ANESTHETIC MANAGEMENT OF PATIENT WITH CARDIAC DEVICES

Essay
submitted for partial fulfillment of master degree in anesthesiology and intensive care

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2005
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فَضُلُّ اللَّهِ عَلَيْكَ عَظِيمًا
النساء: من الآية 113
## Contents

<p>| | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Introduction</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Physiological review</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>Artificial Cardiac Pacemaker</td>
<td>25</td>
</tr>
<tr>
<td>4</td>
<td>Automatic Implantable Cardioverter Defibrillator</td>
<td>40</td>
</tr>
<tr>
<td>5</td>
<td>Ventricular Assist Devices</td>
<td>48</td>
</tr>
<tr>
<td>6</td>
<td>Intraaortic Balloon Pump</td>
<td>57</td>
</tr>
<tr>
<td>7</td>
<td>Anesthetic Management of Patients With Intrathoracic Gadgets</td>
<td>74</td>
</tr>
<tr>
<td>8</td>
<td>Anesthetic Management of Patient With Ventricular Assist Device And of Patient With Intraaortic Ballon Pump</td>
<td>106</td>
</tr>
<tr>
<td>9</td>
<td>Summary</td>
<td>116</td>
</tr>
<tr>
<td>10</td>
<td>References</td>
<td>120</td>
</tr>
<tr>
<td>11</td>
<td>Arabic Summary</td>
<td>4-1</td>
</tr>
</tbody>
</table>
# List of Tables

<table>
<thead>
<tr>
<th></th>
<th>Title</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Factors affecting contractility</td>
<td>15</td>
</tr>
<tr>
<td>2</td>
<td>Effect of increasing heart rate on cardiac function</td>
<td>15</td>
</tr>
<tr>
<td>3</td>
<td>Events associated with initiation of cardiac dysrhythmias during the preoperative period</td>
<td>19</td>
</tr>
<tr>
<td>4</td>
<td>Definition of cardiogenic shock</td>
<td>24</td>
</tr>
<tr>
<td>5</td>
<td>Advantages of electrical treatment compared with drugs as initial treatment for preoperative arrhythmias</td>
<td>25</td>
</tr>
<tr>
<td>6</td>
<td>NASPE – BPEG five – position pacemaker code</td>
<td>26</td>
</tr>
<tr>
<td>7</td>
<td>Pacing modes for bradyarrhythmias</td>
<td>26</td>
</tr>
<tr>
<td>8</td>
<td>Early and late complications following pacemaker or PCD implantation</td>
<td>39</td>
</tr>
<tr>
<td>9</td>
<td>Miscellaneous factors that could affect the success of defibrillation</td>
<td>41</td>
</tr>
<tr>
<td>10</td>
<td>Criteria for ICD implantation</td>
<td>42</td>
</tr>
<tr>
<td>11</td>
<td>Potential complications of ICD surgery</td>
<td>44</td>
</tr>
<tr>
<td>12</td>
<td>Possible interactions between internal cardioverter-defibrillators (ICDs) and pacemakers</td>
<td>47</td>
</tr>
<tr>
<td>13</td>
<td>Ventricular assist devices indications and contraindications</td>
<td>55</td>
</tr>
<tr>
<td>14</td>
<td>Early complications of VADs</td>
<td>56</td>
</tr>
<tr>
<td>15</td>
<td>Late complication of VADs</td>
<td>56</td>
</tr>
<tr>
<td>16</td>
<td>Equipment needed for percutaneous IAB insertion</td>
<td>61</td>
</tr>
<tr>
<td>17</td>
<td>IABP counterpulsation indications</td>
<td>72</td>
</tr>
<tr>
<td>18</td>
<td>Relative and absolute contraindications to the insertion of IABP</td>
<td>72</td>
</tr>
<tr>
<td>19</td>
<td>Intraaortic balloon pump complications</td>
<td>73</td>
</tr>
</tbody>
</table>
List of Figures

<table>
<thead>
<tr>
<th></th>
<th>Figure Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Dual-chambered intraaortic balloon</td>
<td>4</td>
</tr>
<tr>
<td>2</td>
<td>Phases of the cardiac cycle displayed in a pressure-volume diagram</td>
<td>10</td>
</tr>
<tr>
<td>3</td>
<td>Pressure-volume diagram</td>
<td>11</td>
</tr>
<tr>
<td>4</td>
<td>Brutsaert’s proposal for a new definition of systole (S) and diastole (D).</td>
<td>12</td>
</tr>
<tr>
<td>5</td>
<td>Anatomy of the conduction system for conduction of cardiac impulses</td>
<td>17</td>
</tr>
<tr>
<td>6</td>
<td>Schematic diagram of a transmembrane action potential</td>
<td>18</td>
</tr>
<tr>
<td>7</td>
<td>The essential requirement for initiation of reentry excitationis</td>
<td>20</td>
</tr>
<tr>
<td>8</td>
<td>Illustration of available types of transvenous pacing leads</td>
<td>35</td>
</tr>
</tbody>
</table>
List of abbreviation

AICD  Automatic Implantable Cardioverter Defibrillator
ASA   American Society Of Anesthesia
AV    Atrioventricular
BP    Blood Pressure
BPEG  British Pacing And Electrophysiology Group
bpm   Beat Per Minute
BSA   Body Surface Area
Ca++  Calcium Ion.
CABG  Coronary Artery Bypass Graft
CAD   Coronary Artery Disease
CC    Cubic Centimeter
CI    Cardiac Index
CM    Centimeter
CNS   Central Nervous System
CO    Cardiac Output
°C    Celsius Centigrade
CPB   Cardiopulmonary Bypass
CVP   Central Venous Pressure
DC    Direct Current
DFT   Defibrillation Threshold
ECG   Electro Cardiogram
ECP   External Counterpulsation
ECT   Electro Convulsive Therapy
EDP   End Diastolic Pressure
EDV   End Diastolic Volume
EF    Ejection Fraction
EMI   Electromagnetic Interference
EPS   Electrophysiologic Study
Equi-MAC Equivalent To Minimum Alveolar Concentration
ESIS  Electrosurgical Interference Suppression.
ESV   End Systolic Volume
ESWL  Extracorporeal Shock Wave Lithtripsy
FDA   Food An Drug Administration
<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hr</td>
<td>Hour</td>
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<tr>
<td>HR</td>
<td>Heart Rate</td>
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<tr>
<td>IAB</td>
<td>Intra-Aortic Balloon</td>
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<tr>
<td>IABP</td>
<td>Intraaortic Balloon Pump</td>
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<tr>
<td>ICD</td>
<td>Implantable Cardioverter Defibrillator</td>
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<tr>
<td>ICU</td>
<td>Intensive Care Unit</td>
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<tr>
<td>IV</td>
<td>Intravenous</td>
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<tr>
<td>Kg</td>
<td>Kilogram</td>
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<tr>
<td>L</td>
<td>Litre</td>
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<tr>
<td>LBBB</td>
<td>Left Bundle Branch Block</td>
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<tr>
<td>LV</td>
<td>Left Ventricle</td>
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<tr>
<td>LVAD</td>
<td>Left Ventricular Assist Devices</td>
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<tr>
<td>m2</td>
<td>Meter Square</td>
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<tr>
<td>MAC</td>
<td>Minimum Alveolar Concentration</td>
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<tr>
<td>MAO</td>
<td>Monoamine Oxidase</td>
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<tr>
<td>MAOI</td>
<td>Monoamine Oxidase Inhibitors</td>
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<tr>
<td>mg</td>
<td>Milligram</td>
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<tr>
<td>min</td>
<td>Minute</td>
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<tr>
<td>mm</td>
<td>Millimeter</td>
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<tr>
<td>mmHg</td>
<td>Millimeter Mercury</td>
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<tr>
<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
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<tr>
<td>Na+</td>
<td>Sodium Ion</td>
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<tr>
<td>NASPE</td>
<td>North American Society Of Pacing And Electrophysiology</td>
</tr>
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<td>NTP</td>
<td>Noninvasive Trans Cutaneous Pacing</td>
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<tr>
<td>PA</td>
<td>Pulmonary Artery</td>
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<tr>
<td>PAWP</td>
<td>Pulmonary Artery Wedge Pressure</td>
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<td>PCD</td>
<td>Pacemaker Cardioverter Defibrillator</td>
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<tr>
<td>PCWP</td>
<td>Pulmonary Capillary Wedge Pressure</td>
</tr>
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<td>PH</td>
<td>Power Of Hydrogen Ion</td>
</tr>
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<td>PTCA</td>
<td>Percutaneous Transluminal Coronary Angioplasty</td>
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<td>PVR</td>
<td>Peripheral Vascular Resistance</td>
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<td>RA</td>
<td>Right Atrium</td>
</tr>
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<td>RBBB</td>
<td>Right Bundle Branch Block</td>
</tr>
<tr>
<td>RV</td>
<td>Right Ventricle</td>
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<td>Sec</td>
<td>Second</td>
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<tr>
<td>SSRI</td>
<td>Selective Serotonin Reuptake Inhibitors</td>
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<tr>
<td>Acronym</td>
<td>Description</td>
</tr>
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<td>---------</td>
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</tr>
<tr>
<td>SV</td>
<td>Stroke Volume</td>
</tr>
<tr>
<td>SVT</td>
<td>Supraventricular Tachy Arrhythmias</td>
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<tr>
<td>TAH</td>
<td>Total Artificial Hart</td>
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<tr>
<td>TCP</td>
<td>Trans Cutaneous Pacing</td>
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<tr>
<td>TEAP</td>
<td>Trans Esophageal Atrial Pacing</td>
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<tr>
<td>TETS</td>
<td>Transcutaneous Energy Transmission System</td>
</tr>
<tr>
<td>TIVA</td>
<td>Total Intravenous Anaesthesia</td>
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<tr>
<td>V</td>
<td>Volt</td>
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<tr>
<td>VAD</td>
<td>Ventricular Assist Devices</td>
</tr>
<tr>
<td>VF</td>
<td>Ventricular Fibrillation</td>
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<tr>
<td>VT</td>
<td>Ventricular Tachycardia</td>
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<tr>
<td>μg</td>
<td>Microgram</td>
</tr>
</tbody>
</table>
Introduction
INTRODUCTION

The conventional treatment for patients with cardiovascular disease, in whom deterioration is evident, is directed towards; optimization of electrolytes and Acid-base balance state, oxygenation and ventilation, heart rate and rhythm and blood volume. Some patients remain haemodynamically unstable inspite of maximal pharmacological support (Richenbacher and Pierce, 2001).

Heart failure may be seen in the setting of post myocardial infarction, postcardiopulmonary bypass and advanced cardionephropathies. As well as secondary to congenital heart disease and following trauma, therapeutic with cardiac transplantation being a common goal. Because donor hearts are increasingly in short supply and some of these processes are potentially reversible, a variety of cardiac devices and circulatory assist devices have become available for both developmental and general use (Galla et al., 1999).

However, the cardiac devices and the mechanical assist devices are capable of supplementing and replacing cardiac pump function for variable length of time. It is assumed that if the devices used correctly in appropriate patients; mechanical circulatory assistance devices are successful in prolonging life expectancy and improving the quality of that life (Thomas and Kramer, 1993).

