
UNIT 5 CARDIAC EMERGENCIES

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5.0 OBJECTIVES

After studying this unit, you should be able to:

- understand some of the common cardiovascular emergencies;
- know the basics of cardio pulmonary resuscitation;
- select the correct inotropic drug;
- deal with arrhythmias that present as emergencies;
- manage pulmonary edema;
- manage hypertensive emergencies;
- diagnose and manage cardiac tamponade;
- recognize and treat pulmonary embolism; and
- manage cyanotic spells.

5.1 INTRODUCTION

There are several types of cardiac emergencies where immediate action is required as with cardiac arrest or where action could be slightly delayed as with severe hypertension. It is important to be familiar with the basics of cardio pulmonary resuscitation and the use of inotropic and anti arrhythmic drugs. Acute coronary syndromes and aortic dissection are cardiac emergencies and are considered in the respective chapters. Acute pneumothorax also presents as an emergency and one should be familiar with its recognition and management.

5.2 CARDIO PULMONARY RESUSCITATION

Cardiac arrest is defined as cessation of cardiac mechanical activity. It is a clinical diagnosis, confirmed by unresponsiveness, absence of detectable pulse and apnea or agonal respirations. CPR is an attempt to restore spontaneous circulation through any of the broad range of manoeuvres and techniques. It is essential to act immediately as irreversible damage can occur in a short time. Within 15 seconds of cardiac arrest, the patient loses consciousness, electro-encephalogram (EEG) becomes flat after 30 second, pupils dilate fully after 60 seconds and cerebral damage takes place within 90-300 seconds.

The heart rhythm associated with cardiac arrest can be either ventricular fibrillation/ventricular tachycardia (VF/VT) or other rhythms including Pulseless Electrical Activity (PEA) or asystole. Survivability and other outcomes are much better if the associated rhythm is VT or VF. It is extremely important to recognize VT or VF as the underlying rhythm, because these rhythms need defibrillation as quickly as possible for optimal outcomes.

Basic Life Support

The action taken during the first few minutes of an emergency are critical to victim's survival. BLS covers 3 links in the chain of survival. This refers to maintaining airway, supporting breathing and circulation at the site of arrest till further help becomes available. If the victim is unresponsive, his head should be positioned and airway should be opened by performing the Triple Airway Manoeuvre (Head tilt-Chin lift-Jaw thrust).

Supporting Breathing

Breathing is assessed by looking at the chest for any respiratory movements, listening or feeling for breathes sounds. If there are no breathing efforts, initially 2 rescue breaths are provided. In absence of any equipment, mouth to mouth or nose resuscitation remains the best method. The aim is to deliver small volume (400-600 ml) over 1-2 seconds with cricoid pressure if possible. The earlier practice of delivering large volume of air (800-1200 ml) is no longer recommended.

Ventilation, Chest Compressions

Even when the first three defibrillation attempts fail, the best chance of resuscitation still lies in successful defibrillation. Airway is secured by endotracheal intubation if skills are available or, otherwise, by a laryngeal mask airway or a combitube. Ventilation is commenced with 100 per cent oxygen. Fifteen compressions and 2 ventilations are given for a minute and defibrillation is tried again. Once airway is secure by intubation or a laryngeal mask airway, uninterrupted chest compressions should be carried out, without pausing for ventilations. The rate of compression should be 100/minute and ventilation should be carried out at 12 breaths/minute.

Supporting Circulation

No longer than 10 seconds should be spent in assessing circulation. Pulse check for lay rescuer has now been de-emphasized since it takes time and accuracy is only 65 per cent. External chest compressions can produce peak systolic pressure of 60-80 mm of Hg. In adults, a ratio of 15:2 (15 chest compressions followed by 2 artificial breaths) has been adopted for a single rescuer as well as two rescuers performed external chest compressions can produce in children the rate of compression should be maintained close to 100/minute or roughly 2 per second.

Defibrillation

This step is the key to survival. The survival rate is 90 per cent if the patient is defibrillated within 1 minute and only 10 per cent if it is delayed till 10 minutes after arrest. In an adult, one should start with 200 J and increase the energy to 360 J after two shocks. In case of biphasic defibrillators, continuing energy of 200 J is considered optimal. In children, the energy requirement is calculated at the rate of 2-4 J/kg. The optimal paddle sizes are 13 cm (adults), 8-10cm (children) and 4.5-5 cm (infants). The standard paddle position is where one electrode is placed over the right side of upper part of sternum just below clavicle and the other one is placed over the apex.

Advanced Cardiac Life Support

ACLS is best done in a hospital or at the scene by expert paramedical personnel if significant delay is anticipated in transfer of the victim to the hospital. The tasks performed during this phase, include continuation of BLS, use of equipment for ventilation and circulation, 12 lead electrocardiography (ECG) and arrhythmia recognition, establishment of intravascular access and drug therapy and lastly prehospital fibrinolytic therapy for acute coronary syndromes (ACS) and stroke in certain advanced healthcare setups. Venous access in CPR drugs are used with an aim to improve organ perfusion and to protect brain and heart from hypoxia, to facilitate defibrillation and to prevent recurrence of arrhythmias and to normalize metabolic derangements.

Vein Access

A Vein access is important during CPR. Even though a central venous line is ideal, its placement takes time, expertise and it has the potential to interrupt the process of CPR. Therefore, the next best option is a peripheral line. It has been shown that circulation time can be significantly reduced if 20 ml of saline is pushed after a drug bolus in a peripheral line. The other routes of drug delivery are tracheal, intra-osseous and even intra-cardiac/nasal/ Femoral. The tracheal route deserves special mention. The drug (2-3 times the intravenous dose) is diluted in 10 ml of saline and instilled in the tube, followed by 5 ventilations. Intraosseous route may be tried in children in the same dosages as those for the tracheal route. Intra-cardiac, nasal or femoral routes are no longer recommended and instilled in the tube, followed by 5 ventilations. Intraosseous route may be tried in children in the same dosages as those for the tracheal route. Intra-cardiac, nasal or femoral routes are no longer recommended.

Pharmacotherapy

Vasopressors

Adrenaline or epinephrine enhances cerebral and myocardial blood flow by preventing arterial collapse and by augmenting aortic diastolic pressure through alpha 1 and 2 receptors. The optimal dose is 1 mg every 3-5 minutes. In children, it can be given in the dosages of 10 µg/kg or 0.1 ml/kg of 1 in 10000 solution. Dose dependent improvement in regional myocardial and cerebral blood flow has been recorded in various studies but no improvement in hospital discharge and survival has been seen with vasopressin.

As a single bolus of 40 U, vasopressin has been recommended as an alternative to epinephrine in VF/pulseless VT refractory to defibrillation. Further studies are required to evaluate the efficacy of this drug. Vasopressin acts on non-adrenergic V1 receptors and has been recommended in case of fibrillatory arrest (40IU, single dose). The studies have recorded several advantages over adrenaline such as lack of beta effect, no impact of acidosis on its efficacy and even lower incidence of post resuscitation myocardial dysfunction.

Antiarrhythmic Agents

Contribution of lignocaine during arrest, to aid resuscitation from refractory VF remains uncertain. Lignocaine is an alternative in this situation if amiodarone is not available. Other problems associated with it are increase in defibrillation threshold, higher incidence of asystole after its use and a very delicate toxic to therapeutic ratio. Second choice behind amiodarone and procainamide. Amiodarone is useful in treatment of both atrial and ventricular arrhythmias. It is now recommended during ACLS after defibrillation and adrenaline. The dose is 150 mg diluted in 20 ml of 5 per cent dextrose given over 10 min, followed by infusion @1 mg/min for 6 hours and then @ 0.5 mg/min.

Sodium-bicarbonate (NaHCO_3)

There is no definite recommendation regarding its use in cardiac arrest. Whenever possible, bicarbonate therapy should be guided by the bicarbonate concentration or calculated base deficit obtained from blood gas analysis. Sodium-bicarbonate is definitely indicated if there is **hyperkalemia** and tricyclic antidepressant toxicity and pre-existing metabolic acidosis.

