Myths, Pearls, and Pitfalls in Advanced Cardiac Life Support

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Introduction

The American Heart Association's Guidelines 2000 for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care ("ACLS 2000") were published in August 2000 (1). ACLS 2000 represents the collaborative efforts of representatives from multiple United States and international medical organizations. Additions to and deletions from previous ACLS Guidelines were based upon "rigorous evidence-based review" (1).

Health care providers should remember that medical knowledge, including ACLS, is a constantly evolving process. No set of medical guidelines, no matter how evidence-based, withstands the test of time. New studies often cast doubt on old guidelines. Furthermore, the application of even the best science and theory often has shortcomings when applied to actual clinical practice. The ACLS 2000 Guidelines are no exception.

In this article, I've addressed several clinical topics that directly relate to ACLS teaching, either past or present, that merit reconsideration. Some of these represent long-held beliefs in ACLS that have been disproved by more recent studies. Others represent newer recommendations that are unsupported by the current literature. It is my hope that emergency care providers can utilize this information to improve patient care beyond the teaching of standard ACLS.

Pitfall: Reliance on the ECG and clinical information to distinguish between ventricular tachycardia and supraventricular tachycardia with aberrant conduction.

The ECG diagnosis of regular, wide-complex tachycardias (RWCT) can be challenging to even the most experienced emergency physicians and cardiologists. The differential diagnosis of the RWCT includes ventricular tachycardia (VT), sinus tachycardia with aberrant conduction (AC), atrial flutter with AC, and supraventricular tachycardia with aberrant conduction (SVT-AC). Sinus tachycardias with AC and atrial flutter with AC are usually easy to distinguish because of the presence of distinct atrial activity in relation to the QRS complexes on the ECG. However, the distinction between VT and SVT-AC can be very difficult. If the treating physician is considering the use of AV nodal blocking agents, especially calcium channel blockers, this distinction is critical; these agents, while very effective for SVT-AC, can cause significant hemodynamic compromise and adverse outcomes if administered to patients with VT. On the other hand, the administration of drugs usually used for treatment of VT (e.g., lidocaine, procainamide, amiodarone) to the patient with SVT-AC is considered safe and (in the case of procainamide and amiodarone) is often effective. As a result, medical students and resident physicians are classically taught that they should always assume that a RWCT is VT and to treat the rhythm as VT, *not* as SVT-AC. ACLS 2000, however, recommends that when physicians are confronted with a RWCT of unknown type, they should "Attempt to establish a specific diagnosis" through use of a 12-lead ECG (page I-159) and treat accordingly. If the physician concludes that the RWCT is an SVT-AC, traditional medications for SVT may be used. If the physician is incorrect in this determination and the RWCT is actually VT, the results of this treatment can be catastrophic. Given the ACLS recommendation to establish an ECG diagnosis, it is worth

evaluating the evidence regarding whether such a determination can be reliably made at all.

Over the past several decades, many electrocardiographers have tried to identify reliable ECG distinctions between VT and SVT-AC. Indeed, many ECG clues have been identified which essentially *rule in* VT (see Table 1). However, the *absence* of these ECG findings does not *rule out* VT (and therefore rule in SVT-AC). In fact, no electrocardiographer has ever been able to define a reliable set of criteria that *rule in SVT-AC*. The best attempt at defining such a set of criteria was Brugada, et al (2), who in 1991 published a four-step algorithm ("The Brugada Criteria") that reportedly could distinguish between VT and SVT-AC with great accuracy. The algorithm required physicians to evaluate the ECG for (1) absence of an RS complex in all precordial leads; (2) an RS interval >100ms in any one precordial lead; (3) AV dissociation; (4) various QRS morphologies in leads V_{1-2} and V_6. The presence of any one of the four criteria was considered diagnostic of VT, whereas the absence of all four criteria ruled out VT in more than 98% of cases…not perfect, but not too bad.

Attempts at validating the Brugada Criteria have been disappointing, however. In 1996, Herbert, et al (3) studied interobserver agreement between emergency physicians in the use of the Brugada Criteria. Twenty-seven RWCT ECGs were given to three emergency physicians that were trained in the use of these Criteria, and they were asked to decide whether the ECGs represented VT or SVT-AC. The physicians disagreed amongst each other on 22% of the ECGs. More recently, Isenhour, et al (4) studied interobserver agreement as well as accuracy in evaluating 157 RWCT ECGs. The final diagnosis of the ECGs was confirmed electrophysiologically. Two emergency physicians and two cardiologists were asked to use the Brugada Criteria in analyzing the ECGs. The sensitivity of the two emergency physicians in detecting VT was 83% and 79%, respectively, with an interobserver agreement of 82%. The cardiologists fared only slightly better, achieving sensitivities of 85% and 91%, respectively, with interobserver agreement of 81%. In other words, these four physicians using the Brugada Criteria misdiagnosed VT as SVT-AC in 9–21% of cases!