Cardiac pacemaker, ventricular assist devices (VADs), automatic implantable cardioverter defibrillator (AICD), Intra-aortic balloon pump (IABP), are currently available cardiac and circulatory assist devices (Dinardo, 1998).
Historical review:

Mechanical circulatory assistance for the failing circulation has interested and challenged cardiac specialists for decades. The field of mechanical circulatory assistance is dynamic and evolving one. Technological and engineering advances have contributed to the development of mechanical assist devices.

Blood pumps, which are major component in mechanical circulatory assist devices, were taken in consideration for researchers. Brukhonenko and Tchetchuline (1929) have designed a machine that used an excised lung from a donor animal as an oxygenator and to mechanically actuated blood pumps. Their machine was used initially to perfuse isolated organs but later was used to perfuse entire animals. Dale and Schuster (1928) developed a double perfusion pump capable of a variable pulsatile output and intended to carry out whole body perfusion and was the best known at that time.

At the beginning of 1960, cardiopulmonary bypass was sufficiently established to allow open-heart surgery around the world.

Other breakthroughs led to new approaches for assisting the circulation. Improved myocardial perfusion was described by Kantrowitz (1953), when they demonstrated the concept of increasing coronary blood flow by retarding the systolic pressure pulse. This phenomenon, termed diastolic augmentation was further exploited in compression the aorta during diastole using a surgically transferred hemidiaphragm (Kantrowitz and McKinnon, 1959).
Harken (1958) described for the first time the concept of counterpulsation, which is the basis of intraaortic balloon pump (IABP) and as originally proposed involved the removal of blood via the femoral artery during ventricular systole and the rapid reinfusion of the same amount during diastole to increase coronary perfusion pressure. By this method, in normotensive preparations, one could decrease left ventricular work and increase coronary blood flow, thereby, improving the balance between myocardial oxygen supply and oxygen demand. However, Bregman (1978) stated that, this method as described has several drawbacks, among these: appreciable haemolysis, the need for bilateral femoral arteriotomies or subclavian arteriotomy and failure to increase coronary blood flow in hypotensive states.

In the early 1960s, Clauss et al. (1961) and Moulopoulos et al. (1962) conceived the concept of using timed inflation of a balloon, positioned in the central aorta, to generate a positive pressure pulse during diastole to improve coronary blood flow, and then deflation of it during systole to reduce resistance to systolic ejection, thereby reducing myocardial oxygen requirements.

Dennis et al. (1963) and Osbern et al. (1964) conceived the method of external counterpulsation (ECP) for achieving counterpulsation in a relatively noninvasive fashion. Pressure variations are applied to the legs synchronous with the heart beats, thereby using the femoral arterial tree as the pumping chamber in diastole. The initial clinical trials of ECP apparatus produces an effective arterial diastolic augmentations, but the observations concerning systolic unloading are not consistent and some investigators have reported an increase in left ventricular after-load. Major drawbacks seem to be patient discomfort,
haematuria, and varying degrees of injury to the low extremities when ECP is used for any length of time (Bregman, 1978).

However, the first clinical use of intraaortic balloon pump was done by Kantrowitz et al. (1968) to treat cardiogenic shock. The patient was a 45-year old female who had sustained a posterior wall myocardial infarction. She was in deep cardiogenic shock, comatose and anuric. Over a 7-hour period, balloon pumping restored normal circulatory dynamics. The most impressive moment occurred when the urine collection bag began to fill with urine.

Subsequently, Bregman and Goetz (1972) developed a dual-chambered balloon that was designed with a large proximal balloon and a smaller distal balloon. The rationale behind this design was to produce a unidirectional blood flow proximally to the brain and coronary arteries by initial inflation of the distal smaller balloon. This resulted in locking of the distal blood flow and an augmentation of proximal flow. Later on, in the early 1990s Kantrowiz et al. (1992) published clinical studies of a new generation, fully automated intraaortic balloon pump that continuously optimizes diastolic augmentations beat by beat without operator intervention.

Fig. (1): Dual-chambered intraaortic balloon (Bregman, 1977).
As the need for different forms of these devices was more obvious, DeBakey et al. (1966) performed the first successful use of an implantable pulsatile, air–driven, ventricular assist device (VAD) in a patients of poor condition after aortic valve operation. Although the patient died of pulmonary complications after four days of support, the assist device functioned well and successfully improved the circulation and haemodynamic parameters.

In the middle of 1960s, DeBakey (1971) used left ventricular assist device (LVAD) in a female patient with rheumatic aortic and mitral valve diseases and left ventricular failure. She underwent double valve replacement, but her heart could not be weaned from cardiopulmonary bypass. The device was used successfully for 10 days and this woman was probably the first patient to be weaned from an assist device and to leave the hospital.

Subsequently, Normal et al. (1978) performed the first clinical use of a left ventricular assist device as a bridge to cardiac transplantation. The abdominally positioned, externally powered, and single chambered device supported the patient for five days, after which cardiac transplantation was done. Although he died two weeks later of infection, this experience demonstrated that such a device could provide adequate circulatory support.

Few years later, in the middle of 1980s, the first successful bridge to cardiac transplantation with an implantable left ventricular assist device done by over and colleagues using electric type into a 51-year-old patient with ischaemic cardiomyopathy. In the same year, he used the external pneumatic pierce/Donachy left ventricular assist device to support a 47
years old patient with post-infarction cardiogenic shock. The patient was transplanted successfully two days later and survived as the first success with an external left ventricular assist device (Westaby, 1998).

Subsequently, in 1992, food and Drug Administration (FDA) gave the first approval of a pneumatic left ventricular assist device for use in a patient with postcardiotomy cardiogenic shock while the first approval, for use in a patient as a bridge to cardiac transplantation was given in 1994. on the other hand, the first FDA approved clinical trial in which an electric left ventricular assist device was implanted as a permanent form of circulatory support was initiated in 1998 and in the same year the first approval of such a device for use in a patient as a bridge to cardiac transplantation (Richenbacher, 1999).

Since the first pacemaker implantation in 1985, cardiac pacing has continued to grow. So that today more than 500,000 patients in the united states have implanted pacemakers or pacemaker cardioverter defibrillator (PCD), Approximately 400,000 such devices are implanted world wide each year (Barrold and Zipes, 1997).

Pacemakers are used to treat brady arrhythmias, and can restore normal or near normal hemodynamics during rest and exercise. PCDs are used to prevent sudden death from ventricular tachy arrhythmias (Kusumoto and Goldschlager, 1996).

However, a complication – free device suitable for permanent implantation is not yet available thus. The research and development of mechanical assist device supported by a vigorous infra-structures of basic science in biology and medicine, chemistry and pharmacology engineering and computer technology are going to develop new and safe techniques and equipment for circulatory support.
This essay has focused on cardiac physiological review and the development of the cardiac devices and their recent advances, haemodynamic and pathophysiologic changes associated with their use and the anesthetic management of patient with one of these cardiac devices including pre-operative, intraoperative, post-operative and intensive care management.
Physiological Review
PHYSIOLOGICAL REVIEW

In this discussion of cardiovascular physiology, emphasis is placed on the description of recent developments in the understanding of ventricular function. This aspect of cardiovascular physiology is emphasized because cardiac anesthesiologists have acquired, in recent years, a host of new tools to assess ventricular function, as well as new techniques to treat ventricular dysfunction.

Under normal circumstances, the heart acts as a servant by varying the cardiac output (CO) in accordance with total tissue needs (Thys and Kaplan, 1990).

Tissue needs may vary with exercise, infection, heart disease, trauma, surgery, or administration of drugs and anesthetics. Although tissue needs regulate circulatory requirements, the heart can become a limiting factor, particularly in patients with cardiac disease. In this regard it is important to differentiate circulatory function from cardiac and myocardial functions (Brawnnwald, 1977).

Circulatory function refers to the function of the entire circulatory system, including the heart, blood vessels, and blood volume. Failure of any one of these can lead to significant circulatory dysfunction. For example, hypovolemia can lead to circulatory failure and shock in the presence of normal blood vessels and a normal heart.

Cardiac function includes the function of the myocardium, valves, conduction tissue, and supporting structures. Dysfunction of any of these
can lead to cardiac and circulatory failure. The myocardium can be entirely normal, but the heart can fail due to valvular insufficiency.

**Myocardial function** depends on the cardiac muscle itself and its blood supply. Myocardial failure can result from actual muscle damage or myocardial ischemia, leading to inadequate muscle function. Obviously, myocardial dysfunction can produce cardiac failure and circulatory disturbances *(Kaplan et al., 1999).*

**The cardiac cycle:**

The electrical excitation of the myocardium results in a sequence of mechanical events in the heart, ultimately leading to the ejection of the stroke volume (SV) into the circulation. The phases of the cardiac cycle can be described in relation to changes in the heart’s electrical activity, intracardiac pressures, intracardiac volumes, opening and closing of the cardiac valves, or flow into the peripheral circulation.

**Phases of the cardiac cycle:**

1. **Isovolumic contraction phase:**

After the electrical activation of the myocardium, represented by the QRS complex on the electrocardiogram (ECG), individual ventricular fibers begin to shorten. As contraction progresses, the ventricular pressure rises and rapidly exceeds atrial pressure. This pressure reversal results in the closing of the atrioventricular (AV) valves, with coaptation of the edges of the valvular leaflets, ballooning of the pressurized leaflets, and tensing of the chordal apparatus *(Grayzel, 1991).*
As the AV valves have closed, but the semilunar valves have not yet opened, the increase in ventricular pressure is not associated with any change in ventricular volume.

2- Ejection phase:

Once the pressure developed in the ventricle exceeds the pressure in the aorta (or pulmonary artery), the ejection phase is initiated. The actual opening of the semilunar valvular leaflets is the result of the movement of blood across the valve. The ejection phase is marked by a decrease in ventricular volume, while the ventricular pressure increases initially, but then decreases to the dicrotic notch pressure. The ejection phase ends when equilibrium is reached between the ventricular and aortic pressures and volumes, and the aortic valve closes. At this time, the end-ejection or end-systolic point, the ventricular volume has reached its smallest size (*Kaplan et al., 1999*).

*Fig (2): Phases of the cardiac cycle displayed in a pressure-volume diagram (Kaplan et al., 1999).*
If EDV and end-systolic (ESV) volumes can be measured, the difference between these two volumes is the SV. Ventricular function is often expressed as ejection fraction (EF):

$$EF = \frac{[EDV - ESV]}{EDV} = \frac{SV}{EDV}$$

*Fig (3):* Pressure-volume diagram indicating end-diastolic volume (EDV), end-systolic volume (ESV), stroke volume (SV), and the equation for ejection fraction (EF) (*Kaplan et al., 1999*).

### 3- Relaxation phase:

The biochemical process of relaxation begins after peak ventricular pressure has been reached while blood is still being ejected from the ventricle. The relaxant phase usually refers to the phase of the aortic valve and is associated with a rapid, exponential decrease in ventricular pressure in the absence of any change in ventricular volume. During the relaxant phase, the heart returns to its precontractile configuration (*Brutsaert et al., 1980*).

### 4- Filling phase:

While the ventricular pressure decreases, the left atrium is being filled from the pulmonary veins. This filling is manifested by increased atrial pressure and volumes. When atrial pressure exceeds the ventricular pressure (pressure crossover), the AV valve opens and ventricular filling begins. Ventricular filling consists of an early, rapid filling phase and an atrial phase, occurring as a result of the atrial contraction.
Fig (4): Brutsaert’s proposal for a new definition of systole (S) and diastole (D). P = ventricular pressure; V = ventricular relaxation phase; RFP = rapid-filling phase; contr = contraction. The heavy black line indicates peak ventricular pressure. (Brutsaert et al., 1984).
Cardiac output:

The CO is the amount of blood pumped to the peripheral circulation per minute. (Guyton et al., 1972).

