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CLINICAL PAPER

Post-shock myocardial stunning: A prospective randomised double-blind comparison of monophasic and biphasic waveforms☆

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Summary
Introduction: Compared with monophasic defibrillation, biphasic defibrillation is associated with less myocardial stunning and earlier activation of sodium channels. We therefore hypothesised that earlier sodium channel activation would result in earlier restoration of the first sinus beat following elective DC cardioversion.
Methods: Adults undergoing elective DC cardioversion were randomised to receive either monophasic or biphasic escalating transthoracic shocks. The ECG was recorded electronically during defibrillation and the time from delivery of the shock to restoration of the first sinus beat, measured from the beginning of the 'P' wave, was calculated.
Results: Seventy four patients were studied. Data were unavailable from 18 patients. There was no demographic difference between groups. Median time to the first sinus beat following monophasic defibrillation ($n = 25$) was 3.66 s (95% CI 2.55—4.61 s) and following biphasic defibrillation ($n = 33$) was 2.21 s (95% CI 1.76—2.56 s; $P \leq 0.0001$). Linear regression confirmed that the waveform was an independent predictor of time to restoration of sinus rhythm; $P < 0.0001$. The final defibrillation energy level used to achieve cardioversion was not an independent predictor of time to restoration of sinus rhythm; $P = 0.49$.
Conclusion: Biphasic defibrillation for elective DC cardioversion achieved more rapid restoration of the first sinus beat compared with a monophasic waveform. Waveform, but not energy level that achieved defibrillation, was an independent predictor of time to restoration of the first sinus beat. The mechanism for this may be related to the earlier reactivation of sodium channels associated with the biphasic waveform.

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Introduction

The clinical use of electricity for myocardial defibrillation is a well established technique, having first been described for internal cardioversion in 1947 and external transthoracic cardioversion in 1956. The mechanism by which electrical energy is able to terminate atrial and ventricular arrhythmias has not been fully elucidated, although several hypotheses attempt to explain the mechanism of defibrillation. The critical mass hypothesis, the upper limit of vulnerability hypothesis and more recently the virtual electrode polarization hypothesis all assume the delivery of electrical energy of sufficient magnitude to depolarise and render refractory sufficient areas of myocardium to prevent wavefront propagation.[1,2] The amount of electrical energy necessary for successful defibrillation varies depending on the state of the myocardial substrate and increases as the electrophysiological state deteriorates. Delivery of defibrillation energy therefore aims to achieve sufficient current to exceed the threshold necessary to achieve defibrillation, irrespective of the electrophysiological deterioration.

The superiority of a biphasic over monophasic waveform for external DC cardioversion of both atrial and ventricular arrhythmias has generally been established.[3,4] There is general agreement that optimal biphasic waveforms are more efficacious than monophasic waveforms in termination of arrhythmias, particularly long-duration ventricular fibrillation. Over the past decade, biphasic waveforms have generally superseded the original monophasic waveform as the optimal waveform for clinical defibrillation.

Electrical energy delivered during defibrillation may itself disrupt the functioning of the cell membrane, resulting in impaired myocardial function. Shock-induced disruption is characterised by disruption of the lipid matrix and formation of aqueous pores, a pattern of injury known as 'electroporation'.[5] This may disrupt ionic homeostatic mechanisms, causing loss of intracellular potassium and cytosolic sodium and causing calcium overload.[6,7] Functionally, some, but not all studies have shown an energy related impairment in ventricular contractility, presumed secondary to these mechanisms. In animal studies, biphasic shocks with lower energy levels are generally associated with less disruption of myocardial function as evidenced by cardiac output, ejection fraction, systolic pressure and left ventricular end-diastolic volume,[8,9] reduced injury potentials[9] and post-shock arrhythmias.[10,11]

The electrophysiological differences between biphasic and monophasic waveforms have also been studied in relation to the sodium channel. The sodium channel responsible for depolarisation has two gates: an activation gate (m) which is closed at rest but opens rapidly on depolarisation and an inactivation gate (h) which is open at rest but closes slowly on depolarisation. When an electrical stimulus of sufficient magnitude depolarises the myocyte membrane, the m-gate opens causing sodium to enter the cell and triggers an action potential. The action potential is then terminated by the slow closure of the h-gate.[12] Further depolarization is not possible until the h-gate has fully recovered, this phase being the refractory period. In computer models of the ventricular action potential, hyperpolarization by the first phase of a biphasic waveform causes early hyperpolarization that enhances the recovery of sodium inactivation and increases the sodium current to shorten the cellular refractory period.[13,14] These findings are consistent with studies of biphasic waveforms that have reported earlier reactivation of myocyte sodium channels.[13,15,16] In light of these studies suggesting earlier recovery of sodium channel function associated with biphasic waveforms, we investigated patients undergoing DC cardioversion for atrial fibrillation in order to study whether this earlier reactivation was associated with quicker restoration of the first sinus beat following the short period of myocardial stunning that occurs after defibrillation.

Materials and methods

Following local Ethics Committee approval, we studied 74 patients prospectively undergoing elective day case DC cardioversion for atrial fibrillation or flutter. Patients were randomised using an internet-based random number generator (www.randomizer.org) to undergo cardioversion using either a monophasic or biphasic waveform. The waveform allocation was contained in a sealed envelope that was opened at the time of the cardioversion. The cardioversion was performed by a cardiologist who was not involved in the study. Study patients were unblinded at the time of data analysis.

Patients were fasted for at least 6 h prior to general anaesthesia which was administered as per normal protocol for the procedure. Patients continued their normal medications prior to the procedure and no premedication was given. Non-invasive blood pressure and pulse oximetry monitoring were established. The electrocardiogram was monitored directly through the defibrillator