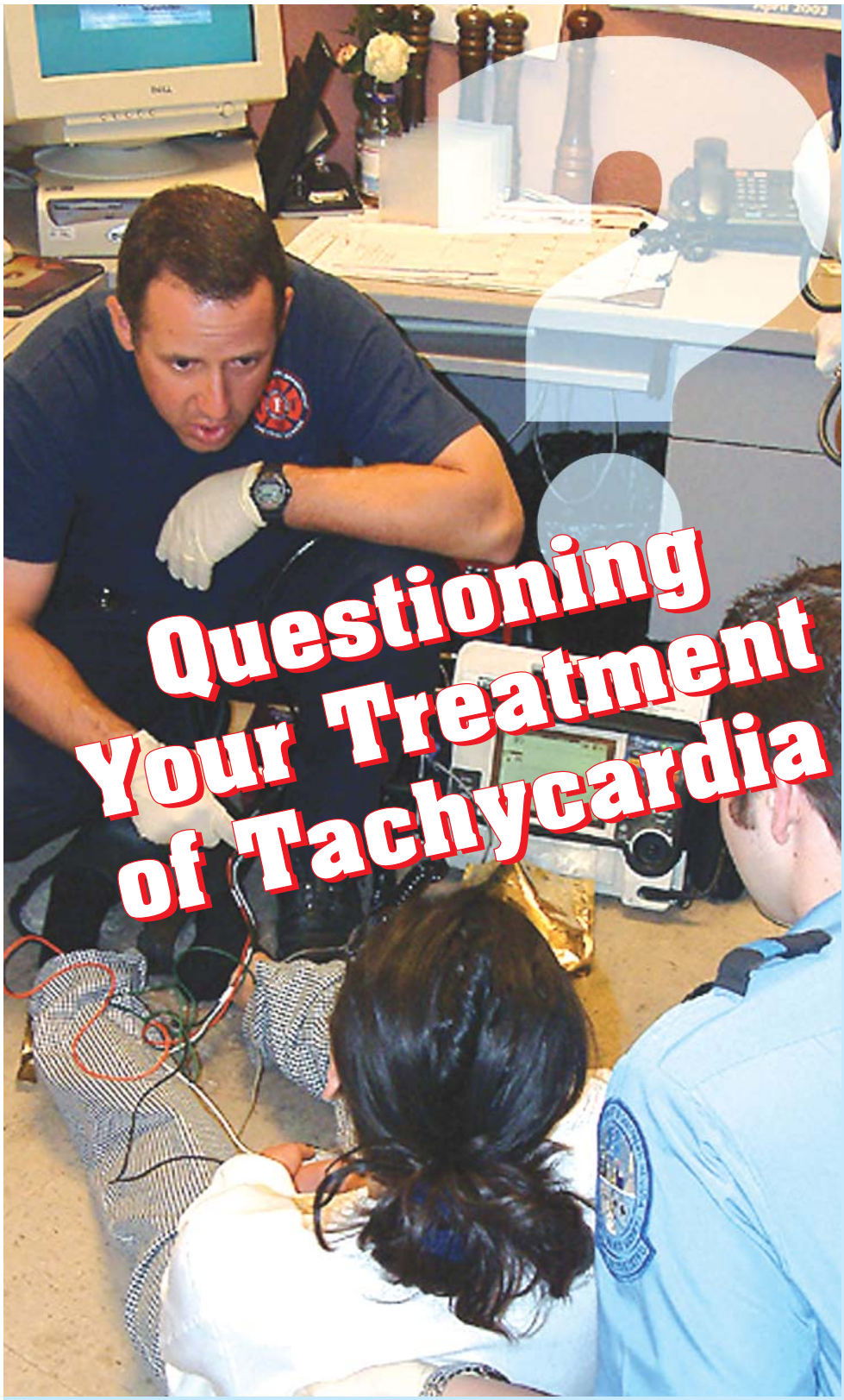


Questioning Your Treatment of Tachycardia

By S. Christopher Suprun Jr., NREMT-P

For students in advanced cardiac life support (ACLS) classes, fear levels seem to rise when we deal with what, in my experience, is the most uncommon of ACLS algorithms: the tachycardias. The multiple treatment algorithm diagrams that lead our efforts to most appropriately treat patients while trying to differentiate cause and effect; ventricular tachycardic rhythms versus supraventricular rhythms with aberrant conduction; and the gray area of whether a patient is stable or unstable leave many students confused. Fortunately, there is an easier way to interpret ACLS tachycardias, especially in the field setting, where drug choices may be limited and transport times may necessitate strong early ACLS.

