Department of
Family Medicine

“The Nightmares Course”

(Acute Care for Family Docs)

Department of Family Medicine

Queen's University

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With thanks to Dr Richard Grainger

Introduction to the Course

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Nightmares is a comprehensive, simulation-based course that will teach you how to respond to emergent situations that you are likely to encounter during your in-patient rotations as a resident, or as a GP who practices in the hospital, either Hospitalist or ER. The course grew out of our residents' concerns that they were being asked to resuscitate patients on the floor and in the ER without having the adequate experience and knowledge to do so effectively. Also, we had a shiny new simulation lab that we wanted to play with.

This manual is the didactic portion of the course that will help you organize and solidify your knowledge ***and is a required reading before you show up for your Nightmares session***. The manual is organized according to the four core topics of the course- arrhythmias, shock, shortness of breath and altered level of consciousness. MI, code blue and team leadership bring up the rear. It was written to be physiology-based and easy to read. I am hoping you will not try to memorize much but instead focus on understanding the simple underlying principles that will guide you to correct action even if you have an incomplete knowledge of a specific disease or clinical situation. Throughout the course, we focus on the same basic, vitals-based approach that should allow you to keep someone alive until more resources arrive or you transfer the patient out, no matter what the situation. We also focus on making this a fun and interesting experience that will make you a better and safer doctor. While knowing the medication dosages is not mandatory, I do urge you to try to commit some of the more important drugs (which are **bolded** in the drugs tables) to memory as they will come up time and again and knowing them will make you more confident and smooth.

I hope you will enjoy the course as much as we enjoy running it!

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About the manual

The manual is divided into three broad sections – background concepts, medical team leadership, and reference.

The background material discusses the physiology and related pharmacology to conceptualize and manage acute critical care situations.

The medical team leadership section outlines good practice, common pitfalls and communication skills necessary to lead an effective team responding to an acute care event.

The reference section contains tabulated information for rapid and easy access.

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"Knowledge dispels fear"

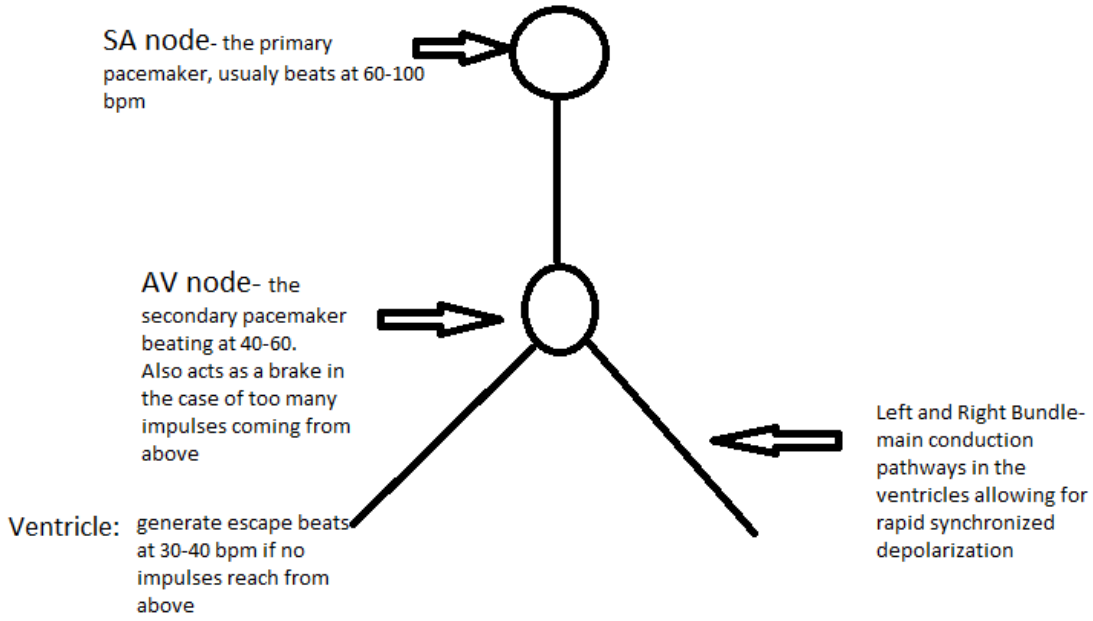
- motto of the Royal Air Force Parachute School

Heart in a nutshell

Before we look at the vascular system when things are wrong, let us remind ourselves how it works when it works well.

Mechanically, the heart is divided into the right side which is relatively weak as it pumps blood into the low pressure pulmonary vasculature, and the much stronger left side which pumps blood into the systemic circulation. Ventricles do most of the heavy lifting, with the atrial kicks contributing about 20% of the output.

Electrically, the heart can be summarized in the picture below:



If left to its own devices, the heart would use its automaticity to perpetually go at whatever that particular heart's intrinsic SA node rate is. But, in response to outside influences, it can also **increase the rate by a factor of 3**, and it can almost **double the strength of its contractions**.

There is a limit to how fast a heart can beat in sinus rhythm while maintaining adequate diastolic filling time, and for an average male it is around 220 beats minus the age, and for an average woman 210 minus the age. ***It is a useful formula to remember as it can help us determine if a particular fast heart rate is sinus or something else.***

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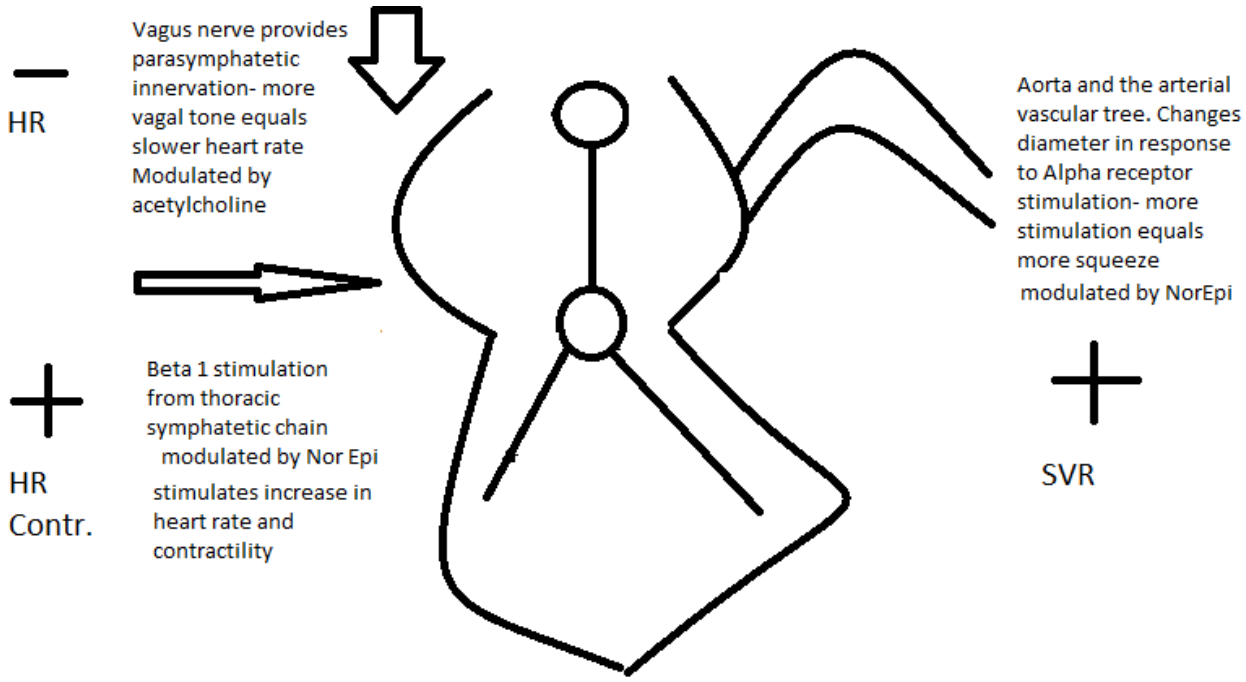
There are two factors that modify the rate- one is the **tone of the vagus nerve** which innervates the SA and the AV node but not the ventricle. Thus, changes in vagal tone can only change the rate, but not the strength of the contractions. The change is inverse, meaning that an increase in vagal tone results in a decrease in heart rate.

The other factor is the **stimulation of BETA1 receptors on the heart,** which are linked to the thoracic sympathetic chain. They innervate both the SA/AV nodes and the ventricles and their stimulation will increase both the rate and the strength of the contractions

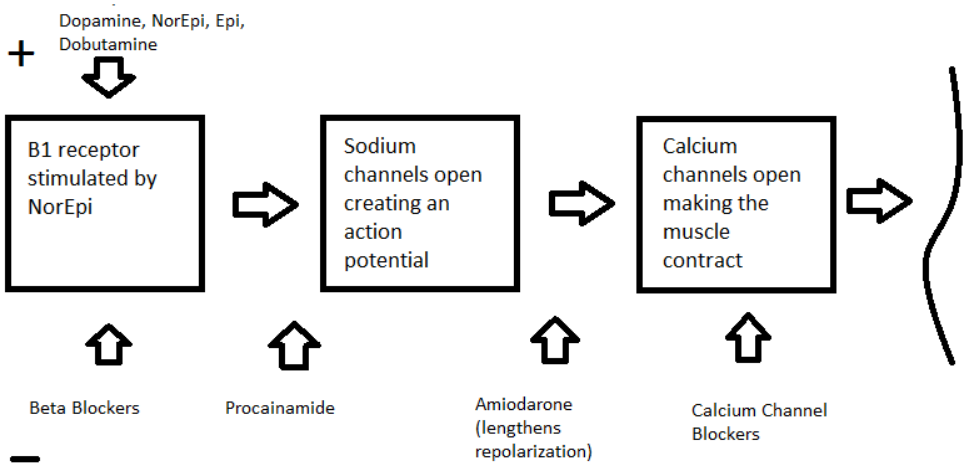
Key Concepts

Vagal tone affects rate only. Sympathetic tone affects rate AND contractility.

The heart pushes the blood into the aorta and the vascular tree which can change the diameter based on the stimulation of the ALPHA receptors, which, like the BETA1s, are linked to the sympathetic system and under its influence can squeeze, increasing the resistance and fluid wave pressure.



Conceptually, when a BETA1 receptor is stimulated, the following chain of events happens that allow first the electrical impulse to propagate and then the cardiac muscle to contract with more or less force. This chain of events is useful to remember when giving drugs that affect it (like antiarrythmics), thus giving us a chance to predict their effect on the heart conduction and contractile force.



SHOCK

SHOCK

Key Concept:
Shock is *lack of adequate tissue perfusion*

In essence, the heart and the vascular system are tasked with one basic function- to provide enough FLOW so that blood reaches the end capillaries and thus oxygen reaches cells. It is when FLOW is disrupted that shock and tissue hypoperfusion occurs. Before we dig into shock, it would be helpful to review basic physiology of circulation.

Unfortunately, we cannot measure flow directly, though we can guess by looking at its surrogates- capillary refill, colour and warmth of extremities, absence of mottling and presence of distal pulses. What we can measure, however, is the driving pressure that creates flow- BP. It is not a perfect determinant of flow as there are other factors that influence it (micro vascular thrombosis in sepsis for example will decrease flow even when adequate blood pressure is present) but it is the best one we have, so we spend a lot of time and thought making sure that BP is within normal limits.

What determines blood pressure?
BP is related to two main factors : Systemic Vascular Resistance (SVR) and Cardiac Output (CO):

$$BP \propto CO \times SVR$$

SVR is the resistance that the whole arterial vascular tree offers to blood flow. It is directly determined by how 'squeezed' or 'open' the arteries are (remember that arteries can change their diameter via their muscular layer). The more squeezed the arteries are, the more resistance there will be in the vascular tree. This is mediated by ALPHA receptors- when they are stimulated by Norepinephrine from the sympathetic system, the vessels will squeeze.

Key Concept
Higher SVR = Higher BP
(for the same CO)
Mediated by ALPHA receptors, so giving α agonists (eg Norepinephrine) will increase BP.

It might seem counter intuitive that more resistance will give us more pressure but it does- think of blowing through a narrow straw- you generate a lot of force with that (the principle behind the blow dart), now try to blow through a wide pipe- it is easy, but it does not generate a lot of pressure.

CO is a bit more complicated. It depends on 2 main factors- Heart Rate (HR) and Stroke Volume (SV).

$$CO = HR \times SV$$

Heart Rate Limits
Time spent in systole is fixed regardless of the heart rate, so as heart rate increases, the duration of diastole and diastolic filling time decreases. So if you go too fast, for example in rapid Afib, your diastolic filling time, and thus stroke volume might suffer and thus reduce blood pressure overall. This is particularly true if the Afib heart rate is above the sinus limit for the age of the person (220-age for males, 210-age for females).

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Heart rate is self explanatory and in general, higher rates will generate more blood pressure and flow. Heart rate is determined first by the intrinsic pacemaker node, modified by BETA1 receptors (up when they are stimulated, down when they are blocked) and by vagal tone (down when the vagal tone is up, up when vagal tone is down) which is modulate by ACETYLCHOLINE.

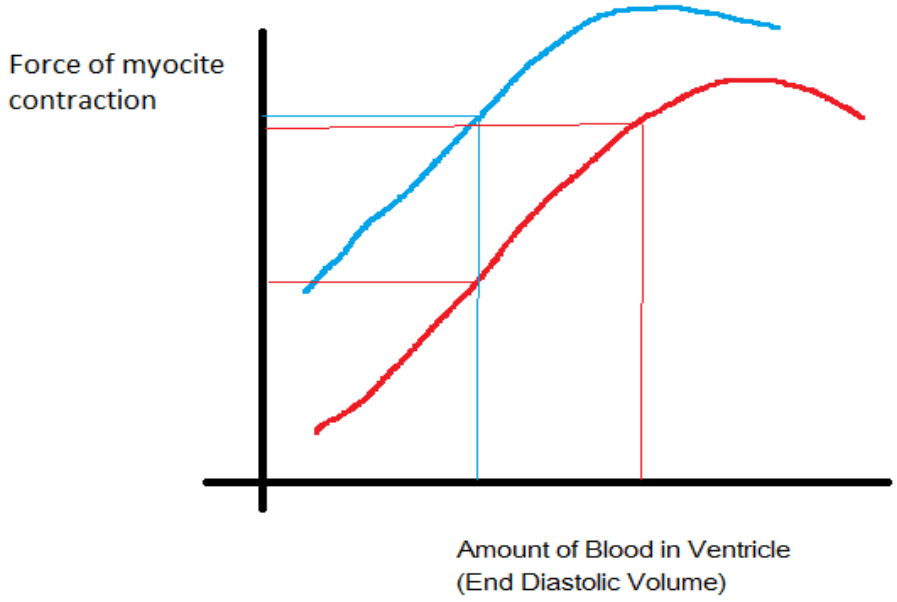
Stroke volume depends on 3 factors: Preload (Pl), Contractility (Co) and Afterload (Al).

SV α Pl x Co x Al

The healthy heart is able to adapt its contractility to the higher or lower forces needed, according to whether SVR goes up or down, but an ischemic heart with a damaged left ventricle might not be able to, leading to pulmonary edema and cardiogenic shock.

Preload, simplified, is the venous pressure that fills the ventricles, assisted by the atrial kicks. It mostly depends on the amount of blood in the venous system and the venous tone, assuming the atrial and working in concert with the ventricles (which sometimes fails, mostly in Afib)

Contractility, simplified, is the force with which the heart pumps the blood. Contractility has two control mechanisms- a passive one where myocyctes will generate more contractile force the more they are stretched (up to a point), and an active one, where BETA1 receptor stimulation results in more contraction force for a given stretch. This is neatly represented by the Frank Starling curves below. BETA1 stimulation moves the curve to the left.



The red line shows a regular contractility, not aided by BETA1 stimulation. Note how the force of contraction rises with volume (up to the point of failure where the curve starts to go back down again. In impaired hearts (low ejection fraction), that point happens sooner than in healthy hearts. Blue line shows what happens when we add BETA1 stimulation- we get more force for the same stretch.

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Afterload is the pressure against which the heart must pump. Normally, afterload is determined by the diameter of the aorta immediately distal to the left ventricle- the more squeezed the aorta is, the more heart has to work. The aorta is more squeezed when ALPHA receptors are stimulated, IE when SVR is higher. Recall that higher SVR means higher blood pressure. But, as seen here, it also means higher workload for the heart. As we will see later, this has implications in cardiogenic shock.

Other conditions can alter the afterload- the commonest one is aortic stenosis. Now, instead of having to pump through a relatively wide aorta, the ventricle has to pump against a very narrow, inflexible opening of the stenosed valve.

The 4 Types of shock

Rapid Infusion Tips

If you leave an infusion through a small IV and just gravity fed, it will take about 10-15 min to infuse a liter of fluid. That can be a very long time when a patient is crashing. Worse, some people think a 'bolus' means putting the infusion through a computerized pump and setting it to "999" which will infuse 999cc over an hour. This is wholly inadequate for resuscitation

Now that we know the 5 factors that influence blood pressure (SVR, HR, Pl, Co, Al), we can look at different types of shock and understand what we need to correct in order to improve things. As mentioned, shock is tissue hypoperfusion, i.e. absence of FLOW. But, as we cannot directly measure flow, we can simplify things and define shock as *low blood pressure, or a clinical picture suggesting impending drop in blood pressure* (think of a febrile, mottling, tachycardic sepsis patient).

There are 4 main types of shock, though it is an imperfect division where many different etiologies are lumped together.

***Hypovolemic*:** simplest, and commonest, type of shock, resulting from low amount of blood in the system. The problem here is low PRELOAD and the solution is 'filling the tank' as fast as possible. To do so we need short, large diameter IV lines (14 or 16 GA) and a PRESSURIZED delivery system.

The table below will illustrate why both a large bore needle AND a pressure system are needed to achieve high flow rates. Good pressure systems are either a pressure bag or a person squeezing the bag with both hands.

	No pressure	**Pressure**
14GA	~250cc/min	~400cc/min
16 GA	~150cc/min	~350cc/min
18 GA	~70cc/min	~100cc/min

From Reddick et al 2011

If you need a temporizing measure while you are filling the tank, ALPHA stimulation will help.

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If the fluid that has been lost is blood, you need to replace it with blood, not crystalloid. As a general rule, if the person has lost enough blood to have persistently abnormal vitals (ie both a low blood pressure AND a tachycardia) they have lost at least 40% of their circulating blood volume and likely will require a massive transfusion in the course of next 24 hours, ie at least 8 units of PRBCs, and accompanying units of Fresh Frozen Plasma and platelets to make the infusate resemble whole blood (this is known as 1:1:1 transfusion, ie equal ratios of PRBC, FFP and platelets).

PRBCs come in 3 general varieties

O+/O- : immediately available and appropriate for everyone but limited in supply. Use O- ve for women of child bearing age, everyone else gets O+

Type specific: matched for ABO/Rh but not minor antibodies. This is what you get when you order a Type and Screen from the blood bank (Type is ABO, Screen is Rh). Can be available within 5-10 min

Crossmatched: matched for ABO/Rh as well as the minor antibodies (of which there are hundreds). Takes about an hour

Fresh Frozen Plasma (FFP) is frozen (doh!) and will need 25-45 min to be thawed

Platelets may or may not be available, depending on the size of the hospital

In practical terms, if you see a bleeding patient with persistently high heart rate and low blood pressure (and no other cause like pain, fever, etc), give O+/O- blood, ask for a Type and Screen so you can continue infusing PRBCs and trigger the massive transfusion protocol so Crossmatched blood, FFP and possibly platelets can become available by the time you need them. There is a 45min-1hr lag from the time you pull this trigger to the time units actually become available, so pull it early.

