Department of
Family Medicine

"The Nightmares Course"

(Acute Care for Family Docs)

Department of Family Medicine

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Introduction to the Course

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, as well as in your future practice if you choose a career that has a hospital-based component. The course grew out of residents' concerns that they were being asked to resuscitate patients on the floor without having the adequate experience and knowledge to do so effectively. Also, we have 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 **a*nd is a required reading before you show up for your first 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. It was written to be physiology-based and easy to read. I am hoping you will not try to memorize anything 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 experienced help arrives, no matter what the situation.

The course itself comprises of 4 sessions: the initial 2 day session and 3 follow-on sessions interspersed throughout your PGY-1 year. The initial 2 day session consists of a half day "basic training" where we will solidify all the basic skills needed for resuscitation, like operating a defibrilator or escalating oxygen therapy, followed by modules that focus on the 4 core topics. Each module will consist of a brief didactic lecture to solidify the content and 4-5 simulation scenarios.

The follow-on sessions each have a different focus. The first one will expose you to scenarios of increased complexity where multiple vitals are compromised and will force your decision-making ability to grow.

The second session involves transporting a critically ill patient from a rural hospital to a tertiary care center and will add logistical challenges to the scenarios

The final session will be a "muscle memory" session where all the previous knowledge will be solidified as we throw a large number of rapid scenarios at you.

Throughout the course, we focus on making this a fun and interesting experience that will make you a better and safer doctor. The onus on learning, however, remains with you and I urge you to review the manual and the algorithms ***before each and every session*** in order to get the most our of them."But this is a pain in the ass!", I hear you say. It might be, but our previous experience with residents shows that if you don't review before a session, we will waste most of it going over the old stuff rather then pushing on. Thus it is in your best interest to come to each session as well prepared as possible. While knowing the medication dosages is not mandatory, I also 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!

Filip Gilic

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"

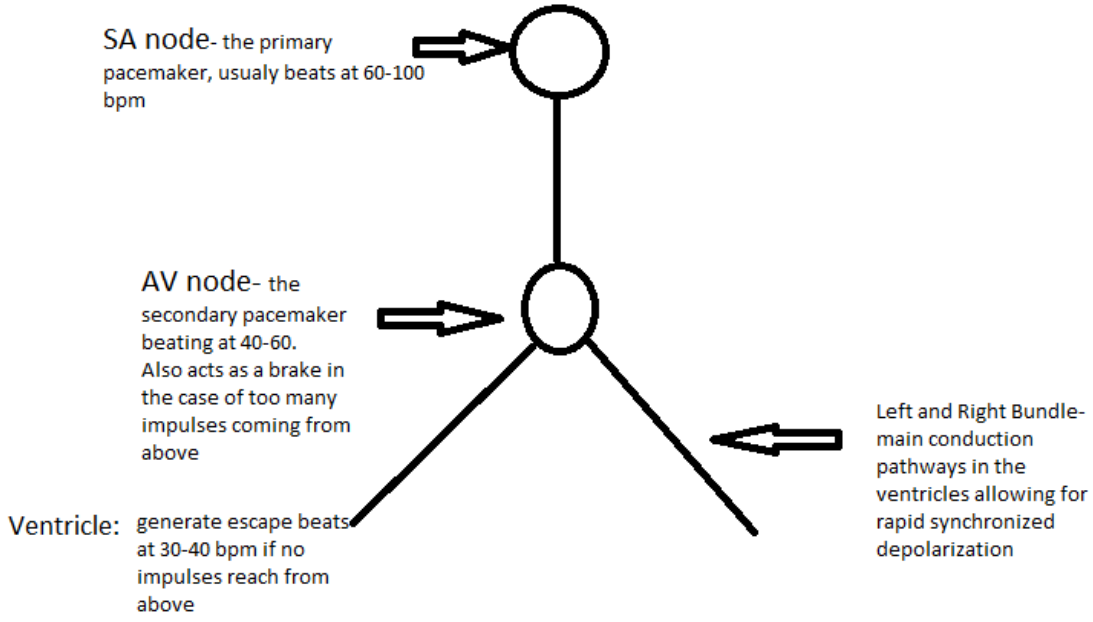
- motto of the Royal Air Force Parachute School

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.***

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

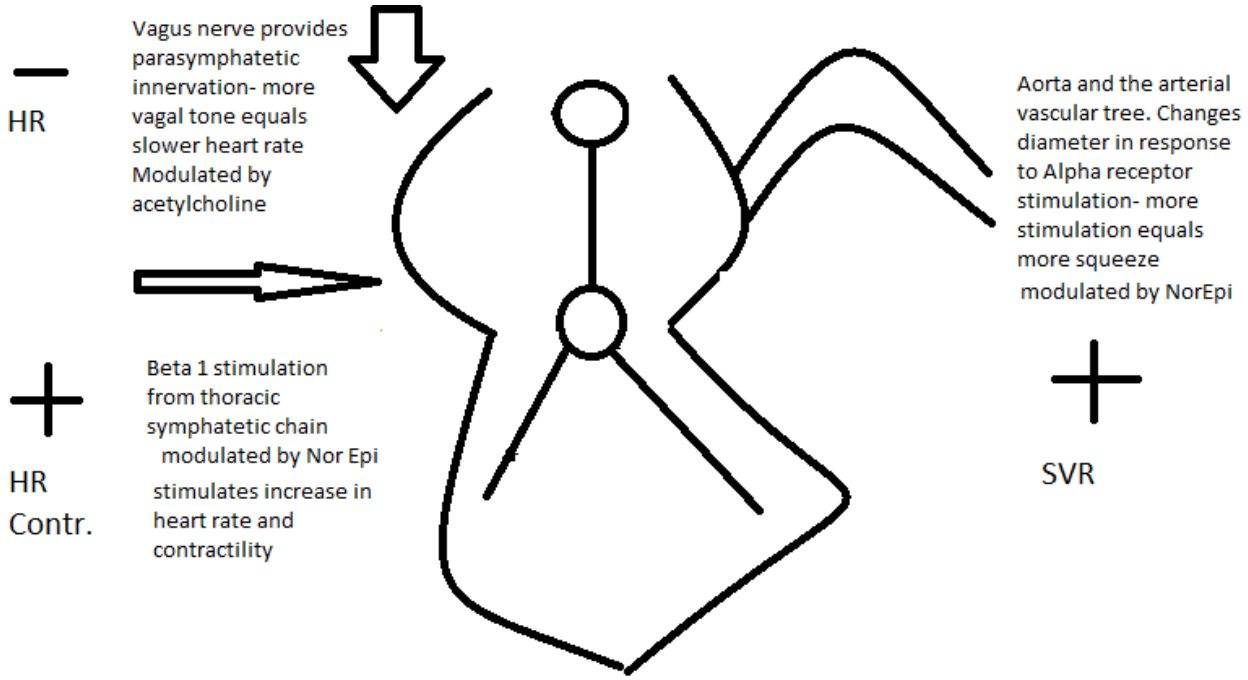
an increase in vagal tone results in a decrease in heart rate.

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

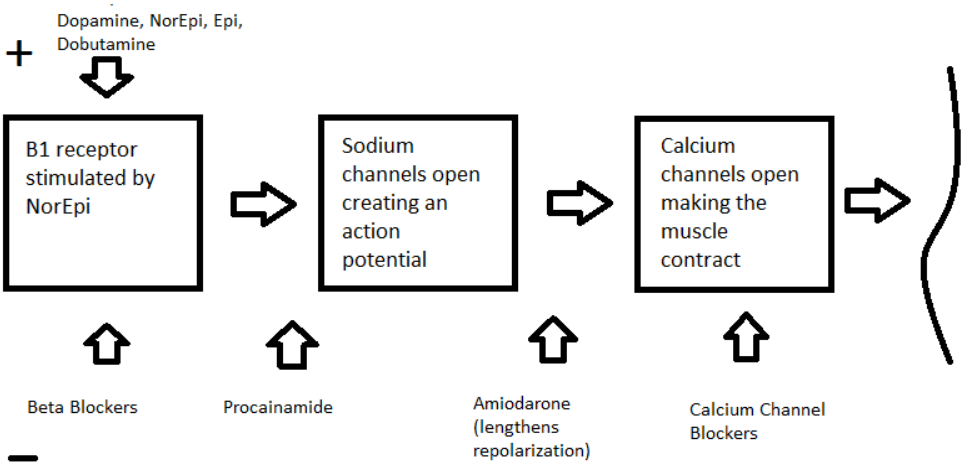
Key Concepts

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

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):

$$BP \alpha CO \times SVR$$

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 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.

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).