My goal here is to make the ACLS tachycardias easier to follow with a simple breakdown of the tachycardia algorithm. My solution does not guarantee perfection, and, like anything else, it can be "what if-ed" ad infinitum, to the point where all we do for our patients is start an IV, attach monitor leads, add oxygen and transport out of fear of doing something wrong, rather than aggressively pursuing good care. Nonetheless, it can be used to simplify the complex algorithm into an easier format as you race your patient to the nearest ED at 3 a.m.

A few quick disclaimers before we begin. Sinus tachycardia (ST) is not discussed in this article. ST is a symptom of something else and will occur secondary to exertion, fear, pain, fever, hypoxia, hypovolemia, etc., but will resolve itself when the primary condition does. Throughout, there needs to be an assumption that the patient's chief complaint is a result of the tachycardia. Additionally, for purposes of treatment, we will assume oxygen is in place, one (or more) intravenous line(s) have been initiated, and the patient's vitals have been taken.

Question 1: Is my patient unstable (with any tachycardia)?

ACLS has not changed its approach to the unstable tachycardic patient for years. Patients who fall into the unstable tachycardia algorithm have signs or symptoms like chest pain, shortness of breath, altered mental status or hypotension, with heart rates of 150 or greater. ACLS teaches that multiple factors should be considered before deciding a patient is unstable, and that a "total clinical picture" should be considered, as opposed to just using a single factor like blood pressure.[1] Obviously, many patients can have heart rates this high with secondary signs/symptoms as described and not all will need immediate cardioversion, but those whose condition is declining should be treated without delay. The concern here is that the patient will deteriorate into a pulseless rhythm without aggressive treatment by synchronized cardioversion.

Synchronized cardioversion at 100 joules, followed by synchronized cardioversion at 200, 300 and 360 joules.

If time permits, sedation should be initiated prior to cardioversion. Sedation options should be discussed.

Photo by Eddie Sperling

Question 2: What if my patient is stable (with any tachycardia) and I don't want to open the drug box?

Here, ACLS has made a dramatic change in its treatment algorithm by lowering the threshold for performing synchronized cardioversion. If you remember back to your initial cardiology training, you will likely have memories of portions of the cardiac cycle that include the refractory period. This was the period in the cardiac cycle where we could limit uptake of calcium, sodium and potassium channels to slow the heart down or increase the threshold for fibrillation to occur. We would change the refractory period waveform with lidocaine, procainamide or magnesium sulfate, depending on what changes we were trying to initiate. We now add amiodarone to our menu of choices.

When you look at all of these different drugs changing the refractory period, you realize that we're not changing the refractory period cycle to its normal self, but to a completely different form that may not help the patient. Much like building the monster Frankenstein, we find we're causing the problem by administering *pro-arrhythmic* medications that are indeed altering the refractory period and causing a number of unintended consequences. In a new set of principles for treating stable tachycardias, the American Heart Association also states that "the use of two or more antiarrhythmics to treat tachycardias is undesirable because it increases the risks of complications to heart function"[2] and that "antiarrhythmics can make an impaired heart worse."[3] Depending on your local protocols and patient's condition, you may consider moving directly to cardioversion with appropriate sedation instead of using medications.

