MANAGEMENT OF CARDiac EMERGENCIES

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INTRODUCTION

Emergencies are clinical conditions caused by life threatening disorders requiring quick and urgent intervention. Cardiac emergencies are part of a long list of emergencies that medical practitioners (general and specialist) are faced with daily. Cardiac emergencies will vary from one continent to the other or from one country to the other. This review discusses some of the cardiac emergencies in our environment and highlights others that are less common in our practice.

DISSECTING AORTIC ANEURYSM

All mechanisms that weaken the aortic wall; the aortic lamina media in particular, lead to higher wall stress which can increase aortic dilatation and aneurysm formation resulting in aortic dissection or rupture. The major inherited disorders include Marfan’s syndrome, Ehlers Danlos syndrome and other inherited familial forms of aortic thoracic aneurysm and dissection. Atherosclerosis is the main cause which leads to compromise of the integrity of the intima. The above leads to intimal rupture, most often at the edge of the plaque. Aortic dissection is classified (stagged) as Type A and Type B. Type A dissection involves the ascending aorta whereas Type B does not involve the ascending aorta. De Bakey’s classification subdivides the disorder into type I (entire ascending aorta), type II (ascending aorta) and type III (descending aorta).

There is a new classification proposed based on further studies that demonstrated intimal haemorrhage, intramural haematoma and aortic ulcer which may be signs of evolving dissection or dissection subtypes. Majority of true cases of dissection starts from the ascending aorta. Symptoms may include sudden pain severe central which radiates to the back or sometimes to the arm and neck, can be difficult to diagnose because of spinal vessels involvement, pain with cerebrovascular accident (CVA), pain with cardiac failure, pain with evidence of myocardial infarction etc.

Diagnosis

Back pain without ECG or enzyme changes of myocardial infarction and chest X-ray will show wide mediastinum. The diagnosis is almost at localizing the tear, extent of dissection, calcification, indications of emergency and aortic arch vessel and side branches involvement. Computed tomography (CT) scan and echocardiography will show double wall of the aorta. Echocardiography involves Trans thoracic (TEE) and Trans esophageal (TEE). Angiography alone or with Magnetic resonance imaging will confirm the diagnosis. An echocardiography machine with colour flow will also identify the area of dissection. Intravascular ultrasound has also been employed the diagnosis of aortic dissection. Tears, false lumen, leaks and calcification are identified using these imaging techniques though the sensitivity and

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specificity differs from one application to the other. The symptom that exceeds four days is regarded as chronic dissection.

Table 1: Three classifications highlighted above

<table>
<thead>
<tr>
<th>Stanford Classification</th>
<th>DeBakey Classification</th>
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<tbody>
<tr>
<td>Type A</td>
<td>Dissection of ascending/descending aorta</td>
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<tr>
<td>Type B</td>
<td>Dissection of descending aorta</td>
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Type I
- Dissection of entire aorta
- Dissection of ascending aorta

Class I
- Classical aortic dissection with an intimal flap between true and false lumen
- Medial disruption with formation of intramural hematoma/hemorrhage

Class 2
- Discrete/subtle dissection without hematoma, eccentric bulge at tear site
- Plaque rupture leading to aortic ulceration, penetrating aortic atherosclerosis ulcer with surrounding hematoma usually subadventitial hemorrhagic and intramural dissection

Emergency room steps

An immediate ECG is mandatory, this will help differentiate between myocardial infarction and dissection, diagnostic imaging highlighted above, chest x-ray might not be sufficient to make a diagnosis, other investigations include C-reactive protein, mild to moderate leucocytosis, slight rise in bilirubin and lactate dehydrogenase. It should noted that sometimes ECG might be abnormal when the dissection involves the coronary vessels. The most current specific indicator is raised smooth muscle myosin heavy chain. Therapeutic decision is base on diagnostic imaging to avoid wrong diagnosis, this entails admitting the patient to intensive care unit, replacement of fluid to haemodynamic deterioration, drugs administered in a separate venous line, unstable patients will require intubation and invasive blood pressure monitoring. The blood pressure should be controlled between 100-120 mmHg, TTE or preferably a TEE will identify pericardial tamponade so that pericardocentesis is done immediately and it does give clearer view of the ascending aorta without interference. The following are signs of severe dissection: severe pain, pulse loss, bleeding stopping, fluid extravasations into pericardium, pleural space and mediastinum, aortic regurgitation murmur, increase LV contraction and signs of myocardial ischemia. Surgery is the ultimate and it is in various forms depending on the complications found. It might require aortic valve replacement.
replacement, CABG and implantation of ascending aortic prosthesis. 

