest.

Increasing PEEP pressure means that the intra-thoracic pressure increases, and that means that any pressure that you read coming out of that patient's chest is going to be artificially raised. It's going to read higher than it really is. Your CVP and wedge pressure numbers are lying to you. (But mom!!)

The way I was taught it, back when the ICU was in the basement of the Great Pyramid: for every 5cm of PEEP after the first five, take away three from the wedge pressure. And presumably, the CVP as well.

So the situation here – this patient is on how much? - fifteen of PEEP? Okay, so we ignore the first five, right? That leaves ten, or two fives, okay? And for each of those, we take three away from the central pressures, okay? So a CVP of 10, and there's two fives of PEEP left over, so that makes actually two threes, so that's six, so we, uh…what was the question?

It's really pretty easy. 15 of PEEP. Take away the first five. That leaves ten, or two fives. For each of those fives, take away three from the wedge and CVP. Two fives – two threes. Got it? Three fives, three threes. See? So the CVP which says 10 actually isn't 10, it's actually 4. See? And the wedge which said 16 is actually 10. See?

The point is: if there's a lot of PEEP, then you have to suspect your central line numbers – they're probably too high. The patient may very well be "wicked dry" (Boston speak). Is he peeing?

The best thing might be to call the surgeon.

Okay – here's Dr. Yakowitz. (The patient's niece?) Orders: normal saline 500cc IV bolus times two over 10 minutes each. (Use the pressure bag trick. **Vent the air first!**) Then run D5 lactated Ringer's (why do surgeons always use Ringer's?) at 300/hour, and give 250cc of 5% albumin every 4 hours until she comes back for morning rounds. Transfuse for a crit less than 30.

So – the patient gets a rapid bolus of a liter of NS, and a bolus of 5% albumin too, or a bag or two of hetastarch (which I understand they make from Jello…kidding!) – and his pressure starts to rise. Wow – look how far we weaned the neo in an hour – let's look at the numbers. Well - first off, the CVP is now 16, and the wedge is 22! Let's talk to Dave from respiratory – yeah, his P02 is 246 – think we can wean the PEEP down?…what do you mean the medical team wants to diurese the patient – we just got hydration orders from the surgeon! (Gnashing of teeth, rending of clothes.)

Let's shoot the numbers: 3.2/ 2.6/ 1700/ 46. Wow! Look at this: CO is 3.8, up from 2.2, index is 2.6, up from 1.5, SVR is down from 2400 to 1700, and the stroke volume is 46, up from 18. And who was the one that wanted to start dobutamine, huh?

So what Mr. Y has done here is to open up, as we filled him up. Make sense? His arteries could afford to loosen, because they were fuller. See that? Isn't that so cool? He's still on the dry side though, isn't he – see, his stroke volume is still low, and he's going to be hiding God-only-knows how much fluid in and around his abdominal wound for the next couple of days, so you need to straighten out your fluid management orders right away.

No – it isn't always that complicated. But wasn't that fun? (Total geek, your preceptor.)

41: What if her abdomen/ arm/ neck/ leg is swelling?

Well, of course, that's the other thing. Postop bleeding happens sometimes – rarely, but it happens. Follow the hematocrit, follow the coags, tell the team, and what I do in belly situations is to measure the abdominal girth every couple of hours with a measuring tape, just as I would for any part of the body that was swelling. Time for an abd CT? Retroperitoneal bleeding, maybe? Last week we had a patient whose neck was swelling after a central line insertion – I've seen it happen after (traumatic) intubation as well, but for different reasons, right? Bleeding vs. subcutaneous air. Either way, that patient is at risk for airway closure – should the patient be tubed? Do you know where your trach kit is? (And the surgeon?)

Non-human example: we took our newly adopted greyhound to get spayed, and brought her home with a lump next to her incision which grew steadily, hour by hour. Went back to the vet, who reassured us repeatedly that this was a seroma, a collection of serous fluid. Seroma my butt. That poor dog wound up with hematomas extending down all four legs, and that was after she spent the night at another vet's hospital with a pressure binder on. The vet had missed a bleeder.

42: What if he pulls out his arterial line?

Oh, well, that's no big deal, right? They can just pop in another one, right? What if the patient is anticoagulated? This can be the source of significant blood loss. Grab the site, compress it, and think about sending a hematocrit. Hold pressure for about 10 minutes, apply a pressure dressing (not too tight!), tell the team, and come back to take the dressing off a few minutes later to see if everything is okay with the hand/ arm/ foot.

43: Central line?

Oh – I don't like this one. Very dangerous, because things could go either way, right? They could bleed outwards, or they could suck air inwards – or they could bleed into their tissues. And what if that's the only access they have? And they're getting their pressors/ sedation/ TPN/ paralysis and antibiotics through it? Nuh-uh: bad.

44: PA line?

Same kind of thing, except that if the line only gets pulled back to the RV – well, somebody tell me, what's the dangerous thing about that? And what if one of the proximal ports is hanging outside of the skin? With the levophed running through it?

Related question – what do you do if your PA line is stuck in wedge?

45: Balloon pump?

Don't let this happen. Make sure that the team knows if your balloon patient is getting confused (they often do), and keep her safe. Sometimes that may even involve intubation, so that the patient can be sedated safely with something like propofol.