***Distributive*:** the second commonest type of shock. It occurs when sufficient amount of blood is available but it is improperly distributed because the vascular tree is inappropriately relaxed and the blood is thus not flowing well to end arterioles. Causes are many, including sepsis, anaphylaxis, neurogenic and adrenal. The problem here is low SVR leading to poor return flow and low PRELOAD. Thus, filling the tank is beneficial, in conjunction with stimulating ALPHA receptors to increase the SVR.

***Cardiogenic*:**

1) When the heart's contractility is insufficient to overcome afterload. In other words, the strength of the pump is inadequate, leading to poor cardiac output forward (towards the aorta) and thus low blood pressure; and backing up of fluid in the pulmonary circulation, leading to pulmonary edema. The problem here is contractility so we

Cardiogenic shock most commonly happens with big left sided STEMIs that kill or stun enough of the myocardium that the left ventricle becomes weak

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have to stimulate the BETA1 receptors to increase it. Adding a lot of fluid would potentially make things worse as it would only lead to more pulmonary congestion. *This is the ONLY time when adding a lot of fluid would make a shock worse*. 500cc to 1L)as long as there are no signs of active CHF) is usually ok as it helps boost the preload and thus help stretch myocites and passively increase contractility (look at the Starling curve in the previous section for illustration why).

2) When the heart rate is low enough (for example with a heart block) that it leads to inadequate cardiac output, despite normal contractility. We will deal with this in the arrhythmia section.

3) When an arrythmia causes the heart rate to become too fast for the age, leading to inadequate diastolic filling time. We will also deal with this in the arrhythmia section.

Obstructive shock: happens when another process blocks the flow of blood to the heart. There are three common causes of this: Tension pneumothorax, cardiac tamponade and massive pulmonary embolus:

Tension Pneumothorax and Tamponade increase the pressure surrounding the heart, increasing the afterload. This will eventually decrease the preload as the pressure surrounding the heart becomes higher than the venous filling pressure. This we can help by 'filling the tank', i.e. giving fluids, in order to keep the venous pressure as high as possible and give ALPHA stimulants to increase venous tone. Eventually, the surrounding pressure will be too much for the heart to pump against, and we can help that by increasing contractility via stimulation of BETA1 receptors. Of course, the definitive treatment is actually relieving the tamponade or the pneumothroax.

Pulmonary embolus prevents the blood flowing from the right to the left side of the heart. Again, increasing the venous pressure by giving fluid and stimulating contractility of the right ride of the heart (BETA1 receptors) will help things.

SHOCK - SUMMARY

	Problem	**To fix**
Hypovolemic (common)	Low Preload	Fluid Possibly blood ALPHA
Distributive (common)	Low SVR	Fluid ALPHA
Cardiogenic (not common)	Low Contractility	A bit of fluid BETA1
Obstructive (rare)	High Afterload	Fluid ALPHA BETA

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Medications For Shock

Drugs that stimulate ALPHA receptors and ***increase SVR*** are called VASOPRESSORS ('pressors').
Drugs that stimulate ***heart rate only***, though blocking of the vagal tone (eg Atropine) are called POSITIVE CHRONOTROPES.
Drugs that stimulate ***both heart rate and contractility*** (IE Beta1stimulants) are called POSITIVE CHRONOTROPES AND INOTROPES.

Don't have a Central Line?
There is a great reluctance to give these drugs through a peripheral IV. In reality, you can safely give any of these meds through a peripheral IV for at least 6 hours and as long as you make sure that the IV is indeed in a vein and not interstitial. If you were to give a VASOPRESSOR interstitially, it would lead to local vasoconstriction and potential tissue death because of that. Even then, there is a way to counteract it (read about Phentolamine rescue, if you have the time and inclination). ***If a person needs a pressor to survive, please do not hold back just because you don't have a central line.***

How to use Pressors - For all pressors, the safest way to use them is to start at the lowest dose and then rapidly titrate to blood pressure. **MAP (mean arterial pressure) of 65 is a reasonable target BP** in all types of shock.

Mean Arterial Pressure
MAP is equal to 1/3 of systolic pressure and 2/3 of diastolic pressure and is generally displayed by monitors in brackets (ie BP 120/75 (90)).

In this course, we focus on two pressors: ***Dopamine*** *and* ***Phenylephrine***. The reasons are that Dopamine is a broad-spectrum pressor/inotrope/chonotrope that is valid in all types of shock and also comes premixed and is present on all crash carts. Phenylephrine is a drug that is also easy to use and can be used in quick boluses, giving it great versatility when you need temporary or continuous blood pressure support for most types of shock.

Dopamine*:* is a mixed-bag drug in the sense that at lower doses (less then 10/mcg/kg/min) it is predominantly a BETA1 stimulant, i.e. chronotrope and inotrope. At higher doses (above 10/mcg/kg/min) it starts having an ALPHA effect in addition to BETA1. ICU people generally don't like it because of this, as they are not sure what effect they are getting, but it is a perfectly reasonable drug to use, especially when you are not sure what is going on as it stimulates everything. Just beware that, due to its BETA1 stimulation, it does produce quite a lot of tachycardia. It is present on all crash carts, so it might be a viable choice as an initial pressor, especially on floor resuscitations where other drugs might not be immediately available. It has inferior outcomes with prolonged use in septic and cardiogenic shock to NorEpinephrine, so the patients should eventually end up on NorEpi.

Phenylephrine*:* is a pure ALPHA stimulant, i.e. a pure vasopressor, with minimal effect on heart rate or contractility. Inject 1-3 cc at a time (100-300mcg) every few minutes. If you want to give it as an infusion, hang the bag you just mixed (100

Phenylephrine Mix
Phenylephrine requires premixing to be done by you, but luckily it is dead simple. It comes in 10mg vials. Take the 10mg in a syringe and inject into a 100cc bag of saline. Shake, not stir. Now you have made a concentration of 100 **mcg**/cc.

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mcg/cc) and let it run at 100-300 mcg/min (1-3cc/min). It can lead to some bradycardia, so don't give it if the patient has a slow heart rate and it is not helpful in cardiogenic shock as it does not stimulate the heart rate or the contractility.

Nor epinephrine: Probably the most-used pressor in the ICU, due to studies suggest it has superior outcomes to other pressors in cardiogenic and septic shock. It is mostly an ALPHA stimulant, i.e. a pressor, but it has some positive INOTROPIC effect as well (increases contractility). Good to use in all shock settings. The usual dose is 2-15 mcg/min (*NOTE: this is NOT mcg/kg/min, like Dopamine, but rather* ***mcg/min***. The per-kilo dose of NorEpi is much lower). It is not premixed like Dopamine, and can't be given in resident-controlled boluses like Phenylephrine. While NorEpi is the pressor that most of the shocky patients will end up down the line, we don't emphasize it in this course due to above mentioned-logistical challenges, as well as floor nurses' lack of familiarity with it and thus reluctance to administer it outside of central line/ICU setting. That said, if you do have the capability to use, it should be your pressor of choice in all types of shock.

Epinpehrine: Strong agonist of both ALPHA and BETA1 receptors. Easily mixed by taking one 1mg amp of Epinephrine (either 1:1000 or 1:10,000) and injecting it into a 1L bag of normal saline, giving a 1mcg/cc concentration. Given in boluses of 2-5cc or infusion at 2-10cc/min

DoBUTamine*:* DoBUTamine is a strong B1 stimulant (positive chronotrope and inotrope) but also a vasodilator. It is commonly used in ICU for cardiogenic shock. It is only mentioned here to distinguish it from Dopamine, to which it has no relation. We will not be using DoBUTamine in this course.

Summary of Medications For Shock

	SVR	HR	Contractility	Types of shock	Dose
Dopamine	Low dose: + High Dose: ++	++ +++	+++ +++	Any	5-10 mcg/**kg**/min (low) ↓ 10-20 mcg/**kg**/min (high)
Nor epinephrine	+++	0	+	Any	2-15 mcg/min
Phenylephrine	++++	0/-	0	Any except cardiogenic	100-300 mcg/min
Epinephrine	+++	+++	+++	Any	2-10 mcg/min

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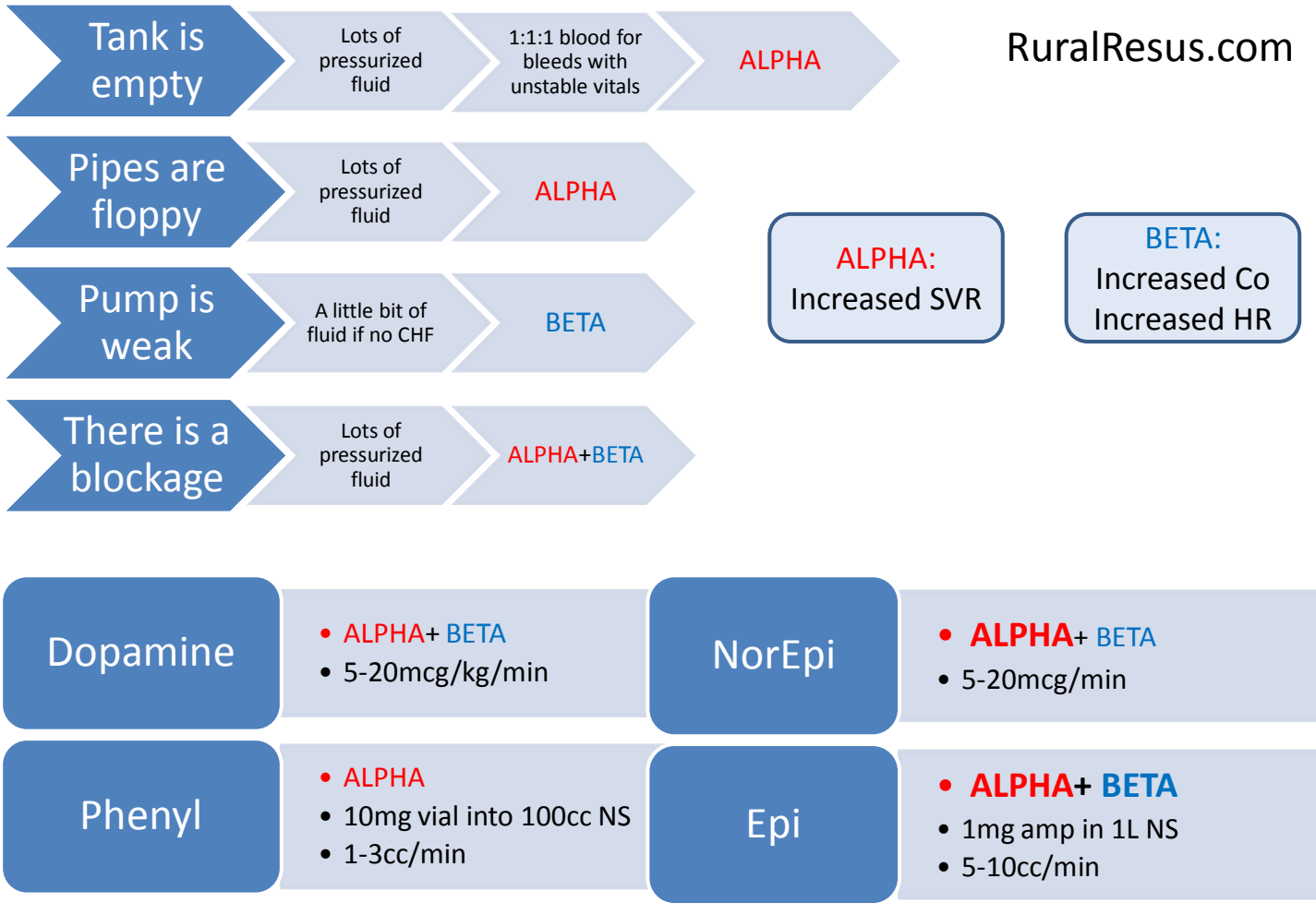
If all this sounds too complicated, remember this:

In hypotensive shock, the problem is an empty tank (low blood volume), in distributive it is floppy pipes (low SVR), *and these two account for most of the shock you will see*. They will respond to fluid/blood and ALPHA stimulation (Dopamine or Phenylephrine).

In cardiogenic shock, the problem is a faulty pump (low cardiac output) either due to low heart rate or low contractility; it gets fixed with a bit of fluid, BETA1 stimulation (Dopamine but NOT Phenylephrine) and can be made worse by excessive fluid administration.

In obstructive shock the cardiac output becomes low because of outside obstruction, and can be improved by fluid, ALPHA and BETA1 stimulation until the obstruction is relieved.



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Arrythmias

Increased heart rate

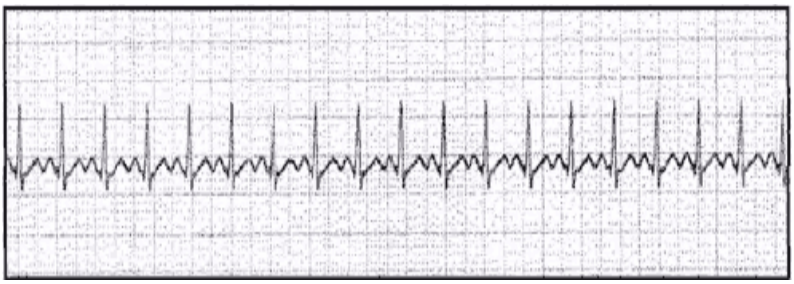
0th step. ABCs

As in all resuscitation scenarios, ABCs rule. Get the patient IV-O2-monitor, a full set of vitals and ensure that the airway is patent.

1st step. Sinus or else?

Sinus tachycardia (or sinus equivalent) or something else? Spend a few minutes deciding if this is what you are seeing- if it is, the fast heart rate is a reaction to something (pain, fever, blood loss, fear, cocaine, etc) and NEVER the cause of the patient's instability.

What is Sinus?
Sinus is defined as ***a p wave before every QRS and a QRS after every p wave, with p wave being positive in leads I and II***. The pacemaking rhythm is coming from the SA node and is being conducted appropriately by the AV node and the His-Purkinje system. Note that the rhythm won't necessarily be perfectly regular, sinus arrhythmia is quite common, especially in young women



Sinus tachycardia: not p before QRS and QRS after every p

Distinguishing Sinus
It is common not to be sure whether a fast rhythm is a sinus or something else, like an SVT or Atrial Flutter. There are a few tricks that can help you in such a situation. First, remember that there is a maximum sinus rate for age (220-age for men, 210-age for women). If the rate is higher than that number (say a rate of 160 in a 70 year old woman), it is likely not sinus. Second, sinus rate tends to have quite a bit of variability with breathing, movement, talking, etc. SVTs and flutter tend to be quite fixed with very little variability. Finally, sinus tachycardia might slow down with treatments such as fluids, pain control, antipyretics and anxiolytics. If what you are seeing is not sinus, or you are not sure, proceed down the subsequent steps. If it is sinus, find and treat the underlying cause.

Adenosine
You can use Adenosine to help you slow down the heart rate transiently so you can see better- distinguishing features are much easier to see at a rate of 100 then at 160. Have a nurse attach the leads for an ECG and be ready to take it. Inject 6mg of Adenosine and as soon as the heart rate slows down, take the ECG. *This is safe in all narrow complex tachycardias*

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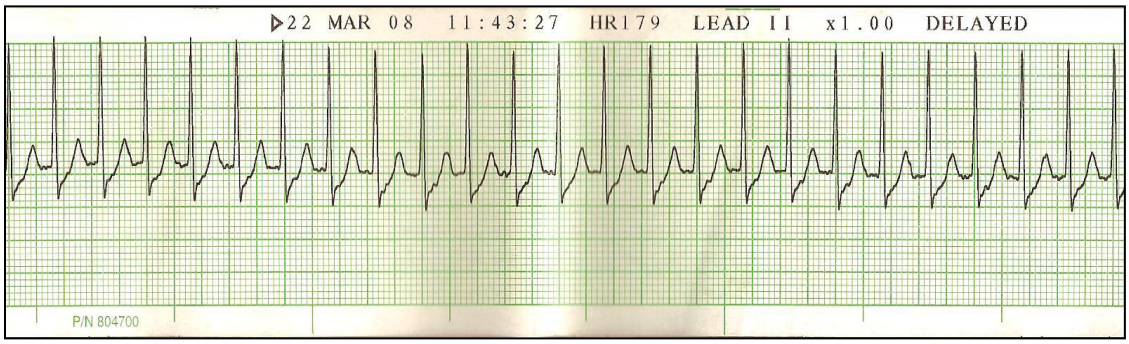

"Something else". In this case SVT. *Note the lack of p waves.*

2nd step. Stable or unstable?

Is the patient stable or unstable?

Instability simply means that the patient is not getting enough blood flow and oxygen to the vital organs, mostly the brain and the heart. A little old lady in chronic A fib who comes with a pressure of 85/55 but is comfortably talking to you and is symptoms-free except for some palpitations is not unstable

The patient is unstable if they

1. Have a low BP AND are confused
2. Have angina type chest pains (not just palpitations) or shortness of breath

Unstable?

The answer to instability, no matter what kind of* arrhythmia *is causing it, is always electricity in the form of synchronized cardioversion. Please do not try to cardiovert sinus tachycardia

Cardioversion is almost the same thing as defibrillation, i.e. a sudden application of large amounts of electricity in order to reset the heart's electrical system, but with one crucial distinction-it is synchronized to the heart's polarization cycles in order to avoid applying the electricity while the heart is repolarizing (IE on a T wave), which could induce Ventricular Fibrillation and kill the patient. As long as you remember to synchronize (IE press the synch button on Lifepack 12), it is perfectly safe to do. The dose should be 150J or above to ensure maximum efficacy.

It is a very painful procedure, so be kind and give the patient some pain medications and sedation before you do it, if you have the time and they can afford teh BP drop that comes with such meds.

3rd step. Narrow or wide?

If the patient is stable, we get to think a bit. We don't have to apply immediate electricity, but we have to decide which chemical (i.e. drugs) treatment we are going to use. The first step is

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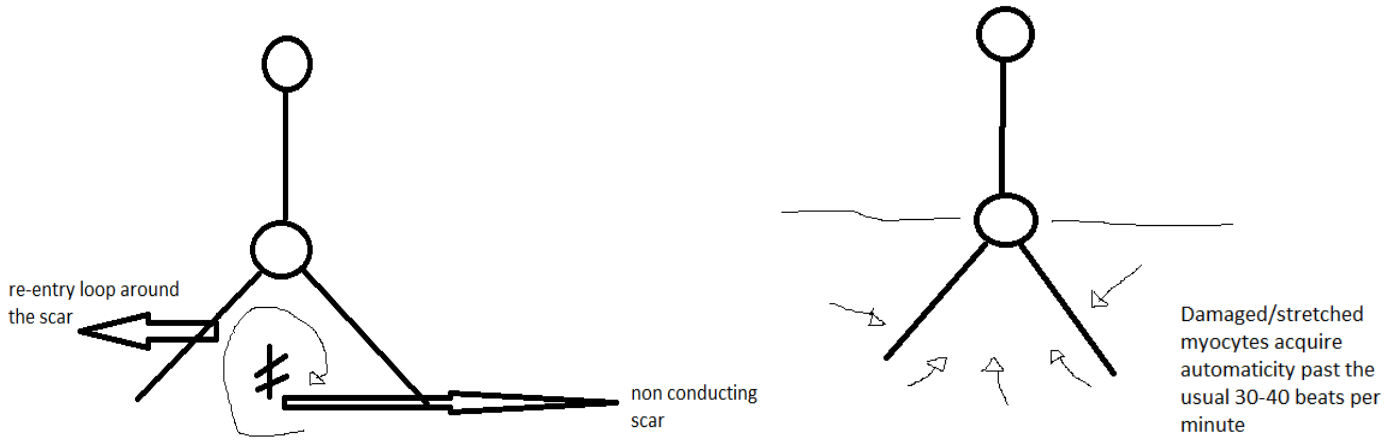
to decide whether the QRS is narrow (<0.12 sec or 3 small squares) or wide (more then 0.12sec). There are four major reasons why a QRS would be wide, as detailed below.