The risk factors associated with dissection of the aorta include hypertension, diabetes mellitus, smoking and any other cause of atherosclerosis. 

Hypertension should be controlled immediately with necessary drugs. Pregnancy is also associated increased risk of dissection. Stents should be taken towards prevention of aortic dehiscence by control or eliminating the factors.

ACUTE HEART FAILURE

This occurs mainly in the setting of hypertension, myocardial infarction, pulmonary thrombo-embolism, sudden failure of prosthetic valve etc. Acute heart failure may be in the setting of acute left or right failure and sometimes the whole heart involved. 

When there is excessive loss of cardiac muscle in myocardial infarction involving the left or right ventricle, the interventricular septum might rupture leading to ventricular septal defect or there could be acute valvular regurgitation. The valvular regurgitation is sequeal to papillary muscle or chordae rupture, producing sudden mitral or aortic regurgitation. Acute endocarditis might lead also lead to sudden regurgitation; acute left or right ventricular failure, which usually may take the form of diastolic dysfunction.

There is usually no peripheral fluid retention but in the case of left ventricular failure there will be severe pulmonary edema causing (described as cardiac asthma) the patient to present with severe shortness of breath. This presentation especially in the setting of hypertension might be mistaken for bronchial asthma. A third of fourth heart sound might be heard in the apex or left lower sternal edge.

Acute pulmonary edema is a common cardiac emergency, which might be the manifestation of acute heart failure. The patient is managed in cardiac position, high concentration of oxygen is administered by facemask, intravenous diuretics (loop diuretics), ventilators (nebulizers), amrinone and vasoconstriction when necessary. Cardiogenic shock results in less tissue perfusion. It is as a result of hypotension and low cardiac output. Because of poor tissue perfusion there will be oliguria, and cold extremities. To confirm a cardiogenic shock, a pulmonary wedge pressure will be normal and mortality is usually very high. Management involves administering 100% oxygen, infusion of fluids if pulmonary wedge pressure is below 18 mmHg; short acting ventilators (glyceryl nitrate), cardiac inotropes, mechanical assisted devices (intra aortic balloon pump).

Management of acute cardiac failure is usually directed to the primary cause, which sometimes might involve surgical intervention. The most common cause in our practice is hypertension, which requires immediate control. It is treated as one of the hypertensive emergencies. Acute cardiac failure in setting of hypertension will manifest as diastolic dysfunction. 

The role of nitrates in the treatment of pulmonary edema is very important apart from diuretics, morphine and oxygen. Pericardial effusion with tamponade, ventricular septal defect from myocardial infarction and sudden mitral or aortic regurgitation from valve failure will require surgical intervention.

CARDIAC TAMпонADE

It is as a result of accumulation of fluid in the pericardial sac that leads to compromise of cardiac function. 

The
fluid could be hemorrhage, suppurative or straw colour. Cardiac tamponade can be detected critically by using imaging techniques like echocardiography, CT scan and magnetic resonance imaging. The clinical signs includes, tachycardia, small volume pulse, pulsus paradoxicus, raised jugular venous pressure with a "Y" descant. Cardiac alox might not be pitiable and heart sounds are usually distant. Echocardiography can detect tamponade even before the manifestation of clinical signs. The echocardiographic signs include, diastolic collapse of right or left ventricular wall and sometimes right atrial wall collapse, apart from detecting the pericardial effusion. Other echocardiographic features include, "swinging heart", left atrial collapse in late diastole and early systole, and mitral E velocity reduction greater than 25% during inspiration. The distance of separation between parietal pericardium and the wall of the ventricle or atrium gives an idea of the severity of the effusion. Management is by pericardiocentesis, which is done under fluoroscopy in the cardiac catheterization laboratory to allow the operator to inventor where the catheter is placed and haemodynamic statues of the patient is also monitored including the ECG and blood pressure. In most third world country set up, pericardiocentesis is done in the open yards hardly with any ECG monitoring needless to talk about haemodynamic monitoring. Sometimes pericardial window might be required especially when there is re-accumulation of fluid. Pericardial effusion and tamponade is not uncommon in our practice, tuberculosis was the main culprit but with the HIV/AIDS emergence the trend seems to be changing. A third of our recent cases, yet to be published were related to HIV/AIDS.