46: What if he pulls out his only IV access and drops his pressure immediately?

Lost your pressor access? Well – do what you can. Get the team in the room – you're going to need quick central access, and for that you want a femoral line so that you don't have to futz around with x-rays and stuff. Or if there's any delay at all, you can try putting in a (hopefully) large-bore peripheral line and running some fluid along with some neosynephrine in a peripheral mix: 10mg in 250cc. We're only supposed to run that for as long as it takes to get a central line in; pressors and peripheral blood vessels really don't go together well. In a code? Do what you have to do, but go to a central line as soon as possible.

47: Needs sedation immediately?

Feeling nervous? Oh, the patient…I think we looked at this question somewhere else, maybe in "Med Tips". Here's a story I heard: a patient, young guy, maybe an OD? He'd been intubated and lined for apnea and hypotension, and I think also maybe had an aspiration pneumonia, so I think it wouldn't have been safe for him to extubate right away. Anyhow, the guy woke up, extubated himself (you know how to work the restraints, right?), yanked his IV's, and was halfway out of the bed by the time the nurses got down the hall. Looking a little blue, too, he was, and no IV's left. That was when they did the nebulized morphine trick. Worked like a charm.

48: Has a rapidly enlarging hematoma at the line site?

At the site where his line pulled out? Or where the new one went in? Not a good sign either way. Is he on heparin? Get the team – if it's really growing quickly, think it might be arterial? Once in a while a central line will wind up in the nearby artery, and if your patient is very hypoxic you may not be able to tell by the color of the blood in the line – likewise if she's hypotensive, it won't come out under pressure the way it normally might. Try hooking it up to a transducer and have a look at the pressure – even if the patient is hypotensive, the pressure will be lots higher in an artery than it will be in a vein.

For the hematoma itself nothing works like pressure at the site. Sandbags seem to have gone out of favor in recent years, and anyway a rapid bleed might need manual pressure, followed by one of those clamps that they use in the cath lab. Once the team takes a look you might want to ask if vascular surgery should take a look at the site; sometimes a patient will need a vessel surgically repaired. Check the distal perfusion – good pulses below? Know how to run a pulse-volume recorder?

49: Has trouble after a paracentesis?

Most of the problems that come after paracentesis have to do with blood pressure dropping after the procedure. The liver is going to start re-effusing ascites (out of the circulation, into the abdomen) as soon as you remove what was there, and it may happen at a pretty rapid rate. Usually the thing to keep in mind is that the patient may need volume replacement: we give one unit of 25% albumin IV for every liter of ascites removed. The albumin tends to stay in the circulation better than IV fluid would, so this works pretty well. Watch out for bloody drainage.

50: Thoracentesis?

This has generally gotten much safer since they got better at ultrasound-guided drainage. Even with really good x-rays, it was just never easy to know where the needle was going, exactly. Obviously the big problem to watch for is pneumothorax – everybody know what a patient with a pneumo looks like? Short of breath – sure. Get a chest film – you're going to get one anyhow to see how well the lungs are re-expanded, right? Know how to page surgery? Know how to needle the chest? Should you? Keep a pleurevac handy.

Arterial Squeeze

51: What if my patient suddenly drops her BP?

How long have you got for an answer? We talked about arrhythmic problems before – of the three parts of the blood pressure, that was "pump". Next would be "volume", and we talked about blood products and IV fluids some. This time it's the third part we're interested in: "arterial squeeze". Some people call this "tone". Not at all hard to grasp – think of the system of arteries as elastic tubes, which is what they are, that can dilate and constrict, which they do. If you have a fixed amount of volume being pumped around in the system of tubes, and the tubes all suddenly dilate, what happens to the pressure? Drops, right? So if you assume that the pump is working okay, and the volume is okay, then what do you do if the squeeze starts to unsqueeze? Anybody remember what an alpha receptor is?

53: Has a sudden rise in BP?

So why are you complaining? Lots of reasons for this – is the patient agitated? Can you tell if your patient is agitated, if he's chemically paralyzed? Not sedate enough maybe? We had a patient a while back who was intubated and who had some kind of expressive neuro deficit, and she really couldn't communicate. She was hypertensive and tachycardic for about two days until someone figured out that she hadn't stooled for a few days…after all sorts of maneuvers with IV meds and drips and this and that, what fixed the problem was a manual disimpaction.

What I worry about more is an inadvertent pressor bolus. There are several ways that this can happen, none of them good for the patient. **You need to try to keep your pressor delivery very constant**. If you're using a background IV flush with the pressors infusing along with it, try connecting the pressor using a manifold (triple stopcock) at the end of the flush line, closest to the patient. If you make a change in the pressor rate and the drip is connected to the flush line two feet away from the patient, she may not "see" the change for a long time if the flush is running at 10cc per hour. For that reason it's usually a good idea to run the flush at a faster rate while you're initially getting the pressors going – the patient will respond more rapidly to changes in the drip.

What you really don't want to do is to bolus the patient with pressor. If your patients' BP drops, yes, turning up the flush rate briefly will get some pressor into the patient. Did his blood pressure just go from 70 to 270 systolic as a result? 320? Not good. But look what else happened – you've neatly washed the flush line clear of pressor, and now the patient may bottom out again before the med gets back down the line. Also not good. Smooth delivery is the only way.