The CO is equal to the product of SV and heart rate (HR):

\[ \text{CO} = \text{SV} \times \text{HR} \]

To compare patients with different body sizes, CO may be corrected in relation to body surface area (BSA). It is then called the cardiac index (CI), which equals the CO divided by the BSA

\[ \text{CI} = \frac{\text{CO}}{\text{BSA}} \]

Factors controlling CO include venous return to the heart, systemic vascular resistance (SVR), peripheral tissue oxygen needs, blood volume, pattern and type of respiration, and body position. The two major determinants of CO are SV and HR.

Stroke Volume

The SV is the amount of blood ejected by the ventricle with each single contraction.

Determinants of st. volume:

- Preload.
- After load.
- Contractility.

1- Preload

Definition: Preload is equal to the ventricular wall stress at end-diastole. It is determined by ventricular EDV, end-diastolic pressure (EDP), and wall thickness. The interaction of these factors is expressed by the Laplace equation.
Determinants: Factors affecting the preload of the heart include the total blood volume, body position, intrathoracic pressure, intrapericardial pressure, venous tone, pumping action of skeletal muscles and the atrial contribution to ventricular filling (Luce, 1984).

2- Afterload:

Definition: Afterload is the second major determinant of the mechanical properties of cardiac muscle fibers and performance of the intact heart. Afterload can be considered either as the stress imposed on the ventricular wall during systole or as the arterial impedance to the ejection of SV (Milnor, 1982).

Impedance: Afterload can also be considered as the external or extracardiac forces (impedance) present in the systemic circulation that oppose ventricular ejection and pulsatile flow. Since the left ventricle is coupled to the systemic circulation through the open aortic valve, the pulsatile flow (SV) and pressure generated by the left ventricle will be hindered and their relationship (pressure/flow ratio) altered by the compliance and resistance of the arterial system. These are determined by the physical properties of the aorta and its side branches (viscoelastic properties and diameter), and by the properties of their content (the viscosity and density of the blood) (Milnor, 1982).

3- Contractility:

Definition: The third determinant of SV is contractility. Contractility is an intrinsic property of the cardiac cell. It defines the amount of work that the heart can perform at a given load.
Table (1) factors affecting contractility:

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<th>Factors decreasing contractility:</th>
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<tbody>
<tr>
<td>Sympathetic stimulation – direct increase of the force of contraction, as well as indirect increases due to increased heart rate (rate treppeo effect, or bow ditch phenomenon)</td>
<td>Parasympathetic inhibition increases heart rate, administration of positive inotropic drugs such as digitalis.</td>
</tr>
<tr>
<td>Parasympathetic stimulation – decreases rate effect sympathetic inhibition via withdrawal of catecholamine or blockade of adrenergic receptors</td>
<td>Parasympathetic stimulation – decreases rate effect sympathetic inhibition via withdrawal of catecholamine or blockade of adrenergic receptors</td>
</tr>
<tr>
<td>Administration of β-adrenergic blocking drugs, slow calcium channel blockers, or other myocardial depressants</td>
<td>Administration of β-adrenergic blocking drugs, slow calcium channel blockers, or other myocardial depressants</td>
</tr>
<tr>
<td>Myocardial ischemia and infarction</td>
<td>Myocardial ischemia and infarction</td>
</tr>
<tr>
<td>Intrinsic myocardial diseases such as cardiomyopathies</td>
<td>Intrinsic myocardial diseases such as cardiomyopathies</td>
</tr>
<tr>
<td>Hypoxia and acidosis.</td>
<td>Hypoxia and acidosis.</td>
</tr>
</tbody>
</table>

*(Kaplan et al., 1999)*

Heart Rate:

Because of its multiple effects on cardiac dynamics, HR is often considered the forth determinant of cardiac function. Heart rate is primarily determined by the rate of spontaneous phase IV depolarization of the sinoatrial (SA) node pacemaker cells.

Table (2) Effect of increasing heart rate on cardiac function

<table>
<thead>
<tr>
<th>Shortening of systole</th>
<th>Shortening of diastole</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decrease left ventricular (LV) myocardial perfusion time</td>
<td>Decrease in ventricular filling.</td>
</tr>
<tr>
<td>Rate – dependent change in stroke volume.</td>
<td>Rate dependent change in cardiac output.</td>
</tr>
<tr>
<td>Rate – dependent positive inotropic effect.</td>
<td></td>
</tr>
</tbody>
</table>

*(Kaplan et al., 1999)*

Control of Heart rate:

Neural and, to a much lesser extent, humoral mechanisms control intrinsic heart rate. Baroreceptors and chemoreceptors and both divisions
of the autonomic nervous system are major components of the neural control. Afferent neural pathways carrying outputs of peripheral sensors reach the brain stem (vasomotor center, nucleus ambiguous, tractus solitarius, and vagal nucleus), where integration of information takes place, efferent neural traffic consisting of input signals to the cardiovascular system leaves the brain stem via the sympathetic neural pathways (cervical sympathetic ganglia, stellate nerve and rami communicantes to thoracic cardioaccelerator nerves from T1 to T4) to stimulate the heart (cardioacceleration, faster AV and intraventricular conduction, increased Contractility), and via the vagal neural pathways (vagus nerve, intramural cardiac ganglia) to restrain the heart (bradycardia, slower AV conduction, decreased atrial and ventricular contractile force, as can occur in response to sudden hypertension, mesenteric traction, and massage or compression of the carotid sinus) (Sarnoff et al., 1969).

Vagal influence usually predominates so that the resting rate of the normal heart is slower (70 bpm) compared with its intrinsic rate, which is about 110 bpm after pharmacologic denervation. The rate of the normal heart is also slower than the rate of the transplanted heart (M. Laughtlin et al., 1978).

Conductivity & arrhythmias:

Transmission of the cardiac impulse is through a specialized conduction system present in the atria and ventricle.
The normal sequence of cardiac depolarization:

Pacemaker cells in the sinoatrial (SA) node spontaneously depolarize and a wave of depolarization spreads over the atria and into the atrioventricular (AV) node. After a brief delay in the node, the impulse enters the bundle of His and right and left bundle branches. The inter-ventricular septum is the first to depolarize, followed by the apical ventricular myocardium, then the bulk of the left and the right ventricular free walls, with the last area activated being the superior portion of the left ventricular free wall or the right ventricular outflow tract. *(Chawhan et al., 2001).*

![Anatomy of the conduction system for conduction of cardiac impulses](image)

*Fig (5):* Anatomy of the conduction system for conduction of cardiac impulses *(Topol et al., 2004).*

The electrocardiogram (ECG) is the corner stone for the diagnosis of cardiac conduction and rhythm disturbances. Cardiac conduction and rhythm disturbances. The normal ECG consists of three waveforms designated the P wave (atrial depolarization), QRS complex (Ventricular depolarization) and T wave (Ventricular repolarization. The P-R interval is the time necessary for the cardiac impulse pass through the atrioventricular (AV) node.
Fig (6): Schematic diagram of a transmembrane action potential generated by an automatic cardiac cell, and of the relationship of this action potential to events depicted on the electrocardiogram (ECG). Phase 4 undergoes spontaneous depolarization from the resting membrane potential (-90 mV) until the threshold potential (broken line) is reached. Depolarization (phase 0) occurs when the threshold potential is reached and corresponds to the QRS complex on the ECG. Phases 1 through 3 represent repolarization with phase 3 corresponding to the T wave on the ECG. The effective refractory period (ERP) is that time during which cardiac impulses cannot be conducted, regardless of the intensity of the stimulus. During the relative refractory period (RRP), a stronger than normal stimulus can initiate and action potential. The action potential from a contractile cardiac cell differs from an automatic cardiac cell, in that phase 4 does not undergo spontaneous depolarization (Topol et al., 2004).

Changes in heart rate are; tachycardia and bradycardia, but changes, in rhythm are called arrhythmias.

Significance of arrhythmias:

Arrhythmias may produce mechanical cardiac dysfunction due to bradycardia, tachycardia or improperly synchronized atrial and ventricular contractions appearance alone does not portray the
Physiologic significance of arrhythmias. For example, ventricular Extrasystoles may have an ominous appearance, yet may have little adverse circulatory effect or effect on outcome (Atlee, 1997).

In contrast, most supraventricular tachyarrhythmias (SVTs) have a less ominous appearance, but due to diastolic encroachment and increased O₂ demand, SVT maybe hemodynamically disadvantageous. Also to be considered regarding significance of arrhythmias are underlying structural heart disease and associated mechanical dysfunction.

Cardiac dysrhythmias that occur during the perioperative period can usually be explained on the basis of abnormalities of cardiac impulse conduction (reentry) or impulse formation (automatic or ectopic) reentry excitation accounts for most premature beats and tachydysrhythmias (Akhtar, 1982).

Table (3) Events associated with initiation of cardiac dysrhythmias during the preoperative period:

<table>
<thead>
<tr>
<th>Arterial hypoxemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Electrolyte disturbances</td>
</tr>
<tr>
<td>Potassium</td>
</tr>
<tr>
<td>Magnesium</td>
</tr>
<tr>
<td>Acid-base disturbances</td>
</tr>
<tr>
<td>Altered activity of the autonomic nervous system</td>
</tr>
<tr>
<td>Increased myocardial fiber stretch</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Intubation of the trachea.</td>
</tr>
<tr>
<td>Myocardial ischemia</td>
</tr>
<tr>
<td>Drugs</td>
</tr>
<tr>
<td>Catecholamines</td>
</tr>
<tr>
<td>Volatile anesthetics</td>
</tr>
<tr>
<td>Co-existing cardiac disease</td>
</tr>
<tr>
<td>Pre-excitation syndrome</td>
</tr>
<tr>
<td>Prolonged Q-T interval syndrome.</td>
</tr>
</tbody>
</table>

(Kaplan et al., 1999)
Fig (7): The essential requirement for initiation of reentry excitations is a unilateral block that prevents uniform anterograde propagation of the initial cardiac impulse. Under appropriate conditions, these same cardiac impulses can traverse the area of blockade in a retrograde direction and become a reentrant cardiac impulse. (Akhtar M., 1982).
Pathophysiology of bradycardia and complete heart block

Complete heart block is the failure of electrical activity from the atrium to progress through. The AV node into the His-purkinje system. When an impulse is not initiated immediately in the bundle of His, arrest occurs for a brief period and stokes-Adams syndrome occurs, which causes light headedness, dizziness, or loss of consciousness sometimes accompanied. By convulsions. During bradycardia, cardiac output is maintained by an increasing stroke volume. When the stroke volume is maximally increased, any further decrease in the heart rate will compromise cardiac output and cause circulatory failure. (Braunwald et al., 2001).

The common causes of heart block:

Organic Disease:

- Disease affecting primary conduction tissue.
  - Lenegre’s disease-sclerodegenerative process of the terminal portions of His bundles.
  - Lev’s disease-fibrous encroachment of the proximal His conduction pathway.
- Disease affecting cardiac tissue.
  - Coronary artery disease (CAD) with ischemia or infarction.
  - Cardiomyopathy.
  - Myocarditis.
- Surgically produced.
- Congenital block.