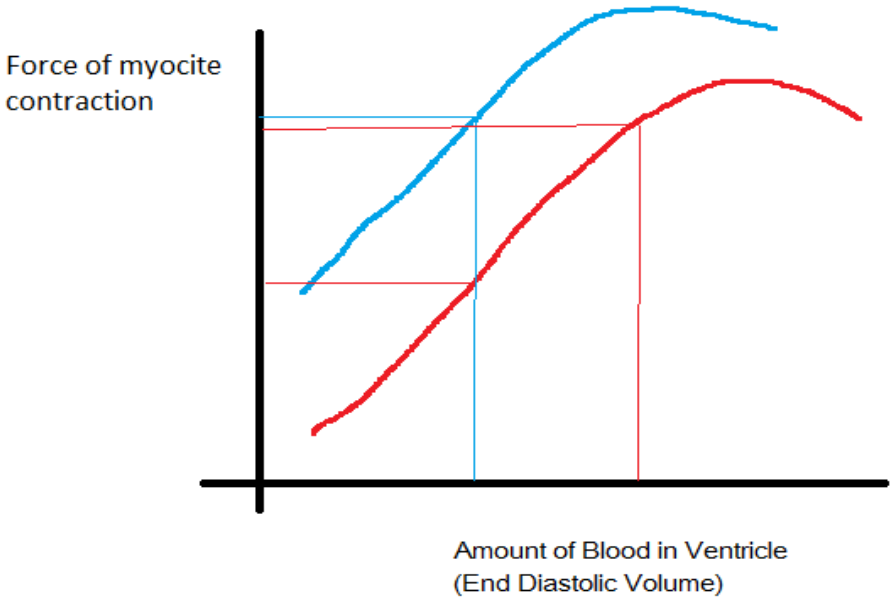
Stroke volume depends on 3 factors: Preload (Pl), Contractility (Co) and Afterload (Al).

SV α Pl x Co x Al

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)

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.

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 - **this down curve only happens in impaired hearts, though-healthy heart can tolerate essentially any amount of fluid and not go into failure**). Blue line shows what happens when we add BETA1 stimulation- we get more force for the same stretch.

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

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. 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 (like 14-18 gauge) and a pressurized delivery system.

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

Rapid Infusion Tips

If you leave an infusion just gravity fed, it will take about 10 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. When you need rapid infusion, the easiest way is to wrap a blood pressure cuff around the fluid bag and inflate the cuff to 300mmHg. With that, you can infuse about 500c/minute through a 16gauge IV in a large vein.

***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*:** cardiogenic shock occurs:

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 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 is usually ok as it helps boost the preload and thus help stretch myocites and thus passively increase contractility.

<< This type of cardiogenic shock most commonly happens with big left sided STEMIs that kill or stun enough of the myocardium on the left side that the left ventricle becomes weak; or sepsis where the infection leads to malfunctioning myocardium.

2) When the heart rate is low enough (for example with a heart block) that it leads to inadequate cardiac output, despite normal contractility. Here, again, we have to stimulate BETA1 receptors to increase the heart rate, or cut the vagal tone with Atropine; and *it is safe to give fluid as contractility is fine*.

3) When an arrythmia causes the heart rate to become too fast for the age, leading to inadequate diastolic filling time. The solution here is to recognize that the heart rate is not a sinus tachycardia and then act according to the type of arrythmia (more on that later).

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

	SVR	HR	Preload	Contractility	Afterload	To fix
Hypovolemic (common)	Normal	Normal	decreased	Normal	Normal	Fluid Possibly blood
Distributive (common)	decreased	Normal	decreased	Normal	decreased	Fluid, Alpha stimulation
Cardiogenic (not common)	Normal	decreased in bradycardia	Normal	decreased	Normal	Beta1 stimulation Ach blocking in bradycardia
Obstructive (rare)	Normal	Normal	decreased	Normal	increased	Fluid, Alpha, Beta 1 stimulation

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.

Not Got 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 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, it usually takes hours for this to occur, and 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. We use Dopamine in this course because it is present on all the crash carts; it comes premixed, and the floor nurses are more comfortable with it and thus more likely to actually administer it.

Phenylephrine*:* is a pure ALPHA stimulant, i.e. a pure vasopressor, with minimal effect on heart rate or contractility. It is my favourite drug to use as a bolus. 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 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.

Phenylephrine Premix

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.

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 use 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.

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	+	Distributive	2-15 mcg/min
Phenylephrine	++++	0/-	0	Distributive	100-300 mcg/min
DoBUTamine	--	+++	+++	Cardiogenic Obstructive	2-20 mcg/**kg**/min

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 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 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.

In any case, giving a moderate amount of fluid (500cc) and, if that doesn't work, starting a Dopamine infusion at 5mcg/kg/min and titrating to MAP of 65 or above is almost never wrong.

Arrythmias

Increased heart rate

0th step. ABCs

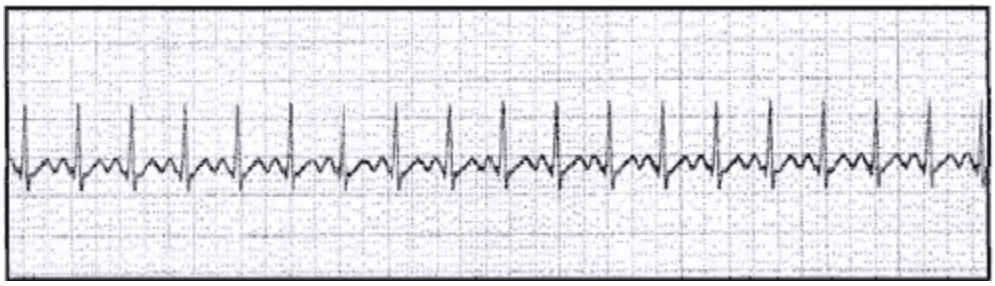
As in all resuscitation scenarios, ABCs rule. Get the patient IV-O2-monitor, a full set of vitals and ensure that the airway is patent.

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, fear, caffeine, etc) and not the cause of the patient's problem.. The one exception to this is an old person with chronic and constant atrial fibrillation for whom a faster then usual A fib might simply be a reaction to the same stimulation that produces a sinus tachycardia in a non-Afibber. Over the last few years, I have solved fast A fib in such patients with a Foley (urinary retention), a chest tube (pleural fluid) and Tylenol (fever).

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



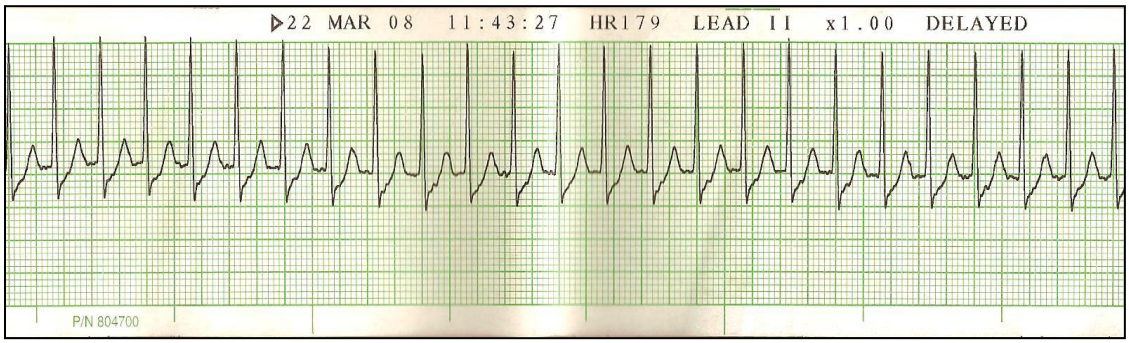
Sinus tachycardia. Note the p before QRS/ QRS after every p.

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. 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 and all* ***regular*** *wide complex tachycardias* (and irregular wide complex tachys are unlikely to be sinus anyways).



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

2nd step. Stable or unstable?

Is the patient stable or unstable?

To me, 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, no matter what the ACLS algorithms say.

Thus, by my definition, if the patient is confused or with a decreased level of consciousness (insufficient blood supply to the brain) or with angina-type pains or shortness of breath (insufficient blood supply to the heart), they are unstable.

> ***Unstable?***
> ***The answer to instability, no matter what kind of tachycardia is causing it, is <u>always electricity</u> in the form of synchronized cardioversion.***

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.

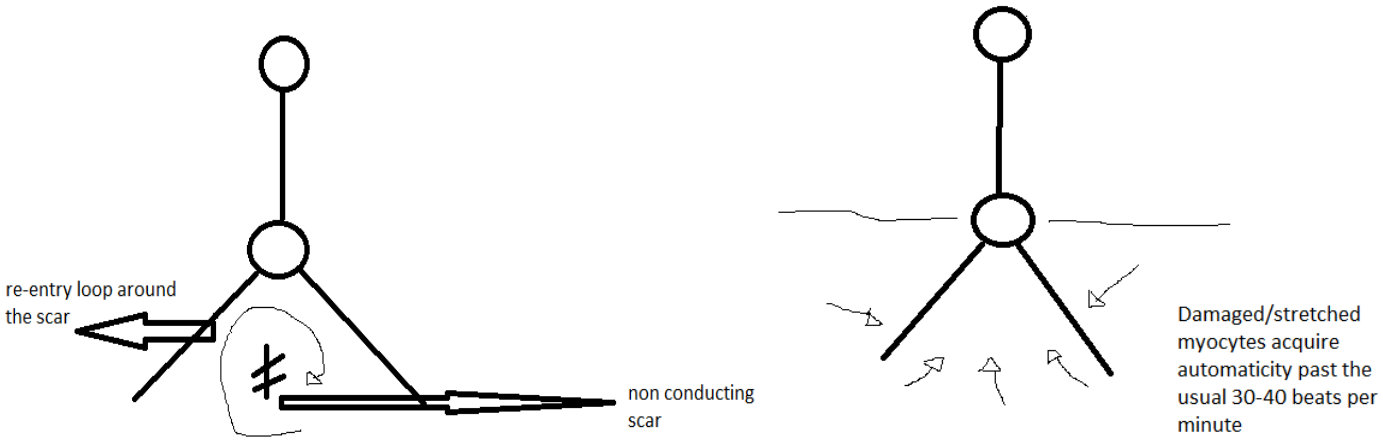
It is a very painful procedure, so be kind and give the patient some pain medications and sedation before you do it, if you have the time.