53: Is becoming septic?

Same problem, right? Dilated arterial bed – bacteremia, endotoxins, (and more lately as the theories say: problems in the clotting cascade? Think Xigris?)

The three rules of sepsis:

1: Fill the tank (fill up the dilated system with volume).

2: Squeeze the tank (that'll be your alpha pressor; probably neosynephrine).

3: Kill the bugs.

You're really going to want both central and arterial lines for this patient. It is not good practice to run pressors without an a-line, and for rapid volume administration nothing but a central line will do. We have large bore introducers – they really work well. (They run "like stink".) Useful for GI bleeds too.

54: What if I turn her in the bed and her pressure drops?

Jayne: Turn her back!

This happens sometimes, in my experience usually with septic patients who are in the really sickest phase of their disease course. The way it was explained to me once was that the patient is probably compressing her septic "focus" – her infectious "pocket", hidden away somewhere, and injecting purulent material into her circulation, causing an acute pressure drop. "Septic showering", they call it. Not a very good sign. Need another abd CT? Maybe IR can find a pocket to drain.

Then there are the patients who get turned in bed and arrest – it seems like the really acute, hypoxic patients who are on all kinds of PEEP, maybe 100% oxygen, maybe on pressors, acidotic, but early on – in the acute phase of whatever it is that they're doing – do this once in a while. Usually a brady arrest, it seems to me. Not a very good sign, but I can remember some patients who got better after doing things like this. I have no real clue why it happens.

55: How do I pick a pressor?

Obviously it varies with the hypotension's cause. Is your patient septic, arterially dilated? They're going to need fluid first, and then something to agonize the **a**lpha receptors, which live in the **a**rteries, right? Neosynephine/ phenylephrine is pure alpha, so it's a good choice for that.

What if they're cardiogenic? Well, which receptors live in the heart – the betas? Which pressor has a "b" in it's name? Dobutamine? Good choice! Except – do you really want to flog this hurtin' heart with something that is going to make it work even harder? I didn't think you did. This patient needs an intra-aortic balloon pump – before they came along, almost 100% of cardiogenic shock patients died.

There's definitely more than you ever wanted to know about this subject in the articles on "Pressors and Vasoactives", and "IABP Review". Don't say I didn't warn you.

56: What if I turn up the pressor and nothing happens?

Always scary. You have to work very hard at being patient. Make sure the flush line is running fast enough that the higher pressor dose is actually getting to the patient. Make sure the pressor is plugged in really close to the patient. Don't give a pressor bolus if it's at all possible – it will only create a whole new set of problems. Don't be afraid to turn the drip rate up on the med itself, but be ready to dial down quickly to avoid overshooting. I usually start cutting back as soon as I see any rise in the patient's blood pressure at all.

57: What if my patient gets a pressor bolus?

Now look - what did I just tell you?

This really isn't a good thing to happen, but it's very clear when it does: usually the blood pressure goes frighteningly high, maybe close to 300 systolic. That pressure surge usually causes a reflex bradycardia, which is the little carotid bodies saying "Slow down!" – exactly the reverse of the usual septic situation, wherein they say "Speed up!" The carotid bodies sit in the aortic arch, looking down into the LV – if the volume reaching them out of the LV suddenly pops up, they send out the message to slow down, and vice versa. That's how the reflex tachycardia occurs in sepsis. The bradycardia that comes with a pressor bolus should not need treatment – the heart rate will come back up as the pressure comes back down.

58: What if my waveforms and numbers just don't make any sense at all?

They're confusing enough when they working properly, aren't they? Sometimes you have to sort things out equipment-wise, especially coming back from a road trip to CT or MRI ; where they seem to have this ability to wrap all the lines and cables around the patient's body in coils – how do they do that? Cables sometimes get plugged into the wrong transducers during a transport, sometimes things get confusing. Start from scratch, and try to sort things out from the monitor to the patient cable by cable, re-zero and re-level everything.

Still not making sense? Here's a common scenario: often the first wedge pressure that gets read is the one that they do during the line placement, while the patient is still in a little bit of Trendelenburg, where they've been maybe for the past hour and a half, maybe getting agitated…yet everyone is surprised when you measure the number again after the patient has had a chance to recover. The fact is that all pressures usually rise and fall together – if your patient is agitated, with a BP of 190/ 110, then the wedge and the CVP will both be elevated too: "Well, yeah, but 32 was his "agitated" wedge. Now that he's sitting back up and gotten his pm ativan, his wedge is 18. Do you still want me to give the lasix?"

CAD

59: What if my patient is having ischemia?

You know this stuff from earlier on – do all that good anti-ischemia stuff.

60: What if my patient has chest pain that won't go away?

Not a good thing. This is often what buys the patient a ticket to the cath lab. The first thing to do is to make sure that it's actually a cardiac process going on – if it's not clear, try doing a Mylanta test. Try to figure out if the pain is actually coming from the chest tube that they put in the patient yesterday… if it turns out to be an ischemic event, do all those nice anti-anginal things that you did before. See if anything specific makes a difference in the patient's pain at all, and let the team know what it is. Make sure the patient is very well oxygenated. Watch for ectopy. Send cardiac enzymes. Do EKGs. If the patient does get a cath, plan for the possibility of a balloon pump.

