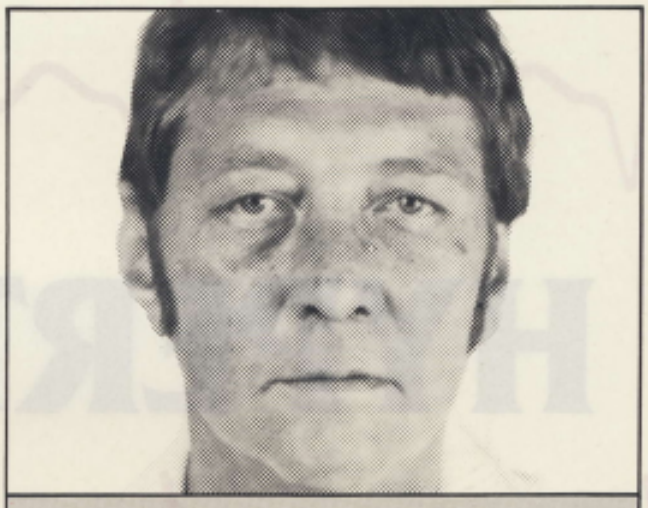
Cardiac Arrest
Diagnosis and management

CJ van der Merwe

Summary

The recognition of cardiac arrest and determination of the mechanism and etiology involved are of paramount importance for resuscitators to instigate the correct management programme immediately. Detailed step by step protocols for managing ventricular fibrillation and asystole are described, including the initial treatment for cardiac arrest in the presence of hypovolaemia and relative hypovolaemia.

KEYWORDS: Death, Sudden; Resuscitation; Ventricular Fibrillation; Heart Arrest; Hypovolemic Shock; Blood Volume; Asystole; Arrythmia; Respiration, Artifical; Infusions, Parenteral; Intubation; Vasodilation; Hypokalemia.



Curriculum Vitae

Prof Coen van der Merwe graduated from UP with the degree MBChB in 1955. After completing his internship and a period as Senior Medical Officer he left for the United Kingdom in 1957. After a period of six months as Senior Medical Officer in Surgery, he was appointed Registrar in Surgery (Birmingham Regional Hospital Council). Upon his return to South Africa he worked for two years as Registrar in Surgery at the King Edward VIII Hospital, Durban, and the Natal University after which he was appointed Senior Surgical Registrar in the Department of Plastic and Reconstructive Surgery at the Wentworth Hospital, Durban. After a second visit to the United Kingdom where he was appointed Senior Registrar in Surgery, he returned to the RSA and practised as a general practitioner in Pretoria. During 1970 he joined the staff of the HF Verwoerd Hospital, and in 1973 was appointed Senior Medical Officer. In 1974 he became Principal Medical Officer and head of the Emergency Unit, of the HF Verwoerd Hospital where he is still employed. In 1977 he obtained the degree of M Prax Med and was appointed Senior Lecturer in the Department of Family Medicine of the University of Pretoria. Various publications followed and in 1980 he was promoted to Associate Professor in charge of the Emergency Unit and Polyclinic of the HF Verwoerd Hospital and the Department of Family Medicine of the University of Pretoria. In addition to academic duties and service to patients in the Emergency Unit, he is currently involved in the training of ambulance personnel. He is a member of the Medical Subcommittee of Civil Defence, Transvaal. He is also responsible for the clinical service at the Laudium Hospital, south of Pretoria.

CJ van der Merwe MB ChB M Prax Med
Dept of Family Medicine
Faculty of Medicine
University of Pretoria
HF Verwoerd Hospital
Republic of South Africa

Abbreviations

CPR: cardiopulmonary resuscitation
VF: ventricular fibrillation
JDC: joules direct current

Irrespective of etiology, the approach to cardiac arrest always involves simultaneous evaluation and management. CPR should be introduced immediately after cardiac arrest has been clinically diagnosed and this is followed by the determination of the exact mechanism and etiological factors involved and prompt dealing with such factors. It is important to determine whether adequate cerebral circulation was maintained within the critical period of four minutes, or whether a longer delay occurred which would mean that there is no justification for a continuation of the resuscitation attempt.