1. The **rhythms are not originating in the SA-AV system, but below it**, i.e. somewhere in the ventricle. This is Ventricular Tachycardia (VT). It is wide because it does not use the His-Purkinje system but conducts cell to cell. We mostly see this soon after heart attacks, caused by a re-entry loop focus around a non-conducting scar, or by damaged ischemic myocytes firing rapidly on their own. VT, even with a pulse, can be lethal.



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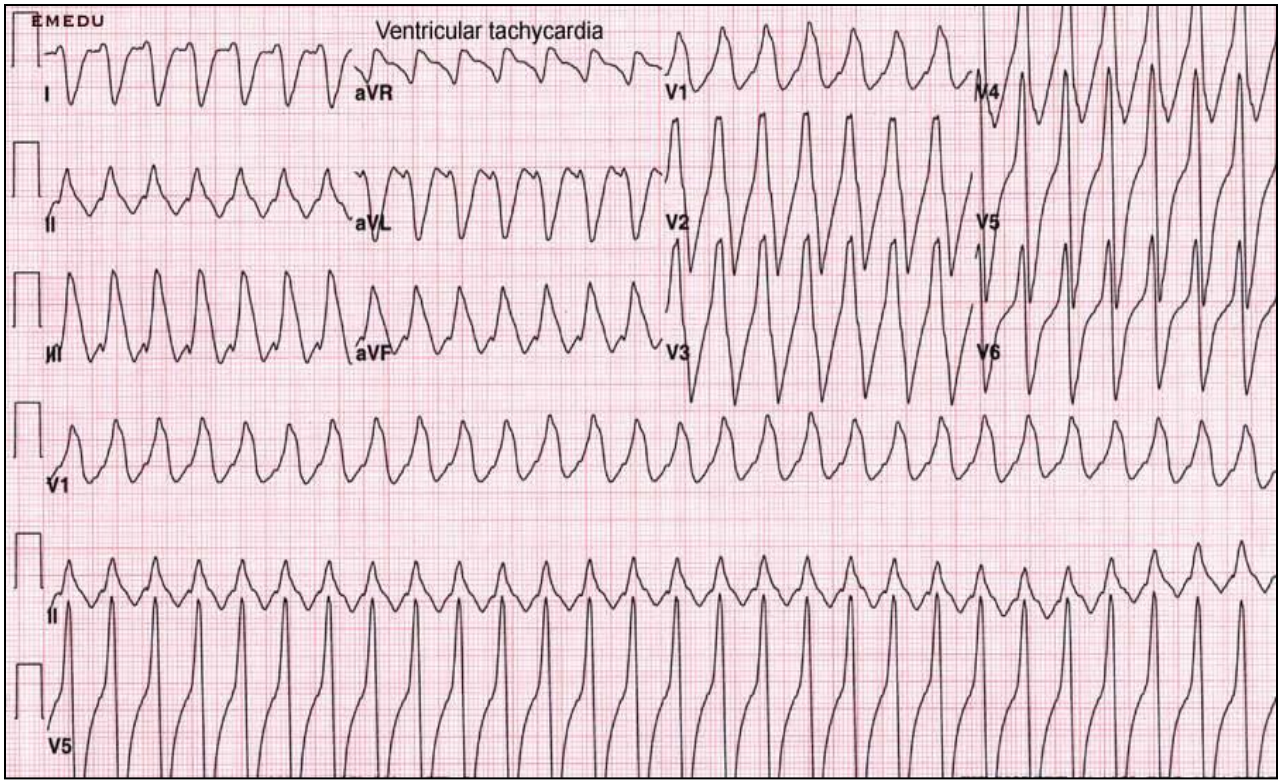

VTach – note wide complex, i.e. cell-to-cell conduction

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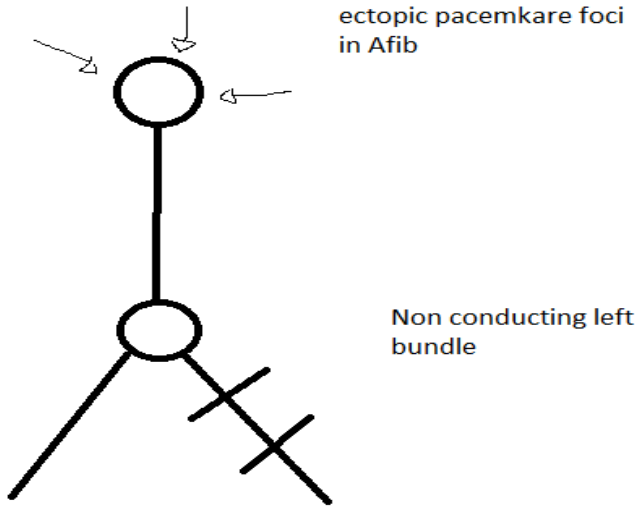
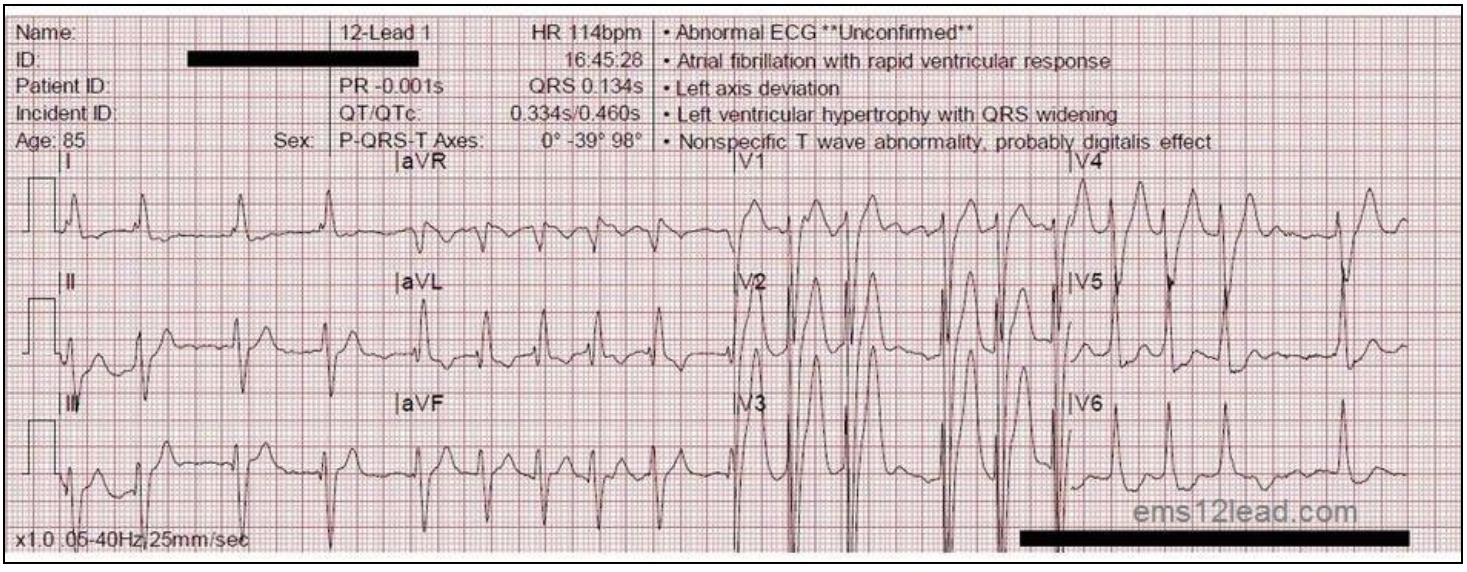
2. The **left bundle of His is blocked**, giving a wide QRS as the impulse from the SA-AV system reaches the ventricle but then has to travel slowly cell-to-cell. This is by far the commonest reason for wide QRS outside of cardiology offices and EPS labs. If this person gets a tachycardic response (fever, pain, etc) or acquires A fib or another arrhythmia, they will end up with a wide QRS tachycardia. In this case, IGNORE the LBBB and treat the underlying rhythm. You might have heard that a new LBBB with chest pain is a STEMI equivalent. It is not.



Left Bundle Branch Block

The rhythm has the characteristics of the left bundle branch block, namely,

1. Positive (upright) QRS in lateral leads (I, aVL, V5-6) and negative (down) in anterior (V1-4)
2. Discordant QRS-ST segments (i.e. where the QRS is positive, ST is negative, and vice versa)
3. Little bunny ears on top of the wide QRS



EKG of Afib with LBBB. Note left axis deviation, QRS-ST discordance and bunny ears in aVL

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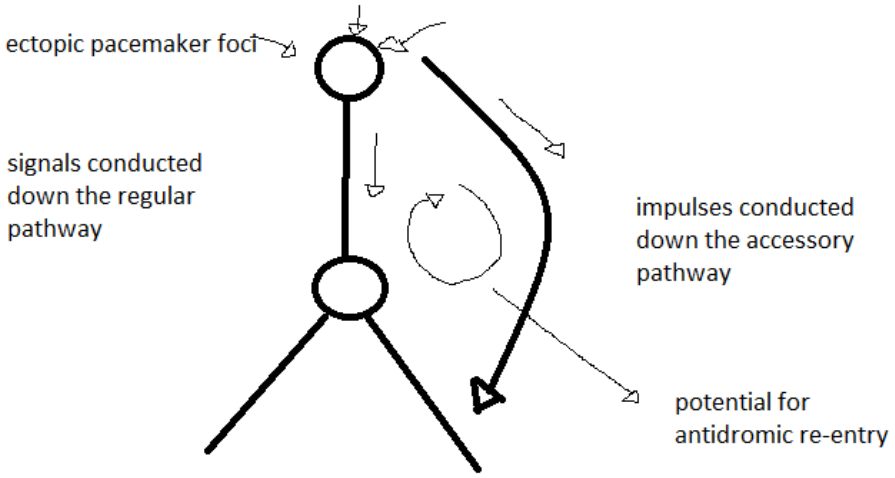
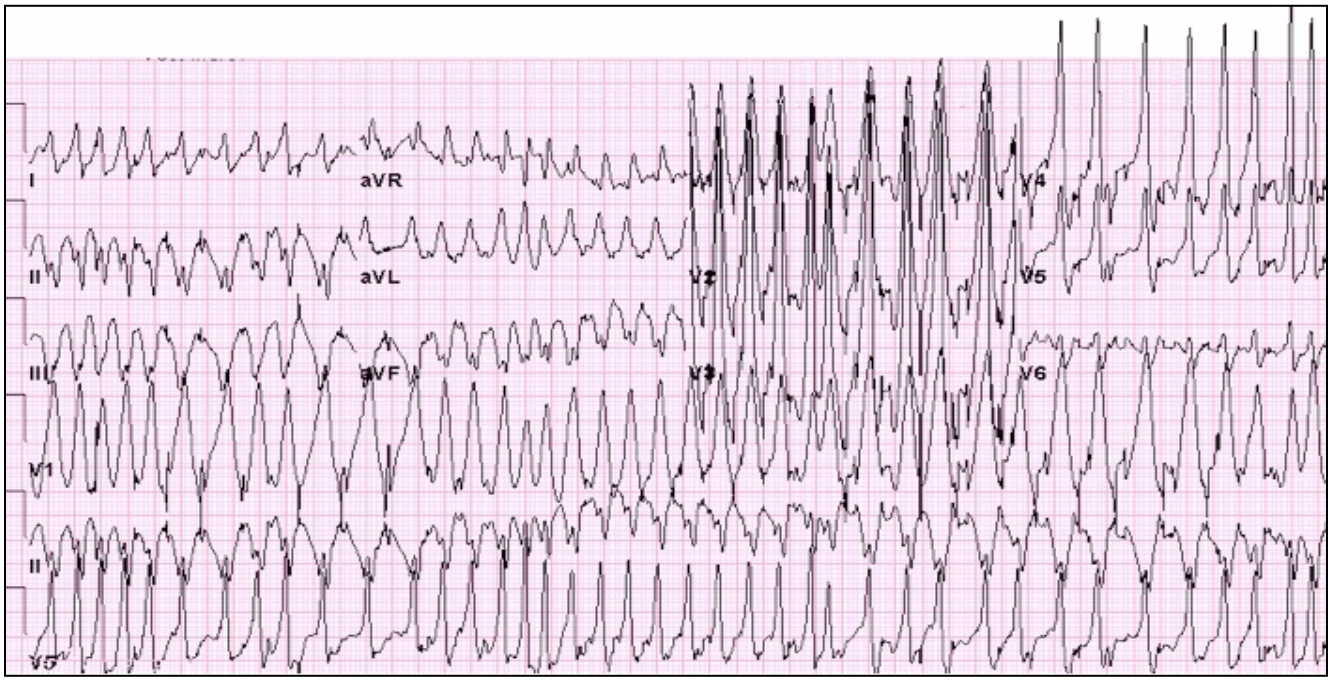
and I

3. **The person has an accessory pathway** (IE WPW) which is capable of conducting anterograde (IE from the top to the bottom) AND the person has acquired another arrhythmia (mostly A fib). We now have a situation where the ectopic pacemakers in the atria are sending signals downstream which can be conducted down the accessory pathway rather then the SA-AV system. Since they are not using the His-Purkinje system, they are conducting cell to cell and the QRS will be wide.



WPW and AV nodal agents

If we block the AV node here, all the atrial impulses will go down the accessory pathway and since there is no brake in that system, we can end up with a ventricular rate of 300-600 which is unsustainable and will lead to Vfib and death. This is why we don't use AVN blockers in wide QRS tachycardias (assuming they are not just a LBBB)



If it looks like dog's breakfast, its likely Afib with WPW!

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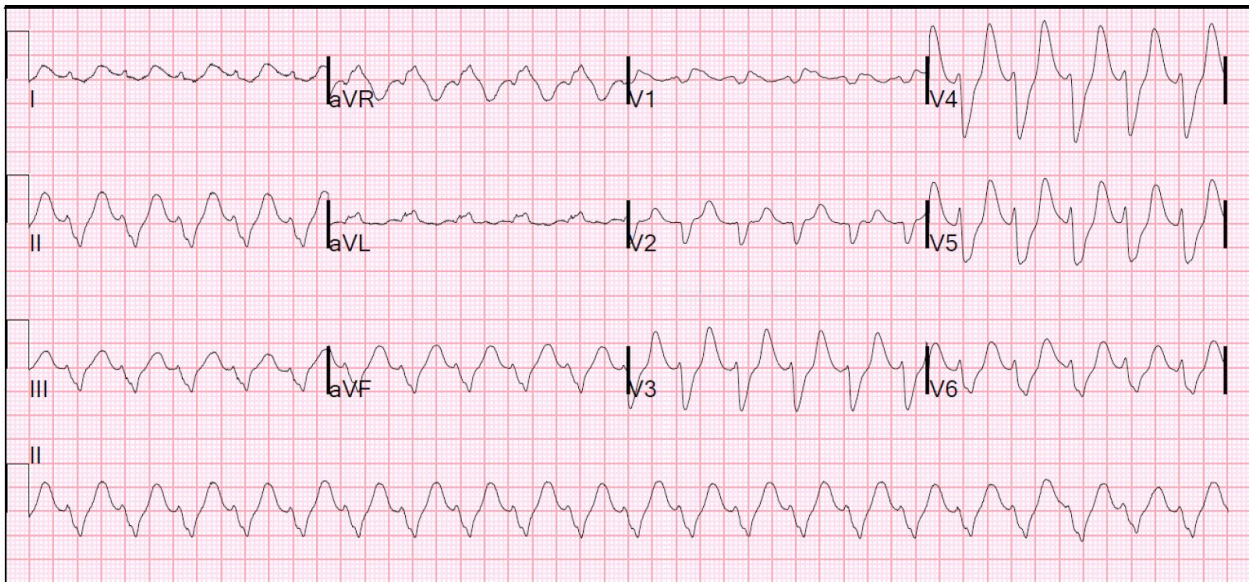
4. The final reason for a wide QRS is the poisoning of the sodium potassium pump by either a sodium channel blocker (or an associated drug like a TCA) or through hyperkalemia. Those patients will eventually end up bradycardic, but they might initially present with a wide QRS tachycardia. Poisonings are beyond the scope of this course and we will cover hyperkalemia in the bradycardia sections.



Hyperkalemia

How do I work out which of the above applies?

It can be quite tricky to figure out definitely which of the three scenarios (we assume no poisoning is present) is causing a fast-and-wide rate. Luckily, it is also not necessary. Leave that to the highly-paid cardiologists. There are two principles to remember

Which Drug to Use?
Amiodarone was in fashion for years, but it has been shown that ***Procainamide (1g given over 1 hr) is safer*** for both VT and WPW with Afib

1. If it is LBBB, ignore the LBBB and look at the underlying rhythm
2. If it is not LBBB, we should avoid giving AV node-specific blockers (i.e. Beta blockers and non DHP Calcium channel blockers) and use a more globally acting conduction blocker, such as Procainamide.

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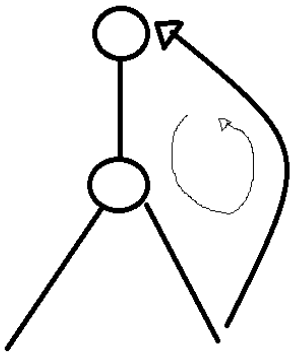
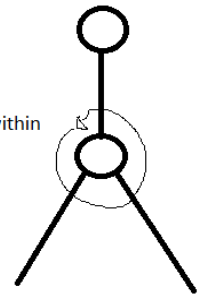
Step 4. Regular or irregular?