Calcium

(Ca^{++}) usually has no role unless patient presents with calcium channel blocker toxicity or if there is evidence of hypocalcemia or hyperkalemia. The dose of calcium gluconate is 0.5 ml/kg (maximum – 20 ml) of 10 per cent solution whereas 10 per cent solution of calcium chloride can be given in a dose of 0.2 ml/kg upto a maximum of 10 ml.

Magnesium

(Mg^{++}) mirrors the action of extra cellular potassium in stabilizing myocardial cell membrane and it is indicated only if hypokalemia or hypomagnesaemia is known to be present or the patient is diagnosed to be having a rhythm called “Torsade de Pointers”. The dose (as magnesium sulphate) is 1-2 gm diluted in 100 ml of 5 per cent dextrose given over 30-60 min followed by an infusion of 0.5-1.0 gm/hour.

Atropine

Enhances automaticity and conduction of both sinoatrial and atrio-ventricular node and is most effective in haemodynamically significant bradycardia because of vagal stimulation. It also has a role in slow pulse less electrical activity (PEA). The recommended dose in PEA and asystole is 1.0 mg IV and repeated 3-5 minutes if required. For bradycardia, the dose is 10 µg/kg repeated every 3-5 minutes up to a total dose of 40 µg/kg.

Assessment of CPR and When to Stop?

Termination of CPR efforts can be a difficult decision to make. It is apparent from the literature that prolonged resuscitative efforts are unlikely to be successful if there is no return of spontaneous circulation within 30 minutes of commencement of CPR.

Check Your Progress 1

1) What is the electrical energy level used for defibrillation?

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- 2) In cardiopulmonary resuscitation what is the number of chest compressions and the number of ventilations prior to establishing an airway?

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- 3) In cardiopulmonary resuscitation what is the number of chest compressions and the number of ventilations after establishing an airway?

-
-
- 4) What is the dose of epinephrine used during CPR?

5.3 INOTROPIC DRUGS

Circulatory failure may be due to failure of the myocardium, inadequate preload (ventricular filling) or pooling of blood for example in venous reservoirs which does not reach the heart. After ensuring that there is an adequate preload with infusions if necessary and hemodynamic monitoring, inotropic agents may be necessary to improve cardiac contractility.

Inotropes

The aim two to improve cardiac contractility. Ideally there should not be undue tachycardia, propensity for cardiac arrhythmias or a significant increase in myocardial oxygen consumption, interaction with other drugs or effect on other organs. The volume status, vasomotor tone whether vasoconstricted or vasodilated and cardiac state should be carefully assessed. An acute inotropic response in the physiological state is a rapid increase in tissue levels of cyclic AMP. The same principle is involved in the use of exogenous catecholamines which stimulate the beta adrenergic receptors and raise cyclic AMP or less commonly when the breakdown of cyclic AMP is inhibited by phosphodiesterase (PDE) inhibitors like Milrinone.

Receptors

There are 2 main types of adrenergic receptors. All catecholamines act directly on the adrenergic receptors. Beta receptor stimulation results in increased ATP conversion to cyclic AMP. Alpha 1 receptor activation acts independently of cyclic AMP and results in release of calcium and increased membrane permeability to calcium.

Norepinephrine is the agonist which acts on Beta receptors. There is depletion of norepinephrine stores in heart failure and a down regulation in the number of Beta receptors. Therefore, Alpha 1 and Alpha 2 receptors are important in maintaining the contractility in the failing myocardium. Epinephrine, Norepinephrine and Dopamine are all naturally occurring compounds. All three are Beta agonists at low doses with increasing Alpha1 receptor activity at higher doses. All increase stroke volume, cardiac output and mean arterial pressure without much change in the heart rate and little by way of arrhythmias. Dopamine is the precursor of Noradrenaline which in turn is the precursor of adrenaline.

Cadiovascular Effects of Catecholamines on Receptors

β_1 +Chronotropy +Dromotropy +Inotropy	β_2 +Inotropy Vasodilation Bronchodil'n	α_1 +Inotropy Vasoconstr'n	α_2 +Inotropy Vasoconstr'n
Noradrenaline	B effects predominate at low dose; Alp at high dose		
Adrenaline	B effects predominate at low dose; Alp at high dose		
Dopamine	B effects predominate at low dose; Alp at high dose		
Dobutamine	β_1+	β_2+	(+)
Isoprenaline	β_1+	$\beta_2(+)$	-

Receptor	Binding by	Effect of Binding
α_1	Epinephrine, Norepinphrine	Increased free calcium
α_2	Epinephrine, Norepinphrine	Decreased cyclic AMP
β_1	Epinephrine, Norepinphrine	Increased cyclic AMP
β_2	Epinephrine	Increased cyclic AMP

Beta Adrenergic Agonists

These drugs cause their positive inotropic effect by activating α -receptors with subsequent stimulation of adenylate cyclase and increased cAMP.

Dopamine

Dopamine is an endogenous catecholamine precursor with selective α_1 activity. However, it also stimulates the release of norepinephrine. At low doses, it stimulates renal dopaminergic receptors, which causes increased renal blood flow and diuresis. This effect has been increasingly questioned. Dopamine is not effectively absorbed orally. It is rapidly metabolized by the body and has a half-life of < 2 min. It is administered IV, by continuous infusion (1-20 $\mu\text{g/kg/min}$). Cardiac arrhythmias may occur due to α -adrenergic activity. Indications include cardiogenic or endotoxic shock and oliguria. In patients with advanced HF, who often have depleted intracardiac norepinephrine stores, dopamine is a less effective positive inotropic drug than other "directly" acting inotropes. At higher infusion rates (5 to 20 $\mu\text{g/kg/min}$), peripheral vasoconstriction occurs as a result of direct alpha-adrenergic receptor stimulation. Increases in systemic vascular resistance are common even at intermediate infusion rates. On initial administration, tachycardia and arrhythmia tend to be more pronounced than with dobutamine and are related to cardiac norepinephrine release.

D o b u t a m i n e

D o b u t a m i n e i s a s y n t h e t i c
d r u g s i m i l a r t o d o p a m i n e , b u t
i t d o e s n o t c a u s e r e l e a s e o f
n o r e p i n e p h r i n e a n d t h e r e f o r e
h a s m i n i m a l e f f e c t s o t h e r
t h a n α - 1 a c t i v i t y . D o b u t a m i n e
i s a m o r e e f f e c t i v e p o s i t i v e

inotrope than dopamine with less chronotropic effects, although it does not dilate the renal vascular bed. Like dopamine, dobutamine is not effective orally and has a plasma half-life of < 2 min. It is the preferred drug for short-term therapy of refractory congestive heart failure. Dobutamine causes an immediate increase in blood pressure due to increased cardiac output. It is given as a continuous IV infusion at $2 - 20 \mu\text{g} / \text{kg} / \text{min}$. The limitations of dobutamine are that it (1) is a relatively weak beta-agonist, (2) only modestly lowers elevated pulmonary artery pressure, (3) eventually produces desensitization phenomena when used chronically, and (4) cannot be effectively used in the presence of high levels of beta-adrenergic receptor blockade. The first three of these limitations can be overcome by combining Dobutamine with a Phosphodiesterase Inhibitor which results in additive effects on myocardial performance, substantial reductions in pulmonary wedge and pulmonary artery pressure.

E p i n e p h r i n e (A d r e n a l i n e)

Epinephrine compared with other inotropic drugs, causes the greatest increase in the rate of energy usage and myocardial oxygen demand. This increase in oxygen need may be detrimental to the failing heart. Epinephrine also causes vasoconstriction and bronchodilation. Epinephrine cannot be given orally. Absorption is more rapid after IM versus SC administration. Epinephrine is available in several preparations and is effective after IV, pulmonary, and nasal administration.

