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# PRINCIPLES OF CRITICAL CARE

Farokh Erach Udwadia

4th Edition

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CHAPTER 4

# Cardiopulmonary Resuscitation and Cerebral Preservation in Adults

## GENERAL CONSIDERATIONS

A sudden unexpected cessation of effective cardiac pump function or beating of the heart is termed "cardiac arrest". This may be reversible if promptly managed, but otherwise invariably leads to death. Cardiac arrest quickly results in a respiratory arrest and resuscitation therefore involves establishing an effective stable circulation as well as maintaining effective ventilation—hence the term cardiopulmonary resuscitation (CPR). Similarly, respiratory arrest if unattended leads within a few moments to cardiac arrest, illustrating the inseparable link between the heart and the lungs. One should in the same breath also consider cerebral preservation, because cessation of effective cardiac pump function results in severe hypoxia due to stoppage or markedly inadequate blood supply to all important organs of the body, most importantly to the brain and the heart muscle itself. Hypoxic brain damage and death therefore always result if resuscitation is delayed, is not implemented, or is unsuccessful. In fact, for several reasons brain damage can result even after successful resuscitation. *The preservation of brain function (cerebral preservation) during and after CPR is therefore crucial to allow a meaningful quality of life.*

It is not the purpose of this chapter to enumerate the numerous causes of sudden cardiac arrest (SCA) or cardiac death. Ischemic heart disease in particular acute myocardial infarction remains the most common cause in Western countries as also in India. Cardiac arrest is a dreaded complication during surgery and anesthesia; it can also complicate investigational procedures, or can occur as a consequence of electrolyte and metabolic abnormalities, or may be caused by drugs—particularly antiarrhythmic drugs such as quinidine, procainamide, and digitalis. It is an important cause of death following trauma, blood loss, asphyxia (due to drowning, carbon monoxide poisoning, and inhalation of food or vomit), sudden large airways obstruction by a foreign body, and electrocution either by electric shock or lightning.

This chapter briefly mentions the diagnosis and the basic mechanisms underlying cardiac arrest. It next describes the various aspects of emergency CPR (basic life support followed as quickly as possible by advanced cardiac life support—ACLS) as currently advised. It then deals with postresuscitation care, directed specifically toward preventing or minimizing brain injury. This is achieved by the prompt use of hypothermia, if a careful neurological assessment so indicates.

## DIAGNOSIS

Diagnosis of cardiac arrest is made if a patient is unconscious, does not respond to a loud shout or call, and has impalpable carotid pulses. Not more than 10 seconds should be spent on assessing pulselessness. In the same 10 seconds in parallel with pulse check, breathing should be checked. If the patient is apneic or is gasping (agonal breathing), he or she should be considered to have cardiac arrest. Therefore, diagnosis of cardiac arrest is appropriate if a major pulse (the carotid) is not felt or if the patient is apneic or has gasping agonal breathing.

## BASIC MECHANISMS

The mechanisms underlying cessation of effective cardiac pump function which if uncorrected lead to rapid loss of consciousness and death are:

- Ventricular fibrillation (VF)
- Ventricular tachycardia (VT) resulting in pulselessness and an unconscious state
- Cardiac standstill or asystole; a markedly severe bradyrhythm has well-nigh the same effect as an asystole or standstill.
- Pulseless electrical activity (PEA)—in this condition though the ECG or cardiac monitor shows a normal or satisfactory electrical activity, the mechanical pumping action of the heart is either extremely feeble or absent.

## OBJECTIVES AND PRINCIPLES OF MANAGEMENT

- The chief objective is to restore and maintain stable, effective cardiopulmonary function resulting in effective transport of oxygen to the tissues of the body.

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- To prevent or minimize brain damage not only during CPR but also after successful restoration of the circulation.
- There is a window of approximately 4 minutes from the cessation of effective cardiac function (with absence of major pulses) after which hypoxic injury to the brain progresses rapidly, ultimately leading to irreversible death. Time is of essence and every second counts. *A diagnosis of cardiac arrest in a patient should therefore be immediately followed by uninterrupted CPR*. Resuscitation after 10–15 minutes of arrest has rarely been found to be successful but most doctors hesitate to resuscitate when >10 minutes have elapsed after an unattended cardiac arrest. Permanent brain damage and vegetative state are the sequel even if CPR is successful.
- Cardiopulmonary resuscitation in terminal malignancy or in terminal hepatic, renal, or pulmonary disease offers a cruel extension of life for a short span, and should be avoided. The ethical issues involved are fairly clear, but should be decided in consultation with the patient's relatives, and one's own colleagues.

## Management of the Individual Patient

It is important that in the hospital setting each doctor, nurse, or paramedical person in the resuscitation team knows exactly what is expected of him or her, and that there is one leader to supervise and direct operations. The leader as far as possible should not participate in the resuscitative efforts. The description that follows is based on the current revised guidelines of ACLS.

*Excellent CPR and early defibrillation of ventricular tachycardia or ventricular fibrillation remain the cornerstone of both basic and advanced life support.*

## Basic Life Support (Flowchart 1)

The steps for CPR in a patient who has suffered an SCA outside the ICU, e.g., in the ward or any department of the hospital are outlined below:

1. If a patient is found unresponsive with unpalpable carotids, shout for help, activate a code (code blue in our hospital) whose sound warns of this emergency, bringing a trained team of doctors and nurses to the site hopefully within minutes. The most important equipment that needs to be rushed to the patient in order of importance are: a defibrillator, bag mask, and an ECG machine. In our hospital, we have in every ward organized a ready (daily checked) emergency trolley containing a laryngoscope, bag mask, laryngeal mask, an endotracheal tube, and emergency drugs. A portable ECG machine and a defibrillator are also kept in the duty room of each ward. An emergency trolley, or at least a defibrillator should be rushed to the site of the emergency.
2. Once the physician at the site of emergency has shouted for help, he or she immediately starts CPR. The CPR should be excellent; *anything short of excellence does not allow significant cerebral and coronary perfusion, thereby reducing the patient's chances for a neurologically good survival.*

   The following points with regard to chest compression need to be stressed if chest compression is deemed to be excellent.