Functional Disturbances:

- Increased vagal tone.
• Drug therapy with quinidine, digitalis, procainamide, verapamil, or potassium.

(Fuster et al., 2001)

Diagnosis of first-, second-, and third-degree AV block, right bundle branch block (RBBB) with left anterior fascicular hemiblock, and left posterior fascicular hemiblock:

First-degree AV block is characterized by a PR interval of greater than 0.20 seconds. Second-degree AV block is subdivided into two types. Mobitz type I, or Wenchebach block, is characterized by a progressively lengthening PR interval, which occurs until an impulse is not conducted and a beat is dropped. Mobitz type II block is characterized by a sudden dropping of the QRS complex, with no progressive lengthening of the PR interval occurring. Third-degree AV block, also called complete heart block, occurs when all electrical activity from the atrium fails to progress into the Purkinje system. The atrial and ventricular contractions have no relationship with each other. The QRS complex is normal in complete AV nodal block. The QRS complex with complete infranodal block is frequently wide, and the ventricular rate is slow, averaging 40/minute. RBBB with left anterior superior hemiblock is indicated when electrocardiogram shows RBBB and left axis deviation. Complete RBBB with right axis deviation is indicative of RBBB and left posteroinferior hemiblock.

Pathophysiology of tachycardia:

The effects of increasing heart rate on cardiac function:

• Shortening of the systole.
• Shortening of the diastole.
• Decrease left ventricular (LV) Myocardial perfusion time.
• Decrease in the ventricular filling.
• Rate-dependant change in stroke volume.
• Rate-dependant change in cardiac output.
• Rate dependant positive inotropic effect.

(Kaplan et al., 1999)

Pathophysiology of Heart failure:
• Systolic heart failure exists when the heart is unable to pump a sufficient amount of blood to meet the body’s metabolic requirements. (Morgan, 2002).
• Clinical manifestations usually reflect the effects of low cardiac output on tissues (e.g., Fatigue, oxygen debt, acidosis.
• Isolated right ventricular failure can occur in the setting of advanced diseases of the long parenchyma of pulmonary vasculature.
• Left ventricular failure most commonly results from primary myocardial dysfunction but may also results from valvular dysfunction, arrhythmias or pericardial disease.
• Diastolic dysfunction can also cause symptoms of heart failure as a result of arterial hypertension, coronary artery disease, hypertrophic cardiomyopathy, and pericardial disease. (Morgan, 2002).
• Systolic and diastolic dysfunction are commonly associated.
• Cardiac output is reduced in most forms of heart failure.
• Heart failure is less commonly associated with an elevated cardiac output. This form of heart failure is most commonly seen with sepsis and other hypermetabolic states. (Morgan, 2002).
Table (4): Definition of cardiogenic shock.

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac output index</td>
<td>&lt; 2.0 liters/min/m²</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>&lt; 90 mmHg.</td>
</tr>
<tr>
<td>Left or right atrial pressure</td>
<td>&gt; 20 mmHg.</td>
</tr>
<tr>
<td>Urine output</td>
<td>&lt; 20 ml/hr.</td>
</tr>
<tr>
<td>Systemic vascular resistance</td>
<td>&gt; 2100 dynes/sec/cm⁵</td>
</tr>
<tr>
<td>PCWP</td>
<td>&gt; 18</td>
</tr>
</tbody>
</table>

(Brownwald et al., 2001).

Compensatory mechanisms of Heart failure:

Major compensatory mechanisms generally present in patients with heart failure include increased preload, increased sympathetic tone, activation of rennin-angiotensin-aldosterone system, ventricular hypertrophy, and release of AVP. Although, these mechanisms can initially compensate for mild to moderate cardiac dysfunction, with increasing severity of dysfunction they may actually contribute to the cardiac impairment. (Morgan, 2002).
Artificial Cardiac Pacemaker
ARTIFICIAL CARDIAC PACEMAKER

Pacemaker design:

Electronic cardiac pacemakers are temporary or permanent (implanted) devices that electrically stimulate the heart. (De Belder et al., 1990).

Table (5). Advantages of electrical treatment compared with drugs as initial treatment for preoperative arrhythmias:

<table>
<thead>
<tr>
<th>Advantage</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Institute or terminate treatment at will immediate and easily titratable</td>
<td>Precise control of heart rate is possible no iatrogencity or myocardial depression proarrhythmia less likely than with drugs avoid adverse drug effect and interactions.</td>
</tr>
</tbody>
</table>

(Kaplan et al., 1999)

Device description:

Pacemakers consist of a power source (battery) that supplies energy for stimulation and other pacemaker functions, circuitry for sensing and regulation of stimulation, and leads that connect the power source and electronic circuitry to electrodes for sensing or stimulation. Epicardial or endocardial electrodes (transvenous) provide invasive (direct) cardiac stimulation, and are used for most temporary and all permanent pacing applications. Noninvasive (indirect) pacing routes include transcutaneous pacing with cutaneous patch electrodes and transesophageal pacing with electrodes behind the left atrium or ventricle. Finally, all temporary or permanent pacemakers are programmable (i.e., capable of reversibly altering stimulus characteristics, timing, and sensing functions) (Kaplan et al., 1999).
Pacemaker Code:

Table (6) NASPE – BPEG five – position pacemaker code

<table>
<thead>
<tr>
<th>I Pacing</th>
<th>II Sensing</th>
<th>III Response</th>
<th>IV Programmability</th>
<th>V Tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>0- None</td>
<td>0-None</td>
<td>0-None</td>
<td>0-None</td>
<td>0-None</td>
</tr>
<tr>
<td>A- Atrium</td>
<td>A-Atrium</td>
<td>I- Inhibited</td>
<td>C-Communicating</td>
<td>P-None</td>
</tr>
<tr>
<td>V- Ventricle</td>
<td>V-Ventricle</td>
<td>T-Triggered</td>
<td>P- Simple programmable</td>
<td>S-Shocks</td>
</tr>
<tr>
<td>D- Dual (A+V)</td>
<td>D-Dual (A+V)</td>
<td>D-Dual (I+T)</td>
<td>M-Multiprogrammable</td>
<td>D-Dual (P+S)</td>
</tr>
<tr>
<td>S-Simple (A or V) §</td>
<td>S-Simple (A or V)</td>
<td>R-Rate modulation</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* antiachyarrhythmia pacing
+ Rate and / or output only
‡Atrial-triggered, ventricular-inhibited
§ Manufacturer’s designation only

NASPE = North American Society of Pacing and Electrophysiology; BPEG = British Pacing and Electrophysiology Group. *(Kaplan et al., 1999)*

Pacing for bradycardia;

Table (7) Pacing modes for bradyarrhythmias

<table>
<thead>
<tr>
<th>Code</th>
<th>Description of mode</th>
</tr>
</thead>
<tbody>
<tr>
<td>AOO</td>
<td>Atrial asynchronous pacing</td>
</tr>
<tr>
<td>VOO</td>
<td>Ventricular asynchronous pacing</td>
</tr>
<tr>
<td>DOO</td>
<td>Dual-chamber asynchronous pacing</td>
</tr>
<tr>
<td>AAI</td>
<td>Atrial-inhibited pacing</td>
</tr>
<tr>
<td>VVI</td>
<td>Ventricular inhibited pacing</td>
</tr>
<tr>
<td>VAT</td>
<td>Atrial –triggered, ventricular pacing</td>
</tr>
<tr>
<td>VDD</td>
<td>Atrial-triggered, ventricular-inhibited pacing</td>
</tr>
<tr>
<td>DVI</td>
<td>Dual-sequential, ventricular-inhibited pacing</td>
</tr>
<tr>
<td>DDI</td>
<td>Dual-chamber, sequential inhibited pacing</td>
</tr>
<tr>
<td>DDD</td>
<td>Dual-sequential, atrial-triggered, ventricular-inhibited or AV universal pacing</td>
</tr>
</tbody>
</table>

AV= atrioventricular *(Kaplan et al., 1999)*

When a pacemaker delivers stimuli at some programmed constant rate, without regard to the heart’s intrinsic or native rhythm, this is asynchronous (fixed-rate) pacing. Pacemakers that respond appropriately to sensed intrinsic atrial or ventricular activity are non-competitive (demand) pacemakers. Responses to sensed intrinsic atrial or ventricular myopotentials include inhibition or triggering of pacemaker output in one or the other chamber.
Single-chamber pacing modes:

Asynchronous pacing modes include AOO and VOO. However, competing rhythms and tachyarrhythmia induction are possible, so that these modes are rarely programmed for permanent pacemakers. They may be used for temporary pacing.

**Dual-Chamber Pacing:**

Dual-chamber pacing modes are intended to preserve the normal relation between atrial and ventricular contractions in patients with symptomatic bradycardia due to AV heart block, but without atrial fibrillation. There is only one asynchronous dual-chamber (OOO) pacing mode, which is primarily used for temporary pacing following cardiac surgery (*Kaplan et al., 1999*).

**Refractory and blanking periods; rate hysteresis:**

Refractory and blanking periods are programmed time intervals during which the atrial or ventricular sensing amplifiers are nonreceptive to myopotentials or extraneous noise.

Ventricular stimulation and the atrial escape interval (AEI) begin if no spontaneous event occurs before the programmed AV interval AVI lapses. Often, AEI is programmed longer than the upper rate interval to maximize the chance for spontaneous atrial rhythm (i.e., rate hysteresis) (*Kaplan et al., 1999*).

**Tachycardia pacing:**

Antitachycardia pacing has limited application to the chronic management of hemodynamically significant supraventricular and ventricular tachyarrhythmias, but is sometimes useful for suppression or termination of tachyarrhythmias in postcardiac surgical patients, or to
reduce QT-interval dispersion/duration and tachyarrhythmia susceptibility in patients with the long QT-interval syndrome (Zipes, 1997).

Most clinically important, recurring tachyarrhythmia (except atrial fibrillation and ventricular tachyarrhythmias with acute ischemia, reperfusion, or infarction) are thought to be due to reentry (Atlee, 1996).

Additionally, antitachycardia pacing may induce new and worse arrhythmias or cause tolerated tachycardias to degenerate to less tolerated ones. Nonetheless, many contemporary PCDs incorporate antiarrhythmia therapy (i.e., pacing first followed by synchronized or non-synchronized shocks as indicated).

Rate-Adaptive Pacing:

For patients with a physiologically responsive sinus node and no atrial fibrillation or quiescence, a pacing mode that tracks atrial activity provides physiologic pacing (VAT, VDD, DDD). The major limitation to wider use of physiologic pacing modes is possible one-to-one tracking of atrial tachycardia. This risk is reduced by programming an upper-rate interval.

Esophageal pacing:

Clinical use:

The value of esophageal ECG (Es-ECG) for arrhythmia diagnosis (amplified P waves) has been recognized for more than 60 years. Transesophageal atrial pacing (TEAP) was first described in 1957 and used for emergency pacing (TEVP) is feasible, but not very reliable in adults. It is more so in infants and small children. TEVP in adults requires somewhat higher currents and possibly longer pulse durations than
supplied by available TEAP pulse generators. TEAP has been used by cardiologists to identify tachycardia mechanisms, test antiarrhythmic drug efficacy, assess risk for rapid atrial conduction with atrial flutter-fibrillation in patients with the Wolff-Parkinson-White syndrome, and to provide controlled rate increases with stress echocardiography (Benson, 1987 and Deal 1995).