However, because of the decreased efficiency of cardiac work, epinephrine is not generally used as a positive inotrope. Epinephrine is an excellent positive inotropic agent in the denervated, transplanted heart because neuronal reuptake is no longer a factor. The dose of epinephrine usually ranges from 0.05 to 0.50 $\mu\text{g}/\text{kg}/\text{min}$. It is used in emergency therapy of cardiac arrest and anaphylactic shock. Ventricular arrhythmias can be expected.

Norepinephrine (Noradrenaline)

Norepinephrine is a mild (10 to 30 fold) β_1 vs. β_2 receptor-selective agonist with relatively high affinity for α_1 receptors. This constellation of properties means that norepinephrine will be a powerful vasoconstrictor, but not a very powerful inotrope in hearts. Norepinephrine does not have any recommended uses in subjects with cardiac decompensation. Those who need peripheral vascular resistance support (such as in sepsis, iatrogenic overvasodilation, or brain injury) are served better by dopamine or dopamine plus phenylephrine administration.

Isoproterenol

Isoproterenol is a synthetic catecholamine. It is predominantly a chronotrope. Increases heart rate and cardiac output without any significant change in the blood pressure. Disproportionate increase in myocardial oxygen consumption. The BP may fall because of predominant Beta 2 receptor induced vasodilation. Tachycardia and the potential for other arrhythmias excludes its use in the cardiac patient. It is used for therapy of bradyarrhythmias or AV block. As a therapeutic inotrope, isoproterenol has only one indication—postoperatively after heart transplantation. Isoproterenol is

useful in this setting because an increase in heart rate is not a problem in the presence of normal coronary arteries, and the chronotropic stimulation is useful in the newly transplanted heart, which often has a sluggish sinus node mechanism. The pulmonary vasodilator properties of isoproterenol are also useful in this setting, where pulmonary artery pressure and pulmonary vascular resistance are usually elevated. The dose of isoproterenol ranges from 0.005 to 0.05 mcg/kg/min.

Phenylephrine

This is a pure alpha-agonist with no beta-agonist activity. Even though alpha₁ receptors can mediate a small inotropic response in the human heart, phenylephrine should be used only to increase systemic vascular resistance in settings in which dopamine is not effective. The usual dose of phenylephrine ranges from 0.3 to 3 µg/kg/min.

Calcium

Calcium is also a positive inotrope but must be given as a slow IV injection or infusion. Calcium must be administered carefully because it can cause cardiac rigor and standstill at high doses. The gluconate form is preferred to calcium chloride. In general should be used only if there is significant hyperkalemia, hypocalcemia or poisoning from calcium channel blockers. When cardiogenic shock is profound, calcium is often added to an epinephrine infusion to produce synergistic increases in contractility and an increase in vascular tone. This combination, made by adding 1 gm of CaCl₂ to 250 ml of intravenous solution containing epinephrine and called "Epi-Cal," has never been subjected to a clinical trial and should be used in resuscitative settings only.

Phosphodiesterase Inhibitors

Milrinone is a Phosphodiesterase inhibitor (PDE) which increases levels of cyclic AMP. The cardiac effects are increased inotropy and improved diastolic relaxation (lusitropy). Also cause potent vasodilation with decrease in pre and afterload. This combined effect has been termed inodilation. Enoximone has less vasodilation. Combined use with catecholamines like Epinephrine and Norepinephrine may be necessary. No significant increase in heart rate or myocardial oxygen consumption. Milrinone: Loading 50 mcg/kg and maintenance of 0.5 mcg/kg/min.

Doses

All used intravenously (IV).

Steady state reached in 5-10 minutes.

Adrenaline, Noradrenaline and Isoprenaline all have side chains which is associated with 100 times greater potency than dopamine or dobutamine.

All catecholamines have very short half lives. Mainly due to reuptake by the tissues and degradation in liver and lung.

Epinephrine (Adrenaline): 1-70 mcg/min.

For CPR given as bolus of 1mg and infusion of 10-100 mcg/kg/min.

In asystole an IV dose of 5 mg has been used.

For anaphylaxis a dose of 0.3-1.0 mg (often 0.5 mg) given SC or IM.

Norepinephrine: 1-70 mcg/min.

Dobutamine: 2 - 20 mcg / kg / min (100 - 2000 mcg / min) .

Dobutamine infusions are initiated at 2 to 3 mcg/kg/min and are titrated upward according to the patient's hemodynamic response (usually not higher than 20 mcg/kg/min).

D o p a m i n e : 1 - 20 mcg / kg / min (100 - 2000 mcg / min) .

Isoproterenol: Range 0.005 to 0.05 mcg /kg/min.

Phenylephrine: Range from 0.3 to 3 mcg/kg/min.

M i l r i n o n e : L o a d i n g 50 mcg / kg
a n d m a i n t e n a n c e o f
0 . 5 mcg / kg / min .

C h e c k Y o u r P r o g r e s s 2

- 1) What are the two main types of adrenergic receptors?

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- 2) Name two naturally occurring adrenergic stimulators.

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- 3) Give any two effects of Beta adrenergic stimulators.

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- 4) Name one phosphodiesterase inhibitor and what do they do to the level of cyclic AMP.

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5.4 ARRHYTHMIC EMERGENCIES

Asystole

Asystole is defined as the complete absence of cardiac electrical activity. Usually this represents extensive myocardial ischaemia due to prolonged periods of inadequate myocardial perfusion with a very grim prognosis.

- 1) Determine unresponsiveness, absence of breathing and pulselessness.
- 2) Maintain an open airway, remove secretions, vomitus, and initiate CPR with supplemental high concentration of oxygen.
- 3) Continually assess Level of Consciousness, ABCs and Vital Signs.

- 4) Every effort should be made to determine the possible causes of asystole in the patient. Obtain appropriate history related to event, including recent and Past Medical History, Medications, Drug Allergies and Substance Abuse including possible ingestion or overdose of medications, specifically calcium channel blockers, beta-blockers and/or digoxin preparations.
- Epinephrine (1:10,000) 1 mg IV push** every 3-5 minutes. Epinephrine may be given via Endotracheal Tube *if IV not yet established*. (**2-2.5 mg of Epinephrine 1:1,000** is preferred (ET), every 3-5 minutes)
 - Atropine 1 mg IV push** every 3-5 minutes to a total of 0.04 mg/kg. Atropine may be given via Endotracheal Tube if IV not yet established (**2.0 mg of Atropine via ETT** is preferred; maximum dose 0.08 mg/kg).
 - Sodium Bicarbonate** 1 mEq/kg IV Push/if known pre-existing hyperkalemia or known pre-existing bicarbonate-responsive acidosis or if overdose with tricyclic antidepressants.

Atrial Fibrillation

Atrial fibrillation is a totally chaotic activity of the atrial muscle fibers manifested by an irregularly irregular rate. In addition, since the atria are fibrillating, there is incomplete (or non-existent) emptying of these chambers and a loss of as much as 20 per cent of the cardiac output. The rate can be variable, itself a problem, but in addition the loss of the “atrial kick” may, in and of itself, result in hypotension or other signs of cardiovascular compromise.

- If the patient is hemodynamically unstable, Systolic blood pressure is unstable (less than 90): Synchronized cardioversion at 50 J, 100 J, 200 J, 300J, and 360 J. Check rhythm and pulse between each attempted cardioversion.
- If Cardioversion is warranted, consider administration of any of the following for sedation:
 - Valium: if patient < 70 kg: 2.5 mg SLOW IV Push, if patient > 70 kg: 5.0 mg SLOW IV Push *or*
 - Morphine Sulphate 5 mg – 10 mg SLOW IV Push

To Control a High Ventricular Rate

- IV beta blocker like Metoprolol 5mg
- IV Diltiazem HCL (Dilzem)
 - Initial bolus: 0.25 mg/kg SLOW IV PUSH over two (2) minutes.
 - If inadequate response after 15 minutes, re-bolus 0.35 mg/kg SLOW IV PUSH over two (2) minutes.
 - IV Infusion 10-15 mg/hr.
 - Note: 5 mg/hr may be appropriate starting infusion for some patients.
Contraindications: Wolff-Parkinson-White Syndrome, second or third degree heart block and sick sinus syndrome (except in the presence of a ventricular pace maker), severe hypotension or cardiogenic shock.
- IV Verapamil, unless contraindicated
 - Initial bolus: Verapamil 2.5 mg – 5 mg SLOW IV push. If inadequate response or after 15-30 minutes may re-bolus Verapamil at 5 mg-10 mg slow IV push.