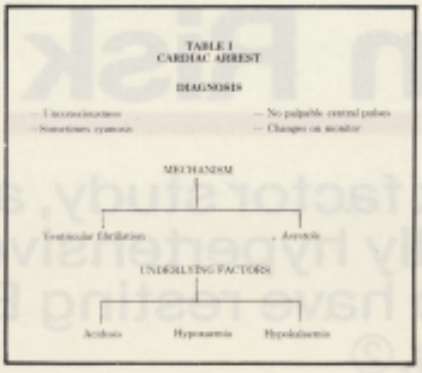
The decision whether to resuscitate or not, and whether to end a resuscitation attempt, is truly a matter of life and death. One can thus understand that resuscitators are unwilling to accept the irreversibility of a situation. Knowledge of the exact time-span is also usually unreliable and for this reason it is good policy to attempt a complete resuscitation before a final decision is made to terminate resuscitation or not.

EVALUATION

1. Recognition of cardiac arrest (Table I)

Cardiac arrest is recognised on the basis of:

- sudden loss of consciousness;
- absence of central pulses (femoral or carotid arteries);
- sometimes cyanosis;
- confirmation with a cardiac monitor.



2. Determination of the mechanism of cardiac arrest

The mechanism of cardiac arrest is determined by means of a cardiac monitor or electro-cardiogram which distinguishes between:

- ventricular fibrillation (fine or coarse types), and
- asystole.

This distinction is essential because it will influence further action radically.

3. Determination and treatment of the etiological condition responsible for the cardiac arrest (Table II)

Evaluation may not interfere with the active resuscitation attempt, however. The most frequent causes are classified as precardiac, cardiac and peripheral causes. Initial action is also influenced by the status of the blood volume, viz normovolaemia, hypovolaemia or relative hypovolaemia.

MANAGEMENT OF A NORMOVOAEMIC PATIENT WITH CARDIAC ARREST:

CPR must be introduced immediately and it is essential to obtain help. A resuscitation attempt requires at least two, and preferably three, informed persons. The following is done:

1. Deliver precordial blow and initiate cardiac massage
2. Ventilation: Oxygen administration with mask and Ambubag brings about adequate ventilation. Intubation should only be attempted after satisfactory mask ventilation has been achieved to prevent a delay in ventilation while the patient is being intubated.
3. Connect the patient to a monitor. (Infusion placing is a second priority — as is intubation.) Now distinguish the mechanism of the cardiac arrest by means of the monitor:

(a) *Ventricular fibrillation* will display fine or coarse bizarre complexes (Fig. 1a and 1b).

(b) *Asystole* will display a straight line.

One should always start by defibrillating any form of cardiac arrest. The reason for this step is that fine ventricular fibrillation may sometimes appear as a straight line and therefore

TABLE II
COMMON CAUSES OF CARDIAC ARREST

PRECARDIAC CAUSES	CARDIAC CAUSES	PERIPHERAL CAUSES
(Volume and respiratory disturbances)	(Direct effect)	(Widespread vasodilatation)
Hypovolaemia	Ischaemic heart disease	Extreme loss of blood
Hypoxia	Structural cardiac abnormalities	(3rd phase shock)
— upper airway obstruction	Electromechanical dissociation	Anaphylaxis
— inhalation of vomitus	Pericardial tamponade	Drug overdose
— drug overdose	Electric shock, hypothermia and vagus inhibition	Severe hypothermia
— deep anaesthesia	Excessive catecholamine — fright — iatrogenic	Septicaemia
— drowning	Septicaemia	Deep general anaesthesia
— serious lung disease	Hypothermia	High spinal block
— inhalation of toxic gas		High epidural block
Hypercapnia		
Electrolyte disturbances (especially potassium)		
Acid-base disturbances		
Tension pneumothorax		
Pulmonary thrombo-embolism		
Fat embolism		
Hypothermia		

Note that many factors (e.g. drug overdose) overlap etiological categories. A further important observation is that the etiology must also be classified under (1) normovolaemic eg ischaemic heart disease (2) hypovolaemic eg loss of blood (3) relatively hypovolaemic eg conditions that result in vasodilation. The priority of intravenous fluid administration at resuscitation is determined by the etiology.

assume the appearance of asystole.