Equipment and methods:

Pill, catheter, and several types of esophageal stethoscope TEAP leads are or will soon be available.

**TEAP leads can be positioned by several ways:**

1- Use The reported distances for position of least TEAP current (inferior alveolar ridge to bipolar atrial electrode center point is about 30cm in adults).

2- During near-maximal stimulation (40 mA x 10 msec), advance lead to TEAP capture, then advance or withdraw lead to point of least current requirement (current – guided insertion); or.

3- Advance lead using ECG guidance until a maximal P wave is observed, also the point of least TEAP current requirement (Deal, 1995).

**Indications for permanent pacemaker:**

Artificial pacing is indicated for treatment of persistent bradycardia of any origin if it compromises hemodynamics or predisposes to ventricular irritability manifested by premature beats or ventricular tachycardia (VT). The two major indications for permanent pacing are failure of impulse formation and failure of cardiac conduction. Clinically, sick sinus syndrome and complete heart block are the most common
indications for pacemakers. The following types of arrhythmias are common indications for pacemakers:

1- **Sinoatrial (SA) node**: sick sinus syndrome, symptomatic bradycardia, hypersensitive carotid sinus syndrome, or vasovagal syncope.

2- **AV node**: second degree or third degree AV block.

3- **Trifascicular block or bifascicular block** with prolonged infranodal conduction.
   - RBBB and left anterior hemiblock with hemodynamic symptoms.
   - RBBB and left posterior hemiblock with hemodynamic symptoms.
   - Alternating LBBB and RBBB.
   - LBBB and first degree AV block.

4- Hemodynamically disabling tachyarrhythmias with resistance to or intolerance of drug therapy or direct current (DC) cardioversion.

5- Congenital long Q-T syndrome.

6- Syncope without an ECG diagnosis.

7- Cardiomyopathies: several symptomatic patients with dilated cardiomyopathy or with hypertrophic obstructive cardiomyopathy may be treated with dual and three chamber pacing (right atrium and both ventricles) (*Atlee and Bernstein 2001, and Braunwald, 2001*).

Indications for temporary and permanent pacing are similar, except that the former is used as a bridge to permanent pacing or for transient bradyarrhythmias (*Kaplan et al., 1999*).
Pacing in children:

Indications for pacing in children are similar to those in adults, but there are some special considerations. As in adults, unequivocal concurrence of symptoms with bradycardia provides. Such concurrence may be determined by Holter or transtelephonic monitoring. Alternative causes (e.g., seizures, breath holding, infantile apnea, disautonomia) must be excluded. Finally, the brady – tachy syndrome is frequently an indication for pacing in children, especially when antiarrhythmic drugs other than digitalis must be prescribed. The use of quinidine, other class I anti-arrhythmic drugs, amiodarone, and propranolol is especially dangerous in children with the brady-tachy syndrome. Any can severely depress sinus node function, and their use may necessitate pacing in children with the brady – tachy syndrome (Kaplan et al., 1990).

Factors that can influence the decision to implant a pacemaker.

1. Overall physical and mental status of the patient, including associated diseases that may result in a limited quality or prognosis for life. Desires of the patient and family.

2. Presence of underlying structural heart disease that may be affected adversely by bradycardia (e.g., dilated cardiomyopathy).

3. Desire of the patient to operate a motor vehicle or the need to use hazardous tools and operate machinery.

4. Remoteness of medical care, including patients who travel widely or live alone who therefore may be unable to seek medical help if serious symptoms arise.

5. Necessity for administering medication that may depress escape heart rates or aggravate atrioventricular heart block. Slowing of basic escape rates due to whatever cause.
6. Significant cerebrovascular disease that might result in a stroke if cerebral perfusion were to suddenly decrease.

**Antitachycardia Pacing:**

The decision to implant a pacemaker for control of tachyarrhythmias is made after careful observation and extensive electrophysiologic study. *(Gregoratos et al., 1998).*

Pacing techniques to terminate tachycardia include underdrive, overdrive, and burst pacing; and extrastimulation. Antitachycardia pacemakers may detect tachycardia and initiate a pacing sequence, or respond to patient-activated instruction. *(Kaplan et al., 1999).*

**Temporary pacing Indications:**

**Bradyarrhythmias:**

For anesthesia and critical care providers, temporary pacing indications continue to evolve and will depend to a large extent on equipment available, familiarity with use, and how aggressive they are with invasive pacing. Cardiovascular anesthesiologists are more likely than the generalists to routinely employ temporary transvenous or epicardial pacing in their practices. Noninvasive transcutaneous ventricular pacing is widely available and easy to use, but may not be feasible or effective in many patients with bradyarrhythmias. *(Birkui et al., 1993).*

**Temporary Pacing Indications:**

**Tachyarrhythmias**

Temporary antitachycardia pacing is most commonly employed after cardiac surgery. With increased availability of effective noninvasive pacing technology, antitachycardia pacing may come to be used in other patients as well. However, some pacing modes used to terminate
tachyarrhythmias (e.g., extrastimulation, burst pacing) are not available on most temporary pulse generators found in operating rooms or intensive care units. Perhaps this is as it should be, in as much as extrastimulation and burst pacing are advanced pacing techniques. Even when used properly, there is definite risk for inducting more dangerous arrhythmias. For this reason, antitachycardia pacing should be performed by or with assistance from physicians with proper training, and always with direct current (DC) cardioverter-defibrillator back-up (Atlee, 1996).

**Temporary Pacing:**

**Invasive (Direct) Cardiac Pacing:**

Direct cardiac pacing methods are the most reliable and preferred for patients having cardiac surgery, especially in the post-bypass period, when epicardial pacing wires are placed as a more or less routine measure by most centers in most patients. For the pre-bypass period, however, especially in patients with or at high risk for hemodynamically significant bradycardia due to AV heart block or sinus node dysfunction, prophylactic transvenous pacing electrodes are recommended. With either epicardial or endocardial pacing, both atrial and ventricular pacing electrodes should be placed, unless the patient has established atrial fibrillation and there is no intent to perform cardioversion or reasonable expectation of spontaneous conversion to sinus rhythm. Whenever possible, a physiologic pacing (atrial-tracking or dual-chamber) mode is preferred for most patients undergoing cardiac surgery. However, while transvenous ventricular pacing electrodes can be positioned fairly easily by most individuals familiar with central venous access techniques, correct placement of transvenous atrial pacing often requires fluoroscopic guidance. (Kaplan et al., 1999).
Pacing leads:

There are a wide variety of temporary pacing leads for direct cardiac pacing. Temporary epicardial pacing leads are usually Teflon-coated, stainless steel wires. They are sutured loosely onto the epicardium and brought out through the chest wall. Usually pairs of electrodes are put on both cardiac chambers along with one or more skin ground wires. Atrial and ventricular wires must be distinguished. A wide variety of bipolar and unipolar leads are used for temporary transvenous pacing. These leads are constructed of relatively rigid woven polyester or plastic. Catheters vary in degree of stiffness; some require guidewires for insertion, and others are sufficiently stiff to permit tailoring of the catheter shape. Some are balloon-tipped for flow-directed insertion, possibly with atrial and/or ventricular ports for insertion of the wire pacing leads. Others permit simultaneous pressure monitoring. Finally, there are a wide variety of introducer sheaths for transvenous pacing electrodes, including those with sideports for intravenous fluid administration. If temporary pacing is the only requirement, however, a peel-away introducer sheath is commonly used (Kaplan et al., 1999).
Fig (8): Illustration of available types of transvenous pacing leads. Top left: Bipolar pacing catheter with tip at right ventricular (RV) apex. Top right: Bipolar J lead positioned in right atrial (RA) appendage. Middle left: Quadripolar catheter lead with one pair of flared electrodes contacting the RV wall, and the other pair in the RV apex. Middle right: Bipolar balloon for atrial pacing, but contact with these is problematical in some patients. Bottom left: Pulmonary artery (PA) catheter with bipolar ventricular pacing wire. Bottom right: PA catheter with bipolar atrial J and ventricular pacing wires (Atlee 1996).

**Lead insertion:**

Cardiac anesthesiologists should become reasonably adept at establishing temporary ventricular (not necessarily atrial) pacing with semi-rigid or flow-directed transvenous leads. In addition, they should be proficient with establishing temporary atrial or ventricular pacing with pulmonary artery catheters adapted for use with atrial or ventricular pacing wires (Kaplan et al., 1999).
Venous Access:

The site of venous access for temporary pacing should take into account the urgency of pacing, desired lead stability, anticipated duration of pacing, and the need to avoid specific complications (Wood, 1995).

Electrodes are positioned most rapidly via the right internal jugular vein, even without fluoroscopy. The left subclavian vein is also useful, but trauma during attempts at cannulation could preclude later use for permanent pacing or PCD leads. Other potential problems with central access include trauma to central arteries, hemorrhage and pneumothorax. The external jugular, brachial, cephalic, and femoral veins are also used for access, but may be virtually impassable without fluoroscopic guidance. Also, extremity routes may permit lead dislodgement with patient movement, and consequent failure to pace (Kaplan et al., 1999).

Lead positioning:

Once central venous access had been achieved, catheters can be positioned under fluoroscopic or electrocardiographic guidance. Concerning the later, the most distal catheter electrode is connected to the V-lead of a 5-lead ECG monitoring system (or V1 of a 12-lead system), and the electrode position will be known by the characteristic waveforms recorded. Once the catheter tip is in the right ventricle, the balloon is deflated and the catheter advanced until marked ST-segment elevation occurs (injury current), indicating contact with ventricular myocardium. In an emergency, the catheter is advanced blindly during synchronous pacing at maximal output until capture is assured. A working electroverter should be available to treat ventricular tacharrythmias induced during lead manipulation. The right atrial (RA) appendage and right ventricular (RV) apex provide the most stable lead electrode position (Wood, 1995).

However, the RA appendage may be deformed or absent from previous cardiac surgery, or the RV apex provide unacceptably high pacing thresholds because of apical scarring following infarction. If so, fluoroscopic guidance will be required to reposition leads (e.g., coronary
sinus, RV outflow tract, ventricular septum). Final catheter position should be confirmed by chest radiographs and ECG recordings (Kaplan et al., 1999).

External Pulse Generators:

Temporary pacing pulse generators are usually constant current output devices powered by disposable commercial 9-V batteries (Wood, 1995).

They provide 12 to 15 V of output, and are intended to function against impedances of 300 to 1000 ohms. Until several years ago, external pulse generators were capable only of SOO, SSI, DVI and DOO modes (S = single – chamber pacing – atria or ventricle). Typically, these generators have programmable rates (30-180 beats/min), sensitivity (0.1 mV – asynchronous), current output (0.1 – 20 mA), and AV delays (0 – 300 msec). Some pulse generators provide rates up to 800 beats/min for pace-termination of atrial tachyarrhythmias. Today, external DDD pulse generators are available. These devices feature extensive programmability of operating modes (AOO, AAI, VOO, VVI, VAT, VDD, DOO, DVI, DDI, DDD), current outputs, sensitivities, refractory periods, and hysteresis, and may also allow high-rate pacing for termination of tachyarrhythmias (Kaplan et al., 1999).