61: What if my patient is having an MI?

What, you mean you left your copy of Braunwald at home? I mean, it only weighs 80 pounds! But we should be able to sum it all up in a couple of minutes…as always, it all depends on the context and the severity of the event. Send enzymes. Think about possible, sometimes even predictable problems: one example might be bradycardias and large fluid requirements in an IMI / RV infarct. Be ready for acute "flashing" of CHF in left-system infarcts. (Why?) Be ready for arrhythmias. Does the patient need lysis? Need an aspirin? You too?

62: What is cardiogenic shock?

Shock is the word used to describe the state that the body gets into when it's not perfusing the peripheral tissues very well, and each of the three components of a blood pressure has it's own version of shock to go with it – this time the name sort of gives it away. Almost by definition a cardiogenic patient is going to be in the middle of a big left-sided MI, diminishing the pumping ability of the LV. Poor pump, poor output, poor pressure, poor perfusion, acidosis, etc. Some patients with a poor EF will always have cardiogenic-looking numbers if you put a PA line in them…and make sure they're not just dry! There's altogether too much material on cardiogenic shock in the Balloon Pump Refresher…

Respiratory

Non- Intubated:

63: What if my patient becomes short of breath?

This is almost never going to happen out of context with the underlying problem – does the patient have heart disease – did she flash? Did she aspirate? Does she have her air boots on? (Why am I asking?) Did she plug? Anaphylax? See Vin Diesel on the TV? (My daughter explains these things to me.)

You won't go wrong by calling for help – get the team, get respiratory, get the resource nurse. Does the patient need to be suctioned? The good thing is that - as always - you don't have to work the problem by yourself. Let me restate this central principle yet again: get help. Ask questions. Work with the team, of which you are a part.

Not actually to be filed under the "short of breath" category, but a nice story anyhow, is the time that I was working on a floor, I think it was during the Pleistocene era (I try to remember which dinosaurs were around then besides myself), and a woman gave me a frantic wave from down the hall. Her roommate, a nice enough lady who never stopped talking, ever, under any circumstances including sleep, had done the classic aspiration thing of a piece of her dinner – just like they teach you in CPR. Obviously before the days of central sat monitoring. She was a very interesting color. I slid into the bed behind her – she was sitting up – did the Heimlich, and out it popped. So that works, anyhow. Or it did then. Good to know.

64: What if she has COPD?

These patients carry around their own set of problems. Oftentimes they'll come in with something that's pushed them over the edge – they're usually sitting there with their feet hanging over the edge anyway. Maybe a URI, maybe pneumonia, maybe a COPD flare. Remember that these folks won't tolerate much oxygen. Nebs, steroids, antibiotics maybe, and remember too that any sedation you give them may be just what they need to stop breathing…

65: Is acutely hypoxic?

Is he wet? Did he plug? Throw a PE? Come disconnected from the vent? Try to think of the possibilities, and try to fit things in with the diagnostic picture: "Oh, he needs to be diuresed again." Or: "Oh, he's just put his chewing gum over his trach again – you can always tell." You'll never go wrong by calling for help, and getting an EKG, a blood gas, and maybe asking for a stat chest x-ray.

66: What if I gave her too much oxygen?

Ah, big deal. So she has COPD and stopped breathing, so what? I mean really, this preoccupation with trivial details…

67: Is acutely hypercarbic?

C02 narcosis is definitely for real. A COPD patient will become unresponsive if they "retain" CO2 when they get too much oxygen. Everybody understands how that works, right? No?

In five minutes or less: When you hold your breath, and your brain begins to scream "Breathe, dopey!", it's the chemoreceptors (also in the brain?) that are doing the yelling, because why? Because your pC02 is rising, up into the 50's maybe. So, you breathe.

Now – if you're dealing with a COPD patient, he probably walks around with a pC02 in the 50's all the time. "50?", he says, "Ha! It is to laugh! I don't worry about no stinkin' CO2! I got plenty of CO2! How about that, huh? What you got? You talkin' ta me?" Taxi driver, is he? Personality might be giving you a clue here…

Of course with a pC02 of 70 he might not be breathing much anyhow, because he'll be narcotized – patients with COPD will still do this – and will become unarousable, barely breathing. Time for a tube? Narcan? Romazicon? Sometimes you can head off intubation with something like a bipap mask – they work, but I hate them. The patients often hate them. And all they need to do is vomit into them…can you imagine? The word is that it takes 20cm of forward pressure from a mask device to start pushing air into the esophagus, inflating the stomach…I wouldn't be so sure about that.

Anyway. In the case of COPDers, walking around as they do with a chronically high pC02, the chemoreceptor thing is deactivated – doesn't work any more, exactly because of that continually high C02, as though they had become saturated, which maybe they are. So these people use a backup system (apparently it was the Great Biomedical Engineer up above who invented redundant systems – I mean was that smart or what?) – they get a stimulus to breathe when they become hypoxic, rather than hypercarbic. You'd think hypoxia would be the primary drive, but it ain't.

"So okay, they breathe when they're hypoxic, big deal." Well, the problem is, see, that if you then give them all the oxygen that they seem to want, why, then they may have no drive to breathe at all, 'cause they aren't hypoxic any more. Which is what they were depending on. And they may stop. Right in front of you. Because you put 100% on them. Bummer.