   - The safest manner of depressing the sternum is with the heel of the rescuer's hand at the nipple line, with the fingers kept off the rib cage. It is best to cover the heel of one hand with the heel of the other, the heels being parallel to the long axis of the sternum. If the rescuer's hands are placed too high or too low on the sternum and if the fingers lie flat on the chest wall, there is a good chance of fracturing the ribs and damaging the viscera, in particular the liver.

     It is important to allow the chest to recoil after each compression, but the hands should not be lifted off the chest or change position.

     The rescuer's elbows should be stay locked, arms straight, with the shoulders directly over the patient's sternum. This allows the rescuer's body to provide a perpendicularly directed force for sternal depression.
   - Chest compressions should be of sufficient depth (5–6 cm, 2–2.5 inch) at a rate of 100–120 beats/min and should allow for complete recoil of the chest between compressions to be effective. Chest compressions are the most important feature of CPR. Coronary and cerebral perfusion pressure and return of spontaneous circulation (ROSC) are maximized when excellent chest compressions are performed. Chest recoil between down strokes (proper depth of at least 5 cm) generates a significant negative intrathoracic pressure resulting in higher cardiac preload resulting in an enhanced coronary blood flow.

     *If the rescuer is single without help, he or she should continue only with chest compression, uninterruptedly till further help arrives.*
   - Once excellent CPR has started and is uninterruptedly in progress, quick attention should be given to the airway. The head is tilted back and the angle of the jaw should be pushed forward, and the tongue should not be allowed to fall back. Any foreign body obstructing the airway must be promptly recognized and removed. This is the one condition which may need a very brief interruption of the chest compressions.

     *The mantra in CPR is circulation (C), airway (A), and breathing (B).*

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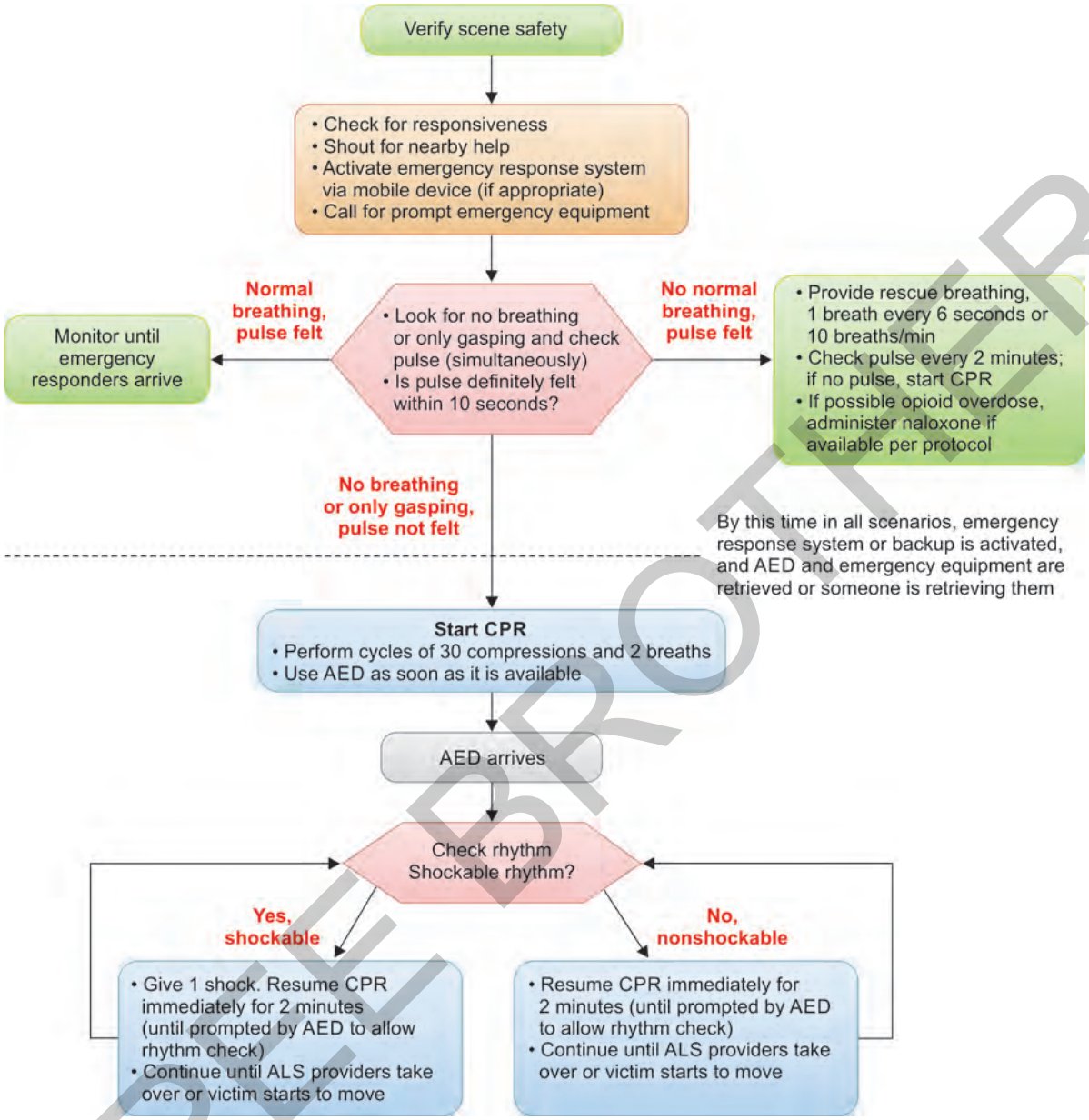
**Flowchart 1:** Adult basic life support algorithm for healthcare providers.




