Amiodarone Supplants Lidocaine in ACLS and CPR Protocols

Anna Mizzi, MD[a,*], Thanh Tran[b], Devanand Mangar, MD[b], Enrico M. Camporesi, MD[c]

KEYWORDS

• Amiodarone • Ventricular tachyarrhythmias • Cardiac surgery • ACLS protocol

Amiodarone is an antiarrhythmic medication used to treat and prevent certain types of serious, life-threatening ventricular arrhythmias. Amiodarone gained slow acceptance outside the specialized field of cardiac antiarrhythmic surgery because the side effects are significant. Recent adoption of amiodarone in the ACLS (Advanced Cardiac Life Support) protocol has somewhat popularized this class of antiarrhythmics. Its use is slowly expanding in the acute medicine setting of anesthetics. This article summarizes the use of amiodarone by anesthesiologists in the operating room and during cardiopulmonary resuscitation (CPR).

SUDDEN CARDIAC DEATH

In a population of 1000, the average annual occurrence of sudden cardiac death (SCD) is approximately 0.2%, but population-related frequency of cardiovascular disease in different areas of the country should be considered. There are approximately 400,000 to 450,000 recorded occurrences of SCD in the United States, which accounts for about 60% of all cardiovascular mortality in this country.[1] Holter studies further indicate that approximately 85% percent of SCDs are caused by ventricular tachyarrhythmias, both pulseless ventricular tachycardia (VT) and ventricular fibrillation (VF).

VT is a critical condition that can lead to VF. VT is characterized as monomorphic when waveforms are at a steady rate and amplitude, and polymorphic when they are inconsistently variable. VF is another critical condition whereby the ventricles tremble rather than contract. VF waveforms are inconsistent in rate and amplitude,

[a] Department of Cardiothoracic and Vascular Anesthesia, San Raffaele Hospital, "Vita e Salute" University, Milan, Italy
[b] Florida Gulf-to-Bay Anesthesiology, 1 Tampa General Circle, Suite A327, Tampa, FL 33606, USA
[c] Department of Surgery/Anesthesiology, Florida Gulf-to-Bay Anesthesiology Associates & University of South Florida, 1 Tampa General Circle, Suite A327, Tampa, FL 33606, USA
* Corresponding author. 1 Tampa General Circle, Suite A327, Tampa, FL 33606.
E-mail address: anna.mizzi@alice.it

Anesthesiology Clin 29 (2011) 535–545
doi:10.1016/j.anclin.2011.05.001 **anesthesiology.theclinics.com**
1932-2275/11/$ – see front matter

often more than 300 beats per minute and more than 0.2 mV in amplitude. The irregular rate and amplitude indicates the inconsistent and hectic electrical activity and contraction when the heart stops pumping. VF waveforms often weaken to asystole within 15 minutes.[2]

In countries with prosperous resources, such as the United States and Europe, cardiac arrest due to VT or VF is mostly caused by myocardial ischemia. As a consequence, major risk factors for SCD include those factors that can accelerate coronary artery disease. Other risk factors associated with SCD include age (typically 45–75 years), male sex, and dilated cardiomyopathy.[2]

Cardiac arrest due to VT or VF causes an interruption in oxygen supply that can lead to critical ischemic damage to the organs. This condition is life-threatening, and leads to death within minutes if untreated. The treatments for pulseless ventricular tachyarrhythmias include use of defibrillation and antiarrhythmic drugs.[3]

CARDIAC ACTION POTENTIALS

Cardiac action potentials are divided into fast-response action potential and slow-response action potential. Fast-response action potential, also known as nonpacemaker action potential, is found in nonnodal cardiomyocytes (atrial and ventricular myocytes, and Purkinje tissue). This action potential type relies on fast sodium channels for depolarization. Slow-response action potentials, on the other hand, also known as pacemaker action potentials, are found in nodal tissue, which consists of sinoatrial and atrioventricular nodes, and depend on calcium channels rather than sodium channels for depolarization (**Fig. 1**).[4]

Cardiac action potentials, in general, use sodium and calcium channels for depolarization, and potassium channels for repolarization. In fast-response action potential, phase 0 represents depolarization, whereby sodium channels open to enhance positive membrane potential. Phase 1 is the initial repolarization, when potassium channels open and allow outward K^+ current. There is, however, a slow increase in inward Ca^{2+} current during this time, impeding the repolarization seen in phase 2. This phase, however, lengthens the period of action potential, and is the only phase that shows a difference between cardiac action potentials and those of the nerves and skeletal muscle. Phase 3 is then the continuation of the repolarization, and phase 4 allows the action potential to return to resting membrane potential.[4]

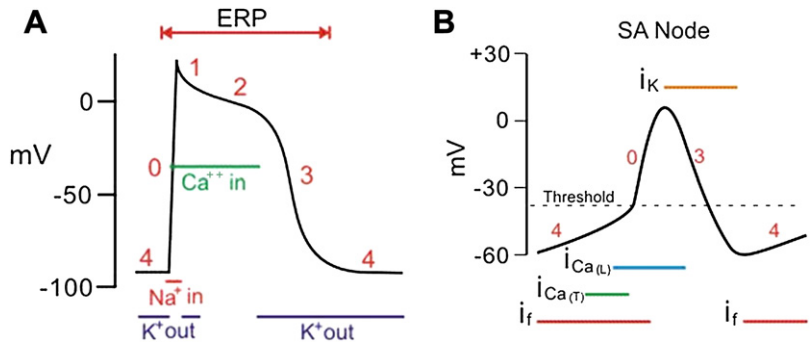

Fig. 1. Cardiac action potential. (*A*) Fast-response action potential (atrial and ventricular myocytes, and Purkinje tissue). (*B*) Slow-response action potential (sinoatrial and atrioventricular nodes). ERP, effective refractory period; SA, sinoatrial.