VENTRICULAR FIBRILLATION (VF)

In the event of ventricular fibrillation (Monitor: bizarre complexes) the following steps are indicated, Table III:

● Defibrillation — starting with 150JDC. Partridge[1] suggests an initial delivery of 200 JDC. The electrodes must be placed correctly (Figure 2). A high dose causes unnecessary structural trauma[2]. However, the dose can subsequently be increased as required.

● Continued cardiac massage and ventilation are essential.

● Administer lignocaine 100 mg IV as a bolus (1 mg/Kg)[3].

● Defibrillate with 200 JDC if sinus rhythm has not been obtained after the first 150JDC and if necessary repeat with 300-400 JDC. CPR must be continued during the periods between DC shocks.

● Lignocaine infusion must be introduced at 4 mg/min[3]. Preparation of the solution: 500 mg Lignocaine in 200 ml 5% dextrose solution (or 0,9% sodium chloride solution). Administration at 80 microdrops per minute supplies about 4 mg per minute (or 2 ml per minute). When an ordinary administration set is used, where 15 drops are equivalent to 1 ml, the administration is adapted to 30 drops per minute. If hyperirritability of the myocardium still occurs (ventricular tachycardia or extrasystoles) the lignocaine dosage is supplemented by intermittent bolus administrations of 50 mgm IV until the arrhythmia is under control or until a maximum of 200-300 mg has been administered.

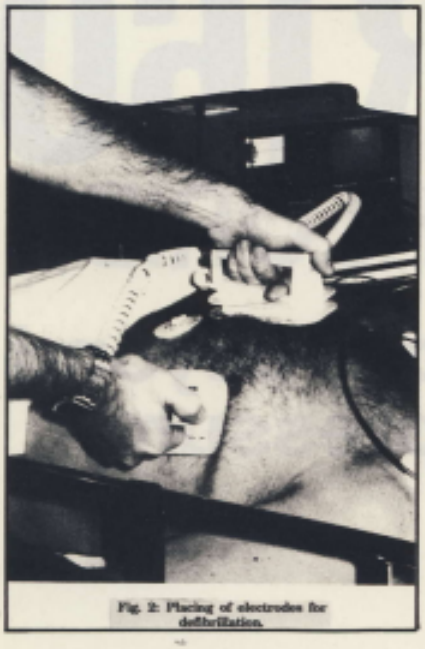


Fig. 2: Placing of electrodes for defibrillation.

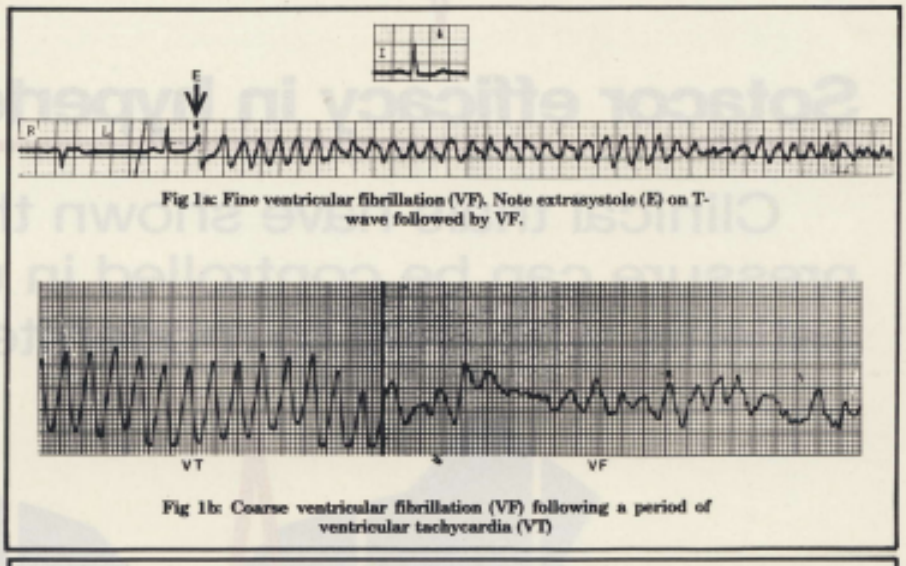


Fig 1a: Fine ventricular fibrillation (VF). Note extrasystole (E) on T-wave followed by VF.