CARDIAC ARREST
Cardiac or cardiopulmonary arrest has a big danger of causing permanent cerebral dysfunction. This is a common situation where medical students and all doctors must know how to manage it. The condition usually causes panic, it is therefore essential that basic procedures are known and followed carefully. Different countries have resuscitation councils that formulate procedures, which enable different health personnel, work together e.g. UK Resuscitation Council.

Basic life support
First step is to ensure whether the patient or victim is unconscious and whether there is pulse. This is done by shaking or shouting at the patient and also palpating the radial or carotid pulse (carotid is preferable). If there is no pulse, call for help, begin basic life support: A (air ways); B (breathing), and C (Circulation).

The airway should be cleared by removing mucus, blood clots and objects. Open thin airways by flexing the neck and extending the head ("sniffing the moving air position"). To establish whether the patient is breathing, look for rise and fall of the chest wall and abdomen. If there is no respiration begins expired air resuscitation. This is done with the head of the victim tilted backwards and the chin pulled forward. The resuscitator takes a deep breath and seals his lips around the victim's mouth and nose. At least four quick puffs are given but mechanical methods of Holger and Nelson. Shawter and Sylvester are thought to be completely useless because they result in the movement of less air than is required to fill the dead space. A firm thrust to the epigastric region can dislocate foreign body that is difficult to remove.
Circulation is achieved by external chest compression. The heel of one hand is placed over the lower part of the sternum and the heel of the second over the first with fingers interlocked. The hands are kept straight and the sternum is rhythmically depressed by 1 1/2 - 2 inches. Chest compression does not massage the heart but the thorax acts as a pump and the heart provides a system of one-way valves to ensure forward circulation. Respiration and compression is continued as follows:

a) Single resuscitator
   Compressions at the rate of 80pm with two respirations after 15 consecutive compressions.

b) Two resuscitators
   Continuous compressions at a rate of 60pm and one respiration given at every 5 compressions.

Mechanisms of sudden cardiac arrest
a) Ventricular fibrillation
b) Asystole
c) Electromechanical dissociation
   In the western experience ¾ of cardiac arrest is due to ventricular fibrillation. Statistics in Nigeria are difficult to come by, but ventricular fibrillation might also rank very high. The absence of well-established intensive care units or medical emergencies will make any available statistics suspect. Cardiac arrest, following pneumonia gives a cardiac or heart rhythm described as “regonal” characterized by inexorable slowing and widening of QRS complexes associated with falling blood pressure and cardiac output. It is a difficult arrhythmia to reverse and usually no attempt should be made because it is a result rather the cause of death.

Cardiac Support
In developed countries by the time simple or basic life support has been established more help would have arrived and advanced cardiac support can begin. These consist of ECG monitoring, endo-tracheal intubation and setting up of fluids. Immediate therapy includes defibrillation, oxygen and cardi-active drugs. The nature of cardiac arrest and the skill of the personnel will determine what type of drugs is to be administered. The ECG will detect malignant arrhythmias e.g. ventricular fibrillation, which must be defibrillated immediately.

Cardiac CPR for up to 2 minutes after 12am on day, do not interrupt for more than 10 seconds, except to defibrillate. If an intravenous line cannot be established, consider giving double dose adrenaline, lignocaine or atropine via an endo-tracheal tube. (Based on recommendation of UK Resuscitation Council 1989).

Management of sudden cardiac arrest
- Ventricular Fibrillation readily treated with defibrillation anti-arrhythmic drug and cardiac stimulants
- Asystole in more difficult to manage. This heart might respond to atropine or adrenaline. If there is any sign of electrocardiographic activity, emergency pacing should be used.
- Electromechanical dissociation. It is usually due to severe mechanical problem like pericardial effusion and tamponade or massive pulmonary embolism. It should be given urgent attention. Other causes include toxic levels of cardiac depressants drugs such as beta-blockers; antidote like adrenaline should be administered.

Sudden Cardiac Death
Natural death due to cardiac cause heralded by abrupt loss of consciousness within an hour of onset of acute symptoms, pre-existing heart disease...
Management of cardiac emergencies. Dambachia S. S.

ECG

Ventricular Fibrillation (VF)

Electro-mechanical Dissociation (QRS without palpable pulse)

Adrenaline 1 mg 1/1

Consider specific therapy
- Hypotension
- Pneumothorax
- Cardiac tamponade
- Pulmonary embolism

Consider calcium
Chloride (10 ml of 10%)
For
- Hyperkalaemia
- Hypokalaemia
- Calcium antagonist

Defibrillate 200 J
Defibrillate 200 J
Defibrillate 200 J
Adrenaline by 1/1
Defibrillate 360 J
Lignocaine 100 mg 1/1
- Consider different paddle position
- Other anti-arrhythmic drug.