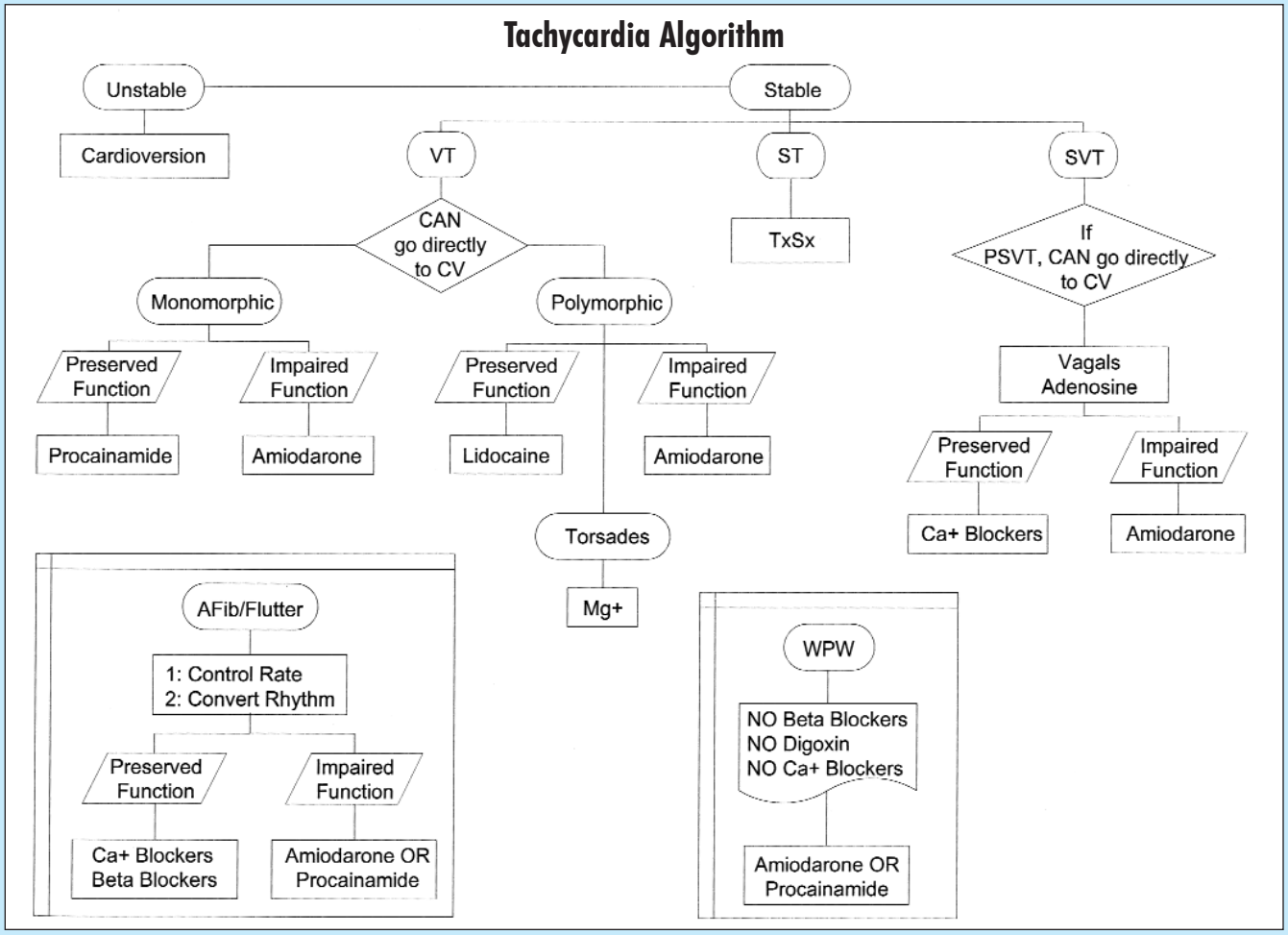
When I first became an ALS provider in 1993, synchronized cardioversion in the field was done infrequently at best, and people recalled these events as though they were remembering historical events from World War II or the signing of the Declaration of Independence. While the American Heart Association straddles the fence somewhat by stating that "in general, stable tachycardias are treated with drugs…,"[4] they also state that "electrical cardioversion is either the intervention of choice or the second 'antiarrhythmic.'"[5] Depending on your patient's history, additional antiarrhythmic treatment may make the impaired heart worse, and cardioversion may be a very real option.

Synchronized cardioversion at 100 joules, followed by synchronized cardioversion at 200, 300 and 360 joules. This should be preceded by appropriate sedation, as discussed previously.