Fig 1b: Coarse ventricular fibrillation (VF) following a period of ventricular tachycardia (VT)

TABLE III
VENTRICULAR FIBRILLATION

- Cardiac massage
- Defibrillate 100-150J
- Lignocaine 100mg IV

Sinus rhythm
- Lignocaine infusion 4mg /min.

With hypotension > 90mmHg systolic
- Dobutamine infusion 1-10µg/kg/min IV
- Blood gas/electrolyte determination. $NaHCO_3$ administration
- 50% magnesium sulphate 2ml IV

With hypotension < 90mmHg systolic (Cardiogenic shock)
- Dopamine infusion 5-10µg/kg/min IV
- and/or Isoproterenol infusion 0,05-0,1µg/kg/min IV
- $NaHCO_3$
  - Empirical 100mmol IV
  - According to blood gas analysis
- Aorta balloon pump

Continued fibrillation
- Continued massage
- $NaHCO_3$ 100mmol IV
- Defibrillate 200J

Sinus rhythm / **Resistant fibrillation**

Coarse fibrillation
- Na HCO_3 100mmol
- Potassium chloride 15% → 5-10ml IV
- Defibrillate 300J if necessary then 400J

Fine fibrillation
- Adrenalin 0,5mg IV

Coarse Fibrillation

Sinus rhythm / **Still resistant**
- Bretilium tosilate 5mg/kg IV
  - after 20mins. repeat 10mg/kg
- Defibrillate 400J

Sinus rhythm / **Still resistant**

J = Joules direct current

● If sinus rhythm has been obtained, but hypotension still exists, dobutamine HCl (Dobutrex)[4] is indicated (1-10 microgram/kg/min). Preparation: 500 mgm Dobutamine in 200 mℓ 0,9 saline solution (not in alkaline solution). Titration at 5 to 30 microdrops per minute is recommended.

● A determination of the blood gas status and electrolyte status is necessary after sinus rhythm has been obtained to determine the exact dose of sodium bicarbonate and potassium needed. The dosage of sodium bicarbonate is determined by the following formula: Base deficiency x Mass in kg x 0,3 = mmoℓ $NaHCO_3$[5]. Half of the dose is administered and a

IF ONLY I COULD GET A GOOD NIGHT'S SLEEP

IF ONLY I COULD GET A GOOD NIGHT'S SLEEP

blood gas analysis is repeated to determine further requirements. Bicarbonate has an alkalising and an osmotic effect. The latter displaces fluid from the interstitial compartment to the vascular compartment. Overloading of the circulation by increased osmotic activity is dangerous to the normovolaemic patient and the maximum dose for an adult should not exceed 200 mmoℓ. Sodium bicarbonate does not penetrate rapidly intracellularly and can cause cardiac arrest as a result of fluid overloading due to osmotic activity. If low potassium values have been obtained or in cases of refractory ventricular fibrillation 5-10 mℓ of a 15% potassium chloride solution (10-20 mmoℓ) must be administered by means of a central catheter or in an infusion solution (severe irritant). Hypokalaemia can be the reason for failed resuscitation.

The approach to cardiac arrest always involves simultaneous evaluation and management.

- Administer 2 mℓ of 50% magnesium sulphate solution IV as this reduces ventricular irritability.

- A resistant ventricular fibrillation may be the result of:
  — acidosis
  — hypokalaemia
  — hypoxaemia

  These conditions must first be rectified by administering 100 mmoℓ sodium bicarbonate and 5 mℓ of a 15% potassium chloride solution IV empirically with constant ventilation.