(AED: automated external defibrillator; CPR: cardiopulmonary resuscitation)
*Source:* 2020 American Heart Association.

Once the airway is secured and kept open, ventilation needs to be provided. Proper ventilation includes:

- Giving two ventilations after every 30 compressions.
- Each ventilation should be no more than 1 second.
- Providing just enough tidal volume to observe the chest rise appropriately (roughly 500–600 mL or 6 mL/kg)
- *Bag-mask ventilation:* Using a bag-mask technique for providing ventilation to work satisfactorily. Ideally, two persons are required to carry this out—one holding and fixing the face-mask, the other squeezing the bag.

  If, however, bag-mask ventilation is ineffective or impossible to perform (during patient transport), ventilation can be performed by a supraglottic airway (e.g., laryngeal mask). This technique allows adequate ventilation and reduces the risk of aspiration compared to bag-mask ventilation technique. Endotracheal intubation is as far as possible avoided during resuscitation from cardiac arrest.
- *Preventing hyperventilation:* Excessive ventilation (excess volume or frequency) is harmful to the circulation as there is an inappropriate increase in intrathoracic pressure which results in decreased venous return, reduced cardiac filling and lower stroke volume so that chest compressions result in poor perfusion.

As soon as a defibrillator is available (and the sooner the better), CPR should proceed as follows—after every cycle of chest compressions lasting 2 minutes the rescuer should check the rhythm. The time to check the rhythm should not

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take >5 seconds. If the rhythm shows VF, pulseless VT or VT with a ventricular rate >150 per minute, the patient should be prompted defibrillated. It is vital that the defibrillator is kept charged and ready to shock well before the rhythm check is done. Biphasic defibrillators are preferred because they have greater efficiency on lower energy level. Chest compressions should be continued till the moment the shock is to be delivered and should be promptly resumed and continued after its delivery without checking the pulse. After every 2 minutes of CPR, if the rhythm is still "shockable" the necessary shock should be given at the full or near full strength.

The above steps in "basic life support" should continue uninterruptedly till a team trained in ACLS arrives and takes over further management.

The following points need to be restressed:

- Cardiopulmonary resuscitation should be nothing short of excellent if good results are to be obtained—chest compressions at 100–120 per minute, continuous uninterrupted.
- If two or more rescuers are present, each rescuer should take over chest compressions after every 2 minutes compression cycle—this is to avoid fatigue which could cause a lapse in the correctness of compressions.
- Defibrillation should be as prompt as possible for good results.
- However, if a rhythm check shows a total arrest (flat line on ECG monitor) or PEA, defibrillation is of no use; chest compression should continue under the above circumstance.
- Any tachyrhythm (besides VF or VT) which is shockable should be shocked in the hope of restoring a stable circulation.

## COMPLICATIONS OF CARDIOPULMONARY RESUSCITATION

Complications are more frequent when incorrect technique is used for chest compression. They, however, can occur despite excellent technique. Complications include rib and sternal fracture; pneumothorax, hemothorax, cardiac and lung contusions; and serious intra-abdominal injury, particularly to the liver.

*An important point to remember is that the most common medical cause of cardiac arrest in an adult is a myocardial infarction or one of the acute coronary syndromes. If there is clear evidence of a myocardial infarct with ECG showing ST elevation even many days prior to an SCA, or if there is a history available of typical cardiac pain before the occurrence of arrest, or if during any of the 2-minute rhythm surveys observed during CPR there are even occasional ECG complexes with ST elevation, an arrangement to shift the patient to the catheter laboratory is imperative and immediate. Resuscitation attempts though difficult should continue till transport to the laboratory is done. Angioplasty and stenting of a blocked left anterior descending (LAD) artery or a dominant right coronary artery (RCA) most often makes the difference between survival and death.*

*There are a number of cardiologists who hold the view that every middle-aged or elderly person with SCA whose cause is not obvious should be given the benefit of cardiac catheterization in the hope that a blocked coronary artery responsible for the arrest could be opened or stented.*

## ASYSTOLE AND PULSELESS ELECTRICAL ACTIVITY

Asystole signifies complete absence of demonstrable electrical and mechanical cardiac activity.

Pulseless electrical activity is defined as one with demonstrable organized electrical rhythm but without sufficient mechanical contraction of the heart to produce a palpable pulse beat or measurable blood pressure.

*The successful management of both depends on (a) immediate and continuing CPR; (b) determining and treating any possible reversible cause of asystole or PEA; (c) administering IV epinephrine every 3–5 minutes until ROSC or a shockable rhythm ensues.*

Reversible causes that need to be carefully looked for and promptly treated are hyperkalemia, severe acidosis, hypoxia, a severe internal bleed, poisoning and most important of all a tension pneumothorax or a cardiac tamponade.

It is important to know that neither asystole nor PEA responds to defibrillation. Atropine is not recommended for asystole or PEA. Cardiac pacing is ineffective and not recommended.

If an SCA occurs in an ICU patient who already has a central venous pressure (CVP) and an arterial line, good feedback information can be obtained. During CPR on such a patient, a reasonable goal is to maintain a diastolic blood pressure >20 mm Hg and maintain the central venous oxygen saturation ($ScVO_2$) above 30%; this provides indirect information about oxygen delivery and cardiac output.