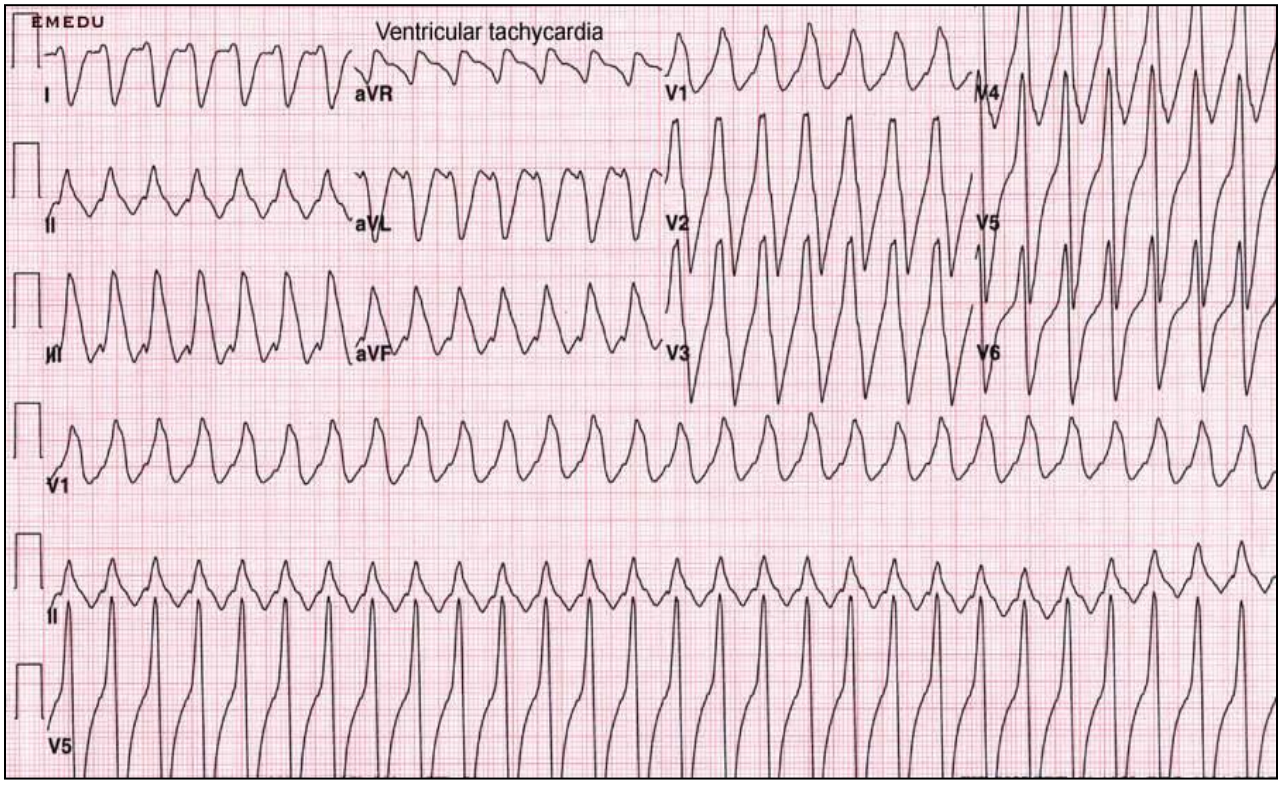
3rd step. Narrow or wide?

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 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 and it can be lethal. There is a subset of people

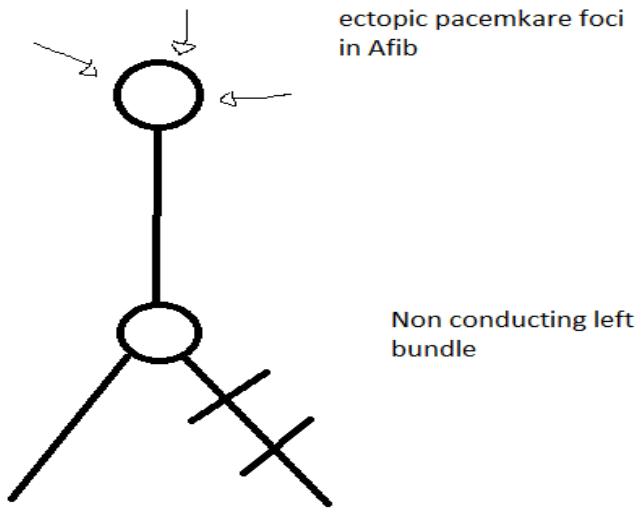
who have never had a heart attack but are predisposed to it due to sodium channel malfunctions and walk among us in intermittent VT and not necessarily any worse for wear.





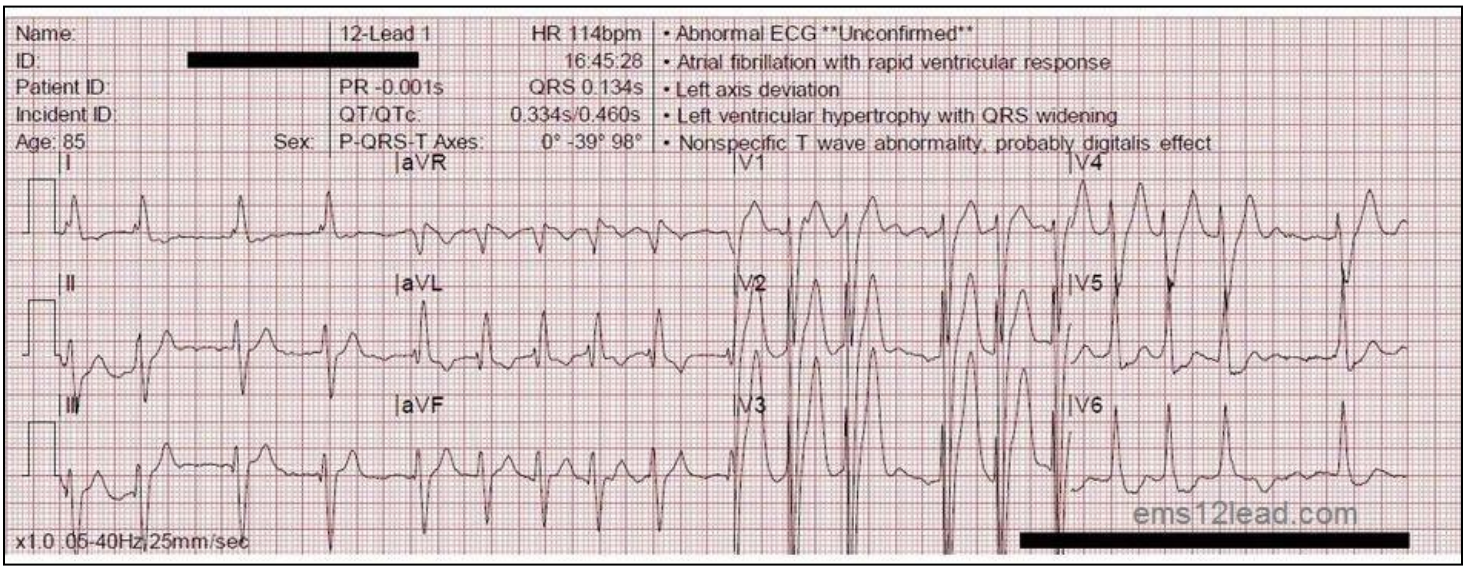
VTach – note wide complex, i.e. cell-to-cell conduction

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. 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.



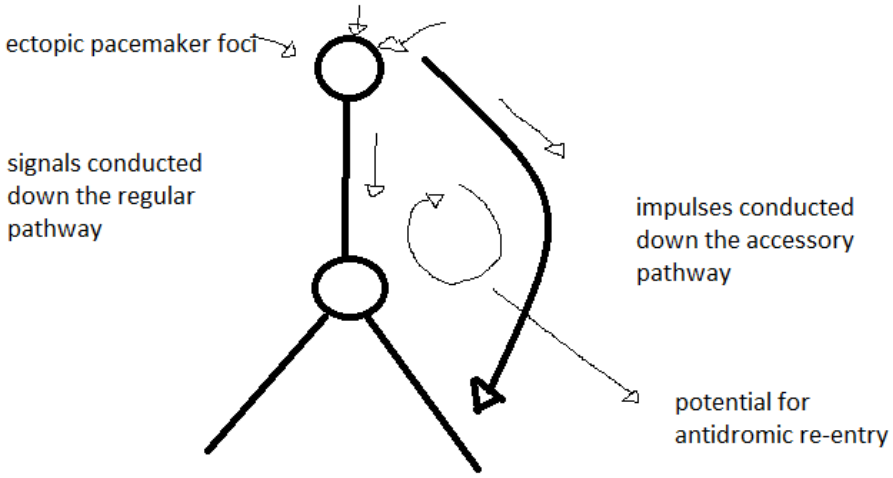
Left Bundle Branch Block

The rhythm has the characteristics of the left bundle branch block- namely, very positive (upright) QRS in lateral leads (I, aVL, V5-6) and negative (down) in anterior (V1-4) with discordant QRS-ST segments (i.e. where the QRS is positive, ST is negative, and vice versa) and can be regular (if it is sinus) or irregular (if the underlying rhythm is Afib). This is treated just like the underlying rhythm (sinus or afib) and the presence of LBBB has no bearing on treatment, the tricky part is recognizing it as such.