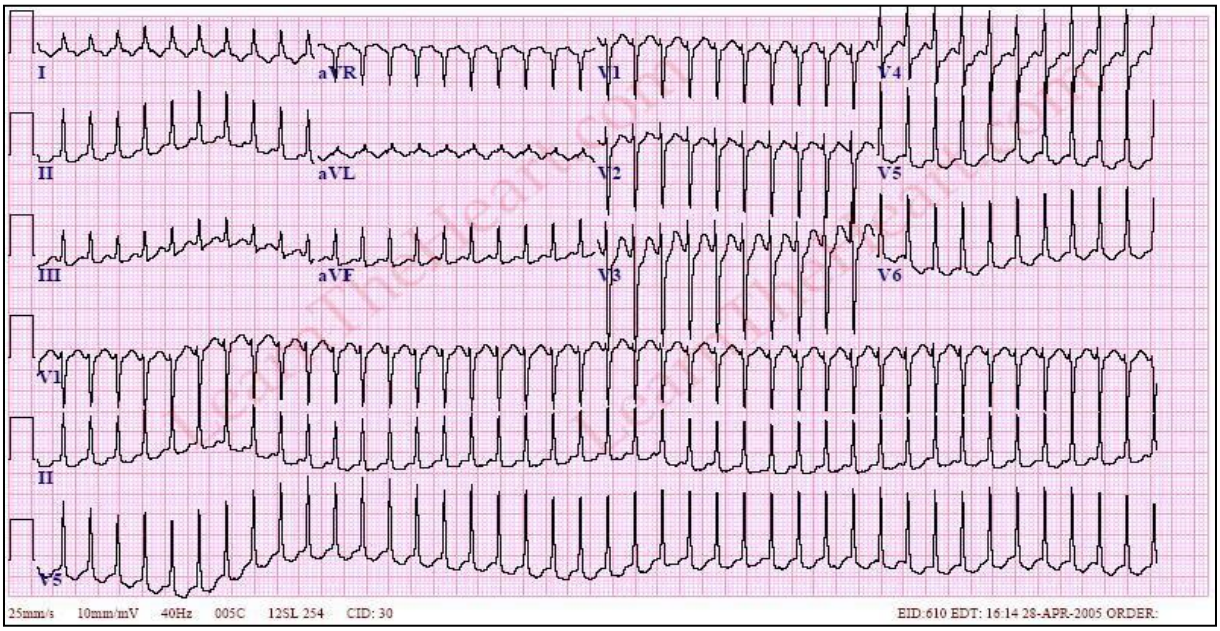
If the QRS is narrow, the final step is to decide whether it is regular or irregular. If it is regular, we assume it is a Supra Ventricular tachycardia (SVT). If it is irregular, we assume it is A fib. SVT is a catch-all phrase to indicate any tachycardia that is not a VT. Sinus tachycardia, A fib and Aflutter are all technically SVTs. In common usage, when we say SVT, we mean AV Node Reentry Tachycardia (AVNRT) and AV Reentry tachycardia (AVRT). As the name suggests, they both are caused by re-entry loops, and the difference is simply in location of those loops. They look very similar and, again, it is not really necessary to distinguish them as they will respond to the same treatment.



AVRT: re-entry loop using an retrograde (upwards) conducting accesory pathway

AVNRT-reentry within the AV node





AVRT

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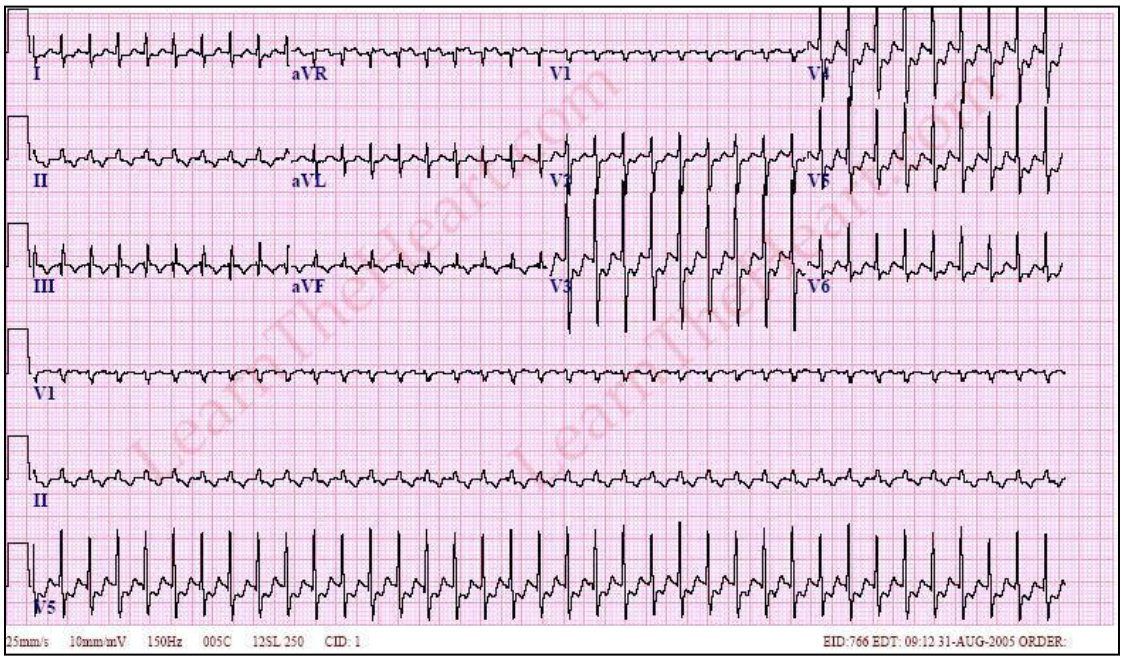

AVNRT

It is a rare occurrence, in a person with an accessory pathway, that the conditions will align that allow a reentry loop to exist and persist. Thus, if we disrupt the loop for even a couple of seconds, we should be able to get our normal conduction back. To that end, we will need to block the conduction through the AV node for a few seconds or minutes to terminate the re-entry. We got two basic options

If none of those are working, it might be time for an elective cardioversion. Since AVNRT/AVRT do not put the patient at the risk of embolic stroke, **cardioversion is essentially always safe to do with an SVT**. Remember pain control and sedation before you do that.

Diltiazem 10-25mg slow infusion over 10min. Works close to 100% of the time and is painless.

Adenosine 6 or 12mg IV. Works very quickly but makes the patient feel awful.

Because it is painless, Diltiazem is probably the superior option.

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AFib – Rhythm or Rate Control?
If the duration of this episode is less than 48 hrs AND all the other episodes have been less than 48 hrs AND the patient is reliable and feels the palpitations every time the A fib comes on (as opposed to the usual story of, " I felt a little funny for a couple of days now, not sure when it started") THEN it is safe to use rhythm control. Note that the risk of stroke is the same whether you use chemical cardioversion (Amiodarone, Procainamide) or electrical cardioversion.

If the narrow QRS rhythm is irregular, you are most likely dealing with **A fib.** Afib is caused by more permanent arrhythmic foci, so a short acting agent like Adenosine will not work in the long run.

If it looks like A fib, because of the potential for embolic stroke, you have to make a decision whether it is safe to use **rhythm control**, or is **rate control** your only option. This has to do with the duration of the A fib (more, or less then 2 days)

The other safe time to cardiovert is in a patient who has been effectively anticoagulated for at least three weeks before the cardioversion and two weeks after (because some strokes happen after cardioversion) or have had a transesophageal echo (not a regular, transthoracic echo) to rule out a clot.

If it is safe to use Rhythm control (<48h), we can accomplish it chemically or electrically. If there are no signs of ischemia or CHF (in which case cardioversion is probably best), we use Procainamide. It is effective around 50% of that time, and within the hour, so if the infusion is done and they are still Afibbing, it might be time to electrically control them, i.e. to cardiovert them. The electricity dose is the same as for SVT or instability- 150-200J.

For **rate control**, the most useful medications is again Diltiazem with close to twice the success rate of Metoprolol

Resting heart rate of **<110** is adequate. If repeated doses of Diltiazem didn't work, you can cautiously try Metoprolol but beware: if they spontaneously convert, they might be quite slow with beta blockers and CCBs on board.

When to Stop?
If none of this is working and they are still symptomatic or above 110, consider sending the patient to Internal Medicine for an admission and, usually, IV Digoxin loading.

CARDIAC MEDICATIONS

Adenosine- very short acting AVN blocker. Acts within seconds and gone within seconds, needs to be given as a sudden push followed by a 10-20cc saline bolus. Safe for anything **<u>except</u> wide** and **irregular** rhythms. Makes the patient feel something awful, although for only 20-30 seconds.

Diltiazem- non-DHP Ca channel blocker that acts mostly on the heart Ca channel receptors (unlike the DHP ones like amlodipine which acts mostly peripherally). Has good evidence of efficacy in Afib for rate control (96% success rate) if given in 2-3 boluses of 15-25mg IV, slow pushed over 1-2 minutes and spaced about 10 min apart. Reaches peak action in ~2 minutes so can judge quickly if it will work or not. If it works, can put the patient on infusion of 10-15mg/hr. Safe for any narrow QRS tachyarrythmia. Can also be used in SVTs with near 100% efficacy with the same dose.

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Metoprolol- Beta blocker. Used in same circumstances as Diltiazem but has much less efficacious in Afib/flutter acute rate control (~50% success rate vs 95% with Diltiazem) and peaks in 20 minutes so need to wait longer to see if it works. Dose is 2.5-5 mg q5-10 min, max of 15mg, all given as slow bolus.

Procainamide- an old Na channel blocking drug that is enjoying a bit of a revival. Safe in wide and irregular rhythms. Good efficacy for Afib conversion and much faster then Amiodarone (55 minutes mean time to conversion versus 6 hrs for Amiodarone). Given as 1gr over 1hr. Is a bit of a vasodilator, so if you get hypotension, give a 250cc bolus and slow down the infusion to half-rate. If that fixes the BP, continue at half-rate until the gram is given. The only time Procainamide is not safe is with CHF/pulmonary edema.

Amiodarone- a complex drug that does a bit of everything (Na channel blockade, Ca channel blockade, Beta receptor blockade), but mostly prolongs repolarization of the cells. It is very much in fashion with the AHA and is the mainstay of the ACLS arrythmias protocols. There is some evidence that it less safe than Procainamide in Torsades de Pointe, VT and WPW with Afib (most likely because of it partial beta blocking capabilities) so the only time you might use it on a live person is if there is presence of acute CHF or the patient can't tolerate Procainamide's vasodilation. Dose for living people is 150mg infused over 10-20 minutes, to be followed by slow infusion of 850mg over 24 hrs (nurses have this protocol in their books). Has decent evidence of efficacy with converting Afib to sinus but the mean time to conversion is 5-6 hours so not very useful in acute settings. Dose for dead people is 300mg IV rapid bolus.

	Mechanism	**Dose**	**Effective in**
Cardioversion	Electricity	100-200J	any
Amiodarone	Multiple	150mg over 10-20min	any >48h except wide and irregular
Adenosine	Intense AVN blocker	6 or 12mg rapid push	Potentially diagnostic in A fib/flutter Curative with SVT
Metoprolol	AVN blocker (Beta 1)	2.5-5 mg IV q10-15 min X3	Avoid in wide QRS Effective any narrow QRS
Diltiazem	AVN blocker (Ca channels)	20-35mg IV bolus, 10-15 mg/hr infusion	Avoid in wide QRS Effective any narrow QRS
Procainamide	Na channel blockade	1g over 1 hr	Any fast arrythmia <48 h

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If all this sounds too much, remember these basic principles:

Spend a minute deciding if fast heart rate is sinus- if it is, it is a reaction to something else and you should try and figure our what it is. *Cardioversion or antiarrythmics will not help you with sinus tach*. You can use Adenosine to help you determine this as long as the rhythm is **not** wide and irregular.

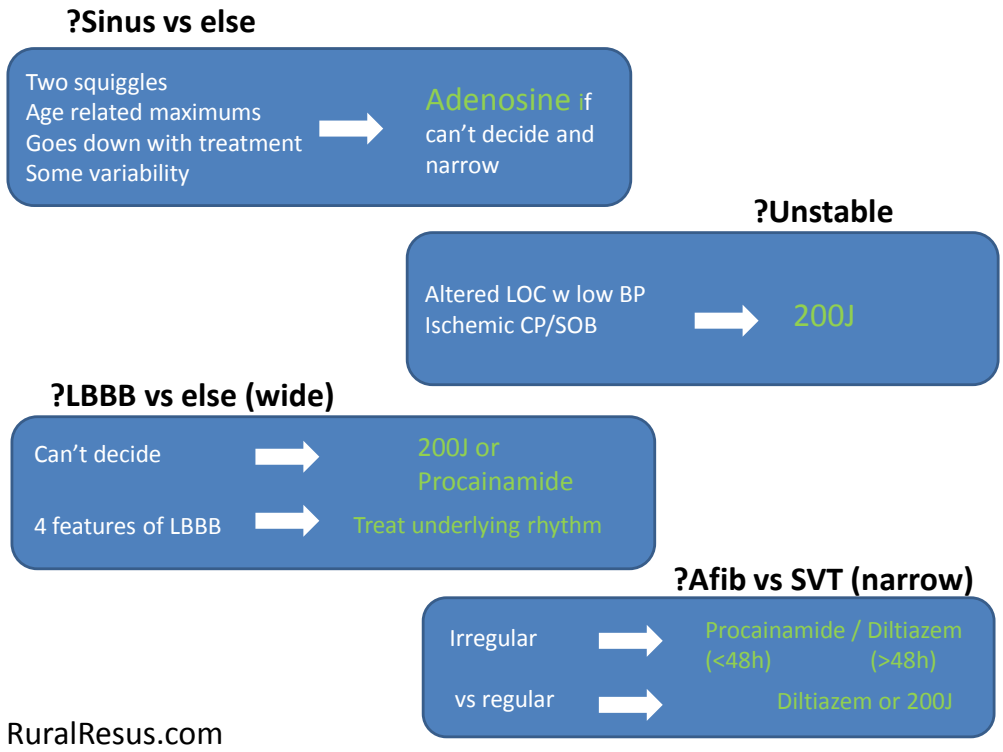
If it is not sinus or you are unsure AND the patient has low perfusion of brain or the heart- cardiovert them.

If you are seeing wide QRS complexes, and are sure it is a LBBB, just treat the underlying rhythm as usual.

If you are not sure, or it is for sure not a LBBB, avoid AV nodal blockers like Beta blockers and Ca channel blockers. Use Procainamide or elective cardioversion instead.

If the arrhythmia is narrow and regular, use Diltiazem or cardivoersion

If it is narrow and irregular, use Procainamide or cardioversion if less then 48hrs, Diltiazem if more then 48hr



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Bradycardia

Bradycardia has a simpler approach than tachycardia.

The first step is whether the patient is **stable or unstable**, using the same criteria as in tachycardia. Note that it is not relevant whether the bradycardia is sinus or not- a sinus brady can drop the blood pressure just as surely as any other type of brady (remember that BP is directly related to heart rate, and there is only so much stroke volume compensation that the heart can do as the heart rate drops).

If the patient is unstable, they should be externally paced. In the meantime, you can try medications. More on medications later.

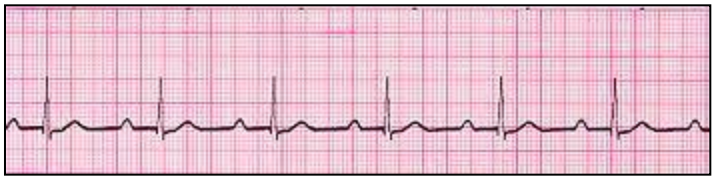
Causes of Bradycardia

Most of bradycardia will be due to old age and wearing out of the conducting systems. However, there are 3 acute causes that must be always ruled out

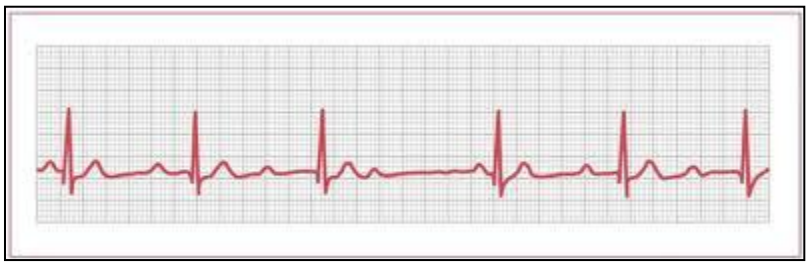
1. Drugs: beta blockers, CCBs, etc
2. Infarct: killing off the conducting tissue
3. Electrolytes: hyperkalemia

Note that the initials make a useful acronym DIE

If the patient is stable, you should determine whether they have a type of block that can lead to asystole or instability, and if so, prepare them for possible pacing and refer them on. ***The potentially dangerous types of AV blocks are 2nd degree type 2 (because it can rapidly lead to 3rd degree) and 3rd degree***.



1st degree AV block – fixed long PR interval



2nd degree type 1- lengthening PR intervals before the QRS is dropped. Benign.

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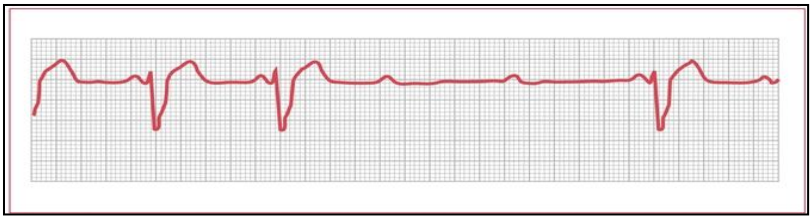
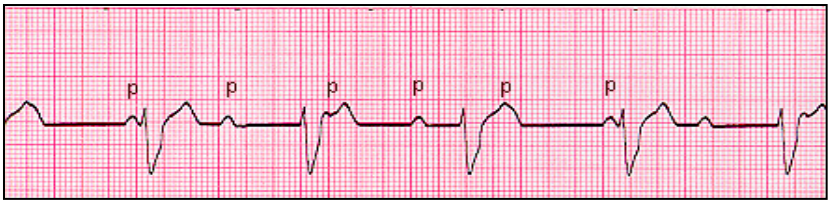


2nd degree type 2- constant PR interval before QRS is dropped. Dangerous.



3rd degree AV block. No relation between P and QRS. Definitely dangerous.

External Pacing

There are 3 ways to externally pace people, only one of which is easily available. That is the trascutaneous, i.e. through the skin. This is pacing achieved by using the pads of a Lifepack 12 or an equivalent machine. It works by supplying regular mini jolts of electricity through the skin to take over the pacing of the heart. It is great when it works, but because it has to deliver the charge through the entire chest wall, it is not very reliable. Also, it hurts a lot, requiring sedation and analgesia.

The other method is transvenous, in which the pacing is delivered through a central line in the neck and a conductive balloon which is jammed in the right atrium. Much more reliable then the transcutaneous as it delivers its charge right next to the heart. This is what most people we send to an ICU with a bradycardia will get while they wait for a permanent, implanted internal pacemaker, which is the third and best method of external pacing.