## MONITORING CARDIOPULMONARY RESUSCITATION

The two most important parameters that need to be monitored are pulse and ECG tracings viewed on a monitor.

1. *Pulse:* The presence of a palpable pulse denotes mechanical activity of the heart and when combined with an original stable rhythm as observed on an ECG monitor denotes successful resuscitation. The importance of not interrupting chest compressions to feel the pulse has already been emphasized.
   It should be noted that satisfactory arterial pressure or a palpable arterial pulse during chest compression does not necessarily reflect coronary perfusion pressure

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because ventricular pressures are elevated simultaneously as well. Therefore, a palpable pulse during chest compression (though a necessary accompaniment to chest compression) may be misleading. The clinician should look for other evidence of improved coronary circulation—as, for example, return of the ECG to an original stable rhythm, a palpable pulse during a break in the chest compression and a rise of end-tidal carbon dioxide ($EtCO_2$).

2. *ECG*: Abnormal rhythms besides VT and VF are commonly encountered during CPR. As discussed earlier shockable rhythms should be promptly shocked. Perfusion may not be absent but very poor in supraventricular tachycardia (SVT) with a ventricular rate >180 bpm or in atrial fibrillation with very fast ventricular rate. Electrical cardioversion is again the treatment of choice to restore a stable rhythm.

### Oxymetry

Monitoring $O_2$ saturation through oxymetry is useful to determine the presence or absence of hypoxemia during resuscitation.

## MONITORING $EtCO_2$ DURING CARDIOPULMONARY RESUSCITATION

Sudden sustained increase in end-tidal $CO_2$ during CPR generally indicates an ROSC; a decreasing $EtCO_2$ may indicate inadequate compression.

Arterial pH and $PaO_2$ and $PaCO_2$ need to be monitored so as to allow if necessary any correction of metabolic or respiratory acidosis.

*Serum electrolytes:* This again may well be important as hypokalemia, hyperkalemia, and hypomagnesemia may well be responsible for arrhythmias or even arrest and need to be corrected for successful resuscitation.

Any blood taken for investigations should be done in the very short break after 2 minutes of a cycle of cardiac compression, when the ECG is being scanned, or during cardiac compression which remains uninterrupted.

The ACLS guidelines also include the following additional features:

- Provide 100% oxygen in patients who have an ROSC, maintaining saturation of 94%; it is believed (rightly or wrongly) that hyperoxia may be harmful to patients leading to myocardial ischemia.
- Oropharyngeal or nasopharyngeal airways can be used as adjuncts.
- In addition to clinical assessment, ACLS guidelines recommend confirming and monitoring endotracheal tube placement, monitoring the quality of CPR, and ROSC. If continuous waveform capnography is not available, a nonwave carbon dioxide detector could be used.

## TERMINATION OF RESUSCITATION EFFORTS

When does one terminate resuscitation in patients with sudden cardiac arrest? The answer is difficult. Rarely, we have seen good recovery after 30 minutes of resuscitation.

The following factors influence the decision to stop resuscitative efforts:

- Duration of resuscitation >30 minutes without a sustained perfusing rhythm.
- Prolonged interval between estimated time of arrest and initial resuscitation.
- Initial ECG rhythm showing asystole and persisting as asystole after vigorous resuscitative efforts for >20 minutes.

Many objective endpoints of resuscitation have been proposed. Perhaps the best predictor of outcome is the $EtCO_2$ level following 20 minutes of resuscitation. A very low $EtCO_2$ (<10 mm Hg) following prolonged resuscitation (>20 minutes) is a sign of absent circulation and a strong predictor of mortality. It is, however, important to note that a low $EtCO_2$ may also be caused by misplaced endotracheal tube. This must be excluded before stopping resuscitation.

Out-of-hospital cardiac arrests in our country and in Mumbai (barring a few exceptional examples) are almost always fatal. It is indeed rare to find a lay person trained in basic life support we can offer help to a patient in cardiac arrest. Even if CPR is given, a well-equipped ambulance with medical personnel may take an hour to arrive at the scene because of the impossible traffic during working hours.

The futility of resuscitation should be realized if the arrest has not been witnessed by emergency medical service (EMS) personnel or by emergency doctors of an arriving ambulance, if the initial rhythm is nonshockable (asystole or PEA) or if there is no circulation after two to three doses of intravenous epinephrine.

## ADVANCED LIFE SUPPORT

The measures constituting ACLS are best illustrated in the management of cardiac arrest in a patient in the ICU. Basic life support in such circumstances merges seamlessly with ACLS. This is best illustrated in **Flowchart 2**. Certain points, however, need to be stressed.

### Use of Antiarrhythmic Drugs

Evidence suggests that antiarrhythmic drugs provide little benefit for refractory VF or pulseless VT. They should, however, be given a try after two attempts at defibrillation have failed. The drugs in use are amiodarone 300 mg IV bolus which can be repeated if necessary or lidocaine 1–1.5 mg/kg bolus then 0.5–0.75 mg/kg every 5–10 minutes. These drugs are given in VF or pVT unresponsive to defibrillation, CPR, and epinephrine.