Apparent asystole (iso-electric ECG)

Where VF Can be excluded

Where VF Cannot be excluded

Defibrillate 200 J
Defibrillate 200 J
Defibrillate 200 J
Adrenaline 1 mg 1/1
Atropine 2 mg 1/1
Consider pacing
If p wave or any other electrical activity present
disease may have been known to be present, but the time and mode of death are unexpected, and about half of this death is almost instantaneous. The clinical condition is usually used as a surrogate for a specific mechanism is involved. In the Western countries, ventricular fibrillation (VF) constitutes 75-80% in adults and the general incidence of sudden cardiac death is put at 0.36-1.26/1000. The causes include: cardiac arrhythmias (ventricular fibrillation or ventricular tachycardia), sudden pump failure (myocardial infarction), acute coronary obstruction (pulmonary embolism) cardiovascular rupture (dissecting aortic aneurysm or myocardial rupture) and Vasomotor collapse (pulmonary hypertension). Most deaths are due ventricular fibrillation and a few due severe bradyarrhythmias. The risk factors include atheromatous coronary artery disease (CAD), increasing age, male gender, family history of CAD, increased LDL cholesterol, hypertension, smoking and alcohol. It is probable that the most common risk in Nigeria is hypertension with atherosclerosis contributing less than half. Specific risk factors for the event are sudden cardiac death, increased heart rate and heavy alcohol consumption. The genetic basis for sudden cardiac death include, inherited genetic abnormality affecting key proteins of the heart e.g. Long QT syndrome, Brugada syndrome, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy, catecholaminergic polymorphic ventricular tachycardia and dilated cardiomyopathy.

Table 2. Recommendations for risk stratification for sudden cardiac death: myocardial infarction and heart failure

<table>
<thead>
<tr>
<th>Demographic variables</th>
<th>Recommendations</th>
<th>Level of evidence</th>
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<tbody>
<tr>
<td>LV ejection fraction</td>
<td>Class I</td>
<td>A</td>
</tr>
<tr>
<td>HR variability or baroreflex sensitivity</td>
<td>Class I</td>
<td>A</td>
</tr>
<tr>
<td>Left ventricular volume</td>
<td>Class II</td>
<td>A</td>
</tr>
<tr>
<td>Ventricular premature beats</td>
<td>Class IIa</td>
<td>A</td>
</tr>
<tr>
<td>Non sustained VT</td>
<td>Class IIa</td>
<td>A</td>
</tr>
<tr>
<td>Resting Heart rate</td>
<td>Class IIb</td>
<td>A</td>
</tr>
<tr>
<td>Late potential</td>
<td>Class IIb</td>
<td>A</td>
</tr>
<tr>
<td>QT interval</td>
<td>Class IIb</td>
<td>B</td>
</tr>
<tr>
<td>Electrophysiological study</td>
<td>Class IIb</td>
<td>B</td>
</tr>
<tr>
<td>T wave alternans</td>
<td>Class IIb</td>
<td>B</td>
</tr>
<tr>
<td>Heart turbulence</td>
<td>Class IIb</td>
<td>B</td>
</tr>
<tr>
<td>Patency of infract related artery</td>
<td>Class IIb</td>
<td>B</td>
</tr>
<tr>
<td>QT dispersion</td>
<td>Class III</td>
<td>B</td>
</tr>
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</table>

The act of defibrillation is very important, the placement of paddles and the amount of joules (energy) to be delivered is important. The sketch below will provide a working guide. Sometimes a temporary pacing is

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event the patient has a pulmonary embolism that requires direct thrombolysis. Intra-aortic balloon pumping is also a very important technique that is used to temporarily assist the failing left ventricle.

MYOCARDIAL INFARCTION

Ischemic heart disease though less common in Nigeria, current reports show increasing prevalence. It has the following risk factors: hyperlipidemia, smoking, hypertension, diabetes mellitus, insulin resistance etc. The following are marks of cardiovascular risk: total plasma homocysteine, fibrinogen, lipoprotein (Lp) a, thrombolytic function as ascertained by IIFPA or PAI-1 antigens, CRP, ICAM and IL6. Myocardial infarction is the most common cause of death in the UK and other developed countries. It is one of the acute syndromes associated with arrhythmoponies. It almost always occurs in patients with coronary atherosoma. It is usually due to fissuring or rupture of the atherosoma plaque leading to thrombosis formation, spasm of the artery, the thrombosis leads to stasis. The myocardium turns pale 6hrs after the event and at 24 hours it becomes necrotic.