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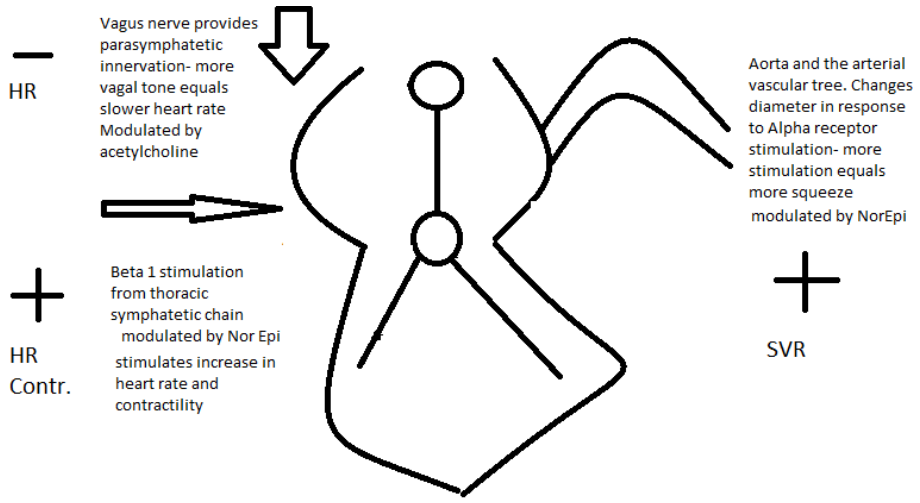
MEDICATIONS

Before we talk about medications, a review of the mechanisms with which the heart controls the heart rate:



Two ways of increasing heart rate with drugs:

One: DECREASE the vagal tone. Since vagal tone is modulated by Acetylcholine, we will need an anticholinergic agent to block it. **Atropine** is one such agent, and the dose is 0.5mg at a time.

Two: INCREASE the BETA1 stimulation. This will increase both the heart rate and contractility, but that is usually OK. **Dopamine** is a great agent to achieve that, at doses of 2-10 mcg/**kg**/min (NOTE: you do not get much more BETA1 stimulation as you go above 10mcg/kg/min, thus the lower max dose than for shock).

DRUGS FOR BRADYCARDIA

	Receptor	**Dose**
Atropine	Ach blocker	0.5mg at a time
Dopamine	Beta 1 stimulant	5-20 mcg/**kg**/min
Fentanyl	Pain killer for pacing	1 mcg/kg (50-75mcg usually)

After you have stabilized the situation, don't forget to check for DIE (drugs, infract and electrolytes). If hyperkalemia is detected as the cause you have two immediate jobs

1. Stabilize the cardiac membrane with Calcium gluconate 1gr IV over 10 min, repeated up to 3 doses
2. Shift the potassium into the cells using high dose Ventolin (10mg neb) and Insulin 0.1u/kg (usually around 6-8 units bolus then an infusion of same per hour. Don't forget to add glucose as you administer it)

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DRUGS FOR HYPERKALEMIA

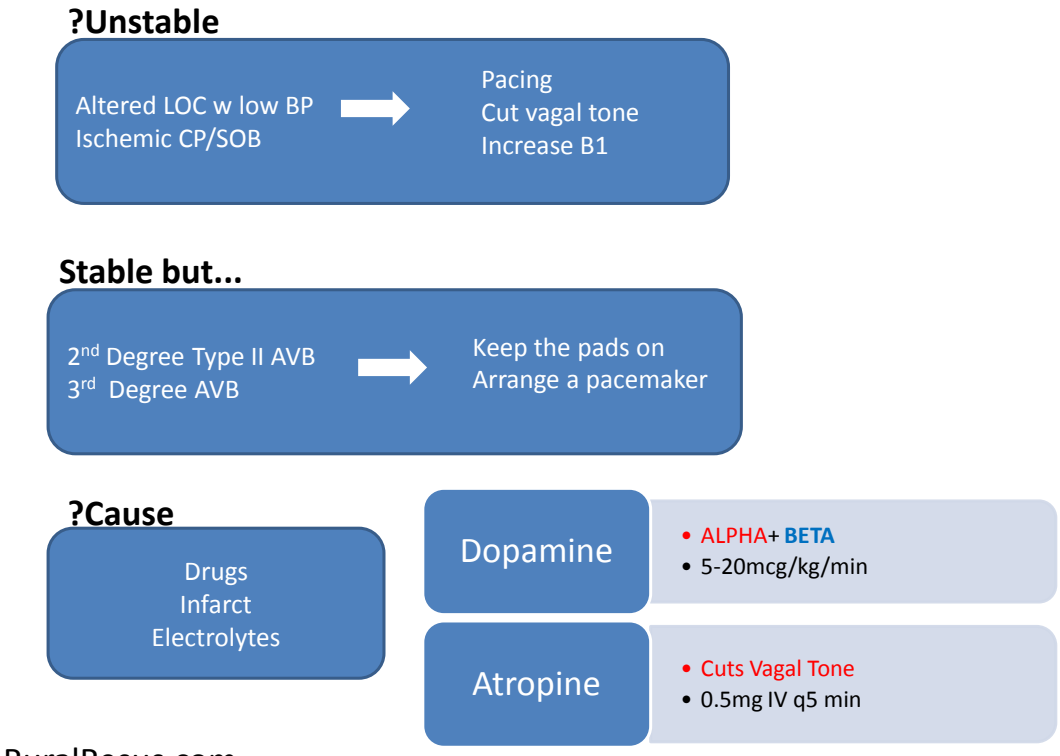
	Role	Dose
Ca Gluconate	Stabilize cardiac membrane	1gr IV over 10 min, up to 3 doses
Ventolin	Shift potassium	10mg neb
Insulin	Shift potassium	0.1mg/kg bolus+ per hour infusion

If the above is too complicated to remember, remember this:

If the low HR is resulting in low BP/confusion/chest pain, ***the patient is unstable and should be paced, no matter what kind of bradycardia it is***. In the meantime, you can try Atropine or a BETA1 agonist infusion.

If they are holding their own with regards to blood pressure but are in 2nd degree type 2 or 3rd degree AV block, you should put the pads on them to be able to pace them if they decompensate, and send them to cardiology for a permanent pacemaker.

Always check for presence of DIE (drugs, infarct, electrolytes)



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Altered LOC (confusion, agitation, coma, seizures)

The brain is the organ that is the most susceptible to changes in the body's homeostasis. Any changes in this homeostasis will lead to altered level of consciousness (LOC). So to diagnose an altered LOC, we just need to look at things that the brain needs in order to function properly and see which one is missing. This is a MUCH simpler approach then trying to decide which of the myriad causes is the cause of this particular alteration.

What Does the Brain Need to Function?

1. **Glucose**. The brain burns pure glucose, and only rarely, when glucose is unavailable, will it convert to feeding on ketones, as it happens in DKA. This however, is a change that happens over time. A sudden drop in glucose will derail a brain very quickly and can produce any kind of change in consciousness- coma, agitation, siezure, anything at all. High glucose is generally better tolerated but let it get high enough and it can produce similar problems- clinically seen as either non-ketotic coma (HONK) or severe DKA.
2. **Oxygen**. Brain tissue is the first tissue to die when we are hypoxic. Acute drops below 80% saturation will cause alteration in cognition.
3. **Blood pressure**. As long as the Mean Arterial Pressure (MAP) stays between about 50 and 150, the brain will dilate and constrict the blood vessels feeding it in order to maintain a constant Cerebral Perfusion Pressure. If the MAP gets too low or too high, however, the compensation gets maxed out and perfusion to the brain changes, usually causing changes in the level of consciousness. See the graphic below.
4. **Temperature**. Brain needs temperatures between 34 and 42 degrees C for optimal conditioning.
5. **Intact brainstem and ONE cerebral hemisphere**. The brainstem houses the Reticular Activating System, which regulates our awakeness. One hemisphere is all you need to maintain "front of the brain" cognition. This is the reason why ischemic strokes rarely produce coma, and intracranial bleeds do it all the time- an ischemic stroke would have to affect both hemispheres (very rare) or the brainstem (also rare) directly. A bleed, on the other hand, blows up one hemisphere then simply has to produce enough of an increase in intracranial pressure to squeeze the other hemisphere against the skull or flush the brain down the foramen magnum and squeeze the brainstem in the process.
6. **Physiologic electrolyte concentrations**. Any electrolyte disturbance can cause an alteration in the level of consciousness but in reality, it usually turns out to be sodium, with calcium playing a significant role in people with cancers and bony metastases.
7. **"Garbage" taken out**- CO2 by the lungs, hepatic metabolites by the liver, uremic metabolites by the kidneys. If these toxic metabolities accumulate, encephalopathy (i.e. altered LOC) follows.
8. **Absence of** any of the myriad **toxins** that can affect the brain. Broadly, we can classify those as infectious toxins (meningitis and encephalitis) or drugs

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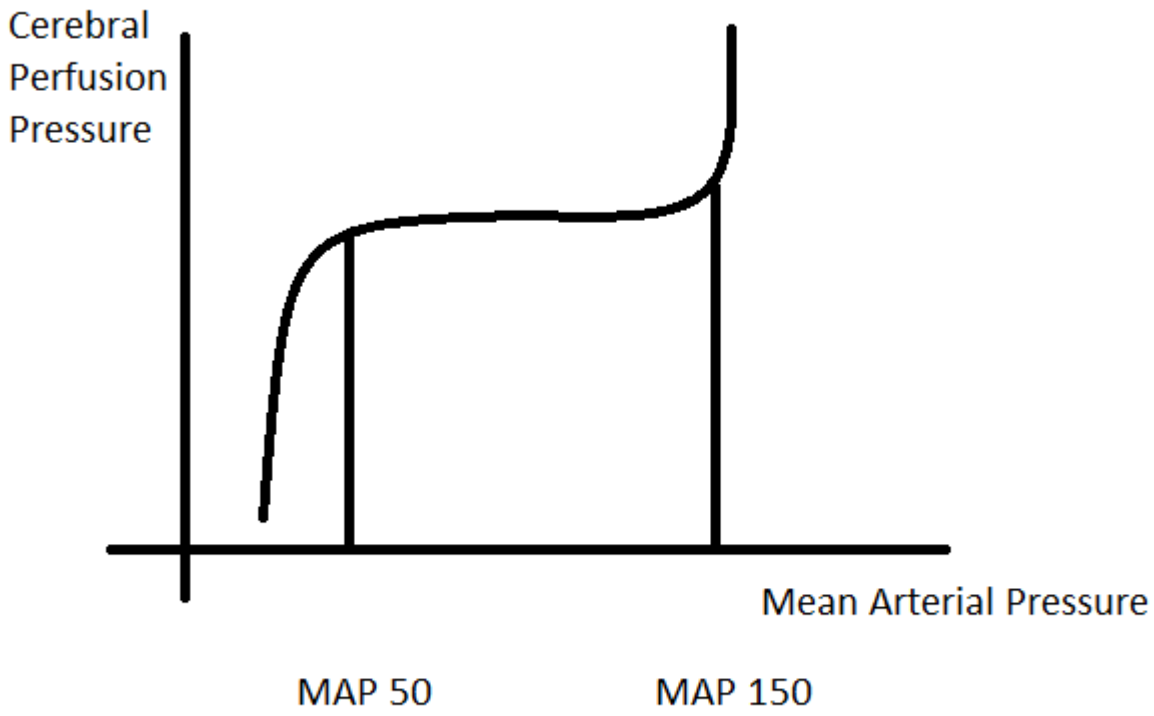

Does that all sound very confusing? Probably. Luckily, there is a not-too-complicated to deal with all this. It mostly revolves around doing a couple of simple things that can help you decide what is the likely culprit and around avoiding hypoglycemia, hypotension and hypoxia.

If you can figure out what is causing the problem, all the better, ***but you will never be wrong focusing on these basics***.

Hypoglycemia, Hypotension, Hypoxia

ANY condition affecting the brain - but especially intracranial bleeds - will get worse if these three are present so your main job will be to ***check the glucose***, supplement if necessary (to normoglycemia only) and then support the ABCs to ***avoid low BP or oxygen saturation.***

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LOC scenario questions.

Of course, the basic IV-O2-monitor/full set of vitals/ABCs apply here, but pay special attention to:

1. ***What is the glucose?*** EVERY patient with a change in consciousness needs to have his glucose checked. If it is less then 4, give 1-2 amps of D50W. If it says high, get serum glucose, a set of VBGs/ABGs and think of DKA/HONK.
2. ***Is the patient's level of consciousness depressed enough that he needs to have his airway secure?*** We like to secure airways (IE intubate) patients who are obtunded because we presume that they will not have adequate self-protection against aspiration and also because it makes it easier to avoid hypoxia, that all-important goal of all brain resuscitation. You can use the Glasgow Coma Scale but it is cumbersome and hard to remember on the spot, so we use AVPU scale instead (Awake, responds to Voice, responds to Pain, Unresponsive). If they are P or U, their GCS is 8 or less so call for help that can intubate them or do it yourself. Vomiting or uncontrollable behavior might push you in the same direction.
3. ***What is their Oxygen saturation?*** As mentioned, acute drops below 80% will cause altered cognition. In addition, hypoxia will worsen outcomes even if it is not the cause. Thus, O2 saturations need to be north of 90 percent if at all possible. Everyone should be put on 100% O2 initially, and if that is not enough, they need to be assisted, either through a Bag Valve Mask, an LMA or an intubation.
4. ***What is their BP?*** As discussed above, brains like their MAP between 50 and 150. If it is outside those parameters, you need to address that. Low MAP gets fluids and a pressor like Dopamine, high MAP gets a Labetalol drip.
5. ***What is their temp?*** Need to be in 34-42 range. Warm or cool with warm or cool fluids as appropriate.
6. ***What are the pupils like?*** Pinpoint pupils bilaterally are a sign of opoid overdose; or too much alcohol and benzos. We cannot reverse the alcohol and benzos, but we can reverse narcotics, so try Narcan and see what happens. If pupils are dilated, it can be either a sympathomimemtic like cocaine or speed, or an anticholinergic like Gravol or Benadryl (silly teenagers do this), or alcohol/benzo wihdrawal. Those all respond to Benzos.
   If the pupils are unequal and/or fixed, the patient likely has a bleed.
7. ***Was the change sudden and are all 4 limbs responding equally to verbal/painful stimuli?*** If not, a bleed is likely.
8. ***What are the electrolytes values*- specifically Sodium and Calcium?**
9. ***Is the garbage being taken out*?** What is the pH and pCO2 on VBG/ABG? Is creatinine suggestive of uremia? Are LFTs and INR/PTT suggestive of liver disease and possible liver encephalopathy.
10. ***If there was fever*** or other signs of infection, what does the LP show?

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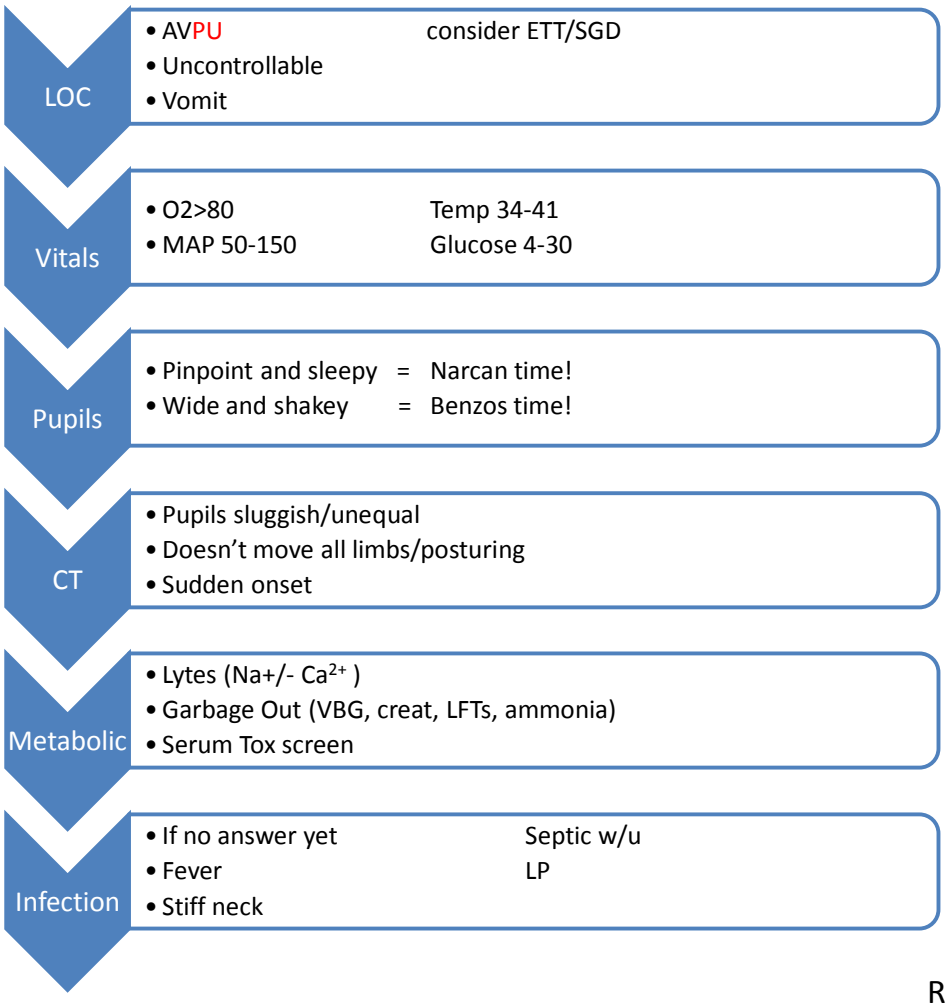

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If this sounds too much to remember, simplify to:	
>>>>> ***IV-O2-monitor, Vitals, ABCs*** <<<<<	
Intubate if P or U on AVPU Full set of vitals+ glucose Keep Glucose >4 Keep O2>90% Keep BP – MAP 50-150	Pupils unequal- bleed- lower ICP Pupils pinpoint or history suggestive of opoids- give Narcan Pupils dilated (and patient agitated, for any reasons)- give Benzodiazepines, lots and often.
>>>>> ***Look at your diagnostic tests!*** <<<<<	

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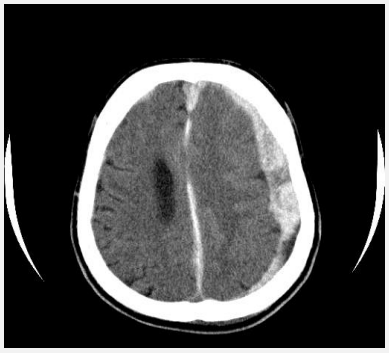
Special Cases

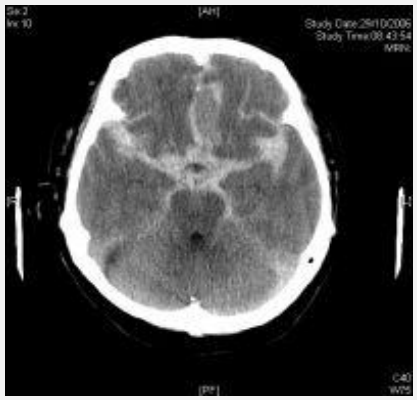
Intracranial bleed.

Intracranial bleeds come in 4 varieties. Subarachnoid (aneurismal or traumatic), subdural (a slower bleed coming from torn venous sinuses), intraparenchymal (ruptured blood vessel in the middle of the brain parenchyma) and epidural (arterial bleeds).



Subdural

There are some differences in prognosis and treatment of these four, but the basic principles are the same: ***avoid hypoxia and hypotension and do what you can to prevent or counteract the rise in intracranial pressure***. Getting a non-contrast CT early is important here as it helps differentiate surgical from non-surgical patients.



Subarachnoid (SAH)

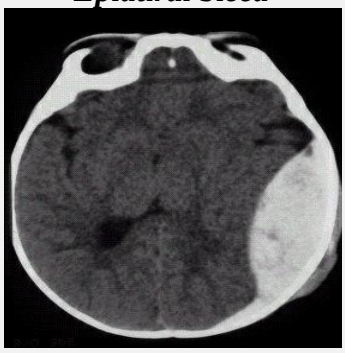
If it looks like the ICP is rising (more obtundation, blown pupils) give them Mannitol 1-2g/kg- it is a potent osmotic diuretic that will, among other things, reduce ICP.