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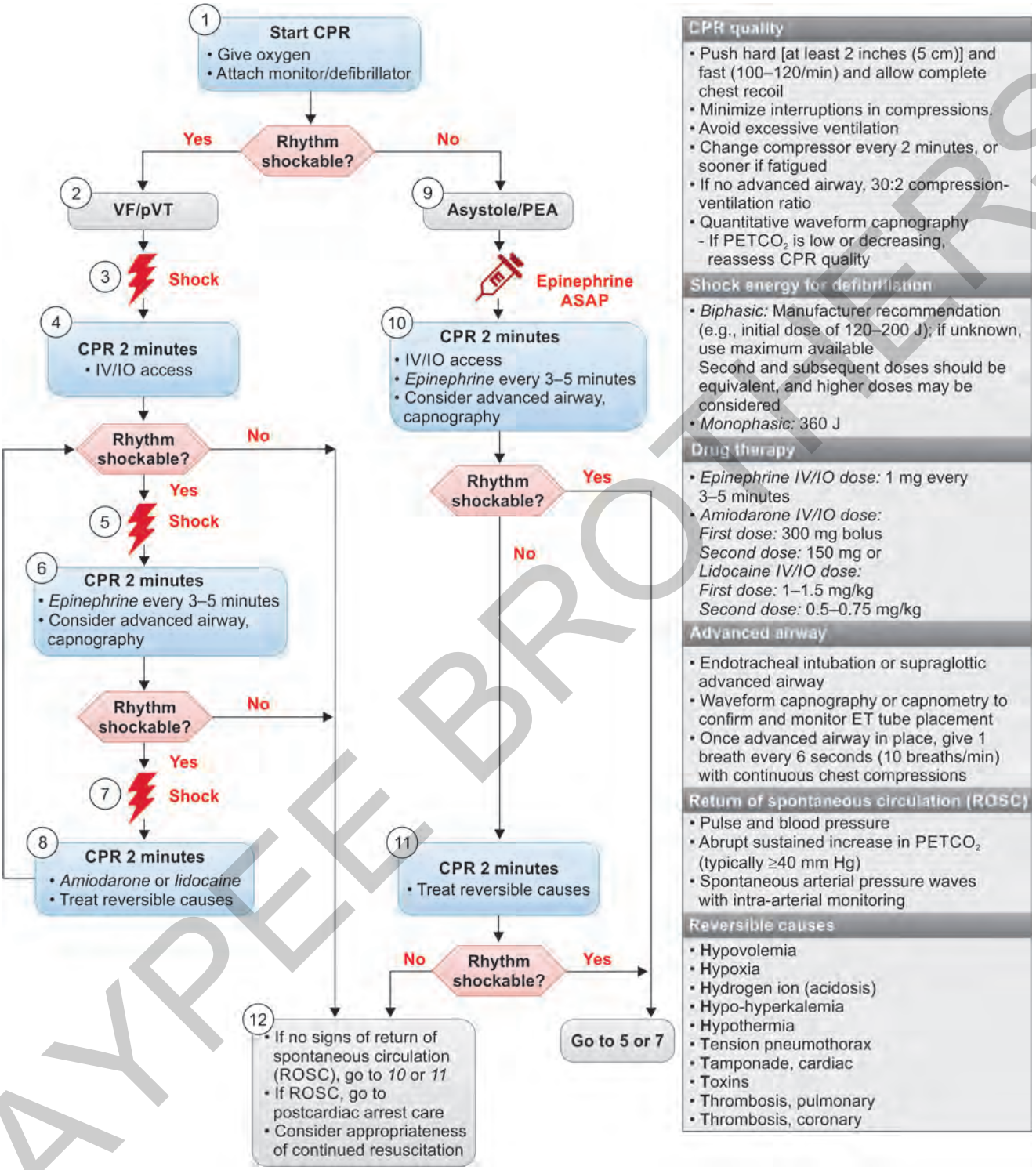
**Flowchart 2:** Adult cardiac arrest algorithm (vF/pVT/Asystole/PEA).




(CPR: cardiopulmonary resuscitation; ET: endotracheal; IO: intraosseous; IV: intravenous; PEA: pulseless electrical activity; pVT: pulseless ventricular tachycardia; VF: ventricular fibrillation)
*Source:* 2020 American Heart Association.

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Magnesium sulfate 2 g intravenous bolus followed by a maintenance dose may be of use to treat polymorphic tachycardia as also torsade de pointes. It is not recommended for routine use in adult cardiac patients **(Flowchart 2)**.

## POSTRESUSCITATION CARE

Postresuscitation care after recovery of spontaneous circulation (ROSC), often needs to address major issues simultaneously headed by an experienced leader.

Issues which necessitate immediate attention and precedence over others may vary from patient to patient. Also many issues may need to be addressed simultaneously.

- The first priority is ensuring hemodynamic stability so that adequate perfusion to vital organs in particular to the heart and brain are maintained.
- The airway needs to be firmly secured. Invariably the patient has been intubated and is on ventilator support. Ventilator settings must ensure that there is no hypoxia, hypocapnia, hypercapnia, and hyperoxia.
- It is crucial to minimize brain injury. In addition to factors 1 and 2 stated above, the prompt use of hypothermia after a careful neurological assessment is of vital importance.
- Global ischemia or reperfusion injury can lead to single or multiple organ failure. These require advanced critical care.
- As far as possible the intensivist team should determine and treat the cause of cardiac arrest.

## Hemodynamic Stability

Ensuring hemodynamic stability after ROSC is a priority issue. An adequate blood pressure must be maintained to ensure good perfusion to the brain and other organ systems. The mean arterial pressure (MAP) should be well above 65 mm Hg, preferably 80–100 mm Hg to optimize cerebral perfusion. Episodes of hypotension even after hemodynamic stability is attained are not uncommon; close monitoring and prompt correction of hypotension is imperative. Volume replacement with isotonic saline should be given to keep the CVP between 8 and 12 mm Hg. Norepinephrine titrated to maintain an MAP of 80–100 mm Hg is often necessary. Inotropic support (dopamine) may also be used. It is believed that inotropic and vasopressor support could well mitigate the myocardial dysfunction observed during the first 24–48 hours after cardiac arrest.

