ABC of Resuscitation

A J CAMM

ASYSTOLE AND ELECTROMECHANICAL DISSOCIATION**Ventricular fibrillation****Asystole****Electromechanical dissociation**

There are three main mechanisms of cardiac arrest: ventricular fibrillation, ventricular asystole, and electromechanical dissociation. About 25% of arrests in hospital and 10% of those outside hospital are asystolic. Less than 3% of sudden cardiac arrests that occur outside hospital are due to electromechanical dissociation, but a very much higher percentage of arrests occurring in hospital are caused by this mechanism. Both asystole and electromechanical dissociation have a much worse prognosis than ventricular fibrillation.

Asystole

ECG monitor — check switches, connections and gain

(Consider intracardiac adrenaline, or pacing if there is any evidence of electrical activity)

Asystole is a form of cardiac arrest characterised by ventricular standstill due to the suppression of natural or artificial pacemakers. Myocardial disease, electrolyte aberration, anoxia, and drugs active on the myocardium may suppress idioventricular rhythms responsible for maintaining cardiac output when either higher or artificial pacemakers fail or when atrioventricular conduction is interrupted. Strong cholinergic activity may suddenly depress the function of the sinus or atrioventricular nodes, especially when sympathetic stimulation is reduced—for example, by ischaemia, infarction, or β blockade.

Asystole is diagnosed when there is no ventricular activity on the electrocardiogram, but a mistaken diagnosis may be made if the electrical recording system is faulty or wrongly connected. The leads, connections, gain, and brilliance of the monitor must be checked. Ventricular fibrillation may be mistaken for asystole, especially if only one lead is monitored or if the fibrillatory activity is of low voltage. Asystole may also give way to ventricular fibrillation caused by anoxia, acidaemia, and hyperkalaemia provoked by the asystole.

Management of asystole

Theoretically atropine should relieve cholinergic depression of function of the sinus and atrioventricular nodes, and there is ample animal and clinical evidence of this during the autonomic chaos after myocardial infarction. This does not necessarily translate into improved survival from asystolic arrest, and in animals atropine does not seem to improve the outcome from cardiac arrest. However, a recent retrospective analysis of 43 asystolic patients given atropine showed successful resuscitation in 14% (compared with none of 41 controls).

The administration of vasopressor drugs such as adrenaline is crucial to adequate cerebral and coronary perfusion during cardiopulmonary resuscitation. α Adrenergic stimulation produces intense vasoconstriction in other vascular beds and prevents coronary and cerebral arterial collapse during chest compression. In addition, the β adrenergic effect of adrenaline increases the rate of discharge of both nodes and the working idioventricular myocardium. The unopposed β adrenergic effect of isoprenaline may confer a further advantage over adrenaline.

Cardiac pacing is undoubtedly effective when applied to the immediate resuscitation of patients with asystole due to sudden and predominant failure of sinus node discharge or to atrioventricular block. However, pacing is not as successful when asystolic arrest occurs because of extensive myocardial or systemic derangement. Generally dismal results have been reported; for example, in 50 asystolic prehospital cardiac arrests managed with transcutaneous pacing ventricular capture was achieved in 26 and a pulse obtained in only five. The traumatic hazards of transcutaneous pacing (through a needle inserted directly into the myocardium) may be avoided by transthoracic pacing (from electrodes applied to the skin of the thorax). However, although transthoracic pacing has proved successful, though often uncomfortable, for elective pacing, it is similarly disappointing for emergency pacing of the victim of asystolic cardiac arrest. Pacing the ventricles via the oesophagus is awkward and almost always unsuccessful. Retrospective studies and recently a prospective study have shown that calcium is not useful for the management of asystolic cardiac arrest.

Electromechanical dissociation

Consider drugs, cardiac tamponade, tension pneumothorax, and other physical causes

Electromechanical dissociation is profound myocardial pump failure despite normal or near normal electrical excitation. It is rare outside hospital and rather more frequent in hospital inpatients. It usually occurs secondary to drugs or mechanical embarrassment such as cardiac tamponade, pulmonary embolism, tension pneumothorax, intracardiac thrombus or tumour, or myocardial rupture or exsanguination. Primary electromechanical dissociation is a failure of excitation-contraction coupling seen most often in the setting of acute inferior wall myocardial infarction. Its mechanism is not understood, but intracellular acidosis and autonomic effects are suspected. It may occur as the endpoint of prolonged ventricular fibrillation and may be produced experimentally by anoxia, acidosis, or perfusion with a calcium free medium.

Management—Once again pressor agents such as adrenaline or methoxamine (both α_1 agonists) are an essential adjunct to cardiopulmonary resuscitation. Theoretically the inotropic effect of β stimulation (adrenaline or isoprenaline) should be advantageous but this has not been confirmed. In electromechanical dissociation calcium may be effective, especially when the QRS complex is wide (0.12 s or more). For example, in a recently reported prospective randomised study eight of 39 such patients who received 5 ml of 10% calcium chloride were resuscitated compared with only one of 31 who received saline. There is some experimental evidence that calcium antagonists may preserve cerebral blood flow and function during cardiac arrest and may be particularly useful in resuscitating patients who have suffered an arrest because of electromechanical dissociation, but there is practically no clinical experience of this.

Conclusions

Resuscitation from cardiac asystole or from electromechanical dissociation is considerably less satisfactory than resuscitation from ventricular fibrillation. Nevertheless, considerable recent progress has challenged old dogma and introduced new and potentially rewarding treatments.

Professor A J Camm, MD, FRCP, is Sir Ronald Bodley Scott professor of cardiovascular medicine, St Bartholomew's Hospital, London (supported by the British Heart Foundation) and a member of the Resuscitation Council (UK).