Perhaps the *only* time that the treating physician can reliably *rule out* VT and *rule in* SVT-AC is when the physician has access to a patient's baseline ECG in sinus rhythm and it demonstrates an identical QRS configuration. For example, Figure 1 demonstrates a RWCT. Figure 2 is the same patient's baseline ECG, showing sinus rhythm with a right bundle branch block pattern. Note that the QRS configurations are identical between the two ECGs, confirming that Figure 1 is an SVT-AC (SVT with right bundle branch block) and *not* VT. When applying this principle, however, physicians must make certain that the morphologies of the RWCT QRS and the sinus rhythm QRS complexes are *exactly identical*. Even slight differences in the QRS morphologies can suggest VT (5).

It should also be mentioned here that the use of "clinical information" (e.g., hemodynamic instability, older age, history of coronary artery disease) is suggested in ACLS 2000 to be helpful in the distinction between VT and SVT-AC (page I-159). While it is true that the presence of any of these factors strongly favors VT, once again the *absence* of these factors cannot *rule out* VT. There are many published cases of patients that are hemodynamically stable, young, and/or have no prior history of coronary artery disease that have VT. Example cases are demonstrated later in this paper.

So what's the bottom line for using the ECG to distinguish between VT and SVT-AC? Remember that even cardiologists have difficulty distinguishing between the two diagnoses when even the best criteria are applied. Failure to diagnose VT can be deadly. If the treating physician incorrectly diagnoses and treats the patient for SVT-AC based on the ECG, there can be disastrous consequences. ACLS 2000's suggestion that the 12-lead ECG (and clinical information) should be used to "establish a specific diagnosis" is dangerous. Stick with what you learned in medical school and residency: all RWCTs (minus the obvious cases of sinus tachycardia or atrial flutter with AC) should be treated as VT!

Table 1: ECG Clues Favoring VT

AV dissociation	Leftward axis
Captured beats	Rightward axis
Fusion beats	Extreme rightward axis
QRS duration > 140 ms.	Specific QRS morphologies in V1, V6
Precordial concordance	

Pitfall: The use of adenosine in distinguishing between ventricular tachycardia and supraventricular tachycardia with aberrant conduction.

Previous editions of ACLS (6) suggested the use of adenosine when treating "wide complex tachycardias of uncertain type." It was placed in the tachycardia algorithm after lidocaine but before procainamide. The idea was that if the RWCT was simply an SVT-AC, adenosine would rapidly convert the rhythm. Many physicians extrapolated this to mean that adenosine would also diagnose the rhythm as SVT-AC. I personally recall ACLS instructors and even some cardiologists saying that adenosine would convert SVTs (in more than 90% of cases) but leave VT unchanged. Adenosine was not only considered therapeutic, but it was also considered a diagnostic maneuver; i.e., it could *rule out* VT if the patient converted to sinus rhythm.

Few physicians are aware, however, that adenosine *will convert some cases of VT to sinus rhythm* as well; this has been reported in both adult and pediatric patients with VT (7-9). Electrophysiologists have known about this "adenosine-sensitive VT" for years, but this phenomenon is not well reported in the general medicine or emergency medicine literature. Further complicating matters is the fact that most of the patients with adenosine-sensitive VT are relatively young patients with no underlying coronary artery disease or structural heart disease (9). These are the patients that are most likely to be misdiagnosed as having SVT-AC — young patients with no underlying cardiac disease who convert to sinus rhythm after adenosine administration! Adenosine can be used safely in most RWCTs, but physicians must be careful not to rely on its ability to distinguish VT from SVT-AC.

Two cases of adenosine-sensitive VT follow. In the first case, a 54-year-old man with no prior history of heart disease was admitted to the hospital for evaluation of intermittent palpitations. When his symptoms recurred, the ECG in Figure 3 was obtained. The ECG demonstrates several classic features of VT, including rightward axis, taller "left rabbit ear" in lead V1, fusion beats, and the presence of atrioventricular (AV)