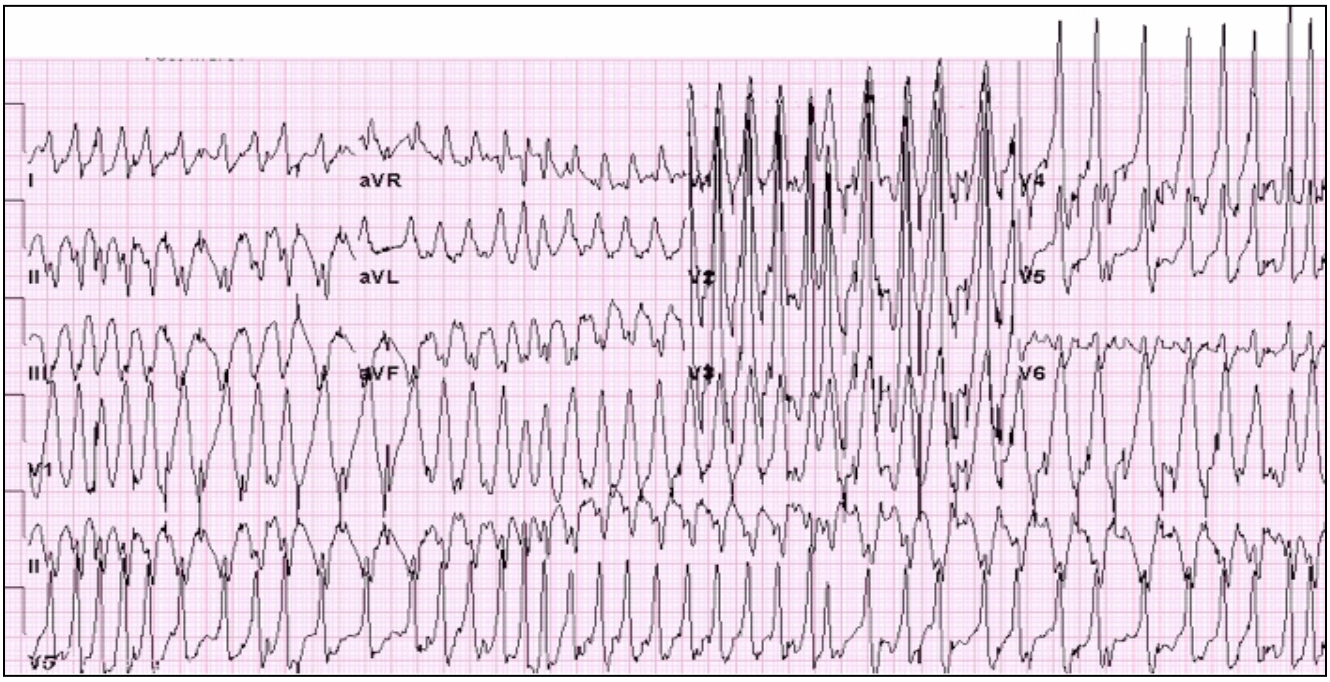
EKG of Afib with LBBB. Note QRS-ST discordance and pronounced left axis

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.



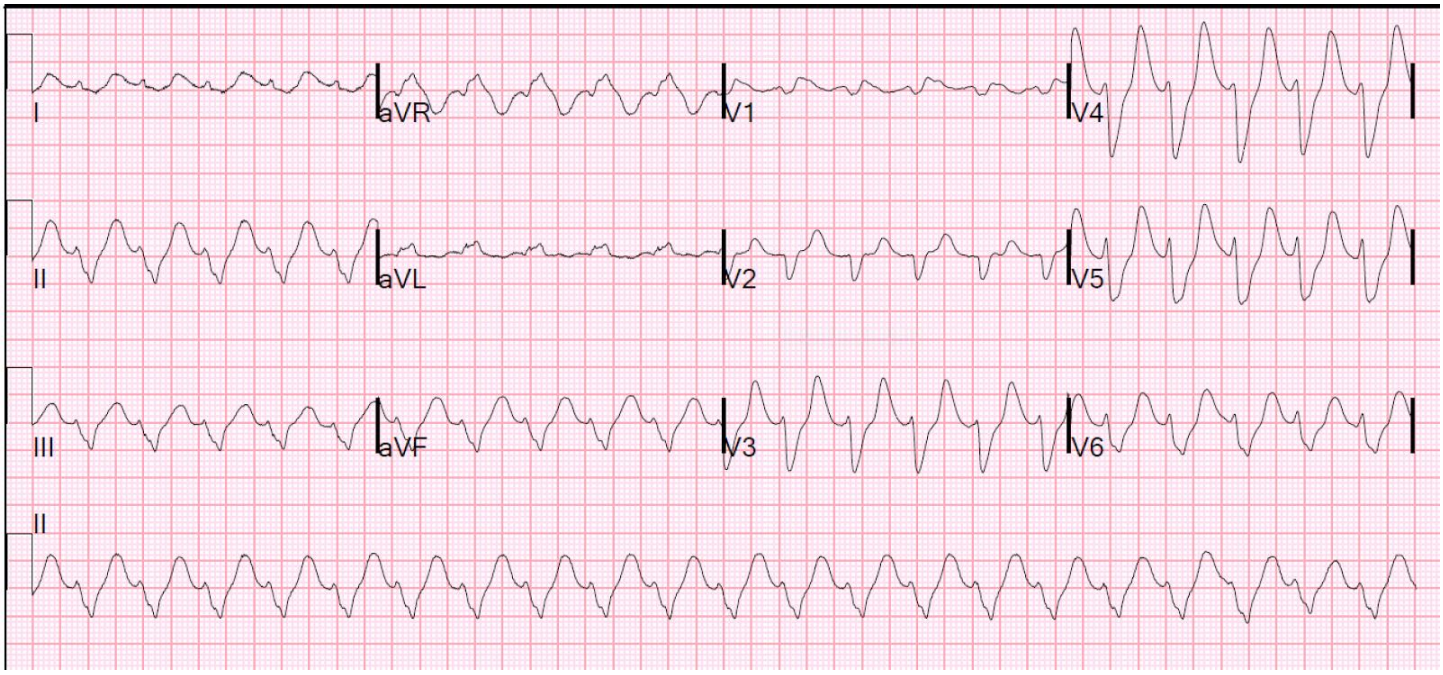
Antidromic Re-entry

Accessory pathways can result in an antidromic re-entry where the impulses are travelling through the accessory pathway, to the ventricle, then *back up the AV system*, to the atria, and back to the accessory pathway. ***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.***



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

4 . The body has been **poisoned by something that affects the heart cells' sodium/potassium pumps**- a trycyclic antidepressant, an anti-arrythmic or high potassium. These patients will generally end up bradycardic over time, but when you see them, they might still be going fast but with a widened QRS. We will not cover the poisoned patient here, just increased potassium. Give an amp of Ca Chloride (which is on the crash cart) or Ca Gluconate (which is not, and needs to be premixed) to stabilize the cardiac membrane, then administer insulin R 0.1u/kg bolus plus 0.1u/kg/hr infusion together with a glucose infusion (~150cc of D10W per hour). Also put them on a Ventolin nebulizer at high doses (10mg). If they are acidotic, can also give 2-3 amps of Bicarbonate (no effect if the patient's pH is normal).



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.

The one principle to remember with the patients with a fast and wide rate is that *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.**

The reason for this is that if the cause of the rhythm is the number 3 scenario (IE a WPW with A fib), blocking the AV node will force the electricity to go through the accessory pathway and might induce VFib. Thus, we want to use something that will slow down the conduction throughout the whole heart.

Which Drug to Use?

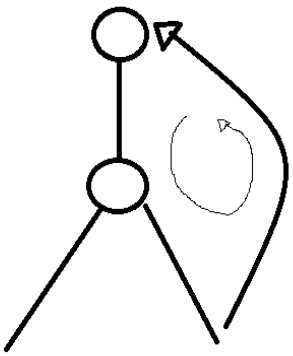
Amiodarone was in fashion for years, but now there is some evidence that it might be dangerous specifically for Afib with WPW and that ***Procainamide (1g given over 1 hr) is safer***. Thus, you can try that, or simply do an elective cardioversion, which is probably safest anyway, assuming the arrythmia has been going on for less then 48 hrs (if it is Afib with LBBB or Afib with WPW, there is a small risk of a stroke if you cardiovert and they have been in it for more than 2 days).

Step 3. 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 or Aflutter. In reality, Aflutter is often quite regular, but, as you will see below, nothing bad will happen if you treat Aflutter as an SVT.