- A resistant fine ventricular fibrillation must be 'made coarse'[6] by the administration of adrenalin 0,5 mg, as defibrillation is more effective when the ventricular fibrillation is coarse[6] by the administration of adrenalin 0,5 mg as defibrillation is more effective when the ventricular fibrillation is coarse.

- If refractory ventricular fibrillation continues and does not react to the abovementioned DC shocks the administration of bretilium tosilate[7,8] should be considered at a dosage of 5 mg/kg which may be repeated if necessary after 15-20 minutes with a dose of 10 mg/kg. The agent increases the ventricular fibrillation threshold value.

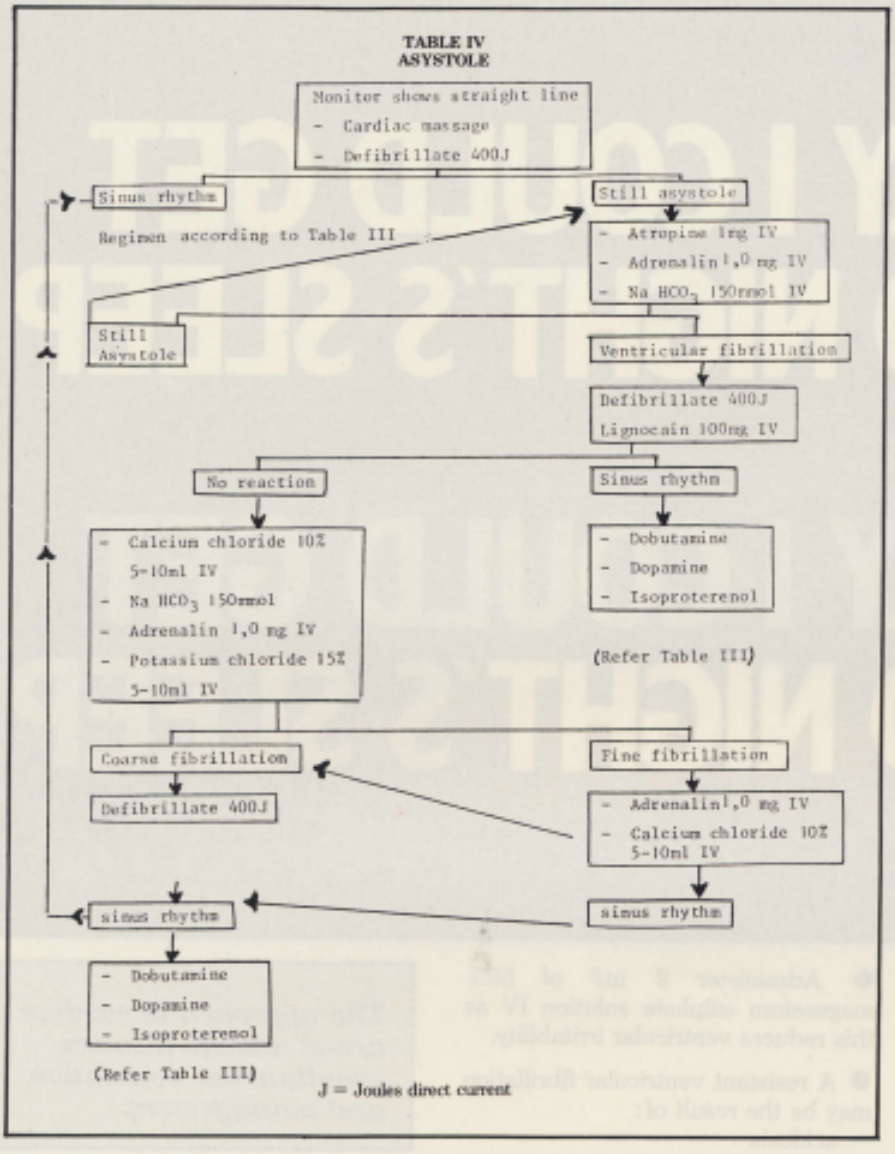
The approach to cardiac arrest always involves simultaneous evaluation and management.