Contraindications: As with Diltiazem above

Bradyarrhythmias

The following can all result in Bradycardia:

Vagal stimulation, intrinsic cardiac conduction system disease, acute myocardial infarction resulting in heart rates from sinus bradycardia to complete, “third degree” heart blocks.

If pulse < 60 and patient is symptomatic, place patient supine and elevate legs.

If patient is symptomatic

- Atropine sulfate 0.5 mg to 1.0 mg IV Push or ET every three (3) to five (5) minutes up to total dose 0.04 mg/kg. If administered via ET, 2.0 mg, followed by 2.0 ml of Normal Saline Solution.
- Temporary pacing if indicated.

The following may be ordered:

- a) Additional Fluid Boluses of Normal Saline as indicated.
- b) Dopamine 5mcg/kg to 20mcg/kg per minute.
- c) Epinephrine Infusion (mix 1 mg in 250 cc Normal Saline) Administer 2mcg to 10mcg per minute.
- d) Glucagon 1.0 to 5.0 mg IM, SC or IV for suspected beta blocker toxicity.
- e) Calcium Chloride 10 per cent 2 - 4 mg/kg IV slowly over five (5) minutes for suspected calcium channel blocker toxicity.

Supraventricular Tachycardia

Supraventricular Tachycardia (SVT) applies to all tachyarrhythmias in which the pacemaker site is originating above the ventricles. Examples of these are Paroxysmal Supraventricular Tachycardia (PSVT), Atrial Fibrillation, Atrial Flutter with a rapid ventricular response, and Junctional Tachycardia with a rapid ventricular response.

Generally these groups of tachycardias identify narrow complex rhythm disturbances and should not be confused with Sinus Tachycardia. Narrow complex SVT with heart rates greater than 150/minute requires immediate intervention under most circumstances.

Vagal maneuvers should be started to terminate or modify AV conduction. These consist of the Valsalva maneuver, carotid massage, eyeball pressure, induced vomiting, etc.

If these fail, IV antiarrhythmic drugs should be used in hemodynamically stable patients.

Administer **Adenosine 6 mg** rapid IV push over 1-3 seconds. If previous 6 mg dose failed to resolve rhythm disturbance. Administer **Adenosine 12 mg** rapid IV push over 1-3 seconds. Repeat **Adenosine 12 mg** rapid IV push over 1-3 seconds if previous doses failed to resolve rhythm disturbance.

Note: Follow all Adenosine with a 20 ml normal saline bolus and elevate extremity.

Diltiazem or Verapamil as Under Section on Atrial Fibrillation.

Unstable Patients

Most patients tolerate SVT well, however, some patients may require emergent treatment. Emergent treatment should be administered when the SVT results in an unstable condition. Signs and symptoms may include: angina, shortness of breath, decreased level of consciousness, systolic blood pressure less than 90, shock, pulmonary congestion, and acute myocardial infarction.

Synchronized cardioversion is done at 50 J, 100 J, 200 J, 300 J, and 360 J. Check rhythm and pulse between each attempted cardioversion.

Ventricular Fibrillation/Pulseless Ventricular Tachycardia

The need for early defibrillation is clear and should have the highest priority.

Since these patients will all be in cardiopulmonary arrest, adjunctive equipment should not divert attention or effort from Basic Cardiac Life Support (BCLS) resuscitative measures, early defibrillation and Advanced Cardiac Life Support (ACLS).

Ventricular Tachycardia with Pulses

Ventricular tachycardia represents a grave, life threatening situation in which the patient requires immediate treatment.

The diagnosis is suggested anytime three or more premature ventricular beats occur in succession. With ventricular tachycardia, cardiac output may drop dramatically or be absent altogether and progress into ventricular fibrillation. Ventricular tachycardia, the patient is considered to be either:

- 1) Pulseless: in essence in Cardiopulmonary Arrest.
- 2) Stable: presents with pulses, conscious, without chest pain, Systolic blood pressure greater than 90.
- 3) Unstable: presents with pulses, but is symptomatic: chest pain, palpitations, shortness of breath (SOB), possible signs and symptoms of congestive heart failure (CHF), hypotension (systolic blood pressure less than 90), decreasing level of consciousness (LOC) or unresponsive.

If the patient is haemodynamically stable, consider pharmacological therapy, for example

- Amiodarone IV
- Lignocaine IV
- Some even favour IV sotalol or procainamide
- A class IC agent IV (??)

Comment: There is much controversy about the best drug for VT. Often the best 'drug' is in fact electricity, as sustained VT often deteriorates into VF if left unmanaged. Recent literature increasingly favours amiodarone, in a dose of 2.5 to 5mg/kg IV over approximately 10 minutes, although some might argue in favour of higher doses or more rapid administration. Lignocaine is falling into disfavour, and class 1C agents are less-and-less favoured because of their negative inotropic activity.

Modalities

- 1) DC current, i.e., Electrical Cardioversion.

- 2) Drugs, i.e, Pharmacological Cardioversion.
- 3) Anti Tachycardia pacing.

If patient is haemodynamically unstable, perform immediate unsynchronised DC countershock starting at 200 J.

Otherwise, if a patient is at any stage haemodynamically unstable or fails to respond to IV medication, then administer synchronised DC cardioversion, starting at 100 J, then 200 J, then maximum settings.

If synchronisation fails due to bizarre QRS morphology, then switch to asynchronous mode (with the attendant risk of ventricular fibrillation).

Check Your Progress 3

- 1) What is the commonest rhythm seen during cardiac arrest?

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- 2) What is the key treatment for success with this rhythm?

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- 3) Name a drug i.e. currently used during refractory VT/VF and its initial dosage.

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- 4) What do you do for a patient with atrial fibrillation who is hemodynamically unstable?

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- 5) How would you administer adenosine to a patient with supraventricular tachycardia.

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5.5 PULMONARY EDEMA

Occurs movement of liquid from the blood to the interstitial space and/or into the alveoli exceeds the return of liquid to the blood and its drainage through the lymphatics. This is a cardiac emergency. The causes and pathophysiology have been discussed in the section under heart failure.

Development of acute pulmonary edema is a terrifying experience with extreme breathlessness developing suddenly, and the patient becomes extremely anxious, coughs, and expectorates pink, frothy liquid, with a feeling of drowning. The patient sits upright, or may stand, exhibits air hunger, respiratory rate is elevated, the alae nasi are dilated, and there is inspiratory retraction of the intercostal spaces and supraclavicular fossae that reflects the large negative intrapleural pressures required for inspiration. The patient often grasps the sides of the bed to allow use of the accessory muscles of respiration. Respiration is noisy, with loud inspiratory and expiratory gurgling sounds that are often easily audible across the room. Sweating is profuse, and the skin is usually cold, ashen, and cyanotic, reflecting low cardiac output and increased sympathetic drive.

Auscultation reveals crepitations and occasionally rhonchi, which appear initially over the lung bases but then extend upward with worsening of the condition. An S3 gallop and loud pulmonic component of the second heart sound are frequently present.

Arterial pressure is usually elevated as a result of excitement and discomfort, which cause adrenergically mediated vasoconstriction. And this usually does not represent chronic systemic hypertension. Optic fundus examination may be useful in differentiating the two conditions.

Sometimes it may be difficult to differentiate between acute pulmonary edema and acute exacerbation of bronchial asthma. Some of the points that may be of clinical use in such a situation are given in the table below.

