Many of you will be aware that worksheets, prepared by international reviewers are now available for public view on the ILCOR website www.C2005.org. This site gives everyone an opportunity to comment or raise questions for consideration at the consensus conference to be held in Dallas, Texas on 23-30 January 2005.

In November 2005, the 2005 International Consensus on Cardiopulmonary (CPR) and Emergency Cardiovascular Care (ECC) Science and Treatment Recommendations (CoSTR2005) will be published simultaneously in the journals Circulation and Resuscitation. This will provide a summary of the science and some treatment recommendations; it will not contain the new resuscitation guidelines. The European Resuscitation Council (ERC) will publish its new guidelines in the journal Resuscitation in December 2005, and the Resuscitation Council (UK) guidelines will be published shortly afterwards.

Resuscitation Council (UK) life support courses

The relevant subcommittees will be able to agree the final content of the new courses only once the ERC and Resuscitation Council (UK) guidelines have been finalized. Because of the vast amount of material that will require changing, the Guidelines Project Group has decided to stage the introduction of these new courses. Once the new material has been prepared, time will be needed for printing and for distribution to course centres and instructors so that they receive the material at least 6 weeks before the date of a course. Therefore, it is planned that the first of each of the new courses shall be held as follows:

ALS and ILS - June 2006.
NLS - July/August 2006.
EPLS - September 2006.

Courses held between now and next summer

Those of you who look at the worksheets on the ILCOR website will see how often the search for evidence on which to base guidelines has ended with conclusions that are less than firm. It is likely that many of the changes in the guidelines, therefore, will be aimed at simplification and better education, rather than the introduction of fundamentally new procedures.

It is important, therefore, to emphasise that instructors should continue to run the current courses right up until the new ones are launched. To do otherwise, perhaps in the mistaken belief that something ‘wrong’ might be taught, would be to change the well-known aphorism into ‘No CPR is better than some CPR’. This would certainly be wrong.
INTRODUCTION
The heart is a mechano-sensitive organ. Its rate and rhythm are affected by intrinsic and extrinsic changes in the mechanical environment. One of the most ‘striking’ illustrations of this is the fact that pre-cardiac impacts may both trigger (commotio cordis) and terminate (pre-cardiac thump, PT) arrhythmias. The use of mechanical interventions, such as PT, to reset disturbed heart rhythms is controversial. This article summarises the means and anti-arrhythmic utility of cardiac mechanical stimulation, and address the mechanisms and potential clinical utility of this intervention.

MEANS OF CARDIAC MECHANICAL STIMULATION

Direct Cardiac Stimulation
Direct mechanical stimulation of cardiac muscle by ‘finger tap’ is a well-established method, used by surgeons to prompt rhythmic contractile activity in hearts after induced arrest during open heart surgery. While this may be one of the most regularly used mechanical interventions to restore the heartbeat, it is also one of the least well characterised, in terms of mechanics and mechanisms. Another form of direct mechanical stimulation occurs during cardiac catheterisation. This is often associated with induction of premature ventricular beats. Interestingly, catheter tip interactions with the cardiac wall can also lead to cardioversion from ventricular tachyarrhythmia and atrial fibrillation.

Intrathoracic Pressure Increase
Several reports have highlighted the link between an abrupt increase in intrathoracic pressure and termination of tachyarrhythmias. This type of mechanical cardioversion can be self-administered, for example by coughing, or via the Valsalva manoeuvre. Given that these interventions also work in heart transplant recipients, it is probable that the haemodynamic unloading of the heart observed in the above settings removes a mechanical substrate for either induction or sustenance of arrhythmias.

Extracorporal Impact: PT
First described in 1920 by Schott as a trigger of competent ventricular contraction in asystolic patients, PT has been used to mechanically pace asystolic hearts and to terminate tachycardia or (more rarely) ventricular fibrillation. Judging by the majority of published reports, PT has the best benefit-to-cost ratio if applied relatively early during the development of serious heart rhythm disturbances.

The universal algorithm for resuscitation includes PT as the first recommended procedure in witnessed cardiac arrest. Using the ulnar edge of the tightly clinched fist, a sharp impact to the lower half of the sternum is delivered with full force from a height of about 20 cm. The fist should be retracted actively after full impact, generating an impulse-like stimulus.

MECHANISMS

General Considerations
The theory underlying PT, developed in the 1970s, is that the mechanical stimulus causes a change in myocardial electrical properties via mechano-electrical feedback (MEF). In particular, excitable myocardium is depolarised upon mechanical stimulation, which either triggers ectopic excitation in quiescent tissue (thereby providing a means for mechanical pacing), or obliterates the excitable gap required to sustain re-entrant excitation (terminating tachyarrhythmias).
Experimental studies have overwhelmingly confirmed that stretch of resting myocardium does indeed cause depolarization. If mechanically-induced depolarization reaches the threshold for action potential generation, it gives rise to an ectopic beat, as seen in cardiac preparations ranging from single cells to tissue, whole hearts, and patients. The mechanically-induced depolarizations may be explained by the activation of stretch-activated ion channels in ventricular cardiomyocytes, which are capable of translating a mechanical stimulus into an electrical trigger.