ASYSTOLE

In the event of asystole (monitor = straight line) the following steps are indicated, (Table IV):

- Cardiac massage, ventilation and defibrillation with 400 JDC must be introduced immediately. Defibrillation is essential as the fibrillation is sometimes very fine and appears as a straight line on the monitor. In these cases defibrillation may be successful. If a defibrillator is available, immediate CPR and defibrillation are priorities while the following measures should be taken thereafter:

- Atropine 1 mg IV is administered first to stop the vagus effect[9]. The anticholinergic effect is dose-dependent and the agent must be administered rapidly. The endotrachael route may be effective.



● Adrenalin 0,5-1 mg IV is administed if no complexes are visible. The agent is a direct myocardial stimulant[10]. If there is no intravenous line 1 mg of adrenalin administered endotracheally may be just as effective[11].

● Sodium bicarbonate 100 mmoℓ IV is administered empirically.

● If ventricular fibrillation occurs, immediate defibrillation must be done with 400JDC shock.

● As soon as a central pulse is palpable the blood gas and electrolyte analysis must be determined and any abnormality must be rectified immediately as was described previously.

● If there is still no reaction to the treatment a solution of 5-10 mℓ of a 10% calcium chloride must be administered intravenously[12]. Administration of this agent is an attempt to transform asystole into a coarse ventricular fibrillation which can be defibrillated effectively.

● In the event of hypotension the administering of the following inotrope agents must be considered:
— Dobutamine HC1 (Dobutrex)
— Dopamine HC1 (Intropin)
— Isoproterenol HC1 (Isuprel)

1. Dobutamine administration (Dobutrex) 1-10 microgram/kg/min
2. Dobutamine HC1 is a beta-adrenergic agent with direct inotropic action and it reduces preload and afterload[13]. Administration increases cardiac output and the haemodynamic index if left ventricle malfunction had occurred. Preparation: 500 mg in 200 mℓ saline solution 0,9%. Titrate at 5-10 microdrops per minute.
3. Dopamine administration (Intropin) 5-10 microgram/kg/min). When this dose is administered dopamine HC1 has beta 1 and beta 2 — receptor stimulation with an improved cardiac output as a result of higher stroke volume and myocardial contractility[4]. If dobutamine does not have the desired effect, dopamine is added. Preparation of the solution: 20mgm dopamine in 200 mℓ normal saline solution which is titrated in average adults at 10-30 microdrops/min according to the reaction desired. It must be realized that the effects of this agent are dose-dependent. With a slower infusion 0,5-2 μg/kg/min dopaminergic effects predominate resulting in selective vasodilatation of renal and mesenteric vascular beds

Always start by defibrillating any form of cardiac arrest.

and increased renal bloodflow and urine output. At rates above 10 μg/kg/per min alpha receptor vasoconstriction becomes apparent with increases in cardiac output, arterial blood pressure and renal blood flow. Rates greater than 20 μg/per kg/per min result in decreased renal blood flow and peripheral vasoconstriction (alpha adrenergic effects). To determine the exact dosage of dopamine to be administered in microdrops, the follwing formula may be used:

$$\frac{\text{Desired } \mu g/kg \times \text{mass (kg)}}{6,7 \text{ or } (7)} = \text{microdrops/min}$$

4. Isoproterenol HC1 (Isuprel).
Side-effects of this preparation are vasodilation and increased oxygen consumption by the myocardium[14]. Adrenalin has replaced this agent to a large extent. The agent is indicated for asystole and bradycardia which is refractory to atropine[9] and also when refractory cardiogenic shock occurs, because this betaselective sympathomimetic agent has a positive inotropic and chronotropic action. Preparation of solution: 1 mg of isoproterenol hydrochloride (Isuprel) in 500 mℓ 0,9% saline solution (final

A resistant, fine ventricular fibrillation must be made 'coarse'.