Maintain BP in the normotensive (MAP 80-90) range with fluids and pressors if needed or labetelol if high (sBP>180)

SAH is the one type of bleed that is **more sensitive** to hypertension, so if the blood pressure is high above sBP>160 or dBP>90, use Labetalol to counteract it.

Intraparenchymal bleed

Epidural bleed



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Hypnotic intoxication

If the patient has ***bilateral pinpoint pupils and depressed level of consciousness***, there is a high likelihood of hypnotic ingestion: narcotics, Benzos or alcohol. Only the Narcotics can be acutely reversed. ***The main danger of hypnotic intoxication is respiratory depression and respiratory arrest***. Our goals are reversal of the narcotic portion of the sedation and airway protection

Narcan Administration

In general, we only want to awaken the patient enough so they are breathing spontaneously and lightly sedated. Full awakening can induce an acute withdrawal which can turn dangerous for the patient and violent for the providers.
Dilute a vial (0.4mg) in a 10cc syringe so you get 0.04mg/cc. Give 1-2 cc at a time, twice q2min. If that has not worked, there is likely a high dose or a high potency narcotic on board, so escalate the dose rapidly: 0.4mg-2mg-4mg-8mg q2 min

If a particular dose works, you will need to put them on a Narcan drip as it only has a 45-60min half-life and ALL the narcotics last longer.
The rule is to give them every hour 2/3 of the dose that was needed to wake them up every hour.

Narcan can be given SC/IM/IV or through an ETT

Agitation with dilated pupils

Lots of different agents or disease states can give you agitation and dilated pupils. Commonest ones are "uppers" (cocaine, amphetamines), anticholinergics (Gravol, Benadryl) and withdrawal from alcohol or opiates. Luckily, they all respond to the same treatment so you don't have to think too hard.

The mainstay of treatment is benzodiazepines. Diazepam, Lorazepam or Midazolam, it doesn't matter. Give early and often and in escalating doses. There are no maximum doses- give them the dose they need to get settled down. Aside from respiratory depression, benzoes have no toxicity- at worst you will oversedate them and then will have to help them breathe through a bag-valve mask or secure their airway with an ETT/LMA.

What About Typical Antipsychotics?
Avoid giving typical antipsychotics in large doses as sedating agents- they have anticholinergic effects and can worsen the agitation as well as increase body temperature, which is often a problem with people in agitated states because of high metabolic outputs.

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Seizures/status epilepticus

Your job with seizing people is two-fold- ***prevent hypoxia, and stop the seizures***. Short-lasting seizures will rarely affect oxygenation, but a longer-lasting one might- thus make sure you have an O2 monitor on these patients and that they are on supplemental oxygen. Also, the longer the seizure lasts, the higher the chance of anoxia-like hyperexcitation permanent brain injury. The chance of this is time-dependent so you really want to get a jump on controlling the seizure. 15 min from the start of the seizure is our absolute cutoff.

As far as seizure control goes, you have several options. As with agitation, ***benzos are first choice***, and at the same doses. Again, there are no maximum doses as benzos have no toxicity outside respiratory depression- in which case you might have to BVM them for a while or ETT/LMA them until they wake up. If benzos are not working and/or 15 min are up, use an RSI induction agent to stop the seizure and intubate to protect the airway.

Hypoglycemia

You generally have two main causes of hypoglycemia: insulin or sulfonylureas. The actual scenarios can vary a lot and in general revolve around either inadequate intake, dosing/timing errors or increased metabolic demand (fever, injury, dehydration, etc). Often there is no identifiable cause and the body just decided to go and use up the sugar.

Insulin Hypoglycemia

Hypoglycemia caused by insulin is fairly straightforward- give the patient some sugar (either orally or as IV Dextrose), make sure that the peak action of the particular type of insulin they are on has passed (30min-1h for Humalog, 2-3 hrs for Regular, 4-10 hrs for NPH) and re-check the glucose after the peak should have passed.

When giving sugar, the easiest way is to get them to eat some simple and complex carbs (a mixture also known as “sandwich and juice”). If the patient's level of consciousness is too impaired for eating, we can use IV dextrose

The initial bolus dose is **1gram/kg**. For an adult, that means 3-4 amps of D50W (50-100grams).

D5W and D10W come in bags and for most types of hypoglycemia, an infusion of 100-200cc/hr of D10W is reasonable (or double that volume of D5W).

Sulfonylurea-caused Hypoglycemia

If you have a sulfonylurea-caused hypoglycemia, things get a touch more complicated. Those medications stimulate the pancreas to release more insulin so if you keep giving them glucose, they will keep stimulating the pancreas to get rid of it, resulting in persistent hypoglycemia. Luckily, we have a perfect solution in Octreotide, a somatostatin analogue. Somatostatin, if you remember from med school, never met a hormone it doesn't like to depress, ie, it will prevent release of insulin. The initial dose is 50mcg bolus and 50mcg/hour infusion. Add a glucose infusion on top of that and you are set. Monitor for 24hrs due to long duration of action of sylfonylureas

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ALTERED LOC DRUGS

	Mechanism	Dose	Use
Narcan	opoid antagonist	Start at 0.04mg, escalate rapidly, no max	opoid overdose affecting LOC or respiration
Diazepam	Sedative	5-10mg IV/IM q2-5min	agitation, seizures
Midazolam	Sedative	2-4mg IV/IM q2-5min	agitation, seizures
Propofol	Sedative	20-40mg IV bolus 20-80 mcg/kg/min drip	agitation, seizures generally will need airway control
Mannitol	osmotic diuretic	1-2g/kg	intracranial bleed with increased ICP
Labetalol	beta blocker	1-2mg/min start then titrate to BP	SBP>160 or dBP>90 in SAH
Octerotide	somatostatin analogue	50mcg bolus and 50mcg/hour infusion	antidote for sulfonylurea poisoning
Dextrose	Sugar	1g/kg glucose 1 amp D50W = 25gr glucose	hypoglycemia

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Shortness of breath

In short, the lungs only have two functions- delivering oxygen to blood (OXYGENATION), and taking away carbon dioxide (VENTILATION). As oxygenation is more important in the short term- let's focus on it for now. To oxygenate, we need to have some O2 in the environment, we need to move enough air into the lungs to reach the alveoli, we need to be able to diffuse the oxygen across the alveolar membrane and we need to have blood reaching the alveoli to pick up the oxygen. The blood should have a decent amount of hemoglobin in it. This gives us 5 main reasons for hypoxia (low oxygenation):

1. There is **inadequate ambient oxygen** (FiO2). Usually not a problem unless at high altitude. In the flatlands, the only common cause of this is carbon monoxide poisoning (it actually displaces O2 from hemoglobin, but the effect is the same)
2. There is **inadequate ventilation** (amount of air moved in and out of lungs).
3. The **alveolar membrane is thickened** because of fluid or inflammatory gunk (CHF vs pneumonia/ARDS), thus preventing easy gas exchange
4. Blood is not going to where the ventilation is happening (**V/Q mismatch**). Usually caused by a PE, but can be the result of any condition where there is poor forward flow from the heart: aortic stenosis, cardiogenic shock, sepsis, poorly perfusing arrythmias - all resulting in poor flow of blood into the lungs.
5. **There is no hemoglobin to uptake the** 98% of the O2 is carried by Hgb. The other 2% is dissolved in the blood (O2 is poorly soluble) and it is what is measured by the pO2 we get from an ABG. The dissolved O2 is functionally irrelvenat. Anemia is a rare cause of breathlessness as it takes a very large and/or sudden drop of Hgb to make the patient dyspneic and it usually gives them other symptoms (weakness or syncope) first.

Now, you might have noticed I called both "moving air in and out of lungs" and "elimination of CO2" ventilation. This is because CO2 elimination depends only on the metabolic rate of production (usually constant in a patient lying on a stretcher) and the amount of air we move into the lungs to accept the CO2. CO2 is vastly (24 times) more soluble then O2and flows down a much steeper gradient then O2 (1000% for CO2, and only 30% for O2), and is,for most part, not protein bound like O2. The end result is that CO2 is much less dependent on "thickness of membrane" problems or V/Q mismatches. Ergo, for a constant metabolic rate, CO2 elimination is only dependant on the amount of air we move in and out of lungs and thus the two terms are used synonymously.

O2 First!

In the short term, accumulation of CO2 is rarely lethal. Acute hypoxia kills lots of people. So, in the first 5 minutes or so of resuscitation, outside of possibly securing the airway (more on that later), ***make sure that the oxygen saturation stays in the reasonable range, preferably above 90%***

How can we support someone's oxygenation? First, we can add supplemental oxygen. The simplest way to do this is through

NPs add about 3% of O_2 concentration for every litre per minute given. Someone on 5L NP will be getting 21% (room air) + 3 x 5% ~ 36% oxygen.

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nasal prongs. If the patient is in more serious trouble, you are better off putting them on a non-rebreather mask, which will deliver close to something around 70% inspired O2 as long as the mask's bag is filled. For true ~100% FiO2, put a set of nasal prongs and crank the O2 past the 15L mark (yes it can deliver >15L/min even though the marking end there) AND a non-rebreather mask attached to another O2 outlet.

Giving supplemental O2 only will work if the patient is awake and making adequate respiratory efforts. If not, the next step is to assist their breathing with a Bag Valve Mask (BVM). This is the quickest way to provide Positive Pressure Ventilation (PPV). We normally breath using negative pressure, i.e. we use the diaphragm to expand the lung, creating negative pressure and letting the air flow in to equalize that pressure.

When we are taking someone's breathing over, we use PPV- IE, we push the air into the lung. This has the benefit of letting the diaphragm and the other muscles of respiration rest, as we are not relying on their work.

With a BVM, we can assist a person' breathing, i.e. having the mask over their face and whenever we feel them taking a breath we squeeze the bag; or we can take it over completely. You can oxygenate and ventilate someone indefinitely using a BVM as long as you know how to use it properly, can get a proper seal and as long as their airway is not closing. The only negatives is that it is hard work and that it can result if air going into the stomach as well, making them vomit and possibly aspirate. Thus, if we anticipate a prolonged BVM, we usually secure the airway to make things easier. More on that later.

Another way to provide PPV is through the use of CPAP/BiPAP machines. There is a lot of confusion with terminology with these. Collectively, using these machines is called Non Invasive PPV (NIPPV). CPAP provided by an anaesthesia machine is called PEEP (positive end expiratory pressure). It is the same thing. CPAP/PEEP simply means that the machine will provide a fixed amount of positive pressure (usually 5-10mm H20) throughout the respiratory cycle. BiPAP means that the machine senses when the patient is taking a breath and increases the PPV it provides in response (usually in the range of 10-20mm H20) to push more air in with each breath. Thus, BiPAP settings are usually something like 12/7 (12mm when inspiring, 7mm at all other times).

NIPPV is only useful, however, with an awake, alert patient who can tolerate the mask (IE who is not claustrophobic), who is not actively vomiting or bleeding into their airway and who is making respiratory efforts- the machine will not breathe for them, it simply makes it easier for them to breathe. The research shows that NIPPV significantly reduces the need for intubation and ICU stay in COPD, CHF and immunocompromised pneumonia, as long as it is applied relatively early in the disease course. There is a protocol for asthma as well which uses much lower inspiratory/expiratory pressures. In reality, I will try NIPPV in almost anyone who is in serious respiratory distress, has no contraindications as above and is able to tolerate it. NIPPV (or any type of positive pressure ventilation) will make a pneumothorax worse and can cause it as well. More on that below.

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The final step of airway/breathing control is intubation or similar method of airway control.

There are 4 main reasons to take over someone's airway:

1. **Their airway is compromised** due to changes in the anatomy (tonsilar or retropharyngeal abscess, airway burn, neck or face trauma, anapxylaxis) or due to "crap" in the mouth (blood, vomit, food).

2. Their **level of consciousness is decreased** to the level where they are not protecting their airway against aspiration. We use P or U on the AVPU scale (see altered LOC section)

3. The patient is facing **imminent respiratory failure** (massive work of breathing, slowing down respirations, persistent hypoxia, etc) and other modes of respiration have failed.

4. They are **hemodynamically unstable** (i.e. in shock) and we want to take over their breathing because we anticipate they will get worse over time and because we want to reduce their metabolic demand as much as possible (sepsis is the usual cause of this).

Of these, the first condition is truly immediately emergent- call for help as soon as you identify one of these situations and prepare for immediate intubation. Closing airway intubations need to be done with an awake patient, are very tough, can deteriorate within minutes and should be attempted by the most experienced person around.

For the second cause, you usually have some time but will eventually need to assume control of the airway. Temporize in the meantime using the Universal Treatment Progression (see below)

For the third, we will asses them using our Respiratory Triangle (see below) and then apply the Universal Treatment Progression until we reach the need for an ETT or temporize with other means successfully

The fourth case usually only calls for an intubation once all the other resuscitative efforts have been completed

Laryngeal masks (LMAs)

LMAs or King LT combitubes (collectively known as supraglottic devices) are excellent rescue devices or airway control devices you can use instead of an ETT or if you fail to place an ETT. It should be a very strong consideration as the primary airway device for a non-expert intubator as it is much simpler to place. It does not provide quite the same airway protection against aspiration as an ETT does since the seal is above the vocal cords, but does provide some. Good for most situations *except:* where the airway is closing (eg anaphylaxis); where there is active bleeding/vomiting/secretions; or when there is severe obstructive lung disease (eg asthma) where the high airway resistance might mean that the air you bag in leaks around the seal rather then go into the tight lungs.
Adult sizes are 3 and 4. 4 is used for most adults, especially male.
Ketamine 100mg-200mg IV should allow most patient to tolerate an LMA.

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One you have placed the ETT or an LMA, you have "bought" that person's breathing. You can provide it through a BVM, but as mentioned, it is hard work, so we usually put them on a ventilator. Since most small hospitals have Resp Techs that live 20-30 min away, it is useful to understand a bit about ventilators so we can start things while we wait for an RT.

Most vents available in ER or on the floor are set to volume control. This means that we set a desired volume that we want every ventilator breath to achieve, a peak airways pressure that should not be crossed (as too much pressure can cause a pneumothorax, a good cap is 30-40mm H20) and the rate of breathing. The ventilation requirements for an adult are 7cc/kg per breath, which for an average 70kg male makes 70 X 7 = 490cc per breath (round up to 500cc). If you remember med school, 500cc is the average tidal breath we take on our own.

The average respiratory rate is 12-16. This gives us a minute ventilation of about 6L per minute (12 X 500cc).

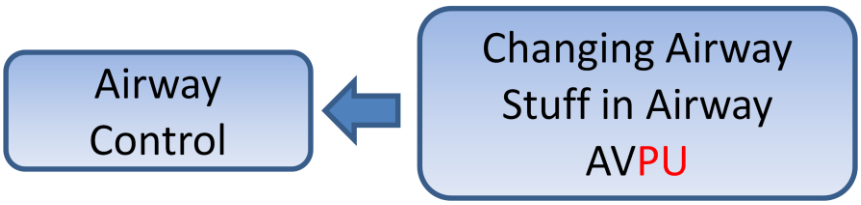
Remember to sedate and paralyze the patent both for their comfort and to prevent complications with the patient fighting the vent or extubating themselves accidentally.

Now that you understand all the methods of respiratory management, let us look at the process of assessing a patient in respiratory difficulty

Evaluation of patient in respiratory difficulty

1. ***Is the airway patent and is the compromised airway the cause of the respiratory difficulty?***
   Any evaluation of SOB begins with an airway assessment. It is very brief:
   1. Is the airway changing?
   2. Is there "crap" in the mouth?
   3. Is the level of consciousness depressed enough to warrant airway protection (P or U on AVPU).

If yes to any of the, start mobilizing resources for an invasive airway in parallel with the rest of your assessment



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If the airway is not an immediate concern, we can assess the function of the lungs. Remember, lungs only get O2 in, CO2 out; and use muscle effort to achieve both. These form the apices of our Respiratory Assessment Triangle, ie the 3 ways in which lungs can fail. Oxygenation is at the top as it is the most important apex in the short term.

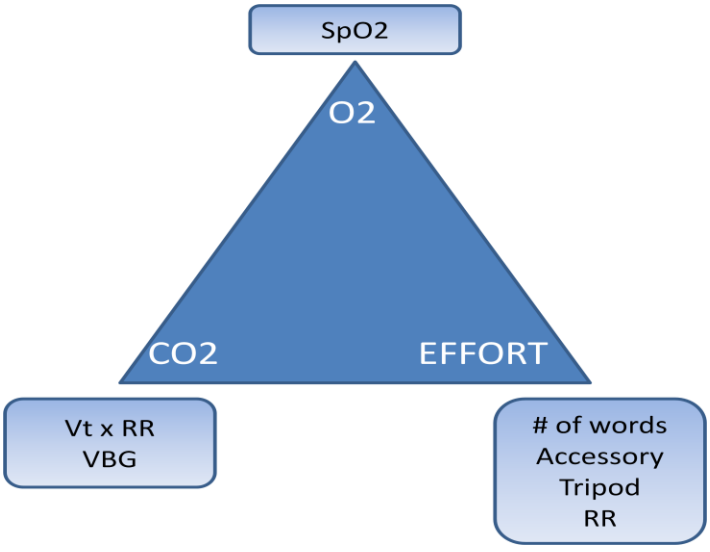
What is their oxygen saturation?
As mentioned above, lack of oxygen will kills a person far quicker then buildup of carbon dioxide. Most of O2 (98%) is carried by Hgb, so saturation is a key measure of oxygenation

Are they moving enough air?
Remember, ventilation only depends on respiratory rate and volume of each respiration. If it is inadequate, it will cause a buildup of CO2 and acidosis over time.

This is a clinical assessment: look at how frequently and how deeply the patient is breathing. As mentioned, the rate should be at least 12-16, and you have a lifetime of experience looking at people moving 500cc tidal volumes. Use auscultation to refine this assessment. We do not need to be exact here- just a gestalt of "moving lots of air" or "not moving a lot of air"

What is their work of breathing?
How many words can they string together? Are they using a lot of accessory muscles (tracheal tug, use of intercostal muscles, abdominal breathing)? Is their respiratory rate very fast or is it slowing down?

If it looks like they are working very hard to breathe, they will likely tire out (you can use up to 75% of your total oxygen/energy consumption on work of breathing) and progress to failure



The advantage of using the respiratory triangle is that it allows us to estimate a patient's

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respiratory status within seconds and with very limited tools (SpO2 monitor, eyes and a stethoscope).
If the patient is failing on any one of the respiratory triangle apices (or multiple ones), we get to apply the **Respiratory Treatment Progression** which is, luckily, identical no matter what the underlying condition is.

We start by increasing FiO2 using nasal prongs (~40%), a non rebreather mask (~70%), or combination of the two (~100%)

If added O2 is not fixing the failure of an apex or apices, we do a quick assessment to see if the dilators could be of use- bronchodilators (Ventolin, Atrovent) for wheezy patients and venodilators (Nitro and Lasix) for bilaterally crackly patients.