Table 5.1: Differentiation between Pulmonary Edema and Bronchial Asthma

Clinical Features	Cardiogenic	Bronchial Asthma
Previous similar episodes and awareness of diagnosis	May/may not be present	Usually present
Profuse sweating	Usually Present	Usually not present
Cyanosis	Frequently cyanotic	Unusual
Chest expansion and resonance	Dull to percussion, no hyper expansion	Hyper expansion and hyper expansion
Adventitious sounds and occasionally prominent	Mainly crepitations crepitations less	Rhonchi present and rhonchi

Management of Pulmonary Edema

Pulmonary edema is life-threatening condition and therefore treated as a medical emergency.

As is the case with chronic stable heart failure, identification and correction of any precipitating causes should be attempted. However, because of the acute nature of the problem, the initial management includes a number of additional nonspecific measures.

- 1) The patient should be in propped up position (provided the blood pressure is adequate) with the legs dangling along the side of the bed, if possible, which tends to reduce venous return.
- 2) 100 per cent O₂ should be administered to improve oxygenation. If patient is not maintaining oxygen saturation with nasal oxygen intubation and mechanical ventilation should be considered. This would increase intra-alveolar pressure, reduces transudation of fluid from the alveolar capillaries, and impedes venous return to the thorax, reducing pulmonary capillary pressure.
- 3) Morphine is the drug of choice. It is administered intravenously, in doses from 2 to 5 mg intravenously. It reduces anxiety, reduces adrenergic vasoconstrictor stimuli to the arteriolar and venous beds, and thereby helps to break a vicious cycle. An antiemetic is usually given along with morphine to reduce chance of vomiting.
- 4) Intravenous loop diuretics produce rapid diuresis, reduce circulating blood volume and hasten the relief from pulmonary edema. Furosemide when administered exerts a venodilator action, reducing venous return. This helps in improving pulmonary edema even before the diuresis is initiated. Given IV in a dose of 40 mg.
- 5) Afterload reducing agents, e.g., IV sodium nitroprusside at 20 to 30 µg/min in patients with systolic BP above 100 mmHg.
- 6) Inotropic support should be provided by dopamine or dobutamine where necessary.
- 7) Sometimes, aminophylline (theophylline ethylenediamine), 240 to 480 mg intravenously, is effective in diminishing bronchoconstriction, increasing renal blood flow and sodium excretion, and augmenting myocardial contractility.
- 8) Rotating tourniquets may be applied in an effort to reduce venous return.
- 9) In unresponsive severe pulmonary edema, endotracheal intubation and positive pressure ventilation may be required.

Once the patient has been stabilized and underlying cause determined, treatment directed at correcting/improving the cause.

Check Your Progress 4

Mention any two drugs used in acute pulmonary edema and the route and dose.

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5.6 HYPERTENSIVE EMERGENCIES

Hypertensive emergencies are acute, severe elevations in blood pressure accompanied by progressive target organ dysfunction such as myocardial or cerebral ischaemia/infarction, pulmonary edema, or renal failure. The patient is critically ill with a blood pressure often greater than 220/140 mm Hg, headaches, confusion, blurred vision, nausea and vomiting, seizures, grade III or IV hypertensive retinopathy, heart failure, and oliguria. Hypertensive emergencies require immediate intensive care admission for intravenous therapy. The common hypertensive cardiac emergencies include acute aortic dissection, acute myocardial infarction and unstable angina and eclampsia. Hypertensive encephalopathy is characterized by severe hypertensive retinopathy (retinal hemorrhages and exudates, with or without papilledema). A new focal neurologic deficit suggests a stroke-in-evolution.

Hypertensive urgencies are acute, severe elevations in blood pressure without progressive target organ dysfunction. Chronically elevated blood pressure, even when severe, does not necessitate urgent treatment. These often can be managed with oral medications and appropriate outpatient follow-up in 24 to 72 hours.

The goal of parenteral therapy is to achieve a controlled and gradual lowering of blood pressure to about 170/110 mmHg. Blood pressure can be reduced to a more normal value over the next 48 hours. However, aortic dissection requires a much more rapid normalization of blood pressure.

Parenteral Agents Available for Hypertensive Emergencies

- 1) Sodium Nitroprusside: 0.25-10 mcg/kg/minute IV. The action is immediate.
- 2) Nitroglycerin 5-100mcg/minute IV. Action in 2 –5 minute. Tachycardia may ensue. Do not use with aortic dissection.
- 3) Hydralazine 5-10 mg as IV bolus or 10-40 mg IM and repeat q 4-6 hour. At times unpredictable.
- 4) Labetalol 20-80 mg as a slow IV injection and effect takes about 5-10 minute.
- 5) Metoprolol 5 mg IV q 10 in (3 doses)
- 6) Esmolol 500 mcg/kg IV over 3 minute. Then 25-100 mcg. Effect in 1-5 minute
- 7) Phentolamine 5-10 mg IV bolus q 5-15 minute. Effect in 1-2 minute

Parenteral Treatment of Specific Hypertensive Emergencies

- 1) Myocardial ischaemia/infarction: β -Blocker + nitroprusside.
- 2) Hypertensive encephalopathy: Nitroprusside.
- 3) Aortic dissection: β -Blocker + nitroprusside to lower SBP to < 120 mm Hg in 20 minutes.
- 4) Heart failure (acute pulmonary edema): Furosemide + nitroprusside.
- 5) Preeclampsia/eclampsia: $MgSO_4$ for seizures and methyldopa + hydralazine to lower diastolic pressure below 90 mm Hg. (oral labetalol is a second-line drug before cesarean section).

Oral Medications for Hypertensive Urgencies

- 1) To manage the patient during the interim period, labetalol is effective in a dose of 200 to 300 mg, which can be repeated in 2 to 3 hours and then prescribed in twice-daily dosing.
- 2) Captopril, the short-acting ACE inhibitor, lowers blood pressure within 15 to 30 minutes of oral dosing. A small test dose of 6.25 mg should be used to avoid an excessive fall in blood pressure in hypovolemic patients; then, the full oral dose is 25 mg, which can be repeated in 1 to 2 hours and prescribed as 25–75 mg twice daily.

Check Your Progress 5

What are the features of hypertensive encephalopathy and mention 2 drugs that can be used IV.

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5.7 CARDIAC TAMPONADE

Cardiac tamponade results from a critical rise in intrapericardial pressure that affects cardiac filling.

Acute cardiac tamponade results from an accumulation of fluid or blood in the pericardial space (cavity). The increased intrapericardial pressure is transmitted to the cardiac chambers and cardiac filling is compromised. The speed of accumulation and the quantity are both important in deciding if tamponade ensues.

The causes and diagnosis of tamponade have been discussed in the section on pericarditis. Common causes presenting as an emergency are malignant effusion, viral or idiopathic pericarditis, renal failure and hemopericardium.

The clinical features are dyspnea, sinus tachycardia, elevated JVP with a further rise during inspiration (Kussmaul sign), pulsus paradoxus and a fall in BP and shock in some cases. Echocardiography is the most important investigation. (See section on Pericarditis).

Pericardial Aspiration

- Obtain an echocardiogram before the pericardiocentesis procedure.
- Perform pericardiocentesis on patients with tamponade and life threatening hemodynamic stability without waiting for any test result.
- Non emergency aspirations should be carried out at a centre where additional investigations can be done on the fluid or a pericardial biopsy performed if necessary.
- Obtain basic blood work from these patients. If time permits, correct any clotting abnormality. Administer fresh frozen plasma if the effusion occurs from thrombolytics, and do not perform pericardiocentesis unless the tamponade is life threatening. Discontinue administration of heparin if the condition is caused by anticoagulation. Pericardiocentesis should be delayed until the clotting profile is normal or protamine is administered, unless the patient is unstable.

