Editorial

Cardiopulmonary resuscitation with active compression-decompression

The son... recommended that we placed toilet plungers next to all the beds in our coronary care unit.1

The case of an Iranian man who had twice been successfully resuscitated from prehospital cardiorespiratory arrest by relatives using a toilet plunger to deliver chest massage1 resulted in the development of an inexpensive adjunct to external cardiac compression. If initial animal and human research, suggesting increased survival, are supported by the findings of larger prospective trials the widespread use of this new device could lead to more effective resuscitation.

Mechanisms of blood flow during closed chest massage

Blood flow during chest massage depends upon fluctuations in intrathoracic pressure. Cerebral perfusion pressure, the difference between the aortic systolic pressure and a combination of the jugular venous and intracranial pressure, is greatest during the compression phase. The coronary perfusion pressure is generated during the relaxation phase and is the difference between the aortic root diastolic pressure and the simultaneous pressure in the right atrium.2 Coronary perfusion pressure appears most important in predicting the success of resuscitation—a gradient of less than 15 mm Hg is rarely associated with a successful outcome.3

During cardiac massage the arrested heart may function as a passive conduit while chest compression and passive relaxation causes the thorax to behave like a bellows: the thoracic pump mechanism. Alternatively, heart valves may remain competent during resuscitation allowing blood to circulate through the cardiac chambers: the cardiac pump mechanism. It is probable that both mechanisms operate to varying degrees.

Methods that augment the cardiac pump mechanism of blood flow are more likely to lead to improved survival than those that augment the thoracic pump mechanism.

Potential advantages of the active compression-decompression device

The CardioPump (Ambu International A/S, Copenhagen) consists of a silicone rubber vacuum cup of 13.5 cm diameter connected to a handle via a connection stem, within which is a spring whose compression and extension is shown, as “compression” and “decompression” force, on a gauge located in the handle. The cup is placed over the midsternum and during the compression phase the central area in contact with the skin is 5–6 cm in diameter. Air is forced out of the vacuum cup and when the handle is lifted upwards, during the decompression phase, the anterior chest wall is pulled forwards.

This active decompression would be expected to result in a relative negative intrathoracic pressure, leading to improved venous return to the heart and increased stroke volume during subsequent compression. This would augment the cardiac pump mechanism of blood flow. The negative intrathoracic pressure would also encourage air to enter the thorax. Significant ventilation caused by the device should be of benefit. Furthermore increased trapping of air in the lungs during the compression phase would lead to a greater rise in intrathoracic pressure and cerebral perfusion pressure.4 This would augment the thoracic pump mechanism.

A further potential advantage of the device is the ability to continuously monitor the force of compression.

Haemodynamic and ventilatory effects

ANIMAL STUDIES

Tucker et al compared active compression-decompression (ACD) with high impulse manual cardiac massage in dogs soon after the onset of ventricular fibrillation.5 ACD was associated with increases in mean aortic pressure, both during compression and decompression, and in coronary perfusion pressure. Pulmonary blood flow was increased and stroke volume was larger because of smaller left ventricular “systolic” volume. Sham ACD (without active decompression) also caused more efficient left ventricular emptying but no change in coronary perfusion pressure. Other animal studies, using radioactive microspheres, showed a significant increase in cerebral blood flow6 and in myocardial blood flow7 during ACD resuscitation. End tidal carbon dioxide concentration which at times of reduced cardiac output depends upon pulmonary blood flow and correlates with coronary perfusion pressure and successful resuscitation,8 was greater in pigs undergoing ACD.9

These findings have not been confirmed by all researchers. Halperin et al reported no difference in peak and mean aortic pressure or in coronary perfusion pressure in dogs,9 and Wik et al showed that ACD increased carotid blood flow but not the calculated coronary perfusion pressure in pigs that had ventricular fibrillation for 3 minutes.10 No increase in end tidal carbon dioxide concentration was seen in intubated dogs, despite an increase in total carbon dioxide exhaled and minute volume.11

HUMAN STUDIES

In the first report of the use of the ACD device in humans (with a mean interval from arrest to entry into the trial of 50 minutes) there was a significant increase in peak femoral artery pressure—from 52.5 mm Hg with standard cardiac massage to 88.9 mm Hg with ACD. The “diastolic” arterial pressure was unchanged. Transthoracic echocardiography showed increased left atrial size, prolonged left ventricular filling time, and wider opening of the mitral valve during active decompression.12 Spontaneous contrast, an indicator of stasis of blood,13 was seen in the cardiac chambers, and cleared during ACD. This latter finding was confirmed by...
another group in seven patients studied a mean 49 minutes after cardiac arrest. Apparent underfilling of the left ventricle was reversed in two of three patients when ACD was used but the degree of left ventricular compression did not differ from that achieved by standard resuscitation. In humans the increased stroke volume associated with ACD is secondary to increased left ventricular decompression volume rather than reduced compression volume.15

Shultz et al compared short bursts of ACD and standard cardiac massage in patients soon after the onset (20–25 s) of ventricular fibrillation induced during implantation of automatic defibrillators. Computer analysis of right atrial and radial artery pressures allowed coronary perfusion pressure to be estimated. Mean coronary perfusion pressure during decompression was significantly higher with ACD (21.7 mm Hg v 18.2 mm Hg). Moreover peak arterial pressure was also higher (60.1 mm Hg v 54.3 mm Hg).16 Again, this improvement in haemodynamic variables has not been seen in every study. Malzer et al reported no significant increase in coronary perfusion pressure when ACD was used in 10 victims of out of hospital cardiac arrest whose heart were brought to hospital.17

In intubated humans, the use of ACD caused a greater end tidal carbon dioxide concentration12 18 and an increase in minute volume (from 1.4 to 7.2 l/min, after subtracting the dead space).19 It is uncertain whether such improvements would also be seen in patients whose airways were not open and protected by endotracheal tubes.