If neither of the above treatment have restored all 3 apices to satisfactory status, it is time to apply positive pressure ventilation (PPV). Before we do that, however, we need to do an assessment (with stethoscope, CXR or bedside ultrasound) to exclude the presence of a pneumothorax as that is the only cause of respiratory difficulty that would get worse with PPV.

There are 4 ways to deliver PPV- in an escalating order-

1. Non Invasive PPV (NIPPV- CPAP or BiPAP)
2. BVM (assists or full ventilation)
3. Supraglottic device (SGD-LMA or King LT)
4. ETT.

There is no fundamental difference in ability to deliver PPV through any of these devices, they simply differ in where in the respiratory tree the PPV is delivered (mouth for NIPPV and BVM, pharynx for SGDs or trachea for ETT) and how much aspiration protection they offer (none for NIPPV and BVM, decent with SGDs, most with ETT). All these methods also offer the ability to provide 100% FiO2

As we apply these methods of PPV delivery, we might find that a hypoxic, hypoventilated, tiring patient is too agitated to tolerate them comfortably. This is where Ketamine can be of help. Have a syringe of 100mg drawn up, and if the patient is not doing well with PPV, inject aliquots of 20mg IV q2-3 minutes until the patient is dissociated sufficiently to tolerate my chosen method. If I find that NIPPV or BVM are still not working well, I would inject another 100mg of Ketamine (and possibly a paralytic) and proceed to an SGD or ETT insertion.

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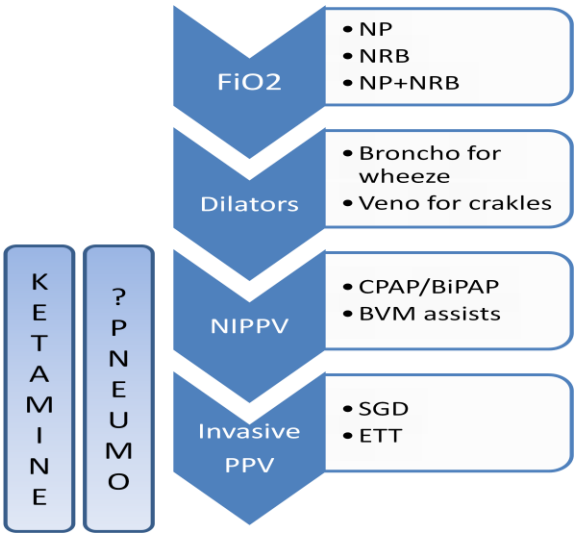

The respiratory treatment progression

Finally, what is causing all this?

Notice that I only care about this after I have done the initial assessments and stabilization as per above. Here I will add CXR to my clinical exam and then go through the 5 reasons for poor gas exchange outlined at the start of the SOB section. Is there inadequate FiO2? Is there poor ventilation? Does auscultation or CXR show any "membrane thickness problems" (CHF or pneumonia). Finally, if none of those are faulty, I will consider V/Q mismatch- PE or other forward flow problems. I will also measure the Hgb in the blood.

I include a CXR (usually portable) in my assessment (mostly because it helps me see "thickness of membrane" problems or rule out a pneumothorax so I can start PPV), but not the arterial gases. Getting arterial gasses while acutely resuscitating an unstable patient is generally a waste of time- the only information it adds are the pO2, pH and pCO2, and as mentioned, pO2 is functionally irrelevant and ventilation can be reasonably assessed clinically, at least in the short term. Acidosis, unless metabolic, will be directly related to lack of ventilation

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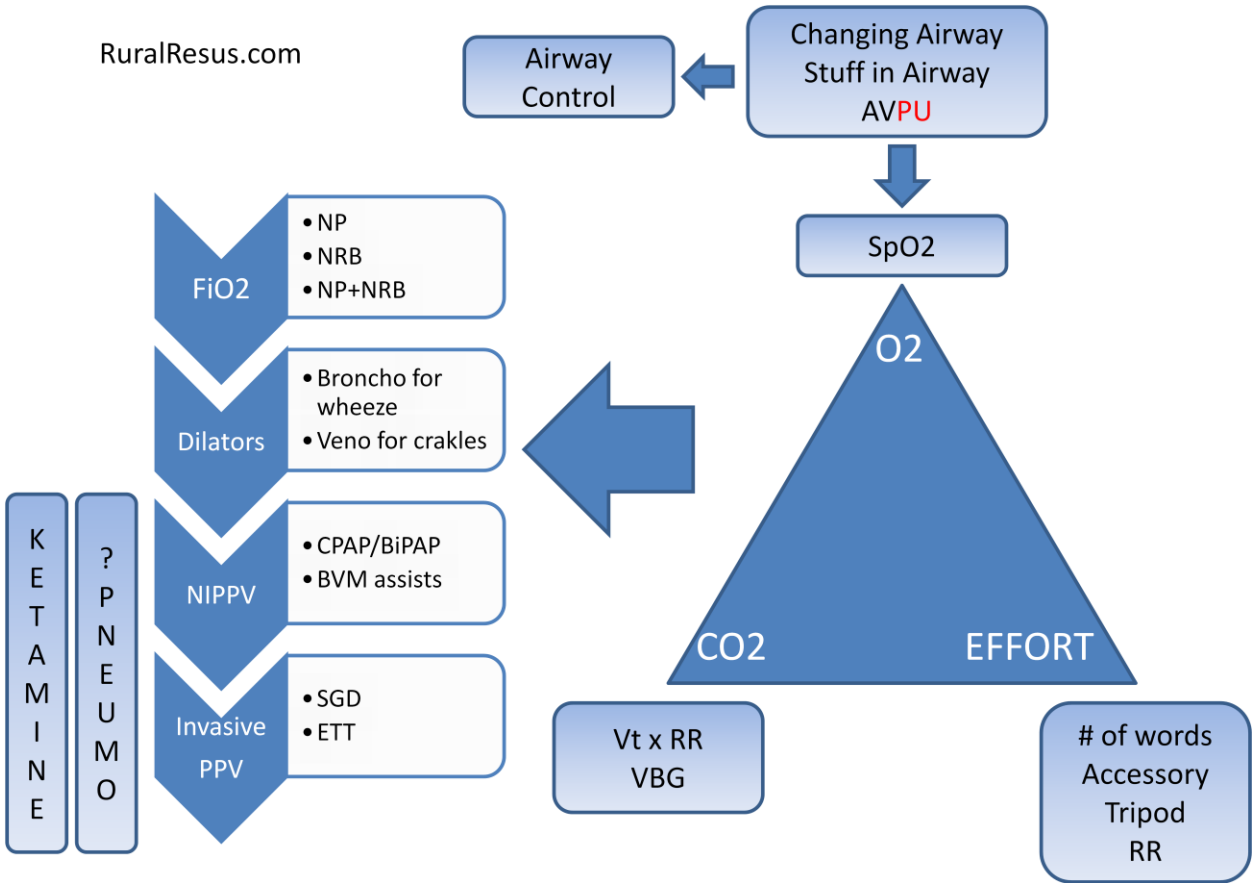
If this is too complicated, remember this:

If the airway is changing for the worse or the patient is unconscious, secure the airway.

Otherwise, assess the focal points of respiratory function (oxygenation, moving air and respiratory effort), and if any of them are unsatisfying, progress the treatment from FiO2 to invasive PPV as needed. Exclude pneumothorax before applying PPV. Once you are happy that the 3 focal points are stable, try to figure out what is causing the trouble using our 5 causes.



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Specific Conditions and Treatments in Shortness of Breath

COPD: standard treatment is Ventolin/Atrovent (5mg/500mcg) nebulized masks, back to back if necessary. 125 mg of IV Solumedrol or 30-50 mg oral Prednisone have similar efficacy, so use oral if they are able to tolerate it. Use antibiotics if the patient has 2/3 or 3/3 of symptoms of: increased dyspnea, increased sputum, increased purulence of sputum; or if the patient is needing hospitalization. For mild-moderate, dischargeable COPD, the first line antibiotics are Septra or Amoxicillin. For severe, hospitalized cases, it is a respiratory fluoroquinolone or a combination of Ceftriaxone and a macrolide.

COPD
Ventolin/Atrovent
Sulomedrol
Antibiotics

COPD and PPV
COPD does really well when assisted with PPV, so consider BiPAP for any serious cases. *Be aware that COPDers, especially those with emphysema, are very susceptible to getting pneumothorax when on PPV, so if they get worse, make that your first suspicion.*

Asthma: again, puffers in nebulized form, preferably back to back are your first line of treatment. If they are being resistant to treatment, adjuncts are: inhaled Epinephrine, 3mg of 1:1000 in a nebulizer, every 15-20 min; 0.3mg intramuscular 1:1000 Epinephrine (like for anaphylaxis); or 2g IV Magnesium sulfate infused over 10-20 minutes. Use Ketamine 1-2mg/kg for intubation. You can try BiPAP but with much lower pressures then in other conditions: start at 8/5 and increase IPAP by 2mmH20 every 15 minutes and EPAP by 1mm H20 every 15 minutes until better or O2 sats improve.

Asthma
Ventolin
Inhaled Epi
IV Mg sulphate

Avoid intubating asthmatics if at all possible as they are very difficult to ventilate properly with mechanical vents.

CHF: Mainstay of treatment is Lasix, usually 60-80 mg IV, higher doses if there is renal failure. Use Nitroglycerin aggressively as long as they have blood pressure to tolerate it. Start at 5 mcg/min and titrate the dose up until they get better or their BP drops below 100 systolic. Once you have figured out the dose they need, you can switch the drip for a patch- for example, if they needed 15mcg/min, that is 900mcg/hr (15 X 60), or 0.9mg/hr, so you can use two 0.4mg/hr patches to achieve a similar dose. Finally, use BiPAP/CPAP aggressively in any serious CHF- it responds really well to PPV.

CHF
Lasix
Nitro
BiPAP/CPAP

Anapylaxis: a rapid allergic reaction, causes SOB when it creates angiodema and bronchospasm. Angiodema happens because the cell-to-cell junctions open with histamine release, leading to leaking of intravascular fluid into intersitital space. This can rapidly obstruct the airway. Thus, the first job in anaphylaxis management is airway assessment and preparation for rapid intubation if the airway looks affected. These are very difficult so call for help immediately.

In the meantime, the mainstay of treatment is IM Epinephrine. We give it IM for speed of administration and less systemic effects then IV dosing. The dose is 0.3-0.5 mg of 1:1000 Epi (the concentrated version, comes in vials. The 1:10,000 concentration we find on crash carts is for IV use). The dose in kids is 0.01mg/kg for a max of 0.3mg. We can repeat this every 5 minutes. Epinephrine, being a vasoconstrictor and a bronchodilator rapidly reverses the

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angiodema and bronchospasm.

For more long term (aka after then the first 1-2 minutes) treatment, we use Benadryl 50mg IV (1mg/kg in kids) for H1 histamine blockade, Ranitidine 50mg IV (1mg/kg in kids) for H2 histamine blockade and Solumedrol 125mg IV to reduce the chance of the "second bump", IE a reactivation of anaphylaxis that occurs in about 10% of cases and can occur 4-72 hrs later. No one who experienced anaphylaxis should leave your care without an Epipen in hand (it contains 0.3mg 1:1000 Epi) or, if a kid, EpiPen JR (0.15mg Epi).

Anaphylaxis
IM Epi 0.3 mg
Benadryl 50mg IV
Ranitidine 50mg IV
Solumedrol 125mg IV

Pneumothorax/pleural effusion: they need a chest tube, or a thoracocentesis in the case of the effusion.
Videos on how to do those at:
http://www.youtube.com/watch?v=hQlt57AyQmg Part 1 chest tube insertion

http://www.youtube.com/watch?v=wuSg_p2Fe0Q&feature=related Part 2 chest tube insertion

http://www.youtube.com/watch?v=2ZRip1STSSQ Thoracocentesis

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SOB DRUGS AND TREATMENTS

	Dose	Conditions
BiPAP/CPAP	10-20mm inspiratory (8 in asthma) 5-10mm expiratory (5 in asthma)	COPD CHF pneumonia asthma
Puffers	5mg Ventolin 500mcg Atrovent	COPD Asthma
Epinephrine	3mg 1:1000 nebulized 0.3mg 1:1000 IM	Asthma
Magnesium	2g IV over 20 min	asthma
Lasix	**60-80mg IV**	**CHF**
Nitroglycerin	**2-20+ mcg/min**	**CHF**
Benadryl	1mg/kg or 50mg IV/IM/PO	Anaphylaxis
Rantitidine	1mg/kg or 50 mg IM/IV, 150mg PO	Anaphylaxis
Solumedrol	125mg IV	COPD Anaphylaxis
Ketamine	**20-100mg IV**	**Sedation** **Induction**
Rocuronium	**100mg**	**Induction paralysis**

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Myocardial Infarction

EKG Territories
II, III, aVf for RCA
V1-3 for LAD
V4-6, I aVL for circumflex

Since you will receive much teaching about this in other forums, I will keep this brief. MI comes in two varieties- NSTEMI- where the EKG looks normal or there are ST depressions, but no ST elevations; and STEMI, where there are ST elevations, usually territorial.

For both, the immediate treatment is to reduce platelet aggregation though use of ASA and Plavix, and anticoagulation through use of IV Heparin or LMWH. LMWH does better then its unfractionated cousin (less bleeding, better outcomes), but if a patient is going for an immediate angiocath, the interventional cardiologsits usually prefer the IV Heparin for its ability to titrate anticoagulation. GpIIbIIIa inhibitors are also used, but rarely outside of cath lab or the CCU, so you don't have to worry about that. Beta blockers are used to reduce the myocardial oxygen demand but they also potentially compromise the heart's ability to respond to cardiogenic shock so we usually let the cardiologists give it in the CCU or the cath lab.

In STEMIs, we have to make a choice of using TNK or going for immediate angioplasty. Since we are supposed to be giving TNK within 30 minutes or the patient showing up, and angioplasty within 90 minutes, if the transport time from the peripheral facility is more then 60 minutes, TNK is usually given, and, when possible, in consultation with the receiving interventionalist. NSTEMIs also go for angiocaths, but usually within 24-48 hrs.

Otherwise, MI care mostly consists of managing its complications, most often arrhythmias and cardiogenic shock, which were covered above.

MI DRUGS

	Dose
ASA	160mg po
Clopidogrel	300 or 600 mg load then 75 mg po od
Enoxaparin	1mg/gs sc q12h
IV Heparin	5000u bolus then per nomogram
Metoprolol	25-50mg po

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Code Blue

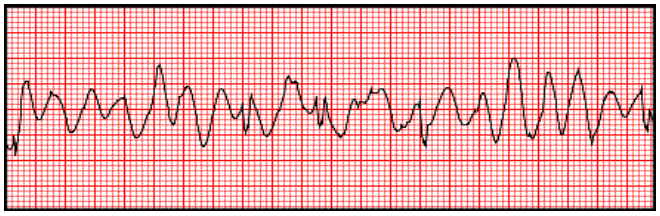
When you are call for a Code Blue, the patient has stopped breathing and/or has lost his pulse. In either case, the patient is now clinically dead. Our job is twofold.

See if it is a "recoverable" death, i.e. a death that responds to electricity applied to the heart, IE defibrillation. This is the case with VT without a pulse or Ventricular Fibrillation. A patient's chance of surviving a witnessed Vfib arrest with rapid defibrillation is 30-40%. In comparison, a non-defibrilable death (eg PEA) only has a 2-4% survival rate. Thus, your priority, after the ABCs and starting CPR, if to put the pads on the patient and analyze the rhythm to see if it is VT or Vfib, and if it is, rapidly defibrilate. If it is not, it is going to be PEA (normal-ish looking EKG but no pulse) or asystole (flatline). The task here is to rapidly identify, from history, whether there is a potentially reversible cause like an MI or a toxin (those Hs and Ts) and act on that.

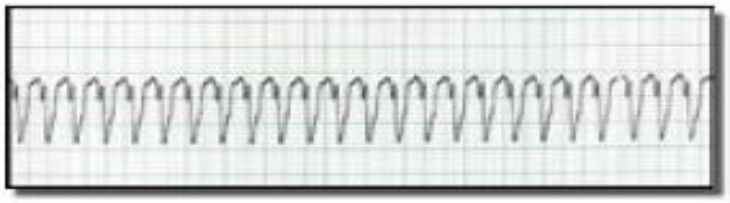
The second task is to **keep the brain alive** while you defibrillate or apply other therapeutics. Brain only has minutes before hypoxia due to lack of blood flow begins to kill it. The way to do that is through EFFECTIVE CPR. CPR, with ventilations, essentially stops or significantly slows down the brain anoxia death timer. Thus, as soon as a Code Blue is announced, ***someone should be doing CPR*** while you attach the pads, get the meds, etc. CPR also helps the heart muscle survive anoxia, making it more likely to respond to defibrillation. It takes a while for CPR to actually build a strong enough pressure wave to create blood flow; and the flow will stop within seconds of CPR stopping. Thus, you must work really, really hard to minimize CPR interruptions. It helps no one if you rescue the heart but the brain is dead. To help us recruit a pressure wave, we use Epinephrine, a strong vasoconstrictor.

Epinephrine in Arrest

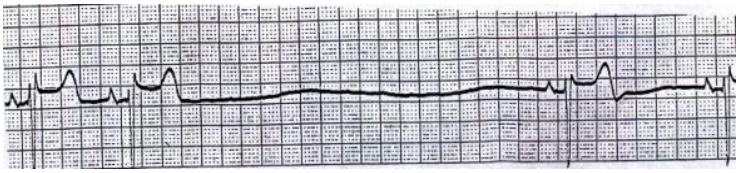
Note that the bolus given in arrest situation is 1mg IV, which is a massive dose that, if given to a live person will likely result in extremely nasty side effects or death. ***Thus, make sure the person is clinically dead before you give***



Vfib



VT



PEA

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Transporting a Patient

Transporting acutely sick patients is inherently risky business.

You leave the relative safety of a nice, warm, spacious hospital where some supports are available, and you put the patient and yourself into a cramped, rocking vehicle with relatively limited equipment and limited support. In addition, if you are going by road, you might find your way blocked by a traffic jam or even end up in a ditch (it has happened); or if going by air, you might find yourself diverted from your goal because of the weather or worse (end up dead in an air crash- it has also happened).

Given these risks, it pays to be as prepared as possible.

First - know your supports.

One paramedic will be riding with you in the back.

Basic care (BCP) paramedics can only give symptom relief meds (like O2, Ativan and nitro), bag a patient or put in a combitube, and operate a defibrilator in an automatic mode. They are not allowed to pace or cardiovert without calling a patch (i.e. calling the base hospital for permission).

Advanced care paramedics (ACPs) are allowed to intubate, can start Dopamine as a pressor (but can't monitor a drip of any kind that YOU have started) and have access to full Lifepack 12 capabilities and more-or-less full scope of ACLS drugs. Finally, there are **Critical Care Paramedics** (CCPs) who belong to ORNGE and who generally do not need a physician to go with them as their scope of practice and skills are quite similar to ours.

