Online 12 Lead Rhythms

Question:
With these given rhythms I just wanted to confirm what CTAS we should be transporting some of these pts. At what point should PVCs be considered a life threat - multi-focal, bigeminal, trigeminal, etc. My understanding of junctional rhythms is that as long as the pt is stable and the rhythm is sustained/mechanical it is not a life threat - is this correct. Should new onset atrial fibrillation be considered a life threat due to the increased risk of clots- if the pt is stable otherwise. Should second degree AV blocks be considered a life threat - again assuming the pt is stable and compensating well.

Answer:
The #1 over-arching point is that the patient condition (blood pressure, LOC, symptoms) dictates the management of the patient. With regards to a junctional rhythm, if a patient is asymptomatic, there is no threat. However, if they are in a junctional rhythm at a low rate and symptomatic (short of breath, hypotensive, chest pain etc.) then they need to be treated based on their symptoms, not the rhythm itself. The same goes for second degree AV blocks and PVCs. You treat the patient based on their symptoms. From a definition standpoint, 3 or more ventricular beats >100 is considered non-sustained VT. Multiple episodes of this (and symptomatic!) would be considered concerning and should be watched closely for signs of decompensation into sustained VT. This then should be treated appropriately based on presence/absence of a pulse and certification.

Question:
Secondly, are there any other polymorphic Vt besides torsades? I took an ACLS course and was told torsades is a polymorphic VT but not all polymorphic VTs are torsades. I asked for clarification but it seems this may have been something the instructor was told but could not back up. I've tried to search out the info myself but I have not been able to find a satisfying answer.

Answer:
Polymorphic VT = VT with multiple different QRS morphologies of the QRS. Torsades = A specific type of polymorphic VT that is caused by an ectopic beat in the setting of a long QT (so the R-on-T phenomenon). In order to be diagnosed, you need to have a preceding long QT and document the extra beat that causes the undulating around the axis thereafter. Note that this is all very much above what you need to know. But now you know!

Question:
Why is treatment considered for SVT at a rate greater than 150 (along with other ECG findings) when SVT occurs at a rate 180-250 bpm as per the 12 Lead Rhythms module?

Answer:
This discrepancy is common when looking at arrythmias. There are different ranges of "expected" rates depending on the resource used. The reason the treatment utilizes 150 or greater AND the associated ECG findings is for safety. Less than 150 and you should be considering another rhythm (ie. sinus tach) rather than SVT. In summary there is no black-and-white cut-off. But, instead ranges of HR and ECG findings that help you determine the rhythm.

Online 12 Lead Basics

Question:
I often look at the QT interval on a 12 lead to see if it is normal. I'm never sure if I should be looking at the QT or QTc. I know QTc mean corrected but what does this mean? Which one should I be looking at?

Great show btw. I teach this stuff to PCP students at Fanshawe but I truly appreciate hearing about it from someone else. It is a good refresher and it seems I always learn something new each time. Thank you.

Answer:
The QTc is a corrected value that takes into account the heart rate and allows for 1) better predictor of arrhythmia, 2) values to be compared for different heart rates.
The formula is QT/(square route of the R-R interval). Thankfully the computer spits it out for us. But the QTc should be used preferentially over the QT for clinical purposes bc of 1) above.

Online ACP Hyperkalemia

Question:
In the incidence of cardiac arrest secondary to hyperkalemia, i would instinctively administer salbutamol through an MDI spacer placed in the BVM circuit. Alternatively, is it acceptable to administer the saline by emptying the nebule down the ETT? I have seen this done by some old school medics when treating cardiac arrest secondary to bronchoconstriction but I am wondering if this route of administration although quick would be as efficacious as the traditional delivery methods?

Answer:
Answer will be sent out to MAC for consensus. There is minimal evidence for Endotracheal Lavage Bolus (EBL). One study published in 2015 noted in answer. PDF available to send

Question:
I am wondering what the role of sodium bicarb is anymore? I do recall getting an order to administer it to a patient in cardiac arrest with a history of hemodialysis (a long time ago - twice in one week actually which was weird) but I can appreciate that calcium gluconate (and salbutamol) are better suited. Is it still useful in the treatment of some overdoses (TCA), crush injuries, arrests with prolonged down time?