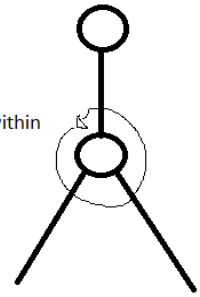
SVT is a catch-all phrase to indicate any tachycardia that is not a VT. Sinus tachycardia, A fib and Aflutter are all technically SVTs. In common usage, when we say SVT, we mean fast, narrow, regular non-sinus tachycardias that *will likely respond to Adenosine*. The commonest ones are AV Node Reentry Tachycardia

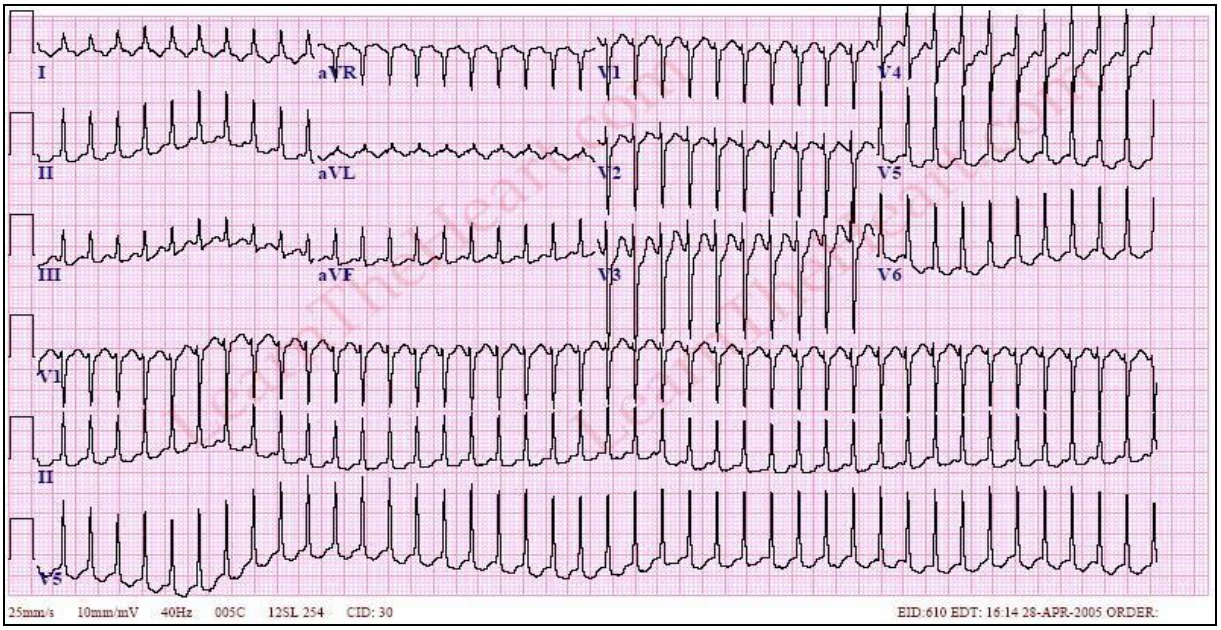
(AVNRT) and AV Reentry tachycardia (AVRT). As the name suggests, they both are caused by re-entry loops, and the difference is simply in location of those loops. They look very similar and, again, it is not really necessary to distinguish them as they will respond to the same treatment.



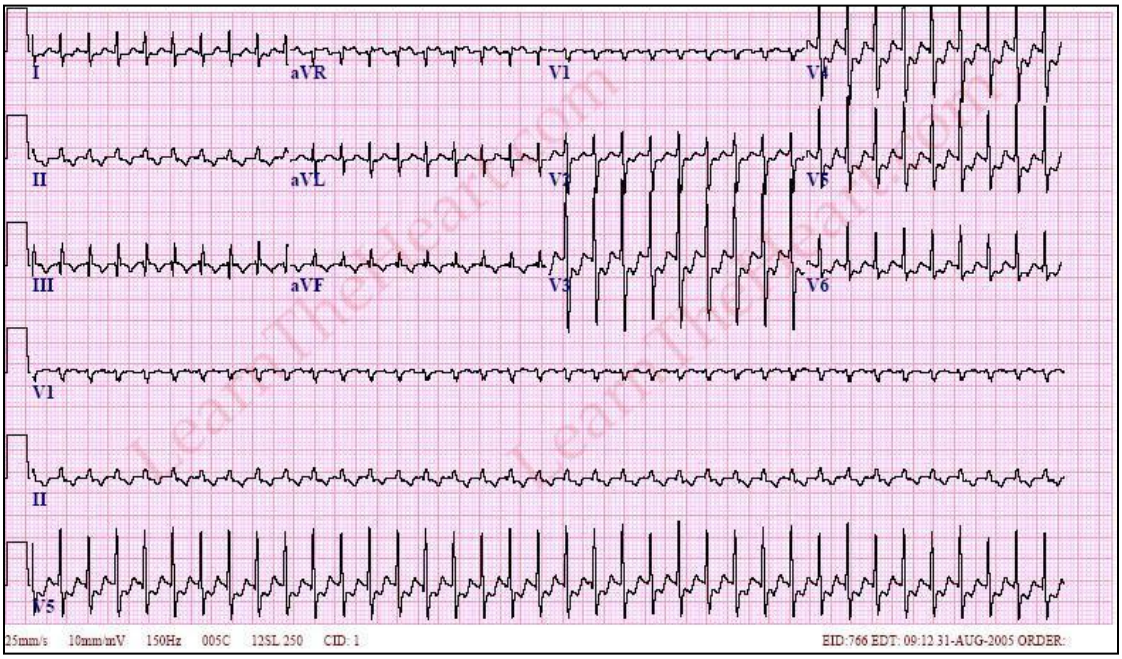
AVRT: re-entry loop using an retrograde (upwards) conducting accesory pathway

AVNRT-reentry within the AV node





AVRT



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 use Adenosine, the most potent AV nodal blocker that we have. Since it is short lasting (5-10 sec), it would not have an effect on arrythmias that are caused by more permanent foci of arrythmias (A fib), but for AVNRT/AVRT it is usually sufficient.

We start at 6mg and try again at 12mg if it is not working within a couple of minutes. If it is persisting, we can try more permanent AV nodal blockers such as Metoprolol (2.5-5 mg IV every 10-15min, for a max of 15mg) or non DHP Calcium channel blockers, usually Diltiazem 15-25mg). You can even try Amiodarone.

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.

If the narrow QRS rhythm is irregular, you are most likely dealing with **A fib/flutter**. They are caused by more permanent arrhythmic foci, so a short acting agent like Adenosine will not work in the long run, though it is not wrong to give it- it will slow things down for a little while.

If it looks like A fib/flutter, 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/flutter.

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 many

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.

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. Chemically, we use Amiodarone 150mg over 10-20 minutes, or Procainamide 1g over an hour. Both can prolong QT intervals, so watch out for that. If the drugs are not working (which will happen in 25-50% of the time) in that time-frame, 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 only medications that have actual evidence are Diltiazem and Esmolol. Esmolol is a very short acting beta blocker that is mostly used in the OR, so we focus on using Diltiazem at a dose of 15-25mg IV over 1-2 minutes, +/- infusion of 10-15mg/hr. Diltiazem reaches serum peak in 2 minutes, so you know pretty quickly if it will work or not. Resting heart rate of **<110** is adequate. If that didn't fully work you can try Metoprolol 5 mg IV at 5-10 min intervals but you should be a bit cautious- if they spontaneously convert to sinus rhythm, they might be quite slow with both BB and CCB 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 **except** **wide** and **irregular** rhythms.

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/flutter for rate control (85-90% success) if given in boluses of 20-35mg IV, slow pushed over 1-2 minutes. 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.

Metoprolol- Beta blocker. Used in same circumstances as Diltiazem but has no evidence of efficacy in Afib/flutter acute rate control 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.

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 it NOT safe in Torsades de Pointe and WPW with Afib (most likely because of it partial beta blocking capabilities), so it is probably best to stay away from it in **wide** and **irregular** rhythms. 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/flutter to sinus but the mean time to conversion is 5-6 hours so not very useful in acute settings. Can be safely used in Afib/flutter patients who are in acute CHF. Dose for dead people is 300mg IV rapid 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/flutter 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 acute CHF/pulmonary edema.

	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
Ca Chloride	Membrane stabilizer	1gr over 10 min	Hyperkalemia

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 the patient is stable, you do not have to cardiovert them, but ultimately can do so if other things fail and duration is <48h.

If you are seeing wide QRS complexes, and are sure it is a LBBB, just treat the underlying rhythm as usual. Check potassium and treat with Calcium and Insulin if high

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, as long as the duration of symptoms is less then 48h. If it is more then 48 hrs, call a cardiologist.

If you are using medications, Procainamide is safe in every tachycardia setting lasting less then 48 hrs. AV nodal blockers are safe in any kind of narrow QRS tachycardia.

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.