dissociation. His blood pressure was 125/65 and heart rate 120 beats/min. Intravenous adenosine was administered, resulting in conversion to sinus rhythm with persistence of AV dissociation and a junctional escape rhythm (Figure 4). Twenty minutes later, the patient reverted to his previous rhythm. His rhythm was eventually successfully treated with intravenous procainamide. Electrophysiologic (EP) testing confirmed that the rhythm in Figure 3 was VT. In another case, a 34-year-old man with no prior cardiac history presented to an emergency department with shortness of breath and palpitations. His blood pressure was 112/64, heart rate 150 beats/min, respiratory rate 22 breaths/min, and pulse oximetry 99%. An ECG was obtained (Figure 5), demonstrating a RWCT. This ECG, like that shown in Figure 3, has several characteristics, any one of which are very strongly suggestive of VT: leftward axis, "steeple" morphology of the QRS complex in lead V1, marked QRS widening (>140 ms), and rS complex in lead V6. However, because of the patient's age, hemodynamic stability, and absence of cardiac history, he was assumed to have SVT-AC and treated with intravenous adenosine. After a 12 mg bolus was administered, he converted to sinus rhythm (Figure 6). The patient was admitted, but reverted to the RWCT after 12 hours. He had not been treated with any prophylactic antidysrhythmics (e.g., lidocaine, procainamide, or amiodarone infusion). He was then treated with amiodarone successfully. Subsequent EP testing confirmed VT.

Pitfall: Use of atrioventricular nodal blocking agents in patients with atrial fibrillation and Wolff-Parkinson-White Syndrome.

Atrioventricular nodal blocking agents (e.g. calcium channel blockers, beta adrenergic blockers, and digoxin) are commonly used for treatment of rapid ventricular rates in the presence of atrial fibrillation (AF). Adenosine is another potent AV nodal blocking agent that is sometimes used when the diagnosis is uncertain and SVT is a consideration. Adenosine will not effectively treat the rate or the rhythm in AF, but it is very short acting and generally considered safe. The use of AV nodal blocking agents in AF patients with Wolff-Parkinson-White Syndrome, however, may be deadly.

Wolff-Parkinson-White Syndrome (WPW) is the most common form of ventricular preexcitation, involving an accessory conduction pathway known as the bundle of Kent. This pathway creates a direct electrical connection between the atria and ventricles, bypassing the AV node (10). As a result, electrical impulses utilizing the accessory pathway are conducted very rapidly directly to the ventricles without the usual slowing or "filtering" (blocking) effect of the AV node. In the presence of AF, WPW patients can achieve ventricular rates in excess of 300 beats per minute. Patients with AF and WPW (AF-WPW) will display three electrocardiographic characteristics that will distinguish them from patients with VT, AF with bundle branch block, and other types of wide complex tachycardias: (1) the rhythm will be irregularly irregular; (2) the QRS morphologies will change in size and shape reflecting some conduction through the accessory pathway, some conduction through the normal pathway, and some fusion beats; (3) ventricular rates in *some* portions of the ECG may exceed 250-300 beats per minute. Figure 7 is an example of a patient with AF-WPW, demonstrating all three of these characteristic findings. The use of AV nodal blocking agents in this condition has been demonstrated to facilitate conduction through the accessory pathway. This results in

acceleration of the ventricular rate producing ventricular fibrillation. Figure 8 panel A demonstrates an example of a patient with AF-WPW that was treated with adenosine; panel B shows the result 10 seconds later. The reason this patient was treated with adenosine is unclear. However, when the ventricular rate in AF-WPW is extremely rapid, the rhythm can have a relatively regular appearance and lead to the misdiagnosis of SVT-AC or VT. A *quick glance* at the ECG (e.g., in Figure 8) may lead a hurried physician to this misdiagnosis. If adenosine is then chosen for treatment, it can produce disastrous results. This problem can be avoided by simply using ECG calipers when evaluating the rhythm. The irregularity then becomes obvious, ruling out VT or SVT-AC and excluding any reason to use adenosine.

Patients with AF-WPW can sometimes be misdiagnosed as having AF with a bundle branch block (AF-BBB). However, patients with AF-BBB rarely have ventricular rates exceeding 200 beats per minute, even transiently. Another important distinction is that patients with AF-BBB will have unchanging QRS morphologies on the ECG. Once AF-BBB has been ruled out, the physician should no longer have *any* thought of using an AV nodal blocking agent.

Treatment of patients with AF-WPW depends on hemodynamic stability. Unstable patients should be treated with electrical cardioversion. Unlike patients with chronic AF, these patients respond well to cardioversion. Hemodynamically stable patients can be treated either with sedation and electrical cardioversion, or they can be treated with intravenous procainamide. Procainamide, a type IA antidysrhythmic, selectively suppresses conduction in the accessory pathway, reduces the ventricular rate, and leads to cardioversion. ACLS 2000 (page I-161) lists amiodarone as an equivalent choice. However, there is little data to support this recommendation. On the contrary, there are case reports (11-13) documenting adverse effects and hemodynamic decompensation in AF-WPW patients that receive amiodarone. Some authorities (10, 14), therefore, advise against using amiodarone in this condition. I recommend using intravenous procainamide in only the most stable of patients. At the first sign of any instability, patients should receive rapid electrical cardioversion (preceded by sedation whenever possible).