Paramedics operate under a base hospital physician's licence (locally, it is KGH) and do not have an independent licence to practice like an MD or an RN or an RT. When they are on their own, they practice under the base MDs licence to the full scope of their practice limits. *When, however, they are in the back with you, they are not the primary care provider and will only follow your orders, up to the scope of their practice.*

A **nurse**, if she goes alone with a patient, can do all of ACLS, use full Lifepack 12 capabilities, can initiate and monitor drips, but needs your orders to do it. So before sending them out, you should write them a set of orders like- "use full ACLS protocols when needed" or "initiate Dopamine at 5 mcg/kg/min if MAP <50 and titrate to MAP>65", " initiate pacing if HR<40 or MAP < 60". Nurses cannot do airway manouvers except bagging the patient and cannot place a combitube, LMA or intubate.

RTs can monitor vents and NIPPV machines independently and can do the full scope of airway manouvers including intubation. They cannot monitor or initiate meds.

Second, know your mode of transport.

Land ambulance is most spacious (hard to believe) and has the advantage that you can stop if you need to do a tricky procedure, like intubate. Helicopters are noisy, cramped, very limited by weather (I believe ORNGE can only fly under visual flight rules, ie when visibility is good) and have a relatively short range- about 400-600km. They fly low enough that gas expansion is not a problem. Fixed wing has the longest range, is relatively spacious and smooth, but limited to landing on airstrips and then going to hospital by land ambulance. Finally, because they fly high,

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any gas that is trapped and unable to communicate with the atmosphere will expand because of its density compared to the thin air at altitude. Thus, pneumothoraces expand and get worse/cause tension, gas trapped in obstructed bowels expand and cause rupture and air in the ET tube cuff can expand and rupture the cuff. Thus, before going on fixed wing, decompress all gas pockets in the body the best you can (NG tube/chest tube) and it is good practice to replace air in all devices relying on air insuflation, like LMAs and ETT cuffs, with saline.

Third, know your equipment.

Ambulances carry drugs that BCP/ACPs can use (and you are welcome to use any of it), which covers basics like Ativan, Lidocaine, Morphine, Adenosine, IV/IM Epinephrine, etc. Ambulances under KGH do not carry Amiodarone, and will only have Dopamine as their pressor (and only if it is an ACP crew). They will not have "fancy" stuff like Ketamine, Propofol or Norepinephrine

Thus, don't find yourself in a situation where you need something critical only to find out that you didn't bring it because you assumed that the medics have it!

Finally, know your patient and their disease.

There are 4 steps to this:

1. ***Anticipate complications***. This is where it pays to know a bit of medicine. For example, if I were getting in an ambo with a guy with a large anterior STEMI, I might think that he might develop: arrythmias with hemodynamic instability, cardiogenic shock, or Vfib/pulseless Vtach.

2. If any of the anticipated complications are likely and will require invasive/complex procedures like intubations or chest tubes, ***do it before you leave the safety of the hospital***, where you got more equipment, space and support and can call an anesthetist or a surgeon to help you. There is always an essential tension before just pilling into the ambo as fast as you can so you can get them to St Elsewhere where definitive management awaits, and doing more to stabilize the patient before heading out into the cold night. I would say that, on average, the balance lies in properly stabilizing before heading out, even for minutes-count scenarios like STEMIs and strokes.

3. ***Make a plan*** in your head before you leave how you will deal with every single one of the anticipated complications, and make the plans at least one step/alternative "deeper" then you normally would, to account for a higher likelihood of failure/difficulty.

For example, dealing with an asthmatic in a hospital, my plan would be: "give asthma treatments, intubate if those fail or are not improving the situation". In a transport situation, my plan might be: "continue with asthma treatment. If it fails, consider intubation. If unable to intubate in the ambulance, place a temporary airway like an LMA or a King LT combitube. If have trouble with LMA, start bagging with a BVM" In other words, if you can help it, don't ever rely on only one method of getting out of trouble. Have at least two, and preferably three choices at every step of your "anticipated complications algorithm" in case one of them fails.

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4. ***Identify all supplies you will need*** for every step of your algorithm and make sure you pack them.. All hospitals have "travel bags" prepared with commonly used items, but they are often not as comprehensive as you need them to be- they might have a size 3 laryngoscope blade, size 8 tube and that's it. Go though that bag, check that it has all the little things you need and add what extra you think might be necessary.

For the asthmatic above, I might pack: a size 3 and 4 laryngoscope blade, one handle, a size 7 and 8 tube ET tube, a stylete, a size 4 LMA and a King LT combitube, as well as the CO2 detector and a 10cc syringe and a functioning BVM.

5. ***Pre-pack and pre-dose your medications***. Many meds come in single dose vials, but some critical ones don't. If I were transporting a patient with anaphylaxis, I might prepare 2 or 3 syringes filled with 0.3mg of 1:1000 epinephrine for IM injections, rather then fuss in the back of a moving ambo to crack an epi vial and draw an appropriate amount while the patient is experiencing rebound and is in the throes of angiodema. Also, ***calculate whether you will have enough medications for your anticipated travel time***. For example, let us say I am transporting that septic guy and I have pre-mixed a bag of 10mg of phenylephrine in a 100cc bag and have put him on a 200mcg/min (2cc/minute or 120cs/hr) drip and I am going from Trenton to Kingston (1hr 15 min anticipated travel time). My 100cc bag will be done in 50 minutes and that is not accounting for the possibility of the patient needing a higher dose or travel delays. In other words, I need to premix another bag before I go to ensure I have enough phenyl for the whole trip.

<u>In Conclusion….</u>

If you consider the above, you will give your patient the best chance of surviving the single riskiest phase of their medical care and save yourself a lot of anxiety. In reality, we often just get in and hope for the best, but even if you are very experienced, you get burned from time to time. When you are starting out, ambo rides with critical patients will probably be the most spinchter-clenching experiences you will have. Prepare for it properly, and your sphincter tone will be much less!

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MEDICAL TEAM LEADERSHIP

Code Ineffective

You arrive breathless into a "Code Blue" patient's room. Several nurses are charging around. One is doing CPR that looks pretty ineffective, no one is bagging. Another is trying to get an IV. One more is saying everyone should be in gowns, gloves and masks. There is no crash cart. Two student nurses are standing wide-eyed blocking the doorway. The sides of the bed are up. Another junior resident is intently staring at the monitor since you arrived, and the rhthym looks wide and strange.

Acute care situations are inherently chaotic. Furthermore, the usual presence of a large number of people - especially medical trainees - leads to a strong bystander effect where no one wants to be the one taking charge and assuming the responsibility for a patient outcome. While this is understandable, it also leads to ineffective resuscitation and poor outcomes for patients. If multiple people are trying to control the situation, the nurses and others will unconsciously divide themselves according to whom they know best or who appears to have the best grasp of the situation or who appears most confident. This can lead to a totally chaotic environment where multiple and often contradictory things are attempting to be done.

A clear chain of command **MUST** emerge if this patient's life is to be saved. Most people look to the MDs to lead, so at a number of points during your residency and beyond, you will have to take the leap and be the leader.

There are 4 rules we will use to effectively negotiate this tricky situation:

1. One and only one boss must emerge, and quickly and **formally.** There are usually several candidates- the first doctor on scene, the doctor whose patient it is, or the most senior doctor. You must quickly decide who among them should be the boss and verbally and loudly declare it so everyone on the team knows. This is often a very socially uncomfortable step leading to "You want to lead this?, "No, why don't you?", "Oh, no I couldn't..." type of time-wasting vacillation that the dying patient can't afford. Make it simple: "Mike, you were first on scene, do you want to lead? No, ok, I am taking over, guys I will be boss, Mike will stay on the airway". **You MUST complete this step.**

2. If you are the boss, **be the boss!** If you have chosen or been put in the leadership situation, then lead, even if it is very uncomfortable and contrary to your personality. Note that this does not mean that you must feel like the most competent person in the room or always be right. It simply means executing leadership tasks (as defined below) and keeping overall control of the situation

3. If you are **not** the boss, **don't try to be the boss!** You just finished an anesthesia rotation and think you can do a much better job then the elected leader? Good, use your skills to support the leader, rather than try to take the spotlight. "Hey boss, you ok if I steal nurse Rose and take over the airway and report to you when it is secured ?" is much better then co-opting half the team without permission and yelling orders so they can help you with the intubation while the leader is trying to accomplish something else.

4. No matter what your position, if you are aware of something that no one else is, do not allow

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a harmful course of action to occur. You are putting IVs and noticed the patient lost the pulse while the boss was distracted troubleshooting the Lifepack? Inform the boss "Hey Heather, there is no pulse" and if no action is taken by the rest of the team, initiate corrective action (CPR in this case) yourself. Once the rest of the team realizes what is going on, inform everyone of the situation then **allow the boss to re-establish control**.

What is leadership?

Leadership is easy to recognize but hard to define. We'll define it by what a leader does. We are going to assume a teaching hospital situation here, where lots of hands are usually available. In rural hospitals, you might need to be more directly involved. A leader:

1. **Keeps situational awareness**, i.e. *knowing what is going on so you can figure out what to do.* To do this, you need to avoid getting bogged down into any one particular task and keep your mind free to graze over the whole situation. To maintain proper situation awareness, you must do frequent re-assesments of the situation. Thus, every 2 minutes or so, go though a full set of vitals (remember, the BP doesn't cycle unless you make it cycle) and reevaluate your priorities using the ABC approach. Two tricks are useful here:
    a. Keep your hands in your pockets or on the patient's femoral pulse. This prevents you from getting too focused on, say, helping with an IV insertion or Lifepack operation. *Free hands lead to a free mind*.
    b. If you do need to focus on a mental task, like interpreting an EKG or an X-ray, keep a mental timer set to 1 minute. Once the minute is up, you need to come out of your deep mental dive and "take a breath", i.e. look around and make sure the global situation has not changed.
2. **Controls and Directs**: You will need to assign roles and responsibilities to others. Best is to divide people in sub teams. If you don't have enough people, you might need to bundle up two or more responsibilities to one person. Usual sub-teams are:
    a. Airway- oxygen delivery, BVM and ETT/LMA placement
    b. Lifepack 12 and electricity- placing the leads and pads, executing defibrilation, pacing or cardioversion
    c. IVs and medications- establishing the IVs and preparing and administering the medications
    d. Documenting and communication- documentation of interventions and doses as well as calling other services like X-ray, RT, ICU, etc
    e. CPR- if needed. Best to have 3 people rotating through this (if available), as it is quite tiring if done well.
3. **Co-ordinates**: You must coordinate your sub-teams to best achieve your overall goals. The airway team wants to intubate, but the patient just went into Vfib? You need to inform the airway team to pause until defibrilation can be performed and the pulse returns. Remember that while you have the global awareness of what is going on, the subteams might be very, very focused on their specific tasks and be blind to everything else. The other part of this job is deciding on the order the tasks will be executed-"Ok team, the Xray is here, we are going to intubate and once the oxygen sats stabilize, we will do the chest x ray."

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4. **Plans ahead**: This is the defining job of the leader. While the subteams are focused on the present, you must think of the future. Figure out the likely evolution of the clinical picture, the steps necessary to stabilize it, and decide on the order of these steps, while frequently re-assesing the situation for radical changes.
5. **Communicates.** Communicates his/hers intentions to the sub-teams and acts as a clearinghouse for the information that the subteams are generating, as, again, they might be too focused on a task to hear what someone else is saying. Tips for effective communication are below.
6. **Prevents emotional contagion.** Panic is hugely contagious, worse then measles at a Jenny McCarthy convention. It makes everyone's job harder as panicked people have very little cognitive capacity. It is the job of the leader to set the emotional tone of the resuscitation, even though he/she is usually the most stressed person of anyone in the room. Many of our stress responses, such as pupil dilation and sweating are completely automatic. The two that you do have the most control over, however, are the one that other people pick up the most on- namely your facial expression and your tone of voice. The face of a panicked person often shows frank fear or is simply frozen in one position, and the voice increases in pitch and gets a tremor. Throughout this course, you must constantly strive to control these two expressions of fear as people are incredibly sensitive to them. It is not an easy task- pilots and military personnel spend years practising voice control alone. It is, however, an **essential task** of the leader. Welcome to the big leagues.

Final notes on leadership

There is much in the way that people are socialized today that prevents good leadership. Standing in front of your peers, saying that you are the one in charge then ordering others about is deeply uncomfortable to a lot of people in modern Western society. While a collaborative, shared decision making model works, and works well, it does so only with very experienced providers who have known each other for a long time. You will have neither of those luxuries in a typical floor resuscitation and with inexperienced providers, such leadership almost always results in chaos and indecision. Thus, for the time being, you must find your inner autocrat and simply tell people what to do. Allow mental space for suggestions from others and incorporate them into your decision making, but you must, at all times, make clear that you are the one in charge.

Effective Communication

Communication is key to effective acute response. Effective communication ensures that the receiver(s) understand what is being communicated, *and that the sender knows that it has been understood correctly*. Effective communication is essential in acute situations. This means it should be:

Clear - Concise - Closed Loop

Clear means it is clear WHO it is directed to, and exactly WHAT you want or mean.
Concise means no wasted words or long explanations.
Closed Loop means that you get some acknowledgement that the communication has been received and understood correctly. Often this means the recipient repeating the essentials back, or you asking a suitable question to **check understanding**.

Common Communication Issues

These are just a few examples of the types of common communication issues that can happen, especially in a pressured situation. Recognize any of these? Think how you can encourage better communication in an acute setting where the team may not know each other.

Issue	Typical Phrases	Better
Not being clear who you are talking to	"can someone.."	"Jim, can you…"
	"I think we need to…."	"You two doing CPR …"
Not being clear about what you want	"We need to press the patient…"	"Jane, give phenylephrine 200 mcg IV now please?"
	"They need epinephrine.."	"Mike, give 0.3mg epinephrine IM now".
Not concise	"Um, I think we might need to help the patient breathe soon.. maybe some supplementary O2… they seem to be having trouble… a bit cyanotic maybe…"	"Deepra, rebreather mask with 15L/min O2 please, and let me know what difference that makes to the patient's O2 sat and shortness of breath in 2 minutes".
Not checking understanding	Give drug order, no acknowledgement.	Recipient repeats drug order back, confirms administration.

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NIGHTMARES COURSE - DRUGS AND DOSES SUMMARY

	SVR	HR	Contractility	Types of shock	Dose	SHOCK
Dopamine	Low dose: + High Dose: ++	++ +++	+++ +++	Any	5-10 mcg/**kg**/min (low) I 10-20 mcg/**kg**/min (high)	
Phenylephrine	++++	0/-	0	Any except cardiogenic	100-300 mcg/min	
Nor epinephrine	+++	+	+	Any	2-15 mcg/min	

	Mechanism	Dose	Effective in	TACHYCARDIA
Cardioversion	electricity	200J	Any	
Amiodarone	multiple	150mg over 10-20min	Most tachys Avoid if wide and irregular Avoid if A fib/flutter >48h	
Adenosine	Intense AVN blocker	6 or 12mg rapid push	Avoid in wide and irregular QRS Diagnostic aid in rapid Afib/flutter Curative with SVT	
Diltiazem	AVN blocker (Ca channels)	20-35mg IV 10-15mg/hr infusion	Avoid in wide QRS Effective any narrow QRS	
Procainamide	Na channel blocker	1g over 1 hr	Any Avoid if Afib/flutter>48h, acute CHF Vasodilator-may need 250cc bolus	
Metoprolol	AVN blocker (Beta 1)	2.5-5 mg IV q10-15 min X3	Avoid in wide QRS Effective any narrow QRS	

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	Receptor	Dose	BRADY CARDIA
Atropine	Ach blocker	0.5mg at a time	
Dopamine	Beta 1 stimulant	2-10 mcg/**kg**/min	
Fentanyl	Pain killer for pacing	1 mcg/kg (50-75mcg usually)	

	Dose	***Side effects* and duration**	HYPER K
Ca gluconate	1gr IV over 10 min, up to 3 doses	30-60min Hypercalcemia	
Ventolin	10mg nebulized	2 hrs Tachycardia	
Insulin R	0.1u/kg IV Bolus+ per hour drip	2-4 hrs Hypoglycemia	

Disclaimer: Use of this table does not absolve physician from ensuring that doses are correct.

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	Mechanism	Dose	Use	ALTERED LEVEL OF CONSCIOUSNESS
Narcan	opoid antagonist	0.2-0.4mg per dose, no max	opoid overdose affecting LOC or respiration	
Diazepam	sedative	5-10mg IV/IM q5-10min	agitation, seizures	
Lorazepam (Ativan)	sedative	2-4mg IV/IM q5-10min	agitation, seizures	
Dextrose	Sugar	1g/kg 25g per D50W amp	hypoglycemia	
Dilantin	antiepileptic	1g IV over 20min	Seizures	
Propofol	sedative	20-40mg IV bolus for sedation 100mg for induction 20-80 mcg/kg/min drip	agitation, seizures generally will need airway control	
Mannitol	osmotic diuretic	1-2g/kg	intracranial bleed with increased ICP	
Labetalol	beta blocker	1-2mg/min start then titrate to BP	SBP>160 or dBP>90 in SAH	
Octreotide	somatostatin analogue	50mcg bolus and 50mcg/hour infusion	antidote for sulfonylurea poisoning	
Midazolam	sedative	1-3 mg IV q30 min	Agitation, sedation	

Drug	Dose	MI
ASA	160mg po	
Clopidogrel	600 mg load then 75 mg po od	
Enoxaparin	1mg/gs sc q12h	
IV Heparin	5000u bolus then per nomogram	
Metoprolol	25-50mg po	

Disclaimer: Use of this table does not absolve physician from ensuring that doses are correct.

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	Dose	Conditions	SHORTNESS OF BREATH
BiPAP/CPAP	10-20mm inspiratory (8 in asthma) 5-10mm expiratory (3 in asthma)	COPD CHF pneumonia asthma	
Puffers	5mg Ventolin 500mcg Atrovent	COPD asthma	
Epinephrine	3mg 1:1000 nebulized 0.3mg 1:1000 IM	Asthma anaphylaxis	
Benadryl	1mg/kg or 50mg IV/IM/PO	Anaphylaxis	
Lasix	60-80mg IV	CHF	
Nitroglycerin	2-20+ mcg/min	CHF	
Magnesium	2g IV over 20 min	asthma	
Rantitidine	1mg/kg or 50 mg IM/IV, 150mg PO	Anaphylaxis	

	Dose	**Conditions**	GI BLEED
Octreotide	50mcg bolus then 50mcg/hr	Variceal bleeding	
Pantoprazole (Pantoloc)	80mg bolus then 8mg/min infusion	PUD bleeding	

	Dose	***Side effects* and duration**	INTUBATION
Ketamine	1-2mg/kg (usual dose 100mg)	2-4 min peak, 40 min duration	
Propofol	1-2mg/kg (usual dose 100mg)	20-40mg IV bolus for sedation 100mg for induction 20-80 mcg/kg/min drip	
Rocuronium	1 mg/kg	40 mins duration.	

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correct.

	Dose		Conditions	ANAPHYLAXIS
	Adult	Peds		
Epinephrine (most important)	0.3mg IM	0.01mg/kg IM	q5-15min	
Diphenhydramine (Benadryl)	50mg IV (max 400mg in 24h)	1mg/kg IV (max 200mg in 24h)		
Ranitidine (Zantac)	50mg IV	1 mg/kg IV		
Methylprednisone (Solu-Medrol)	1-2mg/kg/day		Prevent rebound. Dose 3 days.	
Ventolin	Prn	prn	Bronchospasm not responsive to epinephrine	

Disclaimer: Use of this table does not absolve physician from ensuring that doses are correct.

THE END

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