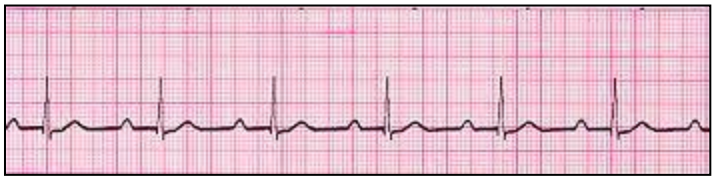
Causes of Bradycardia

1. The heart's conduction system is worn out, leading to sinus bradycardias with the potential for long sinus pauses (sick sinus syndrome)

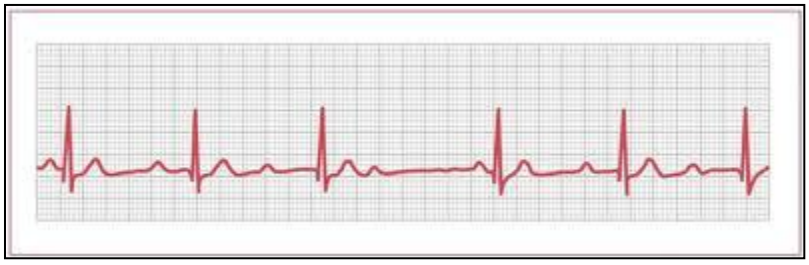
2. Because a degree of separation of conduction has occurred between the atria and the ventricles (AV block)

3. The heart has been poisoned (hyperkalemia, beta blocker overdose, tricyclic overdose, etc). *Again, this is a special case which will not be covered here.*

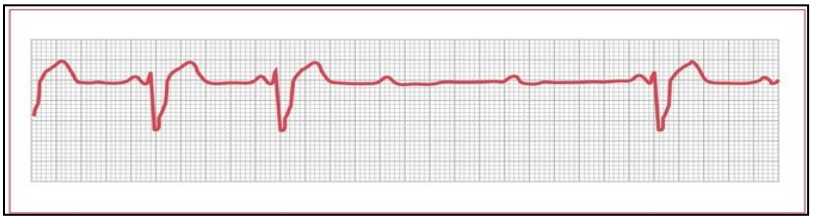
If the patient is stable, you should determine whether they have a type of block that can lead to asystole or instability, and if so, prepare them for possible pacing and refer them on. ***The potentially dangerous types of AV blocks are 2nd degree type 2 (because it can rapidly lead to 3rd degree) and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



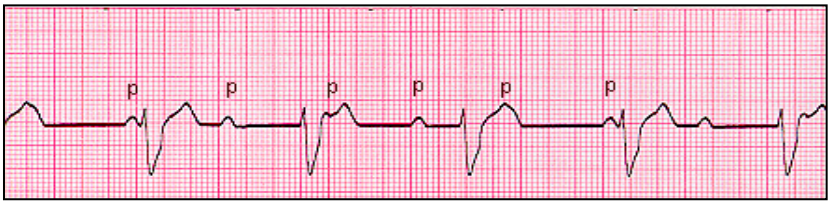
1st degree AV block – fixed long PR interval



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



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



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

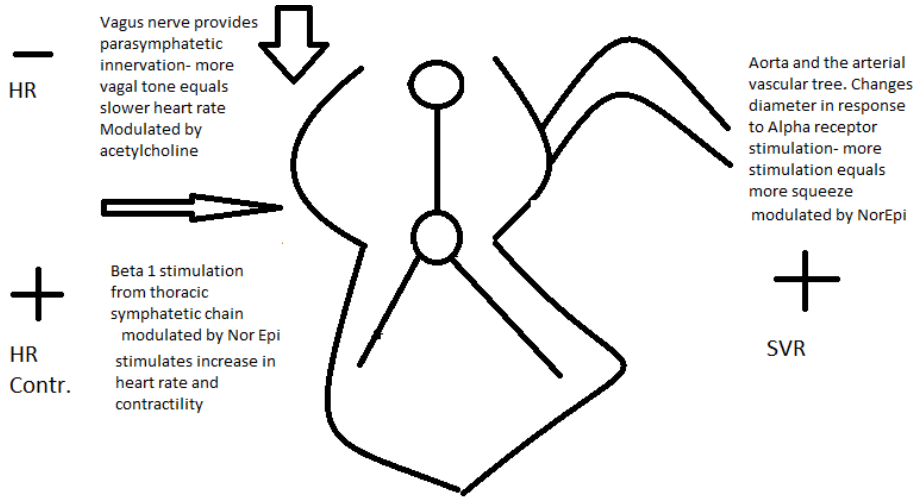
External Pacing

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.

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	2-10 mcg/**kg**/min
Fentanyl	Pain killer for pacing	1 mcg/kg (50-75mcg usually)

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.

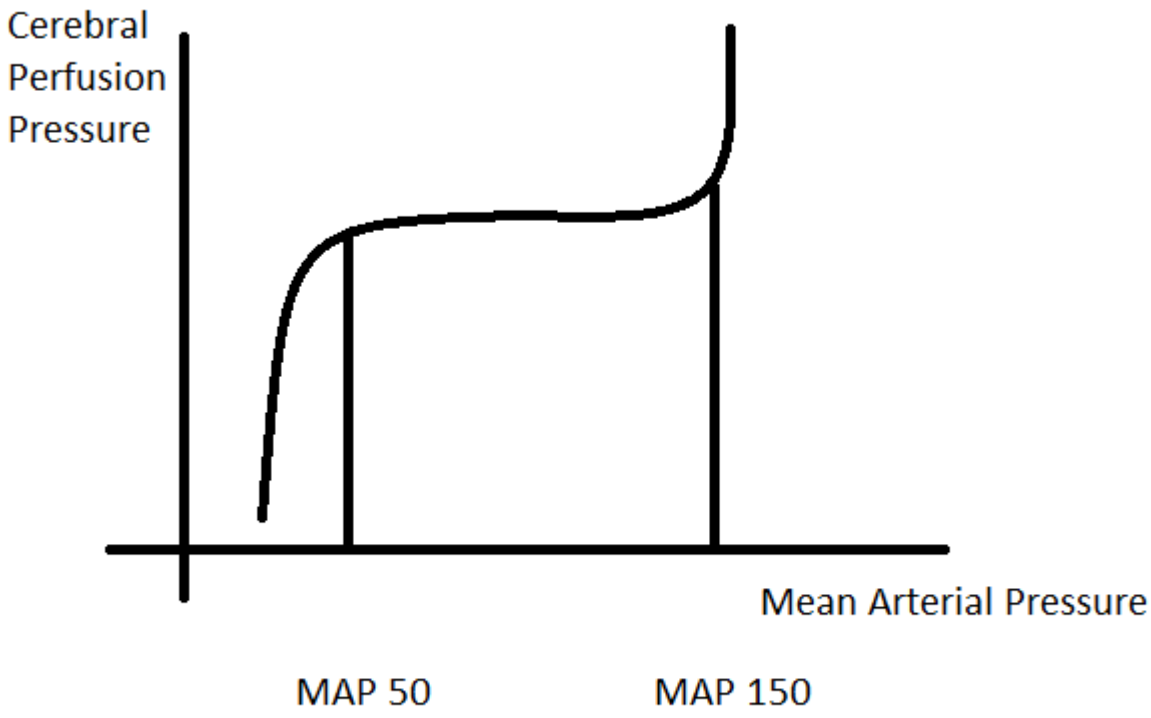
If they are holding their own with regards to blood pressure but are in 2nd degree type 2 or 3rd degree AV block, you should put the pads on them to be able to pace them if they decompensate, and send them to cardiology for a permanent pacemaker.

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.
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 35 and 42 degrees C for optimal conditioning.
5. **Intact brainstem and both cerebral hemispheres**. This is the reason why ischemic strokes relatively 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, simply has to produce enough of an increase in intracranial pressure to 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 (meningitis and encephalitis), medical drugs (benzos, etc), drugs-for-fun (cocaine, EtOH) and over-the-counter drugs (Gravol, Benadryl).



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.***

LOC scenario questions.

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

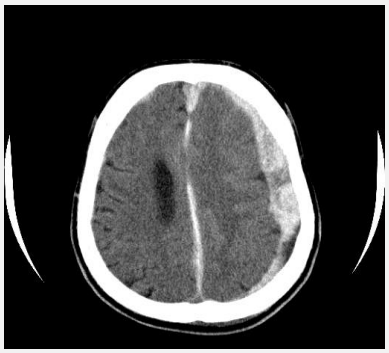
1. ***What is the glucose?*** EVERY patient with a change in consciousness needs to have his glucose checked so that is my first instruction to the nurse as I enter a room. If it is less then 4, give 1-2 amps of D50W. If it says high, get serum glucose, a set of VBGs/ABGs and think of DKA/HONK.
2. ***Is 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 an adequate gag reflex to protect 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 I suggest you use AVPU scale instead (Awake, responds to Voice, responds to Pain, Unresponsive). If they are P or U, call for help that can intubate them or do it yourself. Another trick I use is to put an oral airway in their mouth and if they DO NOT gag, I go ahead and intubate them.
3. ***What is their Oxygen saturation?*** It needs to be north of 90 percent if at all possible. Thus, everyone should be put on 100% O2 initially, and if that is not enough, mostly because they are not making an adequate respiratory effort, they need to be assisted, either through a Bag Valve Mask, an LMA or an intubation. BVM is perfectly adequate if you are not comfortable with intubating. Don't bother with getting an ABG at this stage- acidosis and hypercarbia will kill a brain far slower then lack of O2, so for now just focus on that O2 saturation monitor.
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. Call for help if not comfortable with these drugs.
5. ***What is their temp?*** Need to be in 35-42 range. Warm or cool with warm or cool fluids as appropriate.
6. ***What are the pupils like?*** Pinpoint pupils bilaterally are a pretty sure sign of opoid overdose, 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. If the pupils are unequal and/or fixed, the patient likely has an increased intracranial pressure.
7. ***Are all 4 limbs responding equally to verbal/painful stimuli?*** If not, a stroke or a bleed are 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. ***Is there history of poison ingestion*?** If yes, focus on toxidromes, not specific toxins. Use pupils to guide you: small pupils for narcotic/parasymphatetic and dilated for sympathomimetic/anticholinergic. Urine tox screen rarely adds anything useful that you can't figure out from the clinical picture since treatment is purely dependent on toxidromes rather then specific drugs.
11. ***If signs of bleed or stroke, what does the CT show?*** Note that the CT is nor an immediate priority-ABCs and avoiding hypoxia/hypoglycemia/low BP take precedence. Only take someone to the CT scanner once the above are stable.
12. ***If there was fever*** or other signs of infection, what does the LP show?