Actually it seems to be a progressive thing – they'll breathe less and less as they get a higher and higher FiO2. This is called "retaining", because they retain CO2 in response to getting oxygen - they exchange less gas, breathing more and more shallowly. You can actually document the rise with a series of blood gases, and it can be very abrupt: "Look, on 40% his pCO2 was 62, but on 60% it was 104!" "I guess that was when he stopped breathing, huh?" Narced. Sometimes these patients want something like 1.5 liters/minute of oxygen and no more. Strange but true.

68: When should I get a blood gas?

Our rule is to get a blood gas anytime we make a vent change (about 20 minutes later), or if there's a clinical change in the patient. Here's a story: not too long ago we had a patient with respiratory failure, looking kind of tenuous on 100% face mask O2 (not a CO2 retainer… even some people with COPD, just aren't) – and the intern had sent a blood gas which looked pretty good. The patient became increasingly agitated, which was treated with some Ativan, then some Haldol. He then became unresponsive, and pale, and wasn't breathing much, if at all – when we asked the intern to send another gas, (he was in the room with us), he told us that the last one had been fine – what was the problem? Audience – what was the problem? Actually, more than one person in that room had a problem.

69: Suddenly starts wheezing?

Did the patient just get a new med? Is she having an allergic reaction to something? Is she halfway through her first dose of a new antibiotic? Stop giving whatever it is she's getting, get the team, get some benadryl, maybe some IV hydrocortisone…then there are the people who "flash" in CHF and wheeze – they call this "cardiac asthma": get an EKG. It always depends on the context.

70: What if my patient is "flashing"?

This often comes under what we looked at before under the heading of the "acutely ischemic" thing. The basic rule for any acute situation always applies: get the help you need into the room right away (this always includes the resource nurse.) Try to think ahead a bit: is the patient going to need intubation in the next five minutes? Three minutes? Half hour? Try to remember "LMNOP", ekgs, blood gases, an x-ray, things like that, and suggest them at appropriate times. Then make sure they happen if they need to.

71: What is "guppy breathing"?

It doesn't a whole lot of ICU experience to figure out that a patient breathing shallowly at a rate of 60 isn't going to last very long before needing some kind of intervention: mask vent support, suctioning, diuresis, intubation – maybe all of those, depending, but guppy breathing shouldn't be allowed to go along without some kind of decision about treatment.

72: What if my patient stops breathing?

Well now that doesn't sound very good, does it? As always, it depends on what's going on. Your 92-year-old DNR patient dying of terminal whatever is probably not going to be leaped upon. A patient with too much narcotic on board might get the oral airway-mask-bag plus narcan treatment (don't forget the restraints.) Your task is to see it coming. If you can do that, then you get to wear the invisible golden badge…some of the most experienced nurses never wear extraneous pins, plates, medals, or whatever – just scrub, name and maybe school pin. They're hoping you see the invisible badge.

Note to hospital administrators: if you want your ICU to run like a swiss watch, remember that it's the experienced nurses who are the jewels in the mechanism. Hold on to them.

73: Obstructs her airway?

What with? Did her tongue obstruct? Secretions? Time for a jaw thrust? Oral airway? Nasal airway? Suctioning? All of the above?

74: Has sleep apnea?

These are the people whose tongue will obstruct their airway about 25 times an hour so that they never get any sleep. Tiring. These people often do well with the application of a CPAP mask, or a nasal bipap device – the forward pressure holds things open during the breathing cycle. Sedatives usually a bad idea.

75: Plugs?

This should be the kind of thing that you see coming – the patient who comes in with pneumonia, getting hydrated maybe, who is starting to loosen up all the dry secretions that have been hiding in his lungs for the past week or two. Remember that secretions tend to create a pattern of worsening ventilation – the O2 sat may actually be okay, but the pC02 may be rising rapidly, so the patient's excellent saturation may be fooling you. Use a nasal trumpet for frequent blind suctioning, and say you're sorry.

76: What does a pneumothorax look like?

Sometimes young people will walk into an ER someplace complaining of feeling funny, and it'll turn out that they've been walking around with a dropped lung for who knows how long. Patients in the unit tend to be in rougher shape to start with, and a pneumo is usually pretty obvious – something suddenly looks really wrong. Your main clues still come from the context – did they just get a new neck/ chest central line? How many sticks did it take? How much PEEP is your patient on (are his lung apices up around his teeth – easier to hit?)

Once you have some idea of what the problem could be, the clues will start falling into place – oxygenation worsening abruptly? Any kind of abrupt respiratory change should get a chest x-ray; then hopefully you'll really know.

Another very useful clue is the appearance of a visible pulsus paradoxus on the patient's arterial line waveform. This is easier to understand than it sounds: the patient's blood pressure will drop when the pressure in the chest rises during the breathing cycle – if the patient is intubated, that happens on inspiration; if not intubated, it happens on expiration. If there's a substantial pneumo in there (or a cardiac tamponade – anything that compresses the heart), then the small addition of added pressure during the breath compresses the heart just enough to stop effective pumping – when the pressure comes off, then the pump starts working again. It's very dramatic sometimes, and it means that the situation in the chest is really becoming critical – soon the pressures will rise high enough that the heart will be compressed all the time. Bad.

