Is a biphasic defibrillation waveform “better” than a monophasic waveform for cardioversion from atrial fibrillation?

In a previous issue of Heart, Scholten and colleagues presented data to show that a biphasic external atrial defibrillation shock waveform offers no significant advantage over a monophasic shock waveform in current clinical practice.1 However, there is evidence in this study and elsewhere to suggest that a biphasic defibrillation waveform is “better” than monophasic.2–4 The sense that this should be so may be engendered by the past experience of electrophysiologists with ventricular defibrillators and the decrease in defibrillation threshold that was seen with the advent of biphasic shock waveforms in these devices.5 The question is whether the perceived advantages of biphasic waveforms for atrial defibrillation can be translated into clinical benefit for patients?

SIMILAR EFFICACY

Scholten and colleagues have shown that biphasic and monophasic shocks are of similar efficacy in achieving sinus rhythm but at a lower energy level with biphasic waveform.1 In this sense the study’s outcome is similar to that of the study reported by Page and colleagues,2 who showed that for cardioversion of atrial fibrillation (AF), “a biphasic shock waveform has greater efficacy, requires fewer shocks and lower delivered energy, and results in less dermal injury than a monophasic shock waveform”. Although a multicentre international study the number of patients recruited was less than that in Scholten’s study population. However, as with Scholten and colleagues,1 at lower energy levels biphasic shocks were more successful than monophasic shocks. But over the course of four cardioversion shocks, biphasic performance (maximum of 200 J) was similar to monophasic performance (maximum of 360 J) (success rate 91% vs 85%, p = 0.29). Thus, in terms of achieving sinus rhythm the outcomes were similar in both studies and for both shock waveforms. Whether external paddle orientation (and therefore shock vector) may have a differential impact between shock waveforms has not been tested although paddle placement has been shown to be important in achieving atrial defibrillation success.5

If ultimate atrial defibrillation success rates are similar, does the need for higher energy delivery with monophasic shocks to achieve similar cardioversion efficacy result in a qualitatively important difference between shock waveforms? Page and colleagues2 reported lesser dermal injury with biphasic shocks than monophasic shocks. This is probably of minimal clinical importance. There was no difference in troponin values and the lack of evidence for any myocardial damage is supported by the outcome of other studies.6–8 While it is sensible to investigate whether lower energy delivery may lessen myocardial damage, there is no evidence to suggest that myocardial damage occurs.6–8 An animal model has suggested better maintenance of cardiac function after biphasic compared to monophasic waveform shocks,6 but this has yet to be shown to translate into clinical benefit for patients. It is possible that atrial stunning may be lessened by lower energy defibrillation, but again this is unproven and the benefits may be difficult to verify in a way that is meaningful for clinical practice. Perhaps a lesser period of post-cardioversion anticoagulation with no increased risk of thromboembolic events may be important, but such a study could be difficult to perform and its impact on clinical practice is likely to be marginal.

It is suggested by Scholten and colleagues1 that biphasic external shocks could reduce the need for internal (invasive) cardioversion, which has been shown to offer success when external cardioversion has failed.11 Perhaps a study is needed to compare low energy biphasic defibrillation with internal cardioversion.

ATRIAL DEFIBRILLATION IN CLINICAL PRACTICE

The role of atrial defibrillation in clinical practice is coming under scrutiny. The AFFIRM (atrial fibrillation follow-up investigation of rhythm management) study investigators12 reported no improvement in survival benefit or patient well being when a strategy of maintenance of sinus rhythm was compared in a randomised study to control of ventricular rate response, but the study has flaws related to patient recruitment and management strategies. Nevertheless, the investigators conclude that “management of atrial fibrillation with the rhythm-control strategy offers no survival advantage over the rate-control strategy, and there are potential advantages, such as a lower risk of adverse drug effects, with the rate-control strategy”.12 Others have also reported that rate control is not inferior to rhythm control for the prevention of death and morbidity from cardiovascular causes and may be appropriate treatment in patients with a
recurrence of persistent atrial fibrillation after electrical cardioversion.11 These observations contrast with those of others whose study results demonstrate that an aggressive policy towards persistent atrial fibrillation by means of repetition of electrical cardioversion after early fibrillation recurrence is useful in maintaining sinus rhythm after 12 months.12 The reality is that persistent/permanent atrial fibrillation is a major cause of morbidity and mortality.13 Most clinicians will continue to strive to maintain sinus rhythm in their patients with atrial fibrillation, but the conclusion drawn by Scholten and colleagues’ that biphasic defibrillators are not immediately necessary to help this endeavour is sensible.

There is no immediate need for replacement of external defibrillators with those that deliver biphasic shock waveforms. For such a need to become apparent, there must be evidence for significant clinical benefit: lessened myocardial damage, clinically relevant reduction in atrial stunning and thromboembolic risk, or perhaps increased or comparable efficacy with internal cardioversion shocks.

REFERENCES


IMAGES IN CARDIOLOGY

Crystals in the heart

A 22 year old woman was treated with an implantable cardioverter-defibrillator for ventricular tachycardia (VT) associated with dilated cardiomyopathy. The multifocal slow VTs persisted and were resistant to several antiarrhythmic drugs. Finally, nifekalant, a pure IKr channel blocker, was administered continuously and was effective for 10 months. A round mass was incidentally detected in the right atrium by transoesophageal ultrasonography and was moving to and fro through the tricuspid valves (panels A and B). An emergency operation was performed to remove this mass using cardiopulmonary bypass. The resected mass was a white elastic ball sized 5 × 4 × 3 cm and histopathology revealed a fibrin thrombus containing needle crystals without a foreign body reaction (lower panel, haematoxylin and eosin × 400). This mass contained an extremely high amount of nifekalant and was traced through the superior vena cava up to the right brachiocephalic vein, which coincided with the route of nifekalant administration. These findings suggest that continuous intravenous administration of this antiarrhythmic drug resulted in thrombus and crystal formation in the heart.

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