- *ECG:* A 12-lead ECG should be taken promptly after ROSC. The presence of ST elevation or a new left bundle branch (LBB) block points to a myocardial infarction requiring emergency reperfusion therapy. History of cardiac pain preceding an SCA also warrants prompt catheterization for a block in one or more coronary arteries.

  A prolonged QTc interval may reflect a primary arrhythmia or an electrolyte abnormality. Evidence of right heart strain may suggest pulmonary embolism.

  When the diagnosis of an acute coronary syndrome is uncertain on ECG findings, a bedside ECHO is imperative, it may demonstrate focal wall motion abnormalities suggesting myocardial infarction. Global hypokinesia is frequent after cardiac arrest, but focal hypokinesia does not occur. An ECHO may reveal the presence of a cardiac tamponade, or a dissecting aneurysm responsible for the arrest. *Finally, ongoing cardiogenic shock may be the sole manifestation of acute coronary syndrome in patients with ROSC.*

  Many units suggest that any individual with a past history of ischemic heart disease who suffers an SCA should be sent for catheter study and possible reperfusion after the ROSC. It has already been stated earlier that a number of units which insist that every individual who suffers an SCA and has ROSC should be submitted to catheter study if no other obvious cause of cardiac arrest is evident.

  Imaging studies include a chest X-ray, abdominal ultrasound, and if feasible CT scan of the chest, abdomen, and brain.

  Imaging studies of the chest can help to diagnose a pneumothorax, aortic dissection, cardiac tamponade or even a catastrophic pulmonary embolism. CT of the brain can detect early cerebral edema, or an intracranial hemorrhage that could have been the cause of SCA.
- *Arrhythmias:* The danger posed by the occurrence of arrhythmias after ROSC can be formidable. They may occur during CPR, but more often soon after recovery from cardiac arrest; they may also occur even 24–72 hours after recovery. In an emergent situation (soon after ROSC) there is little time to analyze the ECG with great exactitude.
- Disturbance in rate and rhythm of the heart observed after ROSC are very briefly dealt with here. The reader should refer to the Chapter on Cardiac Arrhythmias and their Management.
- *Bradycardia:* If the bradycardia is symptomatic (hypotension, shock, and chest pain) or marked (at or below 30 beats/min) it needs to be treated. Marked sinus bradycardia or a block at the AV node responds to IV atropine 0.5 mg, repeated to a maximum of 2–3 mg. If still symptomatic, an infusion of dopamine or epinephrine may be of help. A high-grade AV block and in particular a symptomatic complete AV block requires transcutaneous pacing to start with, followed by transvenous pacing after appropriate arrangements have been made.

## Approach to Tachyarrhythmia

The ACLS recommends the following approach in an emergent setting:

- Is the patient in sinus rhythm?
- Is the QRS complex narrow or wide?
- Is the rhythm regular or irregular?
- Regular and narrow QRS complexes mean either a sinus tachycardia or a supraventricular tachycardia.
- Sinus tachycardia requires no medication; attention is focused on treating the cause.
- Supraventricular tachycardia is a regular tachycardia generally caused by a re-entrant mechanism. The treatment is intravenous adenosine or if this fails, a synchronized DC shock.

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- Irregular narrow QRS complex is due to atrial fibrillation, atrial flutter with a changing block, or sinus tachycardia with multiple ectopics. The important initial goal is to reduce the heart rate using appropriate doses of either diltiazem, verapamil or metoprolol. IV amiodarone usually abolishes multiple ventricular ectopics.
- Regular wide QRS complexes could be due to ventricular tachycardia or an aberrantly conducted SVT. Drugs that could be used for conversion are amiodarone or sotalol. Under the prevailing circumstances, particularly if the patient is unstable, one should advise prompt defibrillation.
- *Irregular wide complexes:* These are due to polymorphic ventricular tachycardia (Torsades de pointes), AF with aberrant conduction or atrial fibrillation with Wolff–Parkinson–White (WPW) syndrome. The treatment of choice is electrical cardioversion whichever one of these may be present. It is important that AV nodal blockers (β-blockers, calcium-channel blockers, digoxin, and adenosine) are never given to the patient as they may precipitate ventricular fibrillation and death.

  The above-mentioned arrhythmias may occur during CPR but are most frequently observed after ROSC in patients treated for cardiac arrest. It is for this reason that they are briefly considered here. Time is of essence at this junction, for even patients who are stable after ROSC may become unstable if these rhythm disturbances are not promptly treated. There may be no or little time for elaborate, exact identification of ECG rhythms. Therefore, when approached in the manner described above, prompt treatment becomes organized and feasible.
- *Respiratory considerations: Though endotracheal intubation is best avoided during resuscitation from SCA, once spontaneous recovery of circulation is achieved, it is best to intubate the patient, secure the airway and keep the patient on mechanical ventilation. The stomach should be decompressed through a nasogastric tube.*

The following guidelines are best observed with regard to mechanical ventilation in the postcardiac arrest patient.

- The $pCO_2$ should be targeted to about 40 mm Hg. Hypocapnea can induce cerebral vasoconstriction and should be avoided.
- Oxygen saturation should be >94%. Though hypoxia is clearly harmful and should be promptly corrected, it is advisable to avoid hyperoxia ($PaO_2$ > 300 mm Hg) which is believed to be associated with poor outcomes.
- Hyperventilation should be avoided as it decreases preload and reduces both cardiac output and coronary perfusion pressure.

### *Ensuring Adequate Delivery*

An $ScVO_2$ > 70 in postcardiac arrest is advocated in the belief that the postarrest syndrome may be similar to that of sepsis.

