

20 Questions - Cardiac Arrest

1. You're called to a dialysis unit for "one unconscious." When you arrive, you find a 53M in full arrest. The patient had just arrived and was not yet being dialyzed. He complained of shortness of breath, then collapsed. What rhythm disturbance is likely?
2. In addition to your typical ACLS algorithm, what is special about the treatment of hyperkalemia---i.e. what additional medications will you want to request?
3. You respond to a "one down". You find fire ventilating a patient that has a stab wound to the left chest. They initially reported a pulse, but when you go to assess, the pulse is now gone. What are the four causes of arrest that you should consider in this patient?
4. What are the criteria for attempting to resuscitate a traumatic arrest?
5. Why is epinephrine the current drug of choice in all types of cardiac arrest?
6. What is the purpose of defibrillation, and why can shocking a non-shockable rhythm (e.g. asystole) be harmful?
7. What about pacing in asystole?
8. What is a biphasic defibrillator (vs. monophasic), and why are we using them now?
9. You are called to a "one down" who is found to be in cardiac arrest. Is it advised to perform CPR for 2 minutes then shock or shock as soon as possible?
10. You approach a "one down" in cardiac arrest and are unable to obtain IV or IO access. Which drugs can be given down the ETT?
11. What is Torsades de Pointes? How does it differ from polymorphic VT?
12. What are the causes of PEA arrests?
13. How does the Res-Q-Pod work? Why do we use it?
14. When part of a resuscitation, when your own adrenaline is through the roof, you may observe providers (or do yourself) ventilating patients at a MUCH fast rate than what is indicated. Why is this bad?
15. What is the ideal rate for CPR? Why did we select this rate?
16. What is the current latest news on amiodarone?
17. How well is the brain perfused in CPR?
18. You have a patient who appears drunk/woozy and EKG shows a narrow complex tachycardia at a rate of 150. You've determined this to be unstable SVT (because of the obvious compromised perfusion to the brain). You move to counter shock. Why do we synchronize unstable rhythms vs. defibrillate?
19. You administer a dose of lidocaine. Under what circumstances should you reduce the dose of lidocaine? What is the most common indication of lidocaine toxicity in the unconscious patient?
20. You and your partner are doing CPR on a patient in cardiac arrest. What are some clues that the patient achieved ROSC during CPR?

20 Answers - Cardiac Arrest

1. There is a high probability of VF/VT or PEA secondary to hyperkalemia. This should be of high suspicion in dialysis patients. The increased potassium poisons the cardiac cells, preventing proper conduction. The EKG will begin to change from the ventricles becoming irritated, and just prior to arrest you may see a "sine wave" type pattern that quickly decompensates.
2. Calcium will be one of the first medications you want to give because it stabilizes the cellular membranes / electrolyte gradient of the heart that have been disturbed by the high potassium. Second, bicarb and albuterol nebs may be useful in this setting because both of those medications cause the potassium to shift into the cells and out of the blood where it's able to disturb the heart. Glucose generally won't be as effective without insulin being given with it (insulin acts like bicarb and albuterol, driving potassium into cells). Per the protocols these are considerations for verbal orders.
3. Because this appears to be a trauma patient, causes of traumatic arrest should be considered. These include pericardial tamponade, tension pneumothorax, exsanguination from the stab wound (either into a body cavity or onto the ground), or gas embolism if there is any bronchial

or venous injury. From a prehospital setting, the primary treatment is fluid loading, which will increase right-filling pressures. This will help in all injuries except embolism, but it will not hurt an embolism. If truly sign of tension pneumothorax, needle decompression on the side of the stab wound is also indicated.