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>94% Keep BP – MAP 50-150 (preferably in normotensive range, MAP 80-90)	Pupils unequal- bleed- call help, raise head of bed, intubate, really avoid low O2 and low BP 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!*** <<<<<	

Special Cases

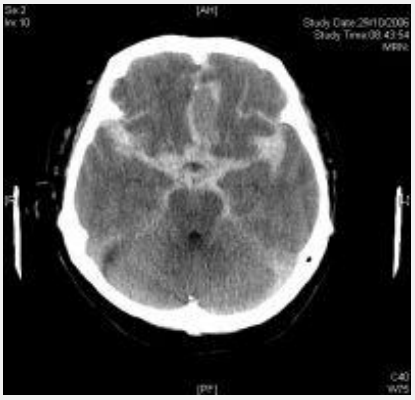
Intracranial bleed.

Intracranial bleeds come in three varieties. Subarachnoid (traumatic SAH is different etiology than a spontaneous, aneurysmal SAH, but is treated much the same), subdural (a slower bleed coming from torn venous sinuses) and epidural (arterial bleeds).



Subdural

There are differences in treating these three, 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.



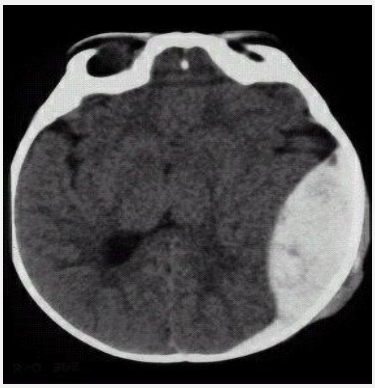
Subarachnoid (SAH)

In the meantime, **call for help**. If there is obtundation, intubate to secure the airway and facilitate proper oxygenation. We use Etomidate to intubate bleeds as it does not cause hypotension like Propofol. Put their head up 30 degrees.

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.

SAH is the one type of bleed that is also made worse by hypertension, so if the blood pressure is high (sBP>160, dBP>90), use Labetalol to counteract the high BP.



Epidural

Opioid intoxication

If the patient has ***bilateral pinpoint pupils and depressed level of consciousness***, there is a high likelihood of opoid intoxication. Be aware that a co-ingestion of "uppers" like amphetamines or cocaine; or anitcholinergics like Gravol and Benadryl can counteract the effect of the opoids and give the patient mid-range pupils.

The main danger of opioid intoxication is respiratory depression and respiratory arrest.

Luckily, we have an excellent antidote in Narcan. Administer as needed until **breathing spontaneously but not agitated and thrashing around, swearing and punching people.** That can be dangerous to you and the sudden massive withdrawal and the resulting symphatomimetic agitation can actually be life threatening to the patient.

Narcan Administration

Dilute a vial (0.4mg) in a 10cc syringe so you get 0.04mg/cc. Give 1-2 cc at a time, every 2-3 minutes. Narcan works within minutes but you might need several doses to achieve the effect, and if you don't get any effect, double the dose each time. If a particular dose works, since Narcan only has a 45-60min half-life you can put them on a Narcan drip. The rule is to give them every hour 2/3 of the dose that was needed to wake them up.

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 5-10mg IV every 5-10 min or Ativan 2-4 mg IV every 5-10 min are good choices. 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.

Avoid giving typical antipsychotics in large doses as your 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.

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.

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.

As far as seizure control goes, you have several options. As with agitation, ***benzos are first choice***, and at the same doses: Diazepam 5-10mg IV every 5-10 min or Ativan 2-4 mg IV every 5-10 min. Again, there are no maximum doses as benzos have no real toxicity- the worst you might do is get them really sleepy and not breathing, in which case you might have to **BVM** them for a while or **ETT/LMA them until they wake u**p. Usually, we also load the patient with a 1g of Dilantin, over 20-30 min, but that takes time. **Dilantin will precipitate if given in the same IV as benzos so use a different line to infuse it.**

Investigations
Once you have the vitals and the patient under control, you should look into what is causing the seizure, and the differential is vast.
Keep in mind key things to rule out: a bleed, an infection (meningitis or encephalitis), a mass, hypoglycemia or hyponatremia. Basic bloodwork and a non contrast CT are a good place to start, with an LP added in a febrile patient.

If the patient is looking like status, i.e. a seizure lasting more then 30 min despite your administrations, you might have to go further and put them on a Propofol drip- this will require you to intubate them for airway control, so make sure to call for help early if a patient's seizure is not ending quickly.

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. Dextrose comes in various concentrations, with D50W (50% solution) coming in 50cc vials (25grams of dextrose per vial) in all crash carts for rapid bolus use on one end, and D5W (5% solution, 50 grams of dextrose per liter) coming in IV bags for infusion mixing on the other end.

The initial bolus dose is **1gram/kg**. For an adult, that means 3-4 amps of D50W (50-100grams). For a child we need to dilute it as, because of their small water volume, you can really mess up their osmolarity with high glucose concentration so we give D25W to kids over 1 year old and D10W to kids under 1 year. How do we get a D25W bolus? Simple- take a D50W ampule and mix it in a 100cc NS bag. That gives you 25 gr of glucose per 100c, aka D25W.
D10W comes in bags so just draw up the required number of grams (remember, it is 10ccs to a gram of D10W) and bolus it. If you can't remember all these calculations, the nurses are pretty used to mixing IV solutions so if you just write **1g/kg** as D10W , and they will be able to figure it out.

For most types of hypoglycemia, an infusion of 100-200cc/hr of D10W is reasonable.

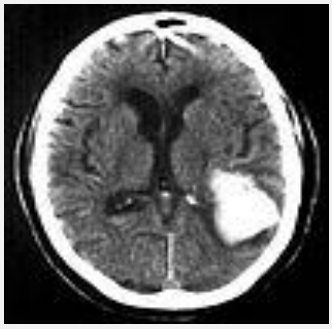
Sulfonylurea-caused Hypoglycemia

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.

Stroke

There are two general types of stroke, hemorrhagic (15%) and ischemic (85%).

Hemorrhagic stroke comes in two varieties, subarachnoid hemorrhage from an aneurysm or an AVM; or intraparenchymal (IPH) bleed, where a blood vessel bursts within the brain tissue (parenchyma). IPH carries a worse prognosis then SAH.



Intraparenchymal bleed

Ischemic stroke comes either from a cardiac embolus, or from carotid artery atherosclerosis. The mainstay of diagnostics for suspected strokes is a non contrast CT, but be aware that CT actually has a fairly low sensitivity for ischemic strokes early on- its real utility is in ruling out a hemorrhagic cause so that the patient can be considered for t-PA treatment.

t-PA window has been extended to 4.5 hours now (from a known time point of onset), so you have a bit more time. The decision whether to use t-PA is made by neurologists based on the NNH stroke scale, but this is not for us to figure out- our job is simply to suspect a stroke (unilateral weakness, paresthesias, face droop, dysarthria, visual loss; vertigo for posterior

ones), do a rapid CT to rule out a bleed if we can and ship them off to a t-PA centre as soon as possible.

ALTERED LOC DRUGS

	Mechanism	Dose	Use
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
Dilantin	antiepileptic	1g IV over 20-30min	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
Etomidate	intubation induction agent	20mg IV single dose	intubation of an intracranial bleed
Octerotide	somatostatin analogue	50mcg bolus and 50mcg/hour infusion	antidote for sulfonylurea poisoning
Dextrose	sugar	1g/kg D50W adults D25W children D10 babies	hypoglycemia

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 lungsto 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 O2.** Very rare as it takes a very large and 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 thus 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 very short term, accumulation of CO2 never killed anyone. 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 (in the 80s or preferably 90s).***

How can we support someone's oxygenation? First, we can add supplemental oxygen. The simplest way to do this is through 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 65% 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.

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. This maxes out at around 40% oxygen

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 intubate the patient 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.

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** or is in danger of closing (tonsilar or retropharyngeal abscess, airway burn, neck or face trauma, anapxylaxis, massive GI bleed/hemoptysis/epistaxis).

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

3.Their **level of consciousness is decreased** to the level where they are not protecting their airway against aspiration. The usual cutoffs are GCS of 8 or less (good luck calculating that on the fly) , or P or U on the AVPU scale (see altered LOC section). I have a simple approach- if I need to inflict painful stimuli in order to get a significant response from a patient, they might need a tube. I then put an oral airway in their mouth- if they gag, they have an intact gag reflex, and I leave them alone (assuming they are not actively vomiting or bleeding). If they do not gag, I go ahead and intubate them.

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, only the first two are 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, assuming you have anticipated the respiratory failure you usually have a bit more time to set up but be aware that, if the patient is on the cusp of failure, they might crash in a hurry.

The other two causes you have time, as long as you can support them by other ways (BVM for decreased LOC; BiPAP/CPAP for hemodynamic instability, as long as they are alert). Use that time to preoxygenate, prepare your equipment, call for help and make a plan of action in case you are having trouble with placing the ETT.