Prophylactic invasive pacing:

The patient with significant cardiovascular disease, but not undergoing cardiac surgery, is more problematic concerning options for temporary pacing indications should arise. Transvenous or epicardial pacing is advised for patients with class I temporary pacing indications. Prophylactic transvenous pacing should be considered for those with class II pacing indications and having extensive or debilitating surgery (Kaplan et al., 1999).
Noninvasive (indirect) cardiac pacing:

Transcutaneous pacing:

Improvements in transcutaneous pacing (TCP) include longer stimulus duration, larger surface area electrodes, and combined pacing – cardioversion defibrillation with ECG monitoring in one device (Wood, 1995). These have made TCP increasingly attractive as an alternative pacing route for temporary or prophylactic management of bradycardia of asystole (Roth et al., 1991). However, with cardiac electrical asystole, if TCP is not instituted early during resuscitation it is not likely to be effective or improve outcome (Cummins et al., 1993).

However, to institute transvenous ventricular pacing in an emergency can be difficult and time consuming, and is not feasible in most out of hospital resuscitations. On the other hand, TCP can be instituted quickly and safely by paramedical personnel. It provides the opportunity to institute pacing treatment for severe bradycardia or asystole early during resuscitation. TCP has also been used to terminate AV nodal reentry and AV reciprocating tachycardia (Altamura et al., 1990).

TCP Thresholds for pulse widths of 20 to 40 msec are lowest (40-80 mA) in healthy volunteers or patients, or when used for prophylaxis. In clinical use, however, thresholds of 20 to 140 mA may be encountered (Wood, 1995).

There is no clear correlation between TCP thresholds and age, body weight or surface area, heart disease, chest diameter, or cardiovascular drug therapy (Klein et al., 1988). Higher thresholds are needed to terminate tachyarrhythmias, up to 24 hours after cardiac surgery, and with emphysema, pericardial effusion or positive – pressure ventilation (Kelly et al., 1989).

As with other forms of pacing, TCP capture may be problematic with myocardial ischemia, severe metabolic derangements, or prolonged resuscitation efforts. Failure to capture most commonly is due to
suboptimal electrode position or patient intolerance to pain (Wood MA, 1995).

Capture Incidence. The incidence of ventricular capture with TCP in healthy volunteers ranges from 50 to 100 percent, but is generally lower during emergent use (10 to 93 percent). Success rates may exceed 90 percent when the method is instituted prophylactically or early (<5 minutes) into bradycardic arrests (Wood, 1995).

Complications and problems: Complications from TCP are said to be extraordinarily rare in the clinical setting. The most frequent problems are the induction of coughing and severe pain during pacing (Wood MA, 1995). Presumably, the former is from stimulation of the diaphragm, either directly or via the phrenic nerve. Other problems with TCP include: (1) inability to obtain TCP capture in emergencies in up to 90 percent of patients; (2) inability to correctly position electrodes due to the nature of surgery or sterile fields; and (3) the possibility that TCP may not restore effective hemodynamics in some patients, particularly those with diastolic dysfunction (Kaplan et al., 1999).

Table (8): Early and late complications following pacemaker or PCD implantation

<table>
<thead>
<tr>
<th>Early</th>
<th>Late</th>
<th>Early or late</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pneumo (hemo) thorax subcutaneous emphysema</td>
<td>Thromboembolism pulse generator erosion lead defects ↑pacing thresholds ↑ pacing thresholds Battery depletion</td>
<td>Lead dislodgement pacemaker arrhythmias pacemaker infection Pacemaker syndrome Generator malfunction Extracardiac stimulation</td>
</tr>
</tbody>
</table>

PCD= pacemaker-cardioverter defibrillator. (Kaplan et al., 1999)
Automatic Implantable Cardioverter Defibrillator
AUTOMATIC IMPLANTABLE CARDIOVERTER DEFIBRILLATOR

Recurrent ventricular tachycardia or ventricular fibrillation that can result in sudden death in the survivor of cardiac arrest may be treated with an automatic implantable cardioverter defibrillator (AICD) that senses the onset of these ventricular dysrhythmias and delivers a synchronized 25-joule electrical discharge (Goldsmith MF, 1991).

Electrophysiologic testing is required before implantation to an AICD, to demonstrate the presence of inducible ventricular tachycardia or ventricular fibrillation that is unresponsive to antidysrhythmic drug therapy. Implantation of an AICD requires general anesthesia for surgical placement of epicardial electrodes, demonstration that the ventricular dysrhythmia can be induced and electrically converted, and placement of the pulse generator in a subcutaneous periumbilical pocket. Periodic follow-up with the aid of a magnet determines the number of shocks that have been delivered to the patient, the battery strength, the status of the pulse generator, and the integrity of the sensing function.

Internal Pacemaker Cardioverter Defibrillator:

Contemporary internal cardioverter-defibrillators (ICDs) have transvenous lead systems and utilize multiprogrammable, triggered therapy for treatment of VT (Cannom Ds, 1995).

Recent or expected improvements to PCD technology include further downsizing of the generator to permit subpectoral as opposed to abdominal wall implantation, dual-chamber pacing, incorporation of
biosensors for in situ hemodynamic monitoring, and addition of an atrial lead for differential diagnosis of tachycardia, atrial pacing and/or defibrillation of the atria (Zipes DP, 1997).

Until recently, most PCD devices required thoracotomy for implantation. Currently most devices employ transvenous spring or coil electrodes with or without subcutaneous patch electrodes for defibrillation (Cannom DS, 1995).

Also, earner devices used monophasic shocks, and were successful in about 70 to 80 percent of patients. Today’s PCDs deliver biphasic shocks, have a lower energy requirement, and are superior for defibrillation (Zipes DP, 1997).

Table (9): Miscellaneous factors that could affect the success of defibrillation

<table>
<thead>
<tr>
<th>Factor</th>
<th>Success</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acidosis and hypoxemia (↑)</td>
<td></td>
</tr>
<tr>
<td>Class Ia antiarrhythmics (↑)</td>
<td></td>
</tr>
<tr>
<td>Amiodarone and phenytoin (↑)</td>
<td></td>
</tr>
<tr>
<td>Prolonged fibrillation (↑)</td>
<td></td>
</tr>
<tr>
<td>Morbid obesity (↑)</td>
<td></td>
</tr>
<tr>
<td>Bretylium, catecholamines (↑)</td>
<td></td>
</tr>
</tbody>
</table>

(Zipes, 1997)

The ICD system is similar to pacemaker in that it incorporates the microprocessor, memory, capacitors, and energy source in a pulse generator that attaches to a lead system interfacing with the heart. New implants are almost exclusively implanted transvenously with the pulse generator placed in a left pectoral location much like a conventional pacemaker. Most systems now incorporate a single right ventricular coil with a hot can active pulse generator or combined right ventricular coil, superior vena cava coil, and active pulse generator can. The “hot can”
serves as an active lead within the shocking configuration. Ventricular sensing and pacing are achieved through two “dedicated bipolar” electrodes at the tip of the right ventricular lead (tip/coil) or via “integrated bipolar” electrodes that incorporate a single right ventricular electrode with the right ventricular coil (tip/coil) (Topol et al., 2004).

**Components of ICD:**

1- Power source “lithium iodide battery.

2- Lead system: usually placed transvenously.

3- Bradycardia. Pacing.

4- Antitachycardia-pacing

5- Memory: used to assess efficacy of therapy or to troubleshoot the system.

**Indications:**

An ICD system is indicated for patients who are at high risk of sudden cardiac death due to ventricular arrhythmias. The combined American College of Cardiology (ACC)/ America Heart Association (AHA) task force has most recently published updated guidelines for implantation of cardiac pacemakers and antiarrhythmias devices in 1998. Although it has been updated with several large prospective randomized trials, it recognizes the ongoing evaluation of ICD indications and remains liberal in its guidelines.
Table (10): Criteria for ICD implantation:

(A) Indications

Class I

1- Cardiac arrest due to VF or VT not due to a transient or reversible cause.
2- Spontaneous sustained VT.
3- Syncope of undetermined origin with clinically relevant, hemodynamically significant sustained VT or VF induced at electrophysiologic study when drug therapy is ineffective, not tolerated, or not preferred.
4- Nonsustained VT with coronary disease, prior MI, LV dysfunction, and inducible VF or sustained VT at electrophysiologic study that is not suppressible by a class I antiarrhythmic drug.

Class II

1- Cardiac arrest presumed to be due to VF when electrophysiologic testing is precluded by other medical conditions.
2- Severe symptoms attributable to sustained ventricular tachyarrhythmias while awaiting cardiac transplantation.
3- Familial or inherited conditions with a high risk for life-threatening ventricular tachyarrhythmias such as long QT syndrome or hypertrophic cardiomyopathy.
4- Nonsustained VT with coronary artery disease, prior MI, and LV dysfunction, and inducible sustained VT or VF at electrophysiologic study.
5- Recurrent syncope of undetermined etiology in the presence of ventricular dysfunction and inducible ventricular arrhythmias at electrophysiologic study when other causes of syncope have been excluded.

(B) Contraindications

Class III

1- Syncope of undetermined cause in a patient without inducible ventricular tachyarrhythmias.
2- Incessant VT or VF.
3- VF or VT resulting from arrhythmias amenable to surgical or catheter ablation e.g., atrial arrhythmias associated with the Wolff-Parkinson-White syndrome, right ventricular outflow tract VT, idiopathic left ventricular tachycardia, or fascicular VT.
4- Ventricular tachyarrhythmias due to transient or reversible disorder (e.g., acute MI, electrolyte imbalance, drugs, trauma).
5- Significant psychiatric illness that may be aggravated by device implantation or may preclude systematic follow-up.
6- Terminal illnesses with projected life expectancy ≤ 6 months.
7- Patients with coronary artery disease with LV dysfunction and prolonged QRS duration in the absence of spontaneous or inducible sustained or nonsustained VT who are undergoing coronary bypass surgery.
8- NYHA class IV drug refractory congestive heart failure in patients who are not candidates for cardiac transplantation.

VT, ventricular tachycardia; VF, ventricular fibrillation; MI, myocardial infarction; LV, left ventricular; EPS, electrophysiologic study; NSVT, nonsustained ventricular tachycardia; ICD, implantable cardioverter-defibrillator.

(Topol et al., 2004)
as with any expensive invasive therapy, candidates for ICD implantation should be carefully screened for appropriateness of the prescribed therapy. The patient should be educated regarding the implantation, maintenance, and follow-up of an implantable device. A thorough discussion of potential therapies including AIT, low-energy cardioversion, and shock defibrillation should ensue. The candidate should be allowed to make an informed choice prior to implantation of a device additionally; as devices incorporate expanded pacemaker capabilities, it will be important to choose the appropriate device for the patient (Topol et al., 2004).