The presentation is usually with chest pain, similar to angina, but usually occurring at rest but lasting for hours. The pain might be severe that it might lead to fear of death (angor animi). There might be restlessness, nausea, and vomiting and sudden collapse. Diabetics, hypertensives and elderly patients might present with silent or painless myocardial infarction. It might go unnoticed or present with hypotension and arrhythmias. Physical findings might include hypotension, assumed precordial pulsation because of bulging infarcted myocardium, forth heart sound, sinus tachycardia, raised venous pressure and pulmonary edema (crepitations). Muscle neurosis might lead to fever; lasting up to 7 days and a pericardial friction rub may develop. Myocardial infarction could evolve right or left ventricles; it could be evolve different regions of the heart e.g. inferior, anterior, inferior lateral, anterior or anterior lateral, septal etc. The worst prognostically subtype is anterior and anterior lateral. Investigations will include. The ST segment elevation and imaging findings are also used to determine the prognosis of the event.

1. Cardiac enzymes
   a) CK-MB, which peaks within 2hrs and back to minimal in 48hrs. It can also be produced by skeletal muscle and brain. The myocardial bound (MB) is an iso-enzyme that is specific to the heart. Carico version can also increase CK-MB.
   b) Aspartate aminotransferase (AST), it peaks 24-48hrs and falls by 72hrs. It is an unspecific because it is also released by damaged blood cells, kidney, liver and lungs.
   c) Lactate dehydrogenase. It peaks at 3-4 days and can last 10-14 days. It is also released by damaged skeletal muscle, red blood cells and liver. It has five iso-enzymes; the cardiac damage causes increase in LDH-1.

2. Electrocardiography
   It is valuable in most cases; the acute event is associated with ST elevation convex up to two days (3days); later the Q wave appears (> 1mm broad and 2mm or more than 25% of the R amplitude). Q wave may also be caused by abnormalities like bundle branch block. It is usually present in full thickness myocardial infarction. The ST segment elevation results from ischemia and injury. The Q wave does not develop in sub-endocardial infarction. The ECG tells which area of
the heart is involved. 3

3. Echocardiography

It is an investigation when performed will show area of infarction and confirm ventricular septal defect and mitral regurgitation from papillary muscle or chordae rupture. Echocardiography can also help in the evaluation left and ventricular function.

Other investigations include chest radiograph and blood biochemistry (Urea and Electrolytes, Blood sugar, lipid profile, unc acid and markers of cardiovascular risk where available.

Management

Patients are admitted into coronary care or intensive care or medical emergency unit immediately. ECG should be monitored continuously because patient develops might develop malignant arrhythmias and ECG also is used to monitor the revascularization therapy. Patient should have above investigations done including the ancillary one. An intravenous canula should be inserted for emergency intravenous medication. The routine medications include oxygen, nitroglycerin, aspirin, analgesics (like morphine or diamorphine), analogetics, anti-emetics and atropine when required. The anti-thrombotic therapy include; glycoprotein IIb/IIIb inhibitors, low molecular heparin and thrombolysis. Thrombolytic therapy or revascularization is very important 22. It is dependent on the time taken before the patient presented. They include IV drugs like streptokinase, urokinase and recombinant plasminogen activating factor (tPA). TPA is usually administered to patients with anterior myocardial infarction, which is worse prognosis. It is possible to salvage a less effective medical revascularization by making rescue angioplasty (PTCA), and sometimes angioplasty is applied as the first step (primary angioplasty). The usual maintenance therapy is administered at the same time and patients are adequately educated on the condition.

Surgical intervention in the form of coronary artery by pass graft (CABG), which is employed when there is recurrent pain even after PTCA and thrombolytic therapy, had been employed. The management of mechanical defects like mitral regurgitation, ventricular septal defect, left ventricular free wall rupture and left ventricular aneurysm would require surgical treatment. Malignant arrhythmias are handled using lidocaine, bretylium. Procardiamide and amiodarone.

