COMMENTARY

Intense dive into the evidence: Epinephrine in cardiac arrest

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DESCRIPTION

Cardiac arrest is known to be the sudden stoppage of cardiac mechanical activity due to dysrhythmia or electromechanical dissociation or it is the abrupt loss of cardiac function in a person who may have or may not have been diagnosed with heart disease. From most of the studies it has been stated that more than 356,000 cardiac arrests occur outside a hospital in the U.S. each year. Cardiac arrest can come on suddenly with resulting in lethal brain and other end-organ damage until and unless it is reversed in within minutes. These cardiac patient's outcomes require not only the restoration of effective cardiac activity but also reversal and mitigation of whole-body ischemia and reperfusion. With rapid initiation of chest compressions, rapid electrical defibrillation of ventricular dysrhythmias and induction of mild hypothermia for 12-24 hours after restoration of pulses would possible to provide better outcome of result. Epinephrine (EPI), also known as adrenaline is a medication and hormone belonging to the class of alpha- and beta-adrenergic agonists (sympathomimetic agents), which works by relaxing the muscles in the airways and tightening the blood vessels. As a medication it is used to treat numerous conditions including anaphylaxis, cardiac arrest, asthma, and superficial bleeding. This drug is known to be the primary drug advocated for patients who are receiving chest compressions during cardiac arrest. But the use of EPI in cardiac arrest has been a source of significant controversy for many years.

Some studies concluded that the use of EPI in high dose consistently showed improvements in rates of Return of Spontaneous Circulation (ROSC) and failed to show improvements in neurologic recovery. But the use of regular-dose EPI in 1-mg IV boluses every 3-5 minutes was continually endorsed by the American Heart Association (AHA) with a Class IIb recommendation (possibly helpful class). Its mechanism of action includes by augmenting coronary blood flow generated by chest compressions during Cardio Pulmonary Resuscitation (CPR). Coronary Perfusion Pressure (CPP), defined as the difference between aortic blood pressure and the right atrial pressure, is the major determinant of coronary blood flow. Whenever the CPR does not generate CPP of more than 15-20 mmHg, the return of spontaneous circulation rarely or never occurs. After more than a few minutes of cardiac arrest the atrial tone collapses and EPI or another vasoconstrictor is essential for restoration of cardiac activity. Where EPI works by increasing the aortic pressure during chest compressions via alpha-adrenergic constriction of arterioles, which thereby increases pressure in the proximal aorta. Hence the Increased aortic pressure may shunt more blood into the coronary arteries and increase the probability of ROSC. From the clinical experiences it has been found that there are no convincing dose-response data for epinephrine use during cardiac arrest and long-term outcome. From some studies it has been concluded that 20-30 kg dose used 1-mg dose of epinephrine, which has been the standard for adult patients ever since. Administration of EPI and increasing EPI dosage are associated with worse survival and neurological outcome after cardiac arrest.

Most of researchers confounded the fact that epinephrine is only administered when initial CPR or defibrillation is unsuccessful and the total dose of epinephrine administered is proportional to how long a patient remains in cardiac arrest, resulting in higher doses for patients who fail to respond to initial treatment. Efficacy of epinephrine declines with duration of cardiac arrest. An observational study conducted in Osaka, Japan including of 3161 cardiac arrests, where the patients compared who received versus those who did not receive epinephrine and analyzed the timing of epinephrine administration. Thereby the study concluded that EPI administered within 10 min of cardiac arrest to patients with VF was associated with better functional outcomes. Hence most of the studies concluded that epinephrine do not provide clear evidence for long-term patient benefit with indiscriminate use of 1-mg boluses (0.05-0.1 mg/kg) of epinephrine during CPR.

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