77: Should we needle the chest or not?

You wouldn't want to be wrong about this, right? (My smart-guy best friend says "I thought I was wrong once but, uh, I was wrong.") Needling the chest may produce the pneumothorax that you are hoping to treat – and that patient will be pretty much committed to a chest tube as a result. Wait for the x-ray if you can, but have your equipment on hand. If the situation approaches a code, then giving your patient a chest tube will probably hurt a whole lot less than half an hour of chest compressions…

Intubated:

78: What if my patient codes during intubation?

This may mean that you waited too long to tube him – the team may hesitate to intubate a patient who might respond to other measures. Judgment call. Patients in the middle of some really acute ARDS-like situation may need more and more oxygen over the course of even a single shift – this kind of rapid movement usually means that intubation is coming, or should be. There's often a great deal of resistance to intubation in a situation that seems crystal - clear to experienced ICU nurses – and who may even be wrong. (I know – hard to believe that ICU nurses can be wrong.) My own feeling is that you can always extubate someone, but if you wait until they're horribly hypoxic you may get into real trouble.

An example: a patient with a sat of 97 on 100% high-flow mask O2, maybe plus a nasal cannula, or maybe on a CPAP mask is not going to do well if you remove all that hardware to intubate him…as in any acutely hypoxic situation where the sat drops, say, below 70, bradycardia can result. Have atropine nearby, but see if the patient's rate comes up first after the tube gets in.

79: Bites the ET tube?

Not good. I think we talked somewhere else about patients coding, or biting through pilot lines and deflating their tube cuffs - biting your airway closed is generally not such a good idea. This patient may need sedation, reassurance, a bite block, or all three.

80: Extubates herself?

Not a good thing – the patient can do herself some vocal cord damage this way, even though the cuffs are very soft and inflated to low pressures. Confused patients can be really determined to pull their ET tubes out, and can get very inventive about getting loose from restraints. A better plan is probably to keep the patient safely sedate until she's ready to extubate. Propofol is a good choice for this situation, as it wears off quickly.

If it looks like the patient might "fly" after a self-extubation, what we do is apply 100% FM 02 (if they're not a known C02 retainer), and watch them very carefully. Clearly some people are going to do worse than others; a patient intubated for airway protection after an opiate overdose will probably do fine if he's awake enough to extubate himself, but someone with a bad pneumonia may need reintubation right away. Keep your intubation equipment handy, and make sure everyone knows that the patient may "de-tune" at any time. Set your alarm limits tight!

Jayne: We keep a bag hanging beside the vent, with a mask O2 setup ready in case this happens.

81: Extubates herself and runs down the hall extubating everyone else?

Hmm. Is she an angry respiratory therapist? Ask her to deflate the cuffs first.

82: What if I can't get the ET tube cuff to seal?

Couple of possiblities: is the tube in the right place? If the cuff is near the cords, it won't seal because it isn't big enough to seal the airway up there. Look at the cm marks on the side of the tube: in average-size people the tube is usually in good position at around 22-24 cm of depth from the lip, or teeth. Make sure the tube is secured to the patient's face and head so it can't shift in and out. No, you can't use crazy glue.

Another possibility, but rare, is that something is wrong with the pilot balloon line. Once in a while the valve cracks, which can happen from somebody inserting an inflation syringe with too much force. Another real fun event is nicking the pilot line while shaving your patient. Either way, the cuff won't hold pressure. The trick: snip the line either at the valve if that's the problem, or at the place where it's nicked. You can insert a #19 butterfly into what's left of the line (try not to poke another hole in it) - hook that to a stopcock and a syringe, inflate the cuff to seal it, tape the whole gadget to a tongue blade, and call anesthesia: the ET tube will have to be changed.

83: How do I know if she needs to be reintubated?

It really should be obvious – she'll be having trouble breathing, not clearing secretions maybe. Sat will be low, respiratory rate will be too high or too low - you'll know.

GI

84: What if my patient pulls his NG tube?

I'd probably pull mine, too. Please make sure I'm getting enough Haldol. Make that fentanyl.

Have I mentioned the DNR tattoo that I'm going to get? I wonder if the shops in New Hampshire have the radio-opaque tattoo dye that shows up on a chest film: "I am a DNR! My attorney's phone number is…". I can just see the scene at the light box.

85: Pulls his NG tube just far out enough to aspirate tube feeds?

Bad. Every now and then you'll walk into a room and say, "Well, this patient is a very short little guy - that NG tube looks way out to me." This is why you want to check the position of the NG tube at the beginning of every shift. In fact, you should keep in mind that you need to check the position of everything at the beginning of every shift (and during the shift!) – last night I noticed that my patient's central line looked like it had taken a yank; not mentioned during report. There was a blood return from all three ports, but the only thing to do was to get a film – it was okay, but who knew?

Question for the group: when do you think you should add methylene blue to the patient's tube feeds?

86: Vomits?

Did he aspirate? Why is he vomiting? Inferior ischemia? Too much tequila before he went down in the airport bar? Did I enter the room? (Why does that happen so much?)

87: Vomits tube feeds?

Did he aspirate? What was the residual last time you checked? Sometimes the end of an NG tube will tuck up into a corner of the stomach – if my patient hasn't got much in the way of bowel sounds and hasn't had much aspirate in a day, I sometimes pull the tube back a bit or advance it a bit. Sometimes you find a 600cc surprise this way.