<table>
<thead>
<tr>
<th>Table (11): Potential complications of ICD surgery:</th>
</tr>
</thead>
<tbody>
<tr>
<td>I. Complications resulting from the subclavian stick technique.</td>
</tr>
<tr>
<td>Pneumothorax.</td>
</tr>
<tr>
<td>Hemothorax.</td>
</tr>
<tr>
<td>Subclavian artery puncture.</td>
</tr>
<tr>
<td>Air embolism</td>
</tr>
<tr>
<td>Bleeding</td>
</tr>
<tr>
<td>Hemothysis</td>
</tr>
<tr>
<td>Brachial plexus injury</td>
</tr>
<tr>
<td>Subclavian arteriovenous fistula</td>
</tr>
<tr>
<td>II. Surgical complications related to the pulse generator</td>
</tr>
<tr>
<td>Pocket erosion</td>
</tr>
<tr>
<td>Pocket hematoma</td>
</tr>
<tr>
<td>Pocket seroma</td>
</tr>
<tr>
<td>Pocket infection</td>
</tr>
<tr>
<td>III. Surgical complications related to the ICD leads:</td>
</tr>
<tr>
<td>Lead dislodgement</td>
</tr>
<tr>
<td>Lead perforation</td>
</tr>
<tr>
<td>Loose set screw</td>
</tr>
<tr>
<td>Failure to isolate the set screw</td>
</tr>
<tr>
<td>Microdislocation</td>
</tr>
<tr>
<td>Malposition</td>
</tr>
<tr>
<td>Diaphragmatic stimulation</td>
</tr>
<tr>
<td>Exit block</td>
</tr>
<tr>
<td>Conductor fracture</td>
</tr>
<tr>
<td>Insulation break</td>
</tr>
<tr>
<td>Venous thrombosis</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
</tr>
</tbody>
</table>
Contraindications:

Implantation of an ICD is contraindicated in any patient who has a remedial cause of ventricular arrhythmias such as acute myocardial infarction, myocardial ischemia, electrolyte imbalance, drug toxicity, hypoxia, or sepsis. Transient ventricular tachyarrhythmias secondary to electrocution or drowning are not indications for ICD placement. Patients with incessant ventricular arrhythmias should not receive an ICD. Additionally, an ICD is contraindicated in any patient with recurrent syncope of undetermined cause without inducible ventricular tachyarrhythmias (Topol et al., 2004).

An ICD system consists of a pulse generator and leads for tachydysrhythmias detection and therapy. ICDs provide antitachycardia and antibradycardia pacing; synchronized (cardioversion) or nonsynchronized (defibrillation) shocks; telemetry; and diagnostics, including stored event electrograms and history logs. Essentially, the pulse generator is a self-powered computer within a hermetically sealed titanium casing (can). One or two in series) 3.2-V lithium-silver vanadium oxide (SVO) batteries with high power density are used to power the pulse generator, circuitry, and aluminum electrolytic storage capacitors. Most ICD designs use two capacitors in series to achieve a maximum voltage for defibrillation. A major challenge in ICD design is the large range of voltages that must be controlled in a very small package. Although monitored intracardiac signals may be as small as 100μV, therapeutic defibrillatory shocks approach 750 V, with a leading edge of 15 amperes (A) and a pulse termination spike of 210 A.

Modern ICDs use transvenous lead systems for sensing, pacing and shocks. Epicardial leads are still used in infants and small children.
Ventricular demand pacing for bradycardia is a standard feature of all single-chamber ICDs. Dual-chamber ICDs have all the capabilities of dual-chamber pacemakers, including adaptative-rate pacing and automatic mode-switching.

Current ICDs have many programmable features, but essentially they measure each cardiac R-R interval and categorize the rate as normal, too fast (short R-R interval). Time (all programmable), it will begin an antitachycardia event. The internal computer and shock. If shock is chosen, an internal capacitor is charged. Most devices are therapy. Typically, ICDs deliver no more than 6 shocks per event, although some can deliver as many as 18. once a shock is delivered, no further antitachycardia pacing can take place. The energy delivered may be programmed to a maximum of 36 joules each shock (Atlee, L, Bernstein AD, 2001 and Rozner MA, 2001).

The NASPE/BPEG generic defibrillator (NBD) code:

Like pacemakers, ICDs have a generic code to indicate lead placement and function. The NBD code is shown in table, however, the most robust form of identification, called the "label form" replaces the fourth letter of the NBD with the appropriate generic pacemaker code.

<table>
<thead>
<tr>
<th>Letter I</th>
<th>Letter II</th>
<th>Letter III</th>
<th>Letter IV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shock chamber (S)</td>
<td>Antitachycardia pacing chamber (S)</td>
<td>Tachycardia detection</td>
<td>Antibradycardia pacing chamber (S)</td>
</tr>
<tr>
<td>O = None</td>
<td>O = None</td>
<td>E = Electrogram</td>
<td>O = None</td>
</tr>
<tr>
<td>A = Atrium</td>
<td>A = Atrium</td>
<td>H = Hemodynamic</td>
<td>A = Atrium</td>
</tr>
<tr>
<td>V = Ventricle</td>
<td>V = Ventricle</td>
<td>(not yet available)</td>
<td>V = Ventricle</td>
</tr>
<tr>
<td>D = Dual (A+V)</td>
<td>D = Dual (A+V)</td>
<td></td>
<td>D = Dual (A+V)</td>
</tr>
</tbody>
</table>

BPEG, British pacing and electrophysiology Group; NASPE, American society of pacing and electrophysiology; NBG, North American and British Genetic.
There are still patients with both an ICD (same as PCD, but without back-up pacing) and permanent pacemaker (Epstein AE, Wilkoff BL, 1993).

Table (12) Possible interactions between internal cardioverter-defibrillators (ICDs) and pacemakers

1- Transient failure of the pacemaker to sense or pace following ICD discharge.
2- Sensing of pacemaker stimuli by the ICD with inappropriate ICD discharge.
3- Oversensing of pacemaker stimuli during VF prevents appropriate ICD discharge.
4- Reprogramming or nonintended pacemaker function caused by CD discharge.
5- Antitachycardia pacing modes trigger inappropriate ICD discharge.

VF = ventricular fibrillation.

(Kaplan et al., 1999)
Ventricular Assist Devices
VENTRICULAR ASSIST DEVICES

Ventricular assist devices (VADs) are true pumps that temporary substitute the left (LVAD), right (RVAD), or both ventricles (Anstadt and Lowe, 1995).

Vascular access is similar for most VAD systems (Pennington and Swartz, 1990; Hahn et al., 1993; Lick et al., 1993 and Magovern et al., 1993). LVADs are most commonly used owing to the predominance of left ventricular failure. In these circumstances, an inlet cannula is positioned in the left atrium or left ventricle and a return cannula in the ascending aorta. Biventricular support requires that an additional set of inlet and outlet cannulas be placed in the right atrium and pulmonary artery. These later cannulation sites are also employed during isolated right ventricular support (Sabiston and Spencer, 1995).

Currently available devices differ in configuration, anatomic locations, flow characteristics, and durability. Many devices are categorized as follows (Chu and Hsu, 2000):

1- Pulsatile or continuous blood flow.
2- Internal (implantable) or external (extracorporeal).
3- Pneumatically or electrically powered.
4- For short-term or long-term support.

Clinical experience has been obtained with ventricular support using four types of VADs which include roller pumps, centrifugal pumps, pneumatic pulsatile pumps, and electric pulsatile pumps (Pae et al., 1996).
I) Roller Pumps:
roller pumps are simple, and inexpensive, but they require systemic anticoagulation, produce nonpulsatile flow, and induce significant blood trauma (Janelle et al., 2002).

Components of the VAD system using roller pump:
The system consists of venous cannula, silicone rubber medical grade pump tubing, and a portable roller pump (Rose et al., 1985).

II) Centrifugal pumps:
Of the various ventricular assist devices, centrifugal pumps have been used most commonly (Pae et al., 1992). They are relatively simple to operate, inexpensive compared with other mechanical assist devices, and less destructive to blood cellular elements compared with roller pumps (Curtis et al., 1999).

Centrifugal pumps are extracorporeal, nonpulsatile E -VADs used in parallel with the native ventricles. These devices are capable of providing outputs of 2 to 6 L/min (Dinardo, 1998).

Description:
Worldwide, numerous centrifugal pumps are in development for clinical use. However, in the United States, only three centrifugal pumps have been commercially available. All are disposable, inexpensive, and simple to operate.

These centrifugal pumps are:
1- The Sarns centrifugal pump.
2- The Saint Jude Medical lifestream centrifugal pump.
3- The Biomedicus BioPump centrifugal pump.
The carmeda BioMedicus BioPump (referred to as BioActive BioPump) has the same appearance as the Biopump, but has heparin bonded to the blood exposed surfaces.

Each of these four disposable pump heads can be magnetically coupled to an electric motor, which is controlled by a computerized console. Control of flow is accomplished by adjusting the revolutions per minute of the spinning pump head (*Curtis et al., 1999*).

**III) Pneumatic pulsatile pumps:**

They are complex, air-driven, pulsatile VADs, which are considerably more expensive than a centrifugal pump but are capable of producing pulsatile flow with no trauma to formed blood elements. Furthermore, integral sophisticated control systems are largely self regulating, and beyond the first few days after device insertion, minimal supervision is required. As drive units become more refined and portable drivers are developed, patient mobility and lifestyle are improved (*Braunwald et al., 2001*). Advantages are significant in that the design may decrease the need for systemic anticoagulation (*Pae et al., 1996*).

**Abiomed BVS 5000 Biventricular support system:**

The Abiomed BVS 5000 is an extracorporeal pulsatile BiVAD used in parallel with the native ventricles (*Jett, 1996*). The device has an effective stroke volume of 80 ml and a cardiac output of 5 l/min. It has a pneumatic driveline attached to a computerized control console on wheels.
The device consists of atrial and ventricular chambers arranged vertically in a rigid polycarbonate housing. The atrial and ventricular chambers are polyurethane bladders. The ventricular chambers contain polyurethane unidirectional trileaflet inflow and outflow valves.

**HeartMate 1000 IP LVAD:**

The HeartMate LVAD is a pulsatile pneumatic implantable bloodpump made of titanium with a polyurethane diaphragm backed by a pusher plate actuated by external pneumatic driver (*Poirier, 1997*).

**Thoratec VAD system:**

The thoratec VAD is the only VAD approved by the FDA for treatment of postcardiogenic shock and as a bridge to cardiac transplantation (*Holman et al., 1994; McBride et al., 1999* and *Korfer et al., 2000*).

**IV) Electric pulstatile blood pumps:**

In the past few years great progress has been made in the development and clinical application of electric VADs. The current generation of electric blood pumps are intracorporeal devices that are capable of providing months, or even 1 or 2 years, of ventricular support. These devices provide left ventricular apex-to-aortic left ventricular assistance and are not designed for right ventricular assistance. Electric VADs are powered by a highly portable external controller and battery pack. These devices employ a percutaneous driveline that connects the intracorporeal blood pump to the external electronics (*Braunwald et al., 2001*).
Novacor N100 LVAD:

The Novacor N100 LVAD is an implantable, electrically powered, wearable system that has been successfully used as a bridge to transplant since 1984. This device contains a polyurethane blood sac that is compressed by dual, symmetrically opposed pusher plates. The pump is actuated by an energy converter. The blood pump and energy converter are contained within a lightweight fiberglass/epoxy housing that is implanted in a peritoneal position in the left upper quadrant of the patient’s abdomen with the percutaneous driveline positioned in the right lower abdominal wall. The inflow and outflow conduits contain unidirectional bioprosthetic valve. Patients require full anticoagulation with sodium warfarin (Robbins and Oyer, 1999).