Pitfall: Over-reliance on lidocaine as an effective treatment for ventricular tachycardia.

Lidocaine has been considered the medication of choice for patients with VT for many years. Many physicians may therefore assume that lidocaine is a very effective treatment for VT. In reality, lidocaine's success rate at treating VT is very poor. Multiple studies have demonstrated that lidocaine is successful in converting VT to sinus rhythm in only 20–30% of cases (15-19). Procainamide and amiodarone, on the other hand, are far more successful at converting VT. The new VT algorithm in ACLS 2000 (page I-163) reflects a shift from previous editions of ACLS by recommending procainamide (or sotalol) as the drug of choice for stable monomorphic VT with unimpaired cardiac function and amiodarone (or lidocaine) in the presence of a poor ejection fraction.

Lidocaine still clearly has a place in the treatment of patients with stable VT. Advantages of lidocaine include ease of dosing and administration (which allow it to be

administered more rapidly than procainamide or amiodarone), rapid effect (when it *does* work), minimal side effect profile, and low cost. Given these advantages, physicians can still feel justified in using lidocaine as the drug of choice in patients with VT. However, physicians that choose to use lidocaine must be prepared to move on to second-line medications rapidly in cases when lidocaine is ineffective.

Pitfall: Use of high-dose epinephrine in patients with cardiac arrest.

Intermediate, escalating, and high dosages of epinephrine (HDE) had been given class IIb classification ("possibly useful and effective") in previous editions of ACLS (6,20), largely based on animal studies and small human trials (21-26). These studies demonstrated an improved return of spontaneous circulation (ROSC) and survival to hospital admission when adult cardiac arrest patients were treated with HDE versus standard dosages of epinephrine (SDE). It was assumed that this improvement in ROSC and survival to hospital admission would translate into increased rates of survival to hospital discharge and neurologic recovery as well. However, this was not the case. Subsequent studies (27-33) confirmed that HDE is not associated with improved survival to hospital discharge, and there is some suggestion that HDE is actually associated with a slightly worse neurologic outcome in survivors. No subgroups of cardiac arrest patients have been identified which benefit from HDE. As a result, HDE is no longer recommended in ACLS 2000.

Pitfall: Over-reliance on amiodarone in patients with refractory ventricular tachycardia/ventricular fibrillation.

Amiodarone is a class III antidysrhythmic (potassium channel blocker) with some additional class I (sodium channel blocking), class II (beta adrenergic blocking), and class IV (calcium channel blocking) effects. This potpourri of physiologic effects has led amiodarone to be effectively used in managing a variety of dysrhythmias, including stable VT, atrial fibrillation, and other types of SVTs. Early small studies (34,35) indicated that amiodarone might also be effective in treating patients with refractory VT and ventricular fibrillation (VF). However, use of amiodarone for this indication was not routinely recommended until 1999 when Kudenchuck, et al (36) published a randomized double blind prehospital trial of amiodarone vs. placebo for shock-resistant VT/VF. In this manufacturer-sponsored study, patients received three shocks, intubation, and 1 mg of epinephrine (EPI), then either 300 mg of intravenous amiodarone or placebo. Subsequent treatment was left to the discretion of the medics or physicians. Kudenchuk found that amiodarone-treated patients were more likely to survive to hospital admission with spontaneous circulation than placebo patients (44% vs. 34%). However, there was no significant difference in terms of survival to hospital discharge (13.4% vs. 13.2%) or good neurologic outcome (7.3% vs. 6.6%).

Further studies will help determine the exact role of amiodarone for patients with refractory VT/VF. The ALIVE study is a manufacturer-supported Canadian prehospital trial of amiodarone vs. lidocaine. The study has already been completed. Preliminary

results were announced at the North American Society of Pacing and Electrophysiology in March 2001, indicating that amiodarone use was associated with increased ROSC and survival to hospital admission compared to lidocaine. However, the researchers did not comment on survival to hospital discharge or neurologic outcome. This information will apparently not be available until the study is published. Another study, ALERT, is an in-hospital study of amiodarone vs. lidocaine. The study results have not yet been released.