Sometimes an NGT will get too far in. You might see a patient losing really enormous amounts of NG drainage, maybe 5 liters a day – the tube may have made it's way into the duodenum. The drainage is usually lighter and clearer than your usual gastric output, and there's really too much of it – if you think that the tube is too far in, you may find that if you pull it back while leaving it to low suction, the drainage may suddenly change color to a nice gastric green. It'll change anyhow, once the tip comes back into the stomach.

88: Vomits and aspirates?

Did he aspirate? Guess so! You were keeping the head of the bed at 45º, right? Checking aspirates, right? Has she stooled lately? Sometimes it just happens, as do many things, no matter how careful or how perfect your care is. Watch the person carefully – almost by definition they're going to have a new pneumonia to deal with. Does she need blind suctioning? Reglan? Intubation?

89: Vomits "coffee grounds"?

Did she aspirate? The classic upper GI bleed scenario. Check a crit, watch her pressure, saline lavage through an NG tube The team definitely inserts this one, esophageal varices can pop if they get poked by an NG tube going down, but how do you know ahead of time? History of previous bleeds? Cirrhosis? Is the patient getting something to block acid secretion?

90: Bright red blood?

"BRB": a little worse than coffee grounds. This person is probably going to need an endoscopy – should he be intubated for airway protection before they do it? Can the patient consent for transfusion? If no one is available for consent, they team can sign the consent themselves, indicating that they couldn't reach any "significant others", and that the situation was emergent.

91: What if he starts passing melanotic stool, or BRBPR?

Same idea, different place. Depending on the severity of the bleed, the patient can be transfused and watched, not transfused and watched, colonoscopized (how exactly do they expect a patient in the midst of an acute abdominal process to drink all that go-lytely, exactly?), or maybe even operated on. Make sure that you're in close communication with the blood bank, and have supplies set up ahead of time. We sometimes "call for the cooler" – which will have all available units of say, A-negative FFP that are due to expire in the next six hours – something like that.

92: What if my patient starts having severe abdominal pain?

This usually happens to me in the car. I need to stay out of Starbucks. Sometimes this can be a whole lot of nothing – other times, some deadly process. Assess carefully, document carefully, drag the resource nurse and the team into the room, follow up. Abdominal CT scan? RUQ ultrasound (what would that be looking for?) Does surgery need a heads-up about the patient?

93: What if he's pregnant?

This almost happened to me in a car. I never got as far as ultrasound though.

94: What does appendicitis look like?

Hurts! I understand that it can show up anywhere in the abdomen. Where is McBurney's Point? –two exits past Dennisport on the Cape, right? I know a good place for lobster rolls.

Jayne: "This whole part is stupid." (Just for the record.)

95: What does a bowel infarct look like?

This is something that we actually do see at times, unlike appendicitis, although all sorts of things are always possible. It's important to remember that hypotension can produce really serious effects in all kinds of places, especially if your patient is a vasculopath to start out with. A patient with high blood pressure at home may have kidneys that go into ATN after just an hour or two of hypotension. (They may have blood pressure like that because their renal arteries are stenosed, and the kidneys are cranking out angiotensin and all, trying to perfuse themselves.) Those renals may be stenosed just like their coronary ones are, and maybe like their carotids, and maybe their mesenteric…)

No bowel sounds, that's for sure. And what do you think their chemistries might be doing?

96: What if my patient has lost bowel sounds, has a K of 6.7 and a pH of 7.10?

See, you already knew! Dead tissue of any size in the body is going to release all the intracellular K it has, and all the poorly perfused/ dead/ almost dead tissue involved is going to go into anaerobic respiration before it dies, producing a big lactic acidosis. These people have lactate levels upwards of 10 – your basic humongous metabolic acidosis. Other things being equal, what will their ABGs look like? (A lactate of ten is high enough to make us old nurses cringe. Saw somebody in the 20's last week, but for different reasons. Your basic Real Bad Sign.)

GU

99: What if my patient stops making urine?

Remember all that stuff about pre-renal, intra-renal, and post-renal?

Pre-renal: ("dry") "Pre" meaning: what's happening in the blood before the kidney gets a chance to see it. Ahead of the kidney. The patient isn't making much urine because he's volume depleted – there's not much water component in his blood. Is his hematocrit 52? Sometimes patients arrrive in the MICU after being diuresed into renal failure from too much volume loss: the BUN will be high, but the creatinine will be normal or heading upwards, maybe something like 70/ 1.2, assuming that they were normal to start with. Some patients certainly look like they need aggressive diuresis – it's usually not wrong to treat someone for CHF if that's what seems to be the problem. Of course if it isn't CHF, or even if it is, they may get a bit too dry. These patients' will probably be making some urine, but very concentrated. One time you might see the pre-renal thing happen is in a patient receiving full-strength tube feeds as her only intake – this stuff is so concentrated that the patient may literally dry out and stop making much urine. Time to dilute that stuff to half strength, and run it at twice the speed. Watch the aspirates.