IF ONLY I COULD GET A GOOD NIGHT'S SLEEP

strength 1 : 500 000). Administration rate is 0,5 to 5 μg per minute (0,25 to 2,5 mℓ per minute of the diluted solution).

5. Potassium chloride
Potassium chloride is administered if it is indicated by the laboratory examination. Normally 5-10 mℓ of a 15% solution is required. The agent is also administered if the resuscitation attempt is ineffective.

MANAGEMENT OF A HYPOVOLAEMIC PATIENT WITH CARDIAC ARREST

The pharmacological intervention for a hypovolaemic patient with cardiac arrest, does not differ from the Previous description. However, rectification of the blood volume is an absolute priority. Cardiopulmonary resuscitation with airway provision and external cardiac massage is introduced. Rapid fluid replacement must be achieved by means of two or more wide-lumen needles placed into peripheral vessels and at least one central catheter should be placed.

Sometimes as many as four and more peripheral infusions are required to ensure satisfactory volume replacement. An incision on the Vena basilica or the Vena saphena (proximal) with placing of a polythene tube of a sterile intravenous administration system is a method for administering large amounts of fluid rapidly. A sterile paediatric feeding tube may also be used as an intravenous catheter placed in the saphenous vein. Incisions can however be time-consuming. A MAST suite could eliminate the necessity of a Vena saphena incision by improving venous filling of the upper extremity to enable peripheral venous cannulation. The placement of a central venous catheter with a wide lumen is the quickest method for administering large volumes of fluid. The standard catheter used for central venous access in most hospitals is a number 16 "Intra-cath". These catheters are suitable for central venous pressure determination but because of the length and small diameter of these catheters, they have a high flow resistance and are therefore inadequate for rapid volume replacement. These catheters have a flowspeed of less than 50% of a standard No 16 needle placed in a peripheral vein! This type of central catheter should only be used for monitoring purposes. A rapid flow can be obtained by placing a wide-lumen catheter into the subclavian vein. Various types of short and wide-lumen catheters (=8,5FR) are available on the market for central placing which will ensure rapid volume replacement. The advantages are that this provides a more rapid inflow and a quicker method for percutaneous placing without an incision. A greater flow is also obtained if administration devices with short tubes are used. Fluid administration may be expedited even further by placing a pressure bag around the plastic container. A pressure bag provides a greater flow than a hand pump.

Rectification of the blood volume is an absolute priority.

It is essential to keep heated crystalloids (37°-40°C) handly for

administration and to warm blood before administration as hypothermia has serious consequences. Hypothermia causes increased oxygen consumption, increased haemoglobin-oxygen affinity, potassium leakage with arrhythmias, platelet sequestration and suppression of coagulation. As bank blood has a deficiency in coagulation factors and platelets, it is essential to administer freshly frozen plasma after every fourth unit of bank blood. Platelet concentrate should be administered after every fifth unit of bank blood to control these harmful consequences.

The standard catheter used for central venous access is inadequate for rapid volume replacement.

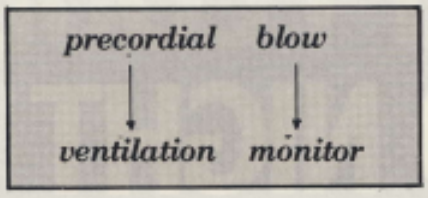
Heating of blood reduces the viscosity which also brings about a more rapid flow. The most important aspect is rapid administration of large volumes of heated Ringer lactate and blood while CPR is continued without interruption.

The use of a pneumatic pressure suit (MAST-suit) is recommended as an aid to intravenous fluid administration. The suit probably displaces 750 to 1 200 mℓ of blood from the periphery to the central blood vessels and it also increases the peripheral resistance. External pressure also

The MAST-suit probably displaces 750-1200ml of blood from the periphery.

changes wall pressure of the injured blood vessels resulting in narrowing of the gap in the injured vessel which assists haemostasis.