The company that produces amiodarone has aggressively marketed this drug for routine use in all patients with refractory VT/VF. ACLS 2000 has given amiodarone a IIb classification for this indication and lidocaine an "indeterminate" classification. This has led some physicians to assume that amiodarone is the drug-of-choice in refractory VT/VF (after EPI). The American Academy of Emergency Medicine has responded by issuing a Position Statement on the use of amiodarone in refractory VT/VF (37). It states that "the use of amiodarone in refractory pulseless VT/VF should not be considered the current 'standard of care' for this condition" and that "Until ongoing or future research clarifies this issue, emergency physicians should use their own discretion regarding antiarrhythmic therapy in patients with cardiac arrest." Several factors should be considered when making the decision regarding which medications to use. Amiodarone costs significantly more than lidocaine or procainamide. The significant cost associated with increased admissions to the hospital ICU should be considered as well, especially given the lack of evidence of improved survival to hospital discharge when using amiodarone. In a national healthcare system that has finite money and resources, could we be using that money for greater benefit elsewhere? Better access to preventive care for the poor? Improved prenatal care? Higher nursing salaries? The results of ongoing and future studies may help answer these questions. For now, however, amiodarone bears strong resemblance to a more expensive version of HDE.

Pitfall: Over-reliance on vasopressin for treatment of patients in refractory ventricular fibrillation.

One of the biggest surprises to many physicians regarding ACLS 2000 was the inclusion of vasopressin in the pulseless VT/VF algorithm (page I-147). A single dose of vasopressin 40 U IV could be given as an alternative to repeated doses of EPI. Vasopressin is a naturally occurring antidiuretic hormone that acts upon receptors in the renal collecting ducts as well as upon receptors in vascular smooth muscle. In high dosages, the vascular smooth muscle effect produces potent peripheral vasoconstriction. This results in the beneficial effect of increased diastolic aortic blood pressure and coronary perfusion pressure, similar to the alpha-agonist effect of EPI. However, vasopressin does not have the adverse beta-agonist effect of EPI, which is associated with increased myocardial oxygen consumption.

Early studies utilizing a pig model for cardiac arrest (38-41) lent support to the idea that vasopressin would produce all the beneficial effects of EPI without the adverse effects. These studies demonstrated improved coronary perfusion pressure, improved vital organ blood flow, improved cerebral oxygen delivery, no increase in myocardial oxygen demand, and improved ROSC. Then in 1996 Lindner, et al (42) published a case

series of eight hospitalized patients with persistent VF that received vasopressin, after which all had ROSC. Three patients survived to hospital discharge.

In 1997, Lindner, et al (43) published the first randomized, double-blind trial of vasopressin vs. EPI. In this prehospital study, 40 VF patients that were resistant to initial CPR and defibrillation were randomized to receive either EPI 1 mg or vasopressin 40 units, then further standard ACLS measures. Vasopressin was associated with statistically significant improvements in ROSC (16/20 patients vs. 11/20 patients) and survival to hospital admission (14/20 patients vs. 7/20 patients). Survivors to hospital discharge included 8/20 patients in the vasopressin group vs. 3/20 patients in the EPI group; however, the difference between vasopressin and EPI in this group was not statistically significant because of the small size of the study.

A larger study published in July 2001 by Stiell, et al (43) showed no benefit of vasopressin vs. EPI. In that Canadian in-hospital study, 200 patient were randomized to receive either a single dose of vasopressin 40 units or EPI 1 mg. Patients in either group that failed to respond after the first dose received subsequent doses of EPI (1 mg every 3-5 minutes). The results of the study showed no difference between the two groups in ROSC, survival to hospital discharge, or neurologic outcome. The authors concluded that they "cannot recommend the routine use of vasopressin for inhospital cardiac arrest patients, and disagree with American Heart Association guidelines, which recommend vasopressin as alternative therapy for cardiac arrest." Ongoing studies should further clarify the role of vasopressin, if any, for patients in VF and other forms of cardiac arrest.

Pearls for Improving Patient Outcomes

- The 12-lead ECG and clinical information are unreliable in distinguishing between VT vs. SVT-AC. Assume all RWCTs are VT!
- Do not give adenosine as a diagnostic challenge to distinguish between VT and SVT-AC.
- Look carefully for the signs of AF-WPW in all wide complex tachycardias, and avoid all AV nodal blocking agents in those patients.
- Consider early use of procainamide or amiodarone for patients with stable VT. Lidocaine can be used, but should but its use should be abandoned quickly if the first 1-2 boluses do not work.
- The use of HDE in adult cardiac arrest patients should be abandoned!
- Until increased survival to hospital discharge and neurologic recovery is demonstrated, the use of amiodarone for refractory VT/VF should not be considered to represent the standard of care.
- Do not rely on vasopressin to improve outcomes in patients with refractory VT/VF. The best benefit still remains with rapid defibrillation.

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