4. If the patient has any signs of life when first responders arrive, they are likely candidates for treatment/transport within our service area given the short transport times. Penetrating injuries tend to have more correctable cause (e.g. tamponade, tension pneumothorax). Blunt traumatic arrests tend to be multi-system in nature and have greater than 99% mortality.
5. Besides brain perfusion, **coronary** perfusion is a huge factor on the chances of ROSC. Epinephrine causes vasoconstriction and higher blood pressures, which will hopefully translate to improved coronary perfusion. The heart requires a certain level of perfusion in order to be shocked out of VF/VT or achieve ROSC. Epi in addition to (more importantly) **high-quality CPR** is key!
6. The point of defibrillation is to depolarize enough of the cardiac cells to hopefully allow the heart's internal pacemaker to take over again in an **organized** rhythm. It cannot "jump start" a dead heart. It can only reset the electrical circuit. Shocking non-shockable rhythms is harmful because the electrical jolt from the defibrillator increases parasympathetic (vagal) tone, decreasing responsiveness to epinephrine. **HOWEVER:** if you are having trouble distinguishing fine VF from asystole, it is advised to shock because the outcome is that much better.
7. No study has ever demonstrated increased survival or ROSC with pacing asystole. In very rare occasions, if the asystole arrest was witnessed, pacing may be appropriate, however this is not strongly supported by evidence. One might consider if the patient's implanted pacer wasn't firing.
8. Biphasic defibrillation sends electrical signals in BOTH directions (i.e. from pad 1a2 **and** pad 2a1). Monophasic just sent electrical shocks one direction. Biphasic defibrillation is the mainstay now and has been slightly more successful in converting shockable rhythms at lower voltages, decreasing the risk of electrical injury (burns).
9. There has been conflicting evidence about this over the last couple of decades, but realistically, it will take a little over a minute to power on the defibrillator, prepare the pads, and defibrillate. It used to be recommended to "prime the pump" with at least two minutes of CPR, but this isn't supported by good evidence. Ideally, with enough personnel, high-quality CPR will be performed while defibrillator setup is taking place and you will defibrillate ASAP if it's a shockable rhythm. In short, if you witness a patient go into VF/VT shock them immediately.
10. Use the mnemonic "LANE": Lidocaine, Atropine, Naloxone, Epinephrine. NOTE that these drugs should be in at least 5cc's of saline and at LEAST 2x the normal dose. There is some evidence that rolling the patient will distribute the drug more and subsequently cause more absorption. However, no matter how ideal, ETT drug administration is far inferior to IV or IO administration.
11. Torsades de Pointes is a form of polymorphic ventricular tachycardia that is associated with prolongation of the QT interval. Not all polymorphic VT is associated with long QT and is caused by different ectopic points on the ventricles producing an electrical signal (vs. just one in monomorphic VT). Any VT should be shocked if unstable, and magnesium can be very helpful in Torsades (and polymorphic VT), so consult medical control!
12. Think of the H's and T's. H's: hypoxia, hypovolemia, hypo/hyperkalemia, hydrogen ions (acidosis), hypothermia. T's: tension pneumothorax, tamponade, trauma, toxins, thrombosis (MI), thromboembolism.
13. In cardiac arrest, CPR is acting as systole (compression) and diastole (decompression, i.e. releasing pressure). The Res-Q-Pod **increases** circulation (especially coronary and cerebral) by preventing the passive entrance of air into the chest through the airway during the decompression phase. The device does allow air to passively leave the chest with compressions. That allows MORE negative pressure inside the chest, allows the vessels/heart to expand with greater force and subsequently more blood flow. The Res-Q-Pod does NOT prevent ventilations, as it will allow air in when the bag is squeezed. It only stops the passive flow of air from entering while the chest is recoiling.
14. Air is good, especially to hypoxic people who need oxygen. But too much of a good thing is never a good thing---and in the case of CPR, it's deadly. Ventilation with a bag is different than how we normally respire because bagging uses POSITIVE pressure, not negative

pressure like we do when we expand our chest to breath when we're normally breathing. By forcing positive pressure into the chest with a bag valve, you're increasing the positive pressure in the chest beyond physiological levels. Ventilating *too fast and too much* will cause the positive pressure to rise *too high* in the chest and crush vital structures (mostly the IVC and right heart), severely compromising blood flow and tanking your chances of ROSC. So, be mindful of your respirations when you're bagging and keep an eye on your colleagues' bagging rates!

15. The proper rate for CPR is 100-120 BPM. Evidence supports this rate because it is the optimal rate at which 1. the heart can pump blood forward and 2. The heart has enough time to *fill*(diastole). Going too fast won't give the heart the filling time it needs, which is also when the coronary arteries are perfused. Compromising blood flow to the coronaries will be detrimental to your rates of ROSC and survival. **However, if using the ResQPump (active compression / decompression device) the rate is 80 bpm.**
16. Amiodarone continues to be the mainstay antiarrhythmic with VF/VT arrest, and it is dosed at 300mg (and subsequently 150mg) during ACLS. Studies have compared amiodarone with other antiarrhythmics, namely lidocaine, but Amio remains the choice at this time. If the patient has pulses, a verbal order could be considered for a **slow push over 10 minutes** of 150mg. Stay tuned for the next update...
17. Even with the best CPR, the brain only sees about 30% of its normal blood flow. This is why ideal CPR is critical. Such devices like the Res-Q-Pod can help increase perfusion to the brain as explained in #13. However, research is trying to push this boundary and this may increase with the advent of heads up CPR...
18. Synchronization of the defibrillator to the EKG is CRUCIAL. Of note, you need to have the 3-lead from the defibrillator on the patient to cardiovert (vs. just reading the rhythm through the pads). The machine will lock onto the peak of the QRS (the R wave) and shock on that point. It's important to synchronize because of the "R on T phenomenon." If the shock is delivered on the T wave (repolarization of the ventricle), you can send that patient into deadly VF. You must re-sync each time you shock. **Look for the white dots over the QRS complex on the monitor**
19. Patients older than 70, or with hepatic disease or CHF are at higher risk to accumulate toxic levels of lidocaine. Whether these restrictions should apply to cardiac arrest states or to the loading dose is a topic of debate. Current Hennepin County ALS protocols do recommend a dose reduction in these circumstances. Arrested patients will not demonstrate toxicity, but are at risk for seizures and other CNS problems after resuscitation.
20. There can be subtle clues that you may have achieved ROSC. A bump in end tidal CO2 (from 18 to 45, for example) may indicate the heart is perfusing again. A change in the EKG is another suggestion that the rhythm changed (not necessarily ROSC), but more difficult to tell because of ongoing CPR. It takes a skilled provider to "see through" the compressions, but is a good clue that the patient may have changed rhythms. However, continue CPR until your next rhythm check as their heart may not be strong enough to perfuse on its own just yet. It should prompt you to be ready to change your algorithm if you see these clues.