In contrast to well-documented mechanical effects on the asystolic heart, there is less data on the mechanisms of mechanical cardioversion of ventricular tachycardia (VT) and fibrillation (VF). Most studies that reported cardioversion of VT or VF did not quantify the mechanical interventions. Nonetheless, the instantaneous conversion from VT or VF to normal sinus rhythm, observed in patients and experimental models, suggests that stretch-activation of ion channels is likely to play a role in these circumstances as well.

NEW INSIGHTS

Clinical Utility of PT: United Kingdom versus United States

There are marked international differences in the approach to mechanical cardioversion. We conducted a small survey on the use of PT in the United Kingdom (UK) and United States (US). There were 100 respondents (UK 57, US 43) reporting 1740 cases (UK 813, US 927). ‘Speed of delivery’ was ranked by 92.5% of the participants as the most important reason for using PT, while ‘perceived inefficiency’ (60.2%), ‘other established procedures’ (45.9%), and ‘unawareness of technique’ (37.8%) were reported to preclude more frequent application of PT. Only 54.3% of professionals were taught PT as part of their curriculum, and no established tools for training or assessment were identified. There was a pronounced difference in opinions on the appropriateness of PT application in VT and VF. UK participants ranked onset of VF (89.5%), followed by VF (54.4%), as the prime indications for PT, with VT in third position (35.1%). In the US, the trend was opposite, with VT (62.8%) narrowly leading over onset of VF (58.1%), with VF a distant third (25.6%). This correlated with a significantly higher success rate of US healthcare professionals, who reported ‘at least temporary cardioversion to normal sinus rhythm’ in 27.7% of PT cases, compared to only 13.3% in the UK. Of note: adverse side effects were rare (<0.5% of cases: UK 0.7%, US 0.2%) and largely of structural nature.

Clearly, this is a limited pilot study comprising just a few retrospective reports; however, the data imply that ‘early application’ of PT (during VT and very early VF) may be most productive.

PT Mechanics

To determine whether PT mechanics might have played an independent role in determining the difference in inter-continental success rates, we developed a ‘thump-o-meter’ to quantify PT impact mechanics. Healthcare professionals in both countries (44 in total: UK 22, US 22) performed three PT-like impacts each. Biomechanical recordings were then correlated to reported individual success rates in the application of PT. Inter-individual differences in pre-impact fist speed ranged from 0.42 to 8.14 m·s⁻¹. Participants with fist speeds of less than 2.25 m·s⁻¹ reported successful cardioversion in 18±3% of PT cases, compared to 36±2% for those who performed faster impacts (p < 0.01). The national average of pre-impact fist speeds was significantly higher among US participants (UK 1.55 ± 0.68 m·s⁻¹, US 4.17 ± 1.68 m·s⁻¹; p < 0.01) Thus, PT success rates are two times higher in the US cohort, compared to the UK. This may be related to differences in the arrhythmia targeted and/or the method used to deliver PT.

A minimum impact force appears to be required to achieve optimal mechanical cardioversion rates, which highlights the need for better procedural instructions and training aids, such as simplified ‘thump-o-meters’.

SUMMARY

Mechanical stimulation affects cardiac electrophysiology via MEF. Like an electrical current discharge, this may either cause or terminate arrhythmia. A potentially important advantage of mechanical energy delivery for cardioversion is that the necessary electrical currents are generated by the cardiac muscle cells. The mechanical stimulus is passed on from pericardium to cardiac tissue quite directly without being dissipated to other parts of the body (note: during trans-thoracic defibrillation in adult humans, only 4% of the applied current actually traverses the heart). PT involves delivery of much lower energy than electrical defibrillation (reducing trauma), and it can be tolerated by the conscious patient.

The understanding of the detailed mechanisms underlying mechanical cardioversion is patchy. Given the instantaneous electrical response of the heart to the mechanical stimulus, it is highly probable that stretch-activation of ion channels plays a key role. Regional differences in the myocardial electrophysiology, variations in the method of PT delivery, myocardial ischaemia, and drug therapy may all influence the potential (continued)
success of PT. Further progress in our understanding of the utility and limitations of mechanical pacing or termination of VT and VF requires novel experimental and computational models, which are being developed by several groups in the UK and US. It would be helpful to furthermore establish a repository of detailed recordings from patients who underwent attempted mechanical cardioversion, such as those that could be acquired from intensive care monitors or automated external defibrillator memory cards. A corresponding initiative is currently being launched at Oxford, and the authors would be delighted to hear from anyone wishing to contribute.

Finally, ALS guidelines should offer clear instructions on how to deliver PT, and training aids should be introduced to increase the probability of successful mechanical cardioversion (either as stand-alone ‘thump-o-meter’ type boxes, or as part of resuscitation mannequin technology).

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