For sedation purposes, many field providers still use 5 mg IV diazepam, while others use 2 mg IV midazolam. Other options include etomidate and ketamine. Some EMS system protocols show a preference for both a sedative and analgesic. They may use midazolam, which will provide both sedation and a retrograde amnesia, as well as morphine for pain. In other words, the patient will have both limited pain and limited memory of the treatment. Other analgesic options include meperidine or fentanyl. Whether sedation is being performed on the stable or unstable patient, care should be taken to not create a hypotensive situation or further intensify an existing hypotension by using sedatives.

Question 3: Is my patient stable (with any tachycardia) and does he/she have signs or symptoms of heart failure?

This question presents the scenario of a patient with a decreased perfusion secondary



Tachycardia algorithm schematic reprinted with permission from Kathleen Camp-Deal, RN, CEN, BSN, MBA, Adjunct Assistant Professor, George Washington University-EHS Programs, Dept. of Emergency Medicine

to lower ejection fractions and signs and symptoms of heart failure. "Amiodarone becomes the antiarrhythmic of choice if the patient's cardiac function is impaired…"[6] While amiodarone does have some hypotensive effects, it is preferable to other anti-arrhythmics due to its unique ability to act on all cardiac tissue. It is believed that it prolongs both the action potential and refractory period without affecting the resting membrane potential.

The action potential is the biochemical stimulation of the myocardial cells that allows sodium and potassium to shift along the heart's cellular wall, causing depolarization and contraction of the heart. The refractory period is the point in the heart's electrical conduction after the ventricles have depolarized and are recharging for the next heartbeat. The resting membrane potential is the normal biochemical state of the heart's cells.

There is some evidence that it also increases coronary blood flow and decreases peripheral vascular resistance, easing the work of the heart while supplying it with oxygen.[7] This occurs through peripheral vasodilation, which lowers the heart's preload, easing the work of the heart, and may dilate coronary blood vessels, feeding the heart additional oxygenated blood.

Intravenous use of 150 mg amiodarone over 10 minutes.

Question 4: Is my patient stable with a supraventricular tachycardia that is not atrial fibrillation (A-fib), atrial flutter or multifocal atrial tachycardia?

In this case, the classic paroxysmal supraventricular tachycardia (PSVT) of the myocardium occurs when rapid atrial firing overrides the sinoatrial node, leading to a rapid heart rate. These patients may have coronary heart disease, but this dysrhythmia could also occur from recent tobacco or caffeine use, overexertion or stress. In some cases, accessory pathway circuits such as Wolff-Parkinson-White (WPW) may exist. Here too, the American Heart Association has not changed its recommendation a great deal. Treatment for the classic PSVT is very similar to before, except there seems to be some preference for diltiazem as the calcium channel blocker of choice over verapamil, after first attempting vagal maneuvers and intravenous adenosine.

A special note of caution regarding WPW: Verapamil is contraindicated in persons with accessory pathways such as WPW, and other medications such as adenosine and calcium channel blockers should be used cautiously, as they "may be harmful."[8] Using atrioventricular node-blocking drugs on dysrhythmias with accessory conduction defects can make it possible to block normal cardiac function and cause the heart to find the "path of least resistance," firing only along the accessory pathway and accentuating the tachycardia rather than correcting it.

Attempt vagal maneuvers, such as valsalva maneuver, followed by sequential dosing of adenosine 6 mg, 12 mg, 12 mg IV, followed by 20cc saline flushes. Adenosine should be given via fast IV push due to its short half-life. If these efforts do not yield success, control the rate and rhythm with a calcium channel or beta blocker. Diltiazem at 0.25 mg/kg, slow IV push, is used widely.

Question 5: Is my patient stable with a supraventricular tachycardia that *is* atrial fibrillation, atrial flutter or multi-focal atrial tachycardia?

In patients with atrial tachycardias that are the result of aberrant foci rather than re-entry circuit defects, we know that adenosine will not break the dysrhythmia because it works by blocking entry into the atrioventricular node. With a multiple-foci dysrhythmia, simply blocking electrical impulses in the hopes that myocardial conduction will get back on track will not occur because there are multiple electrical tracks

that are problematic, as opposed to a single reentry circuit.