## NEUROLOGICAL CONSIDERATIONS

Neurological considerations already mentioned are directed toward minimizing brain injury. These involve:

- A basic neurological examination after ROSC and a careful periodic follow-up examination to assess the neurological state over time.
- The prompt use of therapeutic hypothermia or targeted temperature measurement (TTM) if neurological examination so indicates.

*Basic neurological examination:* This examination is extremely important as it may help to determine the cause, the likely clinical course and outcome and the possible need for neurological intervention (therapeutic hypothermia or targeted temperature measurement—TTM). Sedation and neuromuscular blockade should be avoided to allow a proper examination. Brainstem reflexes should be carefully noted including pupillary, corneal, oculocephalic gag, and cough reflex to stimulation. The presence or absence of these reflexes particularly if their absence persists over 24 hours correlates with poor chances of survival.

The Glasgow Coma Score or Fall Outline of UnResponsiveness (FOUR) score should be determined with particular attention to the motor score, which correlates with neurological outcome.

It is to be noted that asymmetrical neurological findings do not occur following an ROSC and suggest a structural intracranial lesion.

## USE OF THERAPEUTIC HYPOTHERMIA OR TARGETED TEMPERATURE MEASUREMENT

The single most important objective to bear in mind in a patient successfully resuscitated from cardiac arrest is the prevention of brain injury. Though brain ischemia and injury can occur during prolonged CPR, most of it develops as an active process over hours to days after resuscitation. In other words, it is a reperfusion injury.

Several cellular and molecular mechanisms contribute to brain injury. During brain ischemia and soon after reperfusion there is an increased release of stimulatory amino acids, free radicals that harm both neuronal integrity and function. There are focal disturbances in blood flow and an inhibition of protein synthesis. The activation of several extracellular and intracellular signaling pathways leads to specific changes in gene transcription. Most importantly after 24–72 hours of reperfusion there is an activation of specific proteases which lead to neuronal death. Brain injury with neuronal death probably results in a culmination of all these metabolic changes observed after reperfusion is established. There is no drug as yet that has been shown to prevent or counter brain injury. Calcium-channel blockers, magnesium, diazepam, and thiopental have been tried in randomized trials but without benefit.

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This is because there are multiple mechanisms contributing to neuronal injury and death, and a single drug is not likely to act on all of these mechanisms. Perhaps different classes of drugs, each acting on different mechanisms causing brain damage may in the future be discovered. *Today, the focus on postcardiac arrest intensive care rests on (a) achieving therapeutic hypothermia (32–34°C) for 24–48 hours after resuscitation; (b) maintenance of a cardiocerebral perfusion pressure; (c) early cardiac catheterization with a view to opening up and stenting critically blocked coronary vessels if these be present.*

- Hyperthermia should be strongly avoided postcardiac arrest. According to an observational study on 151 patients, the risk of death increases for each degree of rise in temperature over 37° during the first 48 hours after cardiac arrest.
- Data suggest that active control of the core temperature between 32 and 36°C is the best strategy to promote patient survival. This is achieved through therapeutic hypothermia (TH) or targeted temperature management (TTM). Hypothermia refers to a core temperature maintained between 32 and 34°C while temperature control refers to a core temperature not >36°C.
- Patients not following commands or showing purposeful movements following resuscitation should be treated with hypothermia or temperature control. This should be done within 4 hours of ROSC, the sooner the better.
- Either TH or TTM can be used in pregnancy or in hemodynamically unstable patients, as also in those who have had catheter studies (angioplasty with stent insertion) or those who have received thrombolytic therapy. However, hypothermia is associated with a greater risk of bleeding in the last two groups of patients.
- In the postcardiac arrest patients, active control of core temperature should be achieved as soon as possible with control maintained for at least 48 hours. It is suggested that core temperature should be lowered to 36°C for 24 hours for patients who have no complications, patients who have moderate coma with some motor response, no malignant electroencephalogram (EEG) patterns and no evidence of cerebral edema on the CT of the brain. However, it is suggested using 34°C in patients for 24–48 hours who are deeply comatose (no motor response or no brainstem reflexes), EEG showing epileptic form activity or CT brain showing cerebral edema.
- Methods for therapeutic hypothermia and control of temperature:
  - Intravenous infusion of 30 mL/kg of cold 4°C of isotonic saline reduces the core temperature by >1° per hour. Patients with a history of heart failure, or features of severely compromised renal function or signs of pulmonary edema should not receive rapid fluid infusions to induce TH. Surface cooling measures should be used in place of cold intravenous infusions of normal saline.
  - Surface cooling measures consist of using ice packs, cooling blankets, cool water sponges, and fans. These can be used as an adjunct method to induce hypothermia when combined with an infusion of 1–2 L of cold saline.
  - Intravascular cooling devices are also available but they have no proven advantage compared to methods stated above.

  Shivering is a common occurrence during hypothermia; adequate sedation can counter this complication either through a continuous infusion of fentanyl or midazolam.
- Temperature should be monitored continuously during TH or TTM. The gold standard for measuring core temperature is the central venous temperature. Rectal probe may, however, suffice. The core temperature is a measure of the brain temperature.
- Rewarming should be slow, the rate of increase not being >0.2–0.25°C/h. Rapid rewarming could result in hyperkalemia, cerebral edema, and seizures.

  One should be aware of the potential side effects of induced hypothermia. These include a mild coagulopathy, arrhythmias which include bradyarrhythmia, and prolonged QT interval. It can cause hyperglycemia due to insulin resistance. It can also lead to a "cold diuresis" which if marked can cause hypovolemia, hypokalemia, hypomagnesemia, and hypophosphatemia.

  However, the overall benefit of therapeutic hypothermia is now accepted. Many studies have shown that patients treated with TH are more likely to survive compared to those not treated with TH.
- Management of global ischemia during CPR and after ROSC and reperfusion injury during ROSC can lead to single or multiple organ dysfunction necessitating expert critical care.