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.

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.

Most vents available in ER or on the floor are volume controlled. 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, despite nurses always writing 20 resps per minute on charts, is actually 12-16. I usually set mine at 12. This gives us a minute ventilation of 6L per minute (12 X 500cc). Problems can occur if the person attempts breathe and specifically exhale as the machine is giving a mandated breath- the clash of two air columns will skyrocket the airway pressure and set off all kinds of alarms. This is known as ventilator-patient dysnchrony and can lead to pneumothorax. Avoid it by paralysing vented patients, or at least sedating them heavily.

Key Point: You manage respiratory distress/failure on a continuum

Evaluation of patient in respiratory difficulty

First step, as always is **ABCs** - start IV-O2-monitor and get a full set of vitals. Then ask 5 questions:

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 can be very brief- note if there is any drooling or stridor, look quickly into the mouth for a, say, swallowed foreign object, an airway burn or a tonsilar abscess and palpate the neck for swelling. Finally, decide if the patient is conscious enough to protect against aspiration. If you think there is an airway problem, your immediate concern should be securing the airway with

an ET tube. If not, proceed to question 2:

2. What is their oxygen saturation?
As mentioned above, lack of oxygen will kills a person far quicker then buildup of carbon dioxide.

3. Are they moving enough air?
Remember, ventilation 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 bad news in the long run. More importantly, inadequate ventilation can lead to poor oxygenation as well and adding extra FiO2 will not help this.

This is a clinical assessment: look at how frequently and how deeply the patient is breathing (ie respiratory rate and respiratory volumes). As mentioned, the rate should be at least 12-16, and you should have a decent idea what a tidal volume 500cc breath sounds on auscultation and looks like on inspection from listening and looking at normal lungs in healthy patients.

Auscultate if they have decreased air entry in both lungs (chest wall weakness, really bad asthma or COPD), are they wheezy (moderate asthma or COPD), or is there unequal entry (pneumothroax, effusion)

4. What is their work of breathing?
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. When I listen to their chest, are they moving air or is it a "silent chest"?

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

5. 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 4 reasons for poor gas exchangeoutlined 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 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 pH and pCO2, and as mentioned, both of those are "nice to have normal" but your initial time is better spent making sure the oxygenation is OK and that they are not going to crash from the respiratory effort.

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 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

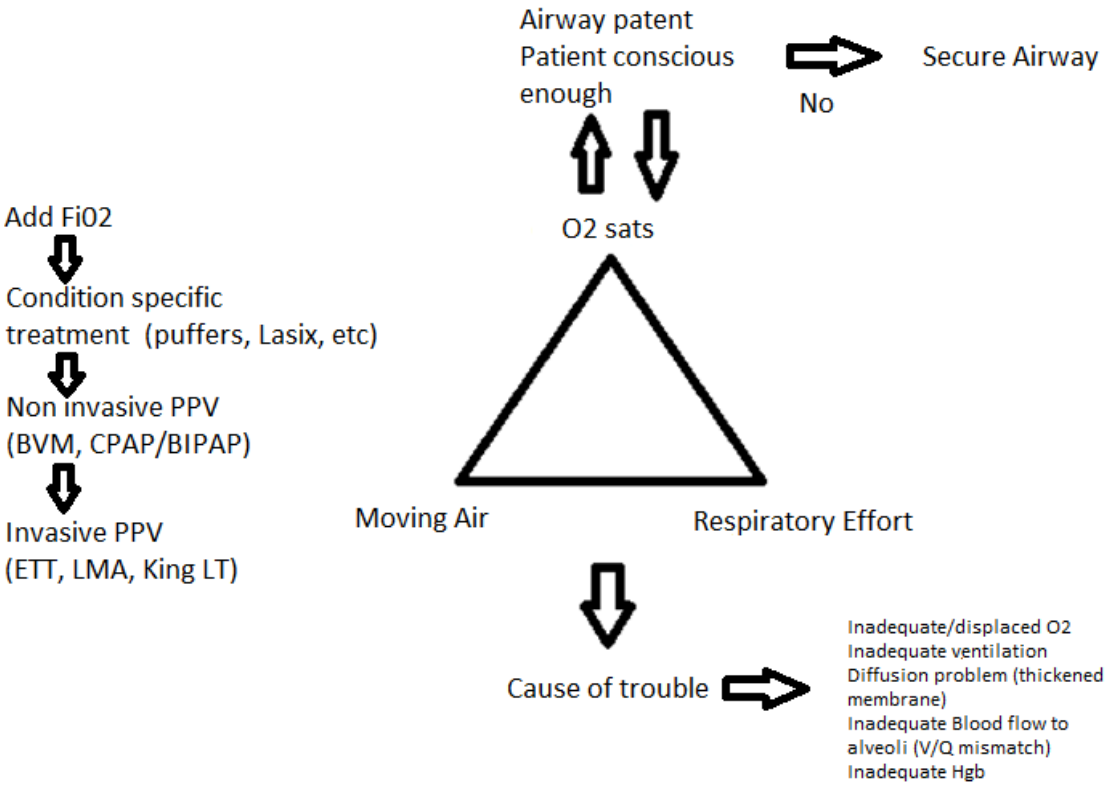
http://www.youtube.com/watch?v=2ZRip1STSSQ Thoracocentesis

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

If this is too complicated, remember this:

Conceptually, the approach to an SOB patient can be summarized with this picture:



If the airway is not patent 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.

Myocardial Infarction

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.

EKG Territories

II, III, aVf for RCA

V1-3 for LAD

V4-6, I aVL for circumflex

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

Code Blue

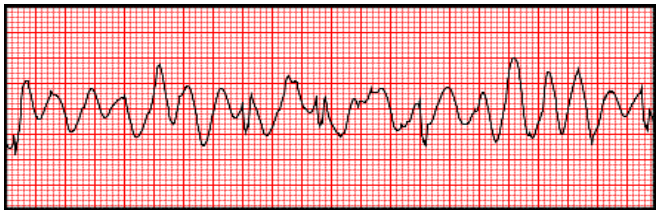
When you are call for a Code Blue, the patient has stopped breathing and/or has lost his pulse. In either case, the patient is now clinically dead. Our job is twofold.

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.

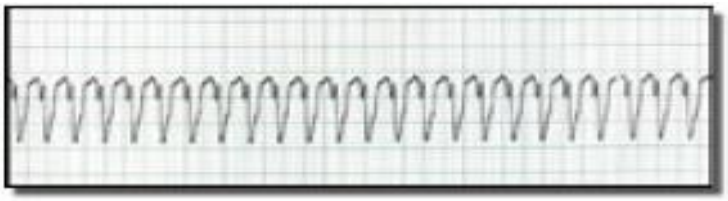
The second task is to **keep the brain alive** while you defibrillate or apply other therapeutics. Brain only has minutes before hypoxia due to lack of blood flow begins to kill it. The way to do that is through EFFECTIVE CPR. CPR, with ventilations, essentially stops or significantly slows down the 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

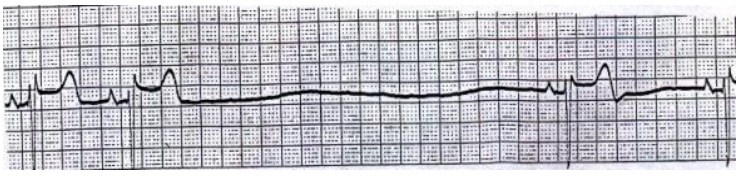
Note that the bolus given in arrest situation is 1mg IV, which is a massive dose that, if given to a live person will likely result in extremely nasty side effects or death. ***Thus, make sure the person is clinically dead before you give such a dose.***



Vfib



VT



PEA

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, 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, Norepinephrine, Phenylehrine, etc.

Thus, don't find yourself in a situation where you need something like Amiodarone 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.

   If the nurses are unable to get more then one small IV in a septic patient, I might choose to get a central line in (either me or an anesthetist can place it) before I leave the hospital, in case the patient develops more profound shock requiring pressors while en route.

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.

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.

In Conclusion….

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!

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 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."
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.." "I think we need to…."	"Jim, can you…" "You two doing CPR …"
Not being clear about what you want	"We need to press the patient…" "They need epinephrine.."	"Jane, give phenylephrine 200 mcg IV now please?" "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.

NIGHTMARES COURSE - DRUGS AND DOSES SUMMARY

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

	Mechanism	Dose	Effective in	
Cardioversion	electricity	200J	Any	**TACHYCARDIA**
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	
Calcium Chloride		**1gr over 10 min**	**Hyperkalemia**	

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

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

	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 D50W adults D25W children D10W babies	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.

	Dose	Conditions	SH
BiPAP/CPAP	10-20mm inspiratory (8 in	COPD	

	asthma) 5-10mm expiratory (3 in asthma)	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

GI BLEED

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

INTUBATION

	Dose	***Side effects* and duration**
Midazolam	0.1 mg/kg IV (usual dose : 7mg)	*Hypotension (mild)*. 30 min peak. 2 hr duration
Fentanyl	0.5-2 mcg/kg (usual dose: 50-200 mcg)	*Hypotension (mild)*. 4-5 min peak, 40 min duration
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.
Succinylcholine	1 mg/kg	*Hyperkalemia*. 5-10 min duration.

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

	Dose		Conditions
	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

ANAPHYLAXIS

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

NOTES:

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