The pericardiocentesis procedure is as follows:

- Ensure that the patient is sitting at 30-45° head elevation, which increases pooling of fluid toward the inferior and anterior surface, thus maximizing fluid drainage.
- Select a site i.e. closest to the pericardial space, avoiding vital structures, such as the internal mammary artery, lungs, myocardium, liver, and vascular bundle at the inferior margin of each rib.
- Shave the skin carefully to avoid any trauma. Anesthetize the local site with lidocaine (1-2 per cent). Make a small incision (approximately 5 mm) to decrease the resistance during needle insertion. Separate the subcutaneous tissue with mosquito grasping forceps.
- Connect the needle with a 3-way stopcock. Ensure that the syringe with 1 per cent lidocaine is connected to the 3-way stopcock on the opposite side of the needle connection. Connect the transducer on the side of the 3-way stopcock. Attach a sterile ECG recorder to the metal part of the needle if available.
- Insert the needle through the subxiphoid approach on the left side under fluoroscopy and direct towards left shoulder. Advance the needle and syringe until the needle tip is posterior to the rib cage. The needle should be advanced toward the shoulder at an angle 15-20° from

the abdominal wall. While advancing the needle toward the pericardial space, aspirate the syringe and inject lidocaine for a better analgesic effect. Continue to advance the needle until fluid is aspirated in the syringe or the ECG monitor shows ST elevation.

- Withdraw the needle slowly with negative pressure on the syringe if the ECG shows ST elevation after clearing the needle with lidocaine. Reinsert the needle in a different direction very slowly until fluid is aspirated in the syringe.
- When the needle tip is inside the pericardial space, a soft floppy-tip guidewire is passed through the needle. Wrap this guidewire around the heart. Remove the needle, and insert a soft catheter with multiple side holes like a pigtail catheter over this wire. Remove the guidewire. Connect the catheter hub with the transducer and syringe with a 3-way stopcock. Place the dressing, and secure the catheter to prevent displacement. Ensure that the catheter is flushed with 1-2 ml of fluid to prevent blockage.
- In a real emergency with hemodynamic collapse aspirate through the needle if a catheter is not available.
- The pericardial catheter can be left in the space for 24 hours with continuous closed drainage occurring, using gravity to increase drainage. The catheter should be removed after 24 hours, if possible, because it increases the chances of infection in the pericardial space. However, keeping the catheter in the pericardial space often is necessary to maintain drainage for longer periods. Negative suction should not be used to maximize the drainage.
- CT-guided pericardiocentesis is a new approach i.e. indicated specifically for patients in whom ultrasound-guided or radiograph-guided pericardiocentesis is unsuccessful.
- Pericardiocentesis with intrapericardial sclerotherapy also is effective in treating patients with malignant pericardial effusion.

Check Your Progress 6

What are the clinical features of cardiac tamponade and what is the most important investigation?

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5.8 PULMONARY EMBOLISM

Acute unexplained dyspnea is often the presenting symptom of PE. The most frequently encountered physical finding is tachypnea, with a respiratory rate at rest greater than 16 per minute. Sinus tachycardia often accompanies dyspnea and can be a clue to the diagnosis of PE. Unexplained pallor, fatigue, apprehension can be other associated symptoms. The diagnosis of PE in this group of patients is many a times missed and requires a high index of suspicion in potential clinical situation.

Pulmonary infarction or hemorrhage is an uncommon presentation of PE. These patients present with pleural pain, haemoptysis, dyspnea, cough and fever. These cases can often be confused as having chest infection, pneumonia or other form of lung disease. Crepitations in lungs and pleural rub are often present in this group of patients.

Acute right ventricular failure can be a presentation in patients with massive occlusion of pulmonary arterial tree. This group usually presents as an emergency. These patients present either with chest pain, syncope, acute dyspnea or in a state of shock with all its signs (pallor,

tachypnea, tachycardia, cyanosis, hypotension, gallop rhythm and other features of circulatory failure). This group of patients may die suddenly, develop refractory shock or improve if provided with necessary medical assistance. In this group of patients the initial differential diagnosis may be a cardiogenic shock secondary to myocardial infarction.

Differential diagnosis of PE is broad and covers a spectrum from life-threatening disease such as acute myocardial infarction to innocuous anxiety states.

Investigations

1) ***X-ray chest***

The chest x-ray is a useful investigation but may not provide a diagnostic information. The x-ray is often helpful in excluding lung disease like pneumonia as a cause of acute dyspnea.

The chest x-ray can be helpful in establishing the diagnosis of pulmonary infarction by demonstrating pleural effusion, an infiltrate, atelectasis, or an elevated hemidiaphragm. However, it should be emphasized that a normal chest x-ray does not rule out diagnosis of PE.

2) ***Electrocardiogram (ECG)***

The ECG is a simple and useful test but may not provide diagnostic information in each and every case. A normal electrocardiogram does not exclude PE.

The presence of right ventricular abnormality, as demonstrated by S1Q 3T3, right bundle branch block, right axis deviation, or atrial abnormality, are seen in only 26 per cent of patients with PE. Atrial fibrillation, supraventricular arrhythmias and ST-T changes in anterior and inferior leads are also seen.

3) ***Arterial Blood Gases (ABG)***

Analysis of ABG is helpful in supporting the diagnosis of PE. Finding of low PO_2 , low CO_2 and respiratory alkalosis are pointers to the diagnosis of PE in a patient with risk factors for its development. It should be remembered that conditions which mimic PE like pneumonia, severe chest infections or chronic obstructive lung disease (COPD) can also cause low PO_2 .

Pulse oxymetry is extremely insensitive in making a diagnosis of PE and can be normal in many patients.

4) ***Echocardiography***

Echocardiography is insensitive for the visualization of thrombi in pulmonary circulation but is a rapid, practical and sensitive technique for detection of right ventricular overload among patients with established and large PE. Echocardiographic detection of right ventricular dysfunction at the time of presentation with PE is useful for risk stratification and prognostication. For those patients in whom transthoracic imaging is unsatisfactory, transesophageal echocardiography may be useful.

5) ***Ventilation Perfusion Lung Scan (VQ scan)***

The VQ scan used to be considered an important investigation for many years. However, the availability of computed tomography (CT) has reduced its utility. At present if facilities

are available, CT is usually preferred for diagnosis. The use of VQ scan is reserved for patients in whom motion artifact or poor right heart function limit the quality of CT examination and those with contraindication to intravenous radiography contrast.

In pulmonary embolism, the ventilation is normal and perfusion in the affected segments is reduced and there is ventilation perfusion mismatch. Certain VQ scan readings are of substantial utility. A normal perfusion scan virtually excludes the diagnosis of PE. On the other hand, presence of multiple perfusion defects strongly favour a diagnosis of PE. The low and intermediate probability scans are non-diagnostic and need further investigation.

6) ***Pulmonary Angiography***

Standard contrast pulmonary angiography has been considered the gold standard for accurate in vivo diagnosis or exclusion of PE. With availability of digital subtraction angiography, nonionic contrast media, improved techniques and experience, it can be performed expeditiously and safely in most patients. Angiography is most useful when a diagnostic dilemma persists despite the use of non-invasive tests. This situation is most common when the diagnostic test results are negative or ambiguous in the presence of high clinical suspicion for PE. Pulmonary angiography is obviously also required when interventions are planned such as suction catheter embolectomy, mechanical clot fragmentation, or catheter-directed thrombolysis.

Diagnosis of PE is entertained by demonstrating filling defects (clots), abrupt cut off or tapering of pulmonary arteries and by ancillary findings such as webs, dilated main and branch pulmonary arteries. Pulmonary artery (PA) pressure should also be recorded during angiography. Elevation of PA pressure further supports the diagnosis of PE.

7) ***D-dimer Assay***

D-dimer is a blood test which can be rapidly performed and is utilized these days as a screening test for venous thromboembolism (deep venous thrombosis, or PE). D-dimer is a fibrin specific degradation product that detects cross-linked fibrin resulting from endogenous fibrinolysis. In the presence of an acute thrombo-embolic event (like DVT or PE) the simultaneous activation of coagulation factors and fibrinolytic enzymes leads to increased concentration of D-dimer.