Intra-renal: In the kidney. If the patient has actually taken a "kidney hit", the problem will probably be something like ATN. This can take a long time to resolve – months, sometimes. Acutely, the scenario is usually a period of hypotension for one reason or another; sepsis maybe. The BUN may not rise right away, but the creatinine will bump up suddenly: 35/ 4.0 . Kidneys really hate to be underperfused – they become "insulted". ("Stupid kidneys!") It seems even easier to insult elderly kidneys that may be perfused by stenotic arteries – if your patient is a vasculopath everywhere else, e.g. coronary arteries, carotid bruits maybe – you can bet they may have renal artery stenosis. These folks' kidneys usually see decent perfusion only at high blood pressure levels, 'cause not a whole lot of blood gets by the stenosis – even an hour or so at a relatively low pressure may make them go into a coma. The kidneys.

Jayne: Keep the patient's MAP above 65 to try to avoid the whole problem.

Post-renal: This is what happens when nobody flushes the foley after the urine output drops. Something is stuck in the urine path after it's been processed by the kidney. Ureteral stone, maybe, or compression of the ureter(s) by something like lymphadenopathy, or tumor? What happens to the kidney if its drainage system is plugged? What does a renal ultrasound look for?

100: Makes too much urine?

Give a little too much lasix, did ya? Sometimes patients will do it on their own; they'll "auto-diurese" for one reason or another – often this has to do with a patient who has high baseline blood pressure and comes in hypotensive. They may not be developing renal failure, but once their pressure gets back up into their normal range they may suddenly start to produce really large hourly volumes. You might see this in a patient who gets sedation for a short period of intubation. While the sedation is on (and the BP is relatively low) the urine output may not be so great – but when the sedation is weaned off – and especially if the patient is a bit agitated (BP up), the output may suddenly become very impressive. Other auto-diuresers: patients who are a couple of days postop may suddenly start mobilizing the five liters of Ringer's that they got during the case.

Then there's diabetes insipidus – this is when you (let me think about this for a second) don't make enough ADH: anti-diuretic hormone. ADH stops you from peeing. Anti diuretic. Not enough ADH, you pee all the time, with a thirst to match. In one end, out the other. Like a siphon. Which is what "diabetes" means. Greek to me, man. Of course, you also turn into a siphon if your blood sugar is 700 for a couple of days…

SIADH is the other way: too much ADH – you don't pee hardly at all. Got something to do with holding onto sodium.

101: Pulls his foley out?

Ow. Was the balloon up? Call urology. Better yet – don't let this happen. (Although even with the best precautions, Houdini down the hall there may find a way.)

102: Twice?

Really ow! Where's urology? If the urethra has been injured, the foley may have to stay in for some time – a couple of weeks?- until it heals.

103: Develops hematuria?

Are you surprised? You spoke to urology, right? Time to start a saline drip through a three-way catheter – and urology put it in, right? Don't let it clot off…

104: With clots?

What did I just say? Are you even listening? Am I just talking to exercise my face? That's the problem with young nurses nowadays. In my day, we had to make our own clots! In the summer! Out of snow! Uphill! Both ways! Yadda yadda! (This doesn't work on my kids, either.)

If the clots are passing through the catheter, good! The problem is: what if they don't? This is why it is absolutely a rule that a saline infusion through a 3-way foley must never, ever be on a pump. Gravity only. Figure it out – what if the pump kept pumping in, and nothing was coming out because a clot was covering the drainage lumen…? If you think that a three-way catheter is clotted, you can try manually flushing it with 30cc of saline, but what often happens is that the clot forms a little flapper over the openings at the tip, which will let you flush into the bladder, but closes when fluid tries to drain. You're going to have to change that tube.

A word about scrotums. Yeah, I know. But somebody has to bring it up. (Maybe the wrong phrase?) We see a lot of patients who are very third-spaced – they have diffuse tissue edema, and the scrotum is not immune; they can really swell up. After getting really worried about one of my patients I checked with the surgeons – simple treatment: an ace wrap. Not too tight. Works like a charm. Like a jewel. Jewels. Something like that.

105: Without clots?

You can usually prevent clots from forming if you run the saline irrigant drip quickly enough – this may involve using bag after bag of saline, but that's just what you have to do. It can be hard to measure exactly what's going in and out – just make sure that what's coming out every hour is more than what's going in! Sometimes we use the little spring measures that are used with PD bags – these tell you how much is infused by the changing weight of the bag.

It's good to have some standard to identify how bloody urine is, besides "Clots or no clots?" Over the years we've gotten comfortable with the "wine-shop" method: "Well, his urine is a nice chardonnay today, but it was definitely a merlot yesterday. Getting better." Often hematuria will be bad enough to require transfusions… what is amicar?

106: What if his BUN and creatinine are doubling every day?

Bad. Foley plugged? Did she spend eight hours hypotensive – heading into ATN? She may be in for a rough month. A useful maneuver that's come up in the past year or so: patients heading into renal failure will usually (duh!) stop making urine – but if you slap them with a big dose of diuretic while they're still on the steep part of the failure curve – just as they're heading into it – sometimes you can get them to change from oliguric ("not peeing much") to non-oliguric ("peeing pretty good") renal failure. What we do is to give some enormous dose of "synergistic diuresis" (whoa! $1.50 for that one) – something like 200mg of lasix and 500mg of diuril, one after the other. If it works, then at least the patient will get rid of water – they won't clear much BUN, but you'll be able to keep them out of CHF.