Clinical trials CARDIAC ARREST IN HOSPITAL In the first published controlled trial, 62 adults in whom the development of cardiac arrest was witnessed in hospital were randomised to receive ACD (29 patients) or standard cardiopulmonary resuscitation (33 patients).19 60% of these patients had terminal conditions before the arrest and only 21 were found to be in ventricular fibrillation or tachycardia. Cardiac activity, sufficient to generate a blood pressure of 90 mm Hg for at least an hour, was subsequently achieved in 18 cases in the ACD group, compared with 10 controls (P < 0.03), and 13 patients survived at least 24 hours, compared with three controls (P < 0.04). Two patients in the study group left hospital alive and all the control patients died.

Tucker et al randomised adults requiring intubation because of severe hypotension or pulselessness developing in hospital.20 Advanced life support measures, including drug administration, and decisions regarding cessation of resuscitation were made at the discretion of the senior medical resident who coordinated the nine-person arrest team. Though this senior resident was not one of the investigators there could be no blinding to the intervention, and a member of the investigation team attended to ensure proper technique. Although there were 308 arrests (60/1000 hospital admissions) during the study period, the trial reported on 25 patients in the study group and 28 patients in the control group. Only 28% were in ventricular fibrillation. More patients in the study group survived at least 24 hours—12 (48%) v 6 (21%), P = 0.041—but there was no statistically significant difference in the chances of being discharged alive—6 (24%) v 3 (11%), P = 0.198. The mean duration of the resuscitation attempt in these successful cases was 15 minutes in the study group and 2–3 minutes in the control group. The implication is that ACD resuscitation allows adequate cerebral perfusion during prolonged periods of cardiac arrest.

CARDIAC ARREST OUT OF HOSPITAL Results of trials in four cities have appeared in abstract form21–23 and in only one of these was there a suggestion of improved outcome.

In a preliminary report of the use of the ACD device by doctors on board emergency ambulances, there seemed to be no significant difference in initial success (35.5% v 52.8%) or discharge from hospital (17.3% v 16.7%) when historical controls were used.24 Four of the 19 survivors during the study period had severe neurological impairment and the authors concluded that the device did not improve outcome.

In a study of American first-responder emergency personnel 53 patients received ACD resuscitation and 77 standard cardiopulmonary resuscitation.25 The mean call-response time was 3.5 minutes, 48 patients received bystander resuscitation, and 80 were in ventricular fibrillation. The mean time to first defibrillatory shock was significantly longer in the study group (9-0 minutes v 6.7 minutes P < 0.03). Twenty one (40%) patients in the study group and 20 (26%) controls were admitted to the intensive care unit, and there was no significant difference in the proportion of patients discharged home without neurological impairment (9 (17%) study group v 12 (16%) control group). Analysis of data from the subgroup of witnessed arrests with a delay to treatment of less than 10 minutes showed a significant increase in likelihood of return of a palpable pulse (22 (69%) v 20 (41%), P = 0.01) and admission to intensive care (19 (59%) v 16 (33%) P < 0.05) but not in the chances of discharge alive (9 (28%) v 9 (18%) P = 0.03) when ACD resuscitation was used.

However, in the most recent report of out-of-hospital cardiac arrest managed by emergency medical services in two Californian cities, 414 patients were randomised to receive ACD and 446 standard basic life support.26 There were no significant differences in the return of spontaneous circulation, hospital admission, or survival to discharge—either overall or in the subgroup described above.

Practicalities and future developments

Doctors, nurses and paramedics can be taught the use of the device efficiently and therefore members of the public should also easily acquire the necessary skills. However, the effort required to perform ACD resuscitation is not inconsiderable. In one recent study a significant proportion of staff in an accident and emergency department were unable to complete one minute of ACD resuscitation.18

Initial publications reported no problems in the use of the device in terms of adhesion to the chest and generation of adequate suction to allow active decompression. None the less there are some instances where obtaining sufficient suction has proved difficult—3 of 33 attempts in one study19 and 1 of 21 patients in another.24 This difficulty has arisen either because an abnormal chest configuration or because defibrillator gel or electrodes on the chest wall interfered with the pump. Survivors of ACD resuscitation have small round ecchymoses where the suction cup has been, but are no more likely to have skeletal injury (for example, rib fractures) than those receiving standard cardiac massage.15 16 21

In May 1993 the US Food and Drug Administration stopped all research into active compression-decompression resuscitation in the United States because there was concern about the inability of researchers to obtain consent from patients.24 25 Those studies that had been
completed have not conclusively shown that use of the ACD device is more likely to lead to successful discharge from hospital, though the numbers of patients included were small and consequently the power to detect a significant difference was weak. Larger randomised trials of both in hospital and out of hospital resuscitation are in progress in Canada, Britain, Germany, and Denmark.

Conclusion
Knowledge of the physiology of cardiopulmonary resuscitation suggests that the active compression-decompression device should have advantages over standard cardiac massage. Most animal studies have confirmed this and demonstrated improvements in haemodynamic variables. Those few randomised controlled trials that have been published have not had the power to detect clinically important benefits. Further research in the United States has been halted and it may be two or three years before other large randomised trials are completed. While there is every reason to expect that ACD resuscitation will compare favourably with standard cardiopulmonary resuscitation there is, as yet, insufficient evidence to advocate the widespread use of these devices.

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Information regarding trials of the active compression-decompression device that are currently in progress was supplied by Ambu International.