ABC of Resuscitation

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VENTRICULAR FIBRILLATION

Ventricular fibrillation

Asystole

Electromechanical dissociation

Sinus rhythm

Coarse ventricular fibrillation

Onset of asystole

Ventricular fibrillation is electrical anarchy within the ventricular myocardium. The normal heart beat is generated by a sequence of depolarisation, mechanical contraction, and active relaxation of the heart muscle that spreads rapidly throughout the ventricular myocardium in an orderly fashion—exquisitely designed to propel blood rather than simply to expel it.

In ventricular fibrillation coordination of the beat breaks down totally, so that electrical depolarisation and contraction of fibres become apparently random. The muscle continues to work after fibrillation supervenes but contraction in one group of fibres is counteracted by relaxation in neighbouring fibres. Inspection of the ventricles would show them to be quivering rather than beating. This movement gradually diminishes owing to progressive ischaemia since no blood flows through the coronary arteries during cardiac arrest. Ventricular fibrillation therefore passes imperceptibly into irreversible and untreatable asystole (asystole occurring as a primary arrhythmia, as in the illustration, or as an abrupt result of therapy does sometimes respond to treatment). The changes are reflected in the electrocardiogram: the distinctive QRS/T complexes of the coordinated heart beat are replaced by a continuous random wave form, which usually starts as a coarse pattern and becomes progressively finer as the quivering of the myocardium ceases.

Ventricular fibrillation is the most common mechanism of cardiac arrest in patients with ischaemic heart disease (the others are asystole and electromechanical dissociation, considered in the next article). Ventricular fibrillation occurs as a complication of myocardial infarction, within the first few hours in uncomplicated cases but less predictably and often later in patients with heart failure or cardiogenic shock. It may also occur unheralded, in the absence of pain or other symptoms, and thus cause sudden cardiac death. The old assumption that a new myocardial infarction is an invariable prelude to sudden cardiac death is incorrect, but most victims do have severe underlying obstructive coronary disease with some recent instability (such as rupture of an atheromatous plaque) that caused localised ischaemia as a trigger for the arrhythmia. Whatever the precipitating cause, consciousness is lost within 10 to 20 seconds of the onset of ventricular fibrillation.

Appropriate first aid (basic life support) or defibrillation must be provided within two to four minutes if cerebral damage is not to be irreversible. It also becomes more difficult to restore the heart beat if resuscitation is delayed.

Electrical defibrillation

Ventricular fibrillation may rarely remit spontaneously or as a result of a precordial thump; but these possibilities exist only very briefly after the onset of the arrhythmia, presumably because the loss of coronary flow exacerbates the electrical instability. No conclusive evidence exists that any drug can abolish ventricular fibrillation in man even if the circulation is maintained by basic life support. The only effective treatment for established ventricular fibrillation is electrical defibrillation, and the quicker this can be provided the better the prospects for long term success.

Defibrillation is a simple treatment. A large electrical impulse ("shock") is delivered to the heart through the chest wall. This causes depolarisation of all the myocardial cells not totally refractory. If enough cells are depolarised then the incessant fibrillatory waveforms of random depolarisation are no longer propagated throughout the ventricular myocardium, and after a brief interval the normal impulses transmitted through the conducting pathways of the heart take over control of the heart beat. Effective mechanical activity usually begins at once but may be delayed for a few moments after restoration of a satisfactory electrocardiogram.

Defibrillators are usually powered by a rechargeable battery but some are mains operated. Few controls are necessary. Apart from an on-off switch, the equipment allows for variation in the energy stored in the capacitor for discharge through the chest wall. Calibration points for delivered energy, including 80 J, 160 J, and 320 J, are usual (older models show calibration in stored energy, 100 J stored being equivalent to 80 J delivered). The shock is delivered by well protected hand held electrodes, or paddles. They have an integral discharge button so that the operator can control the timing of the delivery of the shock.

Means must be available to dump any unused charge. Most portable defibrillators have a monitoring facility with an oscilloscope to show heart rhythm; the defibrillation paddles act also as electrodes for transmitting the electrocardiogram.

For defibrillation one electrode should be placed below the right clavicle and the other over the points designated as V4 and V5 for the electrocardiogram—that is, a little outside the position of the normal apex beat. The polarity of the plates is unimportant so their position is interchangeable. Impedance at the interface of plate and skin must be kept to a minimum. Electrode jelly can be used for this purpose, but it tends to spread during chest compression with the risk that subsequent shocks will arc across the chest surface. Conducting gel pads should be used instead and removed between shocks. The pressure of plate against pad and skin also determines impedance and should therefore be firm. The operator must ensure that neither he nor anyone else is touching the patient when the shock is delivered.