With some patients, in particular those with atrial fibrillation, there is the possible concern of blood clot formation, which, upon cardioversion or conversion to a sinus rhythm, may dislodge and cause an arterial embolus. This creates an increased risk for stroke or other organ damage.

Most experienced field providers have seen multiple patients with atrial fibrillation or, in some cases, atrial flutter. Most of these patients do not require immediate EMS attention. If their dysrhythmia causes higher ventricular responses that are not tolerated, then treatment should focus on controlling the rate and rhythm. Adenosine and/or field 12-leads can be useful as a diagnostic means to better interpret rhythms if the rate is too fast. Occasionally, these rhythms will appear regular, as in the case of an atrial fibrillation or atrial flutter, with fast ventricular response at 180 beats per minute. Some 12-leads may not be able to interpret a rapid rhythm unless it is slowed first, or if the machine can be calibrated to record at a slower speed. Nonetheless, field 12-leads are becoming a much more important component of prehospital cardiac treatment and are encouraged as a useful tool for treatment of arrhythmia.

If the rhythm can be diagnosed and confirmed as a non-reentry circuit tachycardia, then diltiazem or another calcium channel blocker should be used without the delay of drawing up adenosine or attempting other time-consuming procedures.

Control the rate and rhythm with a calcium channel or beta blocker. Diltiazem at 0.25 mg/kg slow IV push is used widely throughout the United States. Many hospitals use doses of 10–20 mg IV.

Question 6: Is my patient stable with ventricular tachycardia?

Finally, if experiencing a ventricular tachycardia (VT), the patient should be treated aggressively and carefully, as the possibility exists for the patient to deteriorate quickly into pulseless ventricular tachycardia or ventricular fibrillation. Additionally, differentiation should be made as to whether the rhythm is monomorphic with a consistent QRS pattern, or polymorphic with a marked QRS pattern change. Evidence is limited, but tends to support procainamide or amiodarone for monomorphic VT. Polymorphic VT represents an altered and possibly deteriorated myocardium that may self-correct or could quickly prove to be fatal. It is often caused by medications or metabolic "derangements," which cause increased QT intervals.[9] Polymorphic VT is also caused by myocardial ischemia, myocarditis and other origins. While lidocaine seems to have fallen out of favor somewhat in VT, it is still acceptable for polymorphic VT.

Field providers should also seek out specific causes of dysrhythmias, as opposed to treating them with a "cookbook" protocol. Two specific cases related to VT that warrant mention are hyperkalemia and tricyclic anti-depressant overdose. Sodium bicarbonate can reduce the lengthened QRS complexes leading to VT and reduce hypotension.[10] In both cases, sodium bicarbonate can assist in correcting the underlying cause of VT, rather than simply utilizing an antiarrhythmic to fix what is a symptom of something else.

For monomorphic ventricular tachycardia, use 20 –30 mg per minute of procainamide.

For polymorphic ventricular tachycardia, use 0.50 –0.75 mg/kg of lidocaine.

Torsade de pointes, a specific form of polymorphic VT, is caused by metabolic disorders such as hypomagnesemia, hypokalemia or hypocalcemia, or drug toxicities such as quinidine, procainamide or tricyclic antidepressants, and is treated, as in other algorithms, by fixing the cause. We generally use magnesium sulfate as a principal treatment method because of its relative effectiveness and margin of safety.[11]

Translated, torsade de pointes means turning of the points. In this variation of polymorphic VT, the tachycardia produces a "peak to trough" and "wide-to-narrow-to-wide-to-narrow rise-and-fall pattern."[12] While the American Heart Association does not consider this a common rhythm, it is not rare either, and they point out the specific need for providers to be familiar with this rhythm and its specific treatment. Treating torsade with typical antiarrhythmics discussed earlier will generally lengthen the QT interval, making the condition worse and "can have disastrous consequences."[13]

For torsade de pointes, use 1–2 grams of magnesium sulfate over 5–60 minutes.

Summary

These six questions may make it easier to guide your steps as critical minutes pass while treating your tachycardic patient. Ultimately, local protocols and the contents of your drug box may limit some of your choices, but good decisions can start the process. Adequate oxygenation, establishing IV lines and diagnosing specific rhythms are the first steps toward successful treatment of your next patient. ■

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