## DETERMINING THE CAUSE AND TREATMENT OF CARDIAC ARREST

Efforts to determine the cause of cardiac arrest should be made pari passu while dealing with the issues stated above. This may or may not be possible. History from a relative or a close associate and a meticulous physical examination after ROSE may give invaluable clues. Laboratory tests in particular CBC, serum electrolyte, arterial pH, blood gases done during CPR and ACLS should be repeated **(Flowchart 3)**.

A two-dimensional (2D) echocardiography is imperative as is an X-ray chest, USG abdomen, and pelvis.

Further imaging should be done if necessary once the patient is hemodynamically stable. Examples of treatable causes of cardiac arrest have already been given earlier.

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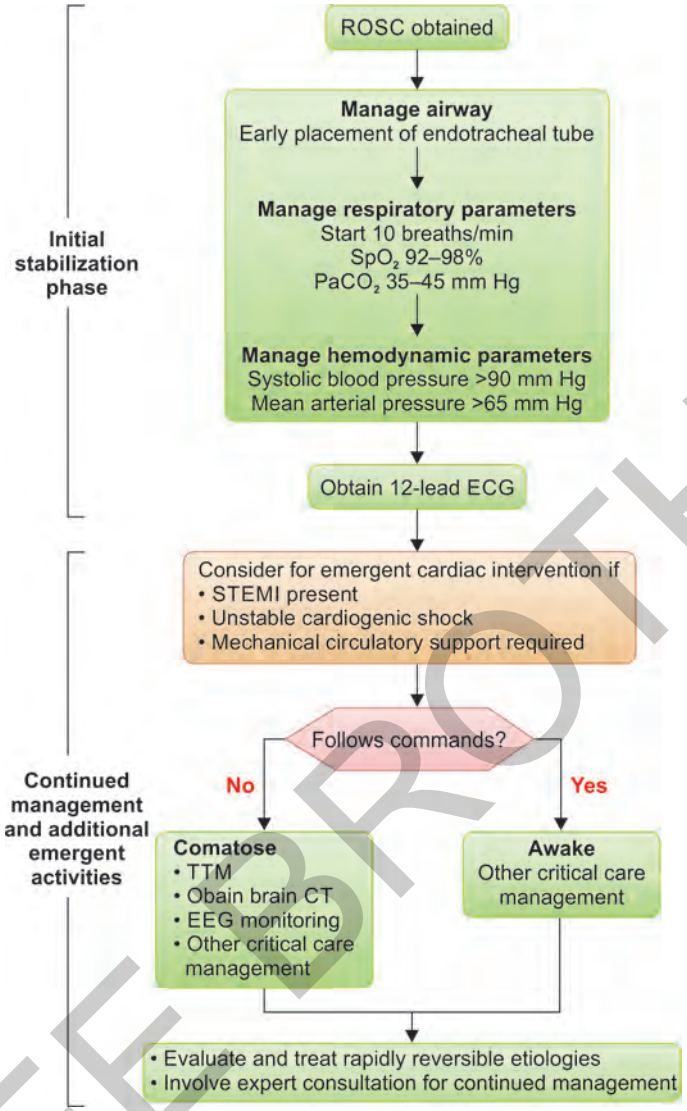
**Flowchart 3:** Adult postcardiac arrest care algorithm.




(EEG: electroencephalogram; ROSC: return of spontaneous circulation; STEMI: ST-elevation myocardial infarction; TTM: targeted temperature measurement)

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# PRINCIPLES OF CRITICAL CARE

The fourth edition of the *Principles of Critical Care* has been long overdue, as it was unfortunately delayed by the COVID-19 pandemic. It is at last ready and I am certain that it will continue to remain the standard text in critical care medicine in this country.

The book on purpose, is not encyclopedic unlike most of the standard Western texts on critical care. It is directed to the practice of critical care as observed and practised in our country and other tropical developing countries. However, it also deals with critical care problems common to both, the East and West.

The critical care of important fulminant infections in the tropics has been revised, emphasized, and will unquestionably prove very useful to intensivists practising in tropical countries such as India, Africa and parts of South East Asia.

The text has been illustrated by new excellent images. To the best of my knowledge, this is the only book on critical care medicine that has a full section devoted to the imaging of various problems pertaining to critical illnesses, thanks to Dr Anirudh Kohli who leads the Imaging Department at the Breach Candy Hospital, Mumbai, Maharashtra, India.

The present edition, just like the previous editions, is based on the author's extensive experience on this subject for over 50 years. Though the book has largely been written by the author to provide an evenness in style, language and content, it also includes guest contributors renowned for their expertise in their chosen fields.

Every chapter has been thoroughly revised. The book also contains new material. To give just two examples—there are chapters on Disaster Management including the COVID-19 pandemic and an excellent description of the COVID-19 pneumonia written by an expert.

The chapter on the Principles, Philosophy, and Ethics of Critical Care is of great importance. It includes the author's reasoned comments on evidence-based medicine.

This comprehensive book is for all doctors interested in critical care medicine and emergency medicine. It should also prove useful to house physicians, registrar, postgraduate students, consultant physicians and surgeons, intensivists, anesthesiologists engaged in the management of life-threatening emergencies.

**Farokh Erach Udwadia** is a Consultant Physician at the Breach Candy Hospital, Mumbai, Maharashtra, India where he is also Director of the Intensive Care Unit. He has authored many books, including *Tetanus, Man and Medicine—A History, Bedside Clinics in Medicine, The Forgotten Art of Healing and Principles of Respiratory Medicine*. He has a number of national and international distinctions and awards to his credit and was conferred the Padma Bhushan by the President of India in 1987.

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