Normal values of this protein have high negative predictive value, i.e. a patient whose D-dimer is less than 500 micrograms is unlikely to have PE. On the other hand, elevated D-dimer levels do not necessarily indicate PE and can be elevated in DVT, infections, inflammation, necrosis, trauma, and cancer, etc.

8) ***Computed Tomographic (CT) Pulmonary Angiography (PA)***

CTPA has gained acceptance as a first – line imaging study in cases of suspected acute PE, replacing traditional V/Q scintigraphy at many institutions. CTPA has also reduced the need for invasive pulmonary angiography.

CTPA provides visualization of the pulmonary arterial system in the axial plane, and multiplanar and three dimensional reconstructions can be generated from raw data to enhance diagnostic accuracy. The cardinal sign of acute PE on CTPA is an intravascular filling defect in a pulmonary artery that partially or completely occludes the vessel and is often associated with increased diameter of the affected vessel. The most specific sign of acute PE is a filling defect that forms acute angles with the vessel wall.

Even spiral CT can diagnose PE. The other advantage of spiral CT technique is its ability to diagnose or exclude other lung disorders which can mimic PE. A reliable diagnosis of lung consolidation, collapse, effusion, abscess, tumors and pneumothorax can be made by CT scan technique.

9) ***Magnetic Resonance (MR) Pulmonary Angiography***

This technique has developed significantly in the last 10 years. However, there are still several problems including acquisition of good images in breathless patients. At the present time CTPA is considered superior to MR angiography.

Management

1) ***General***

Depending on the clinical presentation the patient needs to be treated either in intensive care unit, hospital wards or advanced referral hospitals. Those patients of PE who present with marked symptoms or are hemodynamically unstable (low blood pressure, cyanosis or shock) need to be treated in an intensive care unit. In sick patients, the aim of treatment is to provide symptomatic relief, maintain oxygenation, ventilation and provide inotropic support. Patients who are hypoxic require oxygen and may need ventilatory support.

Dobutamine, a beta adrenergic agonist with positive inotropic and pulmonary vasodilating effects should be considered a first line agent to treat right sided heart failure and cardiogenic shock. The use of diuretics, intravenous fluids and dopamine should be individualized. Sedation should be avoided if patient is showing arterial hypoxemia.

2) ***Anticoagulation***

Anticoagulant therapy has been the mainstay of treatment for VTE. Treatment of patients with uncomplicated PE or DVT involves similar anticoagulant regimens, in part because asymptomatic PE occurs frequently in patients with symptomatic proximal DVT, and vice versa. Treatment of VTE involves initiation of anticoagulant therapy with either UFH or LMWH and considering long term initiation of oral anticoagulation (Warfarin therapy).

The utility of UFH or LMWH in treatment of VTE have been documented in large number of studies. Both these heparins are effective, safe, provide symptomatic benefit and reduce the mortality and morbidity due to this disease. The incidence of PE, its recurrence and the long term sequelae of this illness are reduced by use of heparin. UFH has been in clinical use for over 100 years, is safe and comparatively cheap. LMWH has become popular during the last decade and is gradually replacing the UFH. The primary difference between these two heparins relates to their pharmacological properties and cost. The main side effects of both the heparins is minor or major bleeding. The treatment by either heparin is usually given for 5 to 7 days. Oral anticoagulation is started either simultaneously or soon after depending on patient profile. Before initiating anticoagulation therapy, proper history should be obtained to rule out any history of bleeding (piles, peptic ulcer, hemorrhagic stroke) or related disorders.

Monitoring of the activated partial thromboplastin time (aPTT) is needed and therapeutic range of aPTT ratio (patient/control) of 1.5 to 2.5 is generally recommended. The UFH can be administered intravenously (IV) by infusion or by subcutaneous (SC) route. However, the bioavailability of SC UFH is less than that of IV UFH and larger initial doses of SC heparin are needed to achieve a therapeutic anticoagulant effect. Due to this, intravenous infusion is preferred.

Oral anticoagulation therapy using warfarin or any other agent is advocated in majority of patients for variable duration. The duration of long term anticoagulation is to be individualized depending on risk factors. For patients with VTE associated with a major transient risk factor such as recent surgery, anticoagulation therapy is usually recommended for 3 months. For patients with unprovoked VTE, stopping anticoagulant therapy after 6 or more months of treatment is associated with a high risk of recurrent VTE and justifies long term anticoagulation for such patients. The argument favouring long term therapy is stronger if the unprovoked episode was PE; if a second or subsequent episode of unprovoked VTE occurs; or any hypercoagulable state is diagnosed. Regular monitoring of blood test called as prothrombin time and International Normalized Ratio (INR) is mandatory while administering oral anticoagulation. An INR of 2 to 2.5 is generally recommended. Inferior Vena Cava (IVC) filters are advocated to prevent further episodes of VTE in selected patients and particularly in those with contraindication to long term anticoagulation.

There are newer direct thrombin inhibitors agents like hirudin, hirulog or oral ximelagatran which are investigational at present and may become available in near future as substitute to existing anticoagulants.

3) ***Thrombolysis***

Systemic IV infusion of thrombolytic agents have been proven superior to anticoagulation for treatment of PE. Compared with heparin in hemodynamically stable patients with large PE, systemic thrombolytic therapy reduced mortality (11 per cent versus 4.7 per cent) and recurrent PE (18.7 per cent versus 7.7 per cent, $P = 0.016$) but was associated with higher rates of bleeding complications. In view of the higher incidence of bleeding thrombolysis is not indicated in all patients with large PE.

The use of thrombolysis should be reserved for following category of patients:

- A) Patients with massive PE presenting as a life threatening emergency.
- B) Patients with sub-massive PE and demonstration of right ventricular dysfunction on echocardiography.
- C) Patients who develop recurrent PE despite treatment with heparin. In some centers thrombolytic agents are directly injected into pulmonary arteries (catheter directed thrombolysis) to facilitate clot lysis.

4) ***Newer interventional and surgical treatment***

There are patients who fail to improve despite all available treatment. For these patients interventional (catheter directed thrombolysis, clot fragmentation or thrombus aspiration) techniques used in catheterization laboratories or surgical techniques such as embolectomy are indicated. These techniques are available in some advanced tertiary care hospitals.

The indications for use of these interventions are: 1) Persistent arterial hypotension (systolic blood pressure < 90 mmHg or a rapid decrease of > 40 mmHg). 2) Systemic hypoperfusion and hypoxemia. 3) Need for cardiopulmonary resuscitation. 4) Severe right ventricular failure. 5) Contraindication to anticoagulation or thrombolysis.

Check Your Progress 7

What are the indications for thrombolysis in acute pulmonary embolism?

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5.9 CYANOTIC SPELLS

Hypercyanotic or Cyanotic spell is a paediatric emergency, which requires prompt recognition, and intervention to prevent disabling cerebro-vascular insults and to save lives. A cyanotic spell needs to be taken seriously not just because of the immediate threat but also because it indicates the need for early operation.

How to Recognize a Spell?

- Commonly seen below 2 years (peaks between 2 months to 6 months).
- Onset is usually spontaneous and unpredictable.
- Occurs more often in early morning, although can occur at anytime in the day.
- Infant cries incessantly, is irritable and often inconsolable.
- Tachypnea is prominent and a cardinal feature. Typically these infants have a pattern of deep and rapid breathing without significant subcostal recession.
- Cyanosis deepens as the spell progresses.
- Later gasping respiration and apnea ensues, which leads to limpness and ultimately anoxic seizures.
- Can last from minutes to hours.
- Auscultation reveals softening or disappearance of pulmonary ejection murmur.
- Occasional patient can have profound bradycardia.