Automatic and semiautomatic defibrillators are designed for use by first aid personnel rather than by fully trained medical or paramedical staff. Adhesive electrodes are attached to the chest wall. The electrocardiogram is interpreted by algorithms within the machine, and—depending on the result—a "decision" is made whether or not to deliver a shock. In the semiautomatic models the final decision rests with the operator, but full guidance is provided on a small visual display panel. The energy levels are preset, but otherwise the principles are the same as for manual defibrillation.

Guidelines for defibrillation procedures have been laid down by the Resuscitation Council of the United Kingdom. The sequence of energies and the details of drug administration given here are based on these recommendations.

Procedure

Summary

- (1) The hopeful phase: three shocks at 160 J, 160 J, 320 J (200 J, 200 J, 400 J).
- (2) The struggling phase: three shocks at 320 J (400 J) after, respectively, lignocaine, adrenaline, bicarbonate. Patient intubated if possible.
- (3) The desperate phase: three shocks at 320 J (400 J) with different electrode position or different defibrillator.

While improvements will doubtless be made as skill in resuscitation increases, the guidelines can ensure that relatively few treated patients die in ventricular fibrillation and only those with irreversible myocardial damage are likely to be converted to intractable asystole.

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Unless the onset of ventricular fibrillation is witnessed and a defibrillator is immediately available, the victim should receive basic life support before definitive treatment is given. External chest compression and simple mouth to mouth ventilation should not be interrupted for intubation or placement of intravenous lines if a shock can be delivered within a minute or two. If possible, one rescuer should continue chest compression while a second charges the defibrillator to 160 J (200 J stored) so that interruption of basic life support is minimised before delivery of this and subsequent shocks.

An oscilloscope may take five to 10 seconds to "recover" after delivery of the shock so no interpretation of the electrocardiogram is possible during this period; the procedure after defibrillation should ensure that no time is wasted without circulatory support while the rescuer gazes at an inoperative screen. The following routine is suggested:

- (1) give shock,
- (2) check major pulse within three seconds,
- (3) if no pulse administer 15 more chest compressions,
- (4) only then place paddles to read electrocardiogram.

If coordinated rhythm has returned chest compression is unlikely to precipitate recurrence of fibrillation and it has no serious effect on the beating heart. If fibrillation is still present further shocks are given at 160 J (200 J) and 320 J (400 J) using the procedures outlined above. Basic life support is continued at all times except when the shocks are delivered.

Failure of these initial attempts at rapid defibrillation may be due to hypoxia or acidosis: the ideal treatment for both is hyperventilation after intubation, though outside hospital this will not always be practicable. (Ventilation is more effective than bicarbonate for reversing acidosis at this stage: without efficient elimination of carbon dioxide, bicarbonate may even worsen intracellular acidosis.)

Constant electrocardiographic monitoring will be needed in any prolonged cardiac arrest, and adhesive electrodes should be used for the subsequent phase of the resuscitation attempt.

Drug administration will also be necessary and a venous cannula should therefore be placed as centrally as possible. The sequence recommended is as follows: lignocaine 100 mg intravenously before the fourth shock, adrenaline 10 ml 1 in 10 000 intravenously before the fifth, and sodium bicarbonate 50 ml 8.4% intravenously before the sixth. The endotracheal route may be used for lignocaine and adrenaline (but not for bicarbonate). Transbronchial absorption—at least of adrenaline—is rapid unless pulmonary oedema is present; an effect on the heart may occur relatively quickly because the pulmonary circulation is bypassed. Doses recommended are twice those used for intravenous administration. After each drug has been given chest compression should be continued for a minute or so before the 320 J (400 J) shock is delivered and the usual checks made.

Few patients will remain in fibrillation past this point, but final manoeuvres may include the use of shocks with a different paddle orientation, preferably front to back on the left chest, and—if the equipment is available—a change of defibrillator.

A brief period of asystole is common after defibrillation. This is treated initially only by continued basic life support. Drug therapy for asystole should not be started for a minute or so lest fresh fibrillation is induced unnecessarily. If fibrillation does recur the sequence outlined above is started again from the beginning.