The MAST also controls intra-abdominal haemorrhage to a large extent and may be a substitute for a laparotomy and clamping of the aorta in the emergency unit. Clamping should preferably take place in the operating room. Laparotomy for serious abdominal haemorrhage has an extremely high mortality if it is performed in the emergency unit. The MAST-suit is however not a



substitute for massive volume replacement!

External heart massage in patients with chest injuries is often ineffective and even impossible as in the case of an unstable thorax and cardiac tamponade. In such cases emergency thoracotomy and internal cardiac massage are the only solutions and should be done in the emergency unit.

All haemorrhage must be controlled to ensure successful resuscitation. Any condition that limits the patient's ability to ventilate, must be

IV adrenaline is a priority in the event of cardiac arrest due to anaphylaxis.

terminated. To achieve this, emergency thoracotomy is often the only solution.

MANAGEMENT OF A PATIENT WITH RELATIVE HYPOVOLAEMIA AND CARDIAC ARREST (vaso-dilatation)

Anaphylaxis is an example of this type of cardiac arrest. The pharmacological and electrical intervention are the same as for normovolaemic cardiac arrest in addition to volume replacement to overcome the relative hypovolaemic effects of vasodilatation. Further action is aimed at dealing with the cardiac arrest (VF or asystole) and stabilization of the cardiovascular and respiratory status as was described. Administration of IV adrenaline is a priority in the event of cardiac arrest due to anaphylaxis.

REFERENCES

1. Pantridge JF, Webb SW, Adgey AAJ. Arrhythmias in the first hours of Acute myocardial infarction. *Prog Cardiovasc Dis* 1980-1; 23 (4): 265-78.
2. DiCola VC, Freedman GS, Downing SE, Zaret BL. Myocardial uptake of technetium — 99m stannous pyrophosphate following direct current transthoracic countershock. *Circulation* 1976; 54 (6): 980-6.
3. Wyman MG, Hammersmith L. Comprehensive treatment plan for the prevention of primary ventricular fibrillation in acute myocardial infarction. *Am J Cardiol* 1974; 33: 661-7.
4. Reid PR, Thompson WL. The clinical use of dopamine in the treatment of shock. *Johns Hopkins Med J* 1975; 137: 267-9.
5. Goldberg AH. Cardiopulmonary arrest. *N Engl J Med* 1974; 290 (7): 381-5.
6. De Silva RA, Graboys TB, Podrid PJ, Lown B. Cardioversion and defibrillation. *Am Heart J* 1980; 100 (6) pt 1: 881-93.
7. Holder DA, Sniderman AD, Fraser G, Fallen EL. Experience with bretylium tosylate by a hospital cardiac arrest team. *Circulation* 1977; 55 (3): 541-4.
8. Nowak RM, Bodnor TJ, Dronen S, Gentzkow G, Tomlanovich MC. Bretylium Tosylate as initial treatment for cardiopulmonary arrest: randomized comparison with placebo. *Ann Emerg Med* 1981; 10 (8): 404-7.
9. American Heart Association and National Academy of Science-National Research Council: Standards for cardiopulmonary resuscitation (CPR) and emergency cardiac care (ECC). *JAMA* 1980; 244: 453-509.
10. Livesay JJ, Follette DM, Fey KH et al. Optimizing myocardial supply/demand balance with α — adrenergic drugs during cardiopulmonary resuscitation. *J Thorac Cardiovasc Surg* 1978; 76 (2): 244-51.
11. Roberts JR, Greenburg MI, Baskin SI. Endotracheal epinephrine in cardio-respiratory collapse. *J Am Coll Emerg Phys* 1979; 8: 515.
12. Greenblatt DJ, Gross PL, Bolognini V. Pharmacotherapy of cardiopulmonary arrest. *Am J Hosp Pharmacol* 1976; 33: 579.
13. Sonnenblick EH, Frishman WH, Le Jemtel TH. Dobutamine: a new synthetic cardioactive sympathetic amine. *N Engl J Med* 1979; 300 (1): 17-22.
14. Tarazi RC. Sympathomimetic agents in the treatment of shock. *Ann Intern Med* 1974; 81: 364-71.