Answer:
Best evidence would be in TCA OD. The culture has shifted and it is often not in favor in prolonged arrest nor as prophylactic in crush injury. But, some might use it as a Hail Mary in potential acidotic periarrest or arrest. One of the current London Emerge residents might be giving a podcast or webinar on this very subject this fall... Keep an eye/ear out.

Online ACP VT with a Pulse

Question:
In the VT with a pulse case study it is advised that amiodarone not be mixed with saline but instead with D5W to limit precipitate and that it should be administered with an inline filter. The OBHG latest Companion Document indicates that it is acceptable to mix amiodarone with saline in a 50 ml bag. I have done this in practice previously without issue (that I was aware of). Is the mixing of amiodarone with D5W ideal but since D5W is not typically carried by paramedic services is saline sort of a default? Is it of any benefit to make my own D5W by diluting D50W or D10W?

Answer:
There is a theoretical risk of precipitation of amiodarone with 0.9%NS. Therefore, some pharmacies suggest the use of D5W with amiodarone infusion. Some services are supplied by pharmacies that recommend this practice. This was the case for some of our ACP services when they switched from lidocaine to amiodarone due to drug shortages, recently. However, if your service uses 0.9% NS, this is also acceptable.

Online ACP ECG Extra Syncope

Question:
I have some questions regarding HOCM ECG findings. It was mentioned a few times that this condition would produce dagger like Q waves in the anterior leads but the ECG's you provided didn't resemble that. I did some cross referencing on HOCM via "Life in the fast lane" and read that HOCM produced dagger like Q waves in later leads (V5-V6, 1 and aVL) and inferior leads (2,3,aVF) which resembled what I interpreted via your ECG's.

Answer:
Thanks for bringing this forward. The ECGs are correct. In HOCM you expect to see dagger Q-waves in the LATERAL > Inferior leads with precordial (including anterior) inverted T waves. Sorry for the audio vs pictoral discrepancy. A note will be placed on the portal.

Online Adrenal Crisis

Question:
After administration of hydrocortisone what should we expect in terms of pt recovery - rapid recovery or progressive recovery. Also, if the pt is presenting with hypoglycemia following the administration of hydrocortisone should we initiate our hypoglycemia protocol - and should be start with D50/D10 as per normal or start with glucagon? Similar to the above question should a fluid bolus be initiated for these pts is hypotension is still observed following hydrocortisone administration - as per our normal protocol 20ml/kg to a max of 2L or a modified fluid bolus.

Answer:
Great question! The hydrocortisone WILL take some time be processed. It will take 4-6 hours after administration to have its full effect. Therefore, you should treat, as you have mentioned with your Hyperglycemia Medial Directive and Intravenous and Fluid Therapy Medical Directives, as required. This management will support the patient until the hydrocortisone kicks in.

Interactive Grey Zone

Question:
Should we not continue CPR and not stop to check a pulse unless there is a change in patient condition? This is based on the millionaire questions that reads, "Your patient suffered a cardiac arrest and you just completed your first analysis which resulted in a non-shockable rhythm. What do you do next?
A: Start CPR.
B: Check for a pulse.
C: Get ready to transport.
D. Analyze again to confirm the non-shockable rhythm."

Wherein the answer was listed as B. Check for a pulse

Answer:
Thank you very much for bringing this to our attention. The answer should be Start (or more accurately) continue CPR. In a patient who is confirmed VSA who is non-shockable, you do not want to pause CPR for a pulse check until either a change in patient condition (patient moans, begins moving etc). for you to reassess to see if there is a ROSC, or at the 3rd analysis when you would be calling for further direction (ACP) or TOR (PCP). The reason being we want to reduce peri-shock (or here more accurately peri-analysis) CPR pause. Remember it takes at least 20sec to regain forward flow after a pause in CPR.