Cardiac Lesions which Produce Spells

- Tetralogy of fallot.
- TOF with pulmonary atresia.
- Tricuspid atresia and PS.
- DORV with VSD and PS.
- D-TGA or L-TGA with VSD and PS.
- Single ventricle with PS.
- Atrioventricular septal defect with PS.

Mechanism of spells

Cyanotic spells are due to an acute decrease in pulmonary blood flow, increased right to left shunt and systemic desaturation due to various causes:

- Infundibular spasm due to increased circulating catecholamines as a result of effort of feeding or crying.
- Activation of mechano-receptors in RV due to decrease in systemic venous return or that in LV due to decreases in pulmonary blood flow, leading to peripheral vasodilatation and fall in systemic vascular resistance producing increased right-left shunt and systemic desaturation. Same mechanism can account for occasional episodes of bradycardia. (vaso-inhibitory response).
- Supraventricular tachycardia as a cause of spells in pulmonary atresia.

Management of Spells

- Check airway and start oxygen
If child is uncomfortable with mask or nasal cannula, deliver oxygen via tube whose end is held ½ - 1 inch away from nose. This corresponds to delivering 80 per cent oxygen.
- Knee – chest position.
- Sedate child with subcutaneous morphine 0.2 mg/kg/dose or i/m ketamine (3-5 mg/kg/dose).
- Obtain a reliable intravenous access.
- Soda –bicarbonate 1- 2 ml/kg given as 1:1 dilution or can be diluted in 10 ml/kg of isolyte P which is given bolus as the initial resuscitating fluid.
- Correct hypovolemia (10 ml/kg fluid bolus of isolyte P or dextrose normal saline).
- Keep the child warm.
- Correct anemia by packed cell transfusion. Hemoglobin levels < 12 gm/dl merit correction through a blood transfusion in children with cyanotic spells.
- Start beta –blockade. Beta blockade is fairly safe unless a specific contraindication like bronchial asthma or ventricular dysfunction exists. It should always be given with heart rate monitoring.

Medications and Dosages

- IV metoprolol 0.1 mg/kg, given slowly over 5 minute.
- Can repeat every 5-min for a maximum of 3 doses.
- Can be followed by infusion 1-2 mcg/kg/minute.
- Monitor saturation, heart rates and BP.
- Aim to keep heart rate below 100/minute.

Other Options

- I/V esmolol: 500 mcg/kg over 1 min as loading dose, 50 mcg/kg/min for 4 minutes; if desaturation persists without a significant decrease in heart rate the loading dose will need to be repeated and the infusion rate can be increased in 50 mcg/kg/min increments until 300

mcg/kg/min; this infusion should be maintained at the rate that produces the desired result. Esmolol is relatively expensive but has the advantage of being very short acting.

- I/V propranolol (0.1 mg/kg).

If desaturation persists and there is still no significant trend towards improvement despite maximum beta blockage.

- Start vasopressor infusion.

Methoxamine given i/v at dose of 0.1mg-0.2 mg/kg /dose or i/m (0.1- 0.4 mg/g/dose).

Phenylephrine: 5 ug/kg as bolus and then 1-4 ug/kg/min as infusion.

- If spells are persistent, consider paralysing the child, elective intubation and ventilation and plan for surgery, which can be corrective or palliative (BT shunt).
- If convulsions occur-consider IV diazepam 0.2 mg/kg or IV midazolam 0.1-0.2 mg/kg/dose, as slow push.

Appropriate and timely management of cyanotic spells can save lives and prevent CNS insults.

After a Spell: After a spell is successfully managed, a careful neurological examination is mandatory. In case of suspicion of neurologic insult during a spell, a CT scan is to be done to assess the presence and extent of cerebral infarcts.

- Initiate maximally tolerated beta-blockade (propranolol 0.5-1.5 mg/kg/dose 8 hourly or 6 hourly). The dose can be titrated by the heart rate response. Beta blockade may help improve resting saturation and can decrease frequency of spells.
- Do a detailed segmental analysis by 2D echo for complete diagnosis.
- Plan towards early corrective or palliative operation (depending on the age and anatomy).
- Continue therapeutic (if anemic) or prophylactic iron therapy (if not anemic).

Check Your Progress 8

Mention a drug and a maneuver used in a cyanotic spell.

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5.10 LET US SUM UP

As you have seen there are several types of cardiac emergencies. Some like acute coronary syndromes and dissection are discussed in other sections. Cardio Pulmonary Resuscitation is the key to the patient with cardiac arrest. Defibrillators should be made more freely available. The management of acute pulmonary edema, tamponade and cyanotic spell to name a few are immediate life saving situations.

5.11 ANSWERS TO CHECK YOUR PROGRESS

Check Your Progress 1

- 1) 200 Joules-200 Joules-360 Joules
- 2) 15 compressions and 2 ventilations
- 3) 100 compressions and 12 breaths per minute
- 4) Epinephrine is administered IV in increments of 1 mg diluted in 10 mls of normal saline (1:10,000). It can be given through the endotracheal tube as well in a 2-3 mg dose.

Check Your Progress 2

- 1) All catecholamines act directly on the Beta or Alpha adrenergic receptors.

Beta receptor stimulation results in increased ATP conversion to cyclic AMP.

Alpha 1 receptor activation acts independently of cyclic AMP and results in release of calcium and increased membrane permeability to calcium.

- 2) Epinephrine, Norepinephrine and Dopamine are all naturally occurring compounds.
- 3) All increase stroke volume, cardiac output and mean arterial pressure without much change in the heart rate.
- 4) Milrinone. Raises cyclic AMP level.

Check Your Progress 3

- 1) Ventricular fibrillation
- 2) Electrical defibrillation
- 3) Amiodarone may be considered in VT/VF i.e. refractory to shock. It may be given if the third shock is unsuccessful.

The dose is 300 mg IV in 20 mls 5 per cent dextrose over a period of 15 minutes. A further bolus of 150 mg may be given for refractory VT/VF, followed by an infusion, initially of 1.0 mg/mt for 6 hours, then reduced to 0.5 mg/mt, to a maximum dose of 2.0 G.

- 4) Synchronized cardioversion at 50 J, 100 J, 200 J, 300J, and 360 J. Check rhythm and pulse between each attempted cardioversion.
- 5) Administer Adenosine 6 mg rapid IV push over 1-3 seconds. If previous 6 mg dose failed to resolve rhythm disturbance: Administer Adenosine 12 mg rapid IV push over 1-3 seconds. Repeat Adenosine 12 mg rapid IV push over 1-3 seconds if previous doses failed to resolve rhythm disturbance. Note: Follow all Adenosine with a 20 ml normal saline bolus and elevate extremity.

Check Your Progress 4

- a) Morphine is administered intravenously, in doses from 2 to 5 mg intravenously.
- b) Intravenous loop diuretics like Furosemide when administered exerts a venodilator action, reducing venous return. Given IV in a dose of 40 mg.

Check Your Progress 5

Hypertensive encephalopathy is characterized by severe hypertensive retinopathy (retinal hemorrhages and exudates, with or without papilledema) along with marked hypertension, headache and blunted sensorium.

Check Your Progress 6

The clinical features are dyspnea, sinus tachycardia, elevated JVP with a further rise during inspiration (Kussmaul sign), pulsus paradoxus and a fall in BP and shock in some cases.

Echocardiography is the most important investigation.

Check Your Progress 7

In view of the higher incidence of bleeding thrombolysis is not indicated in all patients with large PE.

The use of thrombolysis should be reserved for following category of patients:

- a) Patients with massive PE presenting as a life threatening emergency.
- b) Patients with sub-massive PE and demonstration of right ventricular dysfunction on echocardiography.
- c) Patients who develop recurrent PE despite treatment with heparin. In some centers thrombolytic agents are directly injected into pulmonary arteries (catheter directed thrombolysis) to facilitate clot lysis.

Check Your Progress 8

Sedate child with subcutaneous morphine 0.2 mg/kg/dose or i/m ketamine (3-5 mg/kg/dose).

Knee chest position.