PULMONARY EMBOLISM

Statistics in Nigeria is not readily available. The heart is affected by the haemodynamic changes in the lungs. The clinical symptoms of unexplained chest discomfort, shortness of breath and light-headedness should raise a suspicion of pulmonary embolism (PE). It accounts for hundreds of thousand hospitalizations in the United States 23. Cardiologist must provide expert treatment of Hemodynamically unstable patients with PE as well as those who have right ventricular failure with stable blood pressure and heart rate. The risk factors include genetic e.g. mutation in factor V gene, resistance to activated C, prothrombin gene mutation, mutation in protein C gene, protein C deficiency, anti-thrombin III deficiency, hyperhomocysteinemia and antiphospholipid antibodies. 27 The factors include surgery and immobilization/trauma, increasing age, cigarette smoking, obesity, systemic arterial hypertension, oral contraceptives, pregnancy, postpartum period, cancer (occult adenocarcinoma), cancer chemotherapy.
Diagnosis is based on symptoms, signs and investigations, but index of suspicion is paramount. The symptoms include shortness of breath and tachycardia and chest discomfort. The presence of severe dyspnoea, cyanosis and syncope indites life-threatening event. The presence of acute cor-pulmonale (right ventricular failure) is established by the features of distended neck veins, S3 gallop, RV F wave and tachycardia. Echocardio-graphic evidence of RV dilatation and hypokinesis or ECG features of acute cor-pulmonale defined by new S1Q3T3, new incomplete right bundle branch block (RBBB) or right ventricular ischaemia. Differential diagnosis includes pneumonia, cardiac failure, myocardial infarction and aortic dissection.

The clinical syndrome of pulmonary embolism is divided into massive, moderate to large, small to moderate, pulmonary infarction, paradoxical embolism and non-thrombotic embolism. Massive embolism syndrome puts the patient at shock stage, the thrombosis usually affects at least half of the pulmonary arterial system. And is present bilaterally. Systemic hypotension and dyspnoea are the major signs and subjects will gain from pressor agent support. The patient with moderate to large does have RV hypokinesis on echocardiography but normal arterial systemic pressures. The lung scan will indicate more than 30% non-pulmonary area and patients are at various stages of RV haemodynamic instability.

Diagnosis
A includes imaging and non-imaging methods. The imaging methods include chest x-ray usually abnormal in about 50% but its role in diagnosis is not well-defined, venous ultrasonography for DVT, nuclear venography for DVT, contrast phlebography for DVT, lung scan for PE, chest computerized tomography (for PE), magnetic resonance imaging (MRI) for PE, MRI pulmonary angiography for PE, LV and RV function. Echocardiography is increasingly its becoming important in the imaging diagnosis of PE and risk stratification, it helps define RV overload. Standard pulmonary angiography is the gold standard method for the diagnosis of PE.

The non-imaging methods include plasma D-dimer Elisa (most promising blood test for pulmonary embolism screening), arterial blood gases, and electrocardiograph and impedance plethysmography for DVT diagnosis.

Management
Rapid risk stratification is of paramount importance. PE patients have a wide spectrum of clinical presentations. It might involve only prevention of further thrombus by administration anti-coagulants, or insertion of filter at inferior vena cava. Clot dissolution or removal is done in patients who have haemodynamic instability, which is done by thrombolytics or embolectomy. Anticoagulation is done by administering low molecular heparin. Other support measures include oxygen, adequate pain relief with non-steroidal anti-inflammatory drugs. The presence of hypoxia and toxic features in subjects call for mechanical ventilation. Intravenous (dobutamine) should be administered to patients that are hypotensive and poor oxygen perfusion. Intravenous nitric oxide is given to patients with pulmonary hypertensive. The most common side effect of heparin encountered acutely is bleeding.

In acute cases emotional support is very important with an educational component. The anatomic extent of the event will determine treatment and the
Management of cardiac emergencies, Bashuachi S.S.  

presence of the underlying cardiopulmonary disease and detection of RV dysfunction.81 Massive pulmonary embolism leads to severe haemodynamic compromise.75 Thrombolytic is an urgent treatment, drugs like streptokinase, which can be delivered directly using cardiac catheterisation. Prevention of causes that can change or modified should be under taken.

OTHER CARDIAC EMERGENCIES

Other cardiac emergencies, which may not be seen in our practice, include acute atrial fibrillation following coronary artery by-pass graft (CABG), tear of the coronary artery during angiography, angioplasty, severe mitral regurgitation following percutaneous transluminal mitral valvuloplasty. Acute atrial fibrillation is managed using cardio version, intravenous amiodarone or sotalol or beta-blocker.57

Cardiac emergencies are part of our every day practice. It calls for training of more cardiologists, having a health policy set up that has a good reaction time to emergency situations. The basic equipments must be provided for any impact to be felt in this field.

REFERENCES


12. Pericardial effusion with evaluation for tamponade must be demonstrated