HeartMate VE LVAD:

It is a pulsatile, electric, implantable blood pump similar to that employed in the pneumatically powered ventricular assist system produced by the same manufacturer (Frazier et al., 1998). It is powered by an internal, electric motor that rotates once for each pulse, during which the pusher plate of the diaphragm is displaced. Air displaced by the movement of the diaphragm within a nondisplaceable housing is vented to the atmosphere through a percutaneous vent line. The external controller is connected to the internal pump through a percutaneous lead, textured as with the pneumatic version. Two batteries provide power for 4 to 7 hours depending on the age of the batteries and pumping conditions. The vent line is incorporated into the percutaneous lead and serves the dual purpose of venting or pneumatic actuation.
Arrow LionHeart LVAD:

It is completely implantable, sealed system developed at the Pennsylvania State University and Arrow International. It contains a segmented polyurethane blood sac that is contained in a rigid housing (Pierce et al., 1993). An inlet and outlet valves provide unidirectional blood flow. The blood sac is compressed by a pusher plate driven by a direct current motor. Air displaced from the pump housing during VAD diastole is managed by a polyurethane compliance chamber. Control electronics and a 30 minute battery pack are contained in an implantable canister that receives power from a subcutaneous energy transmission coil. The external battery pack carried by the patient transfers energy to the implanted coil using transcatheter energy transmission system (TETS) (Weiss et al., 1989).

V. Other Types of VAD:

1) Hemopump:

The Hemopump or Nimbus pump is a nonpulsatile rotary pump that uses axial flow technology to withdraw blood from the left ventricle and pump it into the aorta. It is a miniature axial flow pump at the end of a catheter which can provide flow rates up to 3.5 L/min. the pump is inserted through a 12 mm woven graft that is sutured to a femoral or iliac artery. The cannula is advanced into the aorta, across the aortic valve, and the tip is positioned at the apex of the left ventricle under fluoroscopic guidance. the axial pump sits in the descending aorta in a 7 × 16mm cylindrical housing at the end of a 20cm long flexible infl ow cannula. It aspirates blood from the left ventricle and pumps it directly into the descending aorta (Rosado et al., 1996).
2) Jarvik 2000 VAD:

The Jarvik 2000 VAD is an electrically powered, intraventricular, axial flow LVAD. It has been designed to normalize the cardiac output by augmenting the function of the chronically failed heart for extended periods. Clinical trials have recently begun. The Jarvik 2000 blood pump is constructed of titanium and has a 16mm outflow graft. The pump weight 90 g, measures 2.5 cm in diameter, and displaces 25 ml.

3) MicroMed BeBakey VAD:

It is a miniature implantable axial blood flow pump for use as a VAD. The pump is 1.2 inches in diameter, 3 inches in length and weights 95 grams. It is the first long-term axial flow circulatory assist device to be introduced into clinical trials as a bridge to transplantation. Clinical trials began in Europe in November 1998 and in the United States in June 2000 (Noon et al., 2001). The MicroMed DeBakey VAD could go from the left atrium to the descending aorta or from the left ventricular apex to either the ascending or descending thoracic aorta (Franco, 1999).
Indications VADs:

Indications of VADs include postcardiotomy cardiogenic shock, bridge to cardiac transplantation, acute myocardial infarction, and long-term bridge to recovery (Braunwald et al., 2001).

Table (13): Ventricular assist devices indications and contraindications:

<table>
<thead>
<tr>
<th>Indications for LVADs:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Post-CPB cardiogenic shock with inadequate response to pharmacologic support and IABP.</td>
</tr>
<tr>
<td>- Bridge to heart transplantation.</td>
</tr>
<tr>
<td>- Cardiogenic shock with myocardial infarction.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indications for RVADs:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Isolated post-CPB right ventricular failure.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Indications for biventricular assist devices:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Severe post-CPB biventricular failure.</td>
</tr>
<tr>
<td>- Severe biventricular failure after myocardial infarction.</td>
</tr>
<tr>
<td>- Failure of LVAD to adequately improve, haemodynamics as result of right ventricular failure.</td>
</tr>
<tr>
<td>- Bridge to transplantation.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Contraindications to VADs:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Nor reasonable expectation of ventricular recovery in a patient not considered a candidate for heart transplantation.</td>
</tr>
<tr>
<td>- Sepsis.</td>
</tr>
</tbody>
</table>

(Janell et al., 2002)

Complications of VADs:

Patients who require mechanical circulatory support are critically ill. Thus, complications that occur during the period of mechanical ventricular assistance may be related to the patients clinical conditions prior to VAD insertion, the device implantation procedure or develop postoperatively either as a result of the patient’s hospitalization or device malfunction. Early complications occur in the operating room at the time of blood pump insertion or in the immediate postoperative period. Late complications occur any time the patient is being supported with a VAD (Richenbacher, 1999).
Table (14): Early complications of VADs:

<table>
<thead>
<tr>
<th>Inadequate LVAD flow:</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Hypovolemia.</td>
</tr>
<tr>
<td>- Right ventricular dysfunction.</td>
</tr>
<tr>
<td>- LVAD inflow cannula obstruction.</td>
</tr>
<tr>
<td>Right ventricular failure.</td>
</tr>
<tr>
<td>Patent foramen ovale with systemic desaturation.</td>
</tr>
<tr>
<td>Haemorrhage.</td>
</tr>
<tr>
<td>Thromboembolism.</td>
</tr>
</tbody>
</table>

(Richenscher, 1999)

Table (15): Late complication of VADs:

<table>
<thead>
<tr>
<th>Thromboembolism.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection.</td>
</tr>
<tr>
<td>- Not related to the VAD.</td>
</tr>
<tr>
<td>- VAD related:</td>
</tr>
<tr>
<td>- Percutaneous driveline or cannula colonization.</td>
</tr>
<tr>
<td>- Pocket infection (implantable VADs)</td>
</tr>
<tr>
<td>- Endovascular.</td>
</tr>
<tr>
<td>Multisystem organ failure.</td>
</tr>
<tr>
<td>Pump related:</td>
</tr>
<tr>
<td>- Device malfunction.</td>
</tr>
<tr>
<td>- Costal margin pain.</td>
</tr>
<tr>
<td>- Gastrointestinal compression (implantable VADs)</td>
</tr>
<tr>
<td>Pump dependency.</td>
</tr>
</tbody>
</table>

(Richenscher, 1999)
Intraaortic Balloon Pump
INTRAORTIC BALLOON PUMP

Intraaortic balloon pump (IABP) counterpulsation is a technique that provides haemodynamic support and/or control is haemia both before and after surgery. In contrast to most inotropic agents, IABP provides physiologic assistance to the failing heart by decreasing myocardial oxygen demand and improving coronary perfusion. (Torchiana et al., 1997).

Device description:

The IABP consists of a catheter-mounted balloon, a pump, a gas source, and a microprocessor console.

- The balloon:

The balloon is thin-walled, cylindrical-shaped tube about 2cm x 20 cm) (Dinardo, 1998). It is formed of distensible non thrombogenic polyurethane. The ideal balloon should be 90-95% of the diameter of the aorta when inflated. If is too large, aortic intimal damage or balloon perforation may result without increasing in the efficiency of counterpulsation and, if it is too small, there will be ineffective counterpulsation. The balloon is available in sizes ranging from 35-50 ml; the standard size balloon is 40 ml. the optimal position of the cephalic tip of the balloon is 1-2 cm distal to the origin of the left subclavian artery and the caudal tip of it should be above the origin of the renal arteries (Hartnett and Gaffney, 1993).
- The catheter:

The balloon is mounted on a vascular catheter available in sizes ranging from 8.5 to 12 French. It has multiple pores through which the gas shuttled from the balloon pump console escapes from the catheter to inflate the balloon. The tip of the catheter is radiopaque rectangle, 3x4 mm (Quaal, 1993). The catheter has a hollow central lumen, which allows percutaneous placement over a guidewire, as well as recording of central aortic pressure (Richenbacher, 1999).

- The pump, gas source and console:

The pump inflates and deflates the balloon at precisely timed intervals, with either CO₂ or helium as the inflating gas. CO₂ carries less of an embolic risk if the balloon ruptures because it is absorbed quickly. Helium has a lower density, allowing faster inflation and deflation, and currently is the preferred gas (Dinardo, 1998).

The console itself consists of a pressurized gas reservoir, a monitor for electrocardiogram (ECG) and pressure wave recording, adjustments for inflation/deflation timing, triggering selection switches and battery back-up power sources (Oberwalder, 1999).

Principles of IABP counterpulsation:

Counterpulsation is the term that describes balloon inflation in diastole and deflation during isometric contraction of early systole. In a mechanical sense, balloon inflation causes volume displacement. Blood volume around the IAB is displaced both proximally and distally concomitant with balloon inflation. Balloon inflation can be conceptualized as "compartmentalizing" the aorta. The proximal
compartment includes aortic root and coronary arteries. The proximal compartment includes aortic root and coronary artery. The aortic segment extending beyond the distal balloon tip together with the systemic circulation comprise the distal compartment (Qua I, 1993). The IAB is positioned in the descending thoracic aorta and operates displacing blood within the aortic lumen. Balloon inflation immediately after aortic valve closure augments the diastolic blood pressure, thereby increasing systemic and coronary artery perfusion pressure. Balloon deflation immediately before ventricular ejection decrease the intraaortic pressure, thereby reducing the impedance to left ventricular ejection (Cheung et al., 1996).

The net effect is a favorable shift in myocardial oxygen supply-oxygen demand ratio, with a small increase in systemic perfusion (Braunwald et al., 2001). Unlike the VAD and TAH, the IABP is not designed to completely replace the function of the native ventricle. Rather, the IABP function in concert with the native heart (Richenbacher, 1999).

**Insertion of IAB:**

Early balloon catheter design required a surgical cutdown into the femoral or iliac artery. IAB insertion by this technique is associated with a high rate of femoral vascular complications and marked prolongation of insertion time and required subsequent operative removal of the surgically inserted catheter. Advances in balloon catheter design now permit percutaneous insertion of IAB catheter (Diver, 1993).

Percutaneous insertion is performed by Seldinger technique, placing the balloon through a sheath and over a guidewire. The sheath
can be left in place or removed from the artery. Sheathless insertion minimizes occlusion of the femoral vessels, but can lead to balloon shearing in patients with significant aortoiliac atherosclerosis (Shinn and Joseph, 1998).

**Routes and techniques of IAB insertion:**

There are different routes for insertion of IAB including:

- Femoral arterial route.
- Balloon catheter insertion by the ascending aorta or aortic arch.
- Other insertion sites.

**A- Femoral arterial route:**

The IAB is inserted most commonly through the femoral artery (Hazlerigg et al., 1992). It can be inserted percutaneously at the patient’s bedside. Unfortunately, this approach requires the patient to remain on bed rest with head of the bed elevated no more than 30 degrees (Stavarski, 1996). Also, it is associated with high risk of limb ischaemia in patients with known peripheral vascular disease (Kvilekual et al., 1991).

**1- The standard percutaneous technique for IAB insertion (Sheathed insertion):**

Since 1979, a percutaneous placement of IAB via the femoral artery using a modified Seldinger technique allows an easy and rapid insertion in the majority of situations (Oberwalder, 1999). By this method, IAB can be inserted by the invasive cardiologist, the surgeon, or the critical care specialist using reliably prepackaged, sterilized devices which became available after 1981 (McKiernan and Wehramacher, 1997).
Table (16): Equipments needed for percutaneous IAB insertion:

<table>
<thead>
<tr>
<th>Standard equipment</th>
<th>Sterile insertion kit (items included may vary slightly among manufacturers)</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Sterile 4x4 sponge.</td>
<td>- 18-gauge angiographic needle.</td>
</tr>
<tr>
<td>- Ring forceps.</td>
<td>- J-tipped 120 or 145-cm guidewire.</td>
</tr>
<tr>
<td>- Povidone-iodine solution in sterile container.</td>
<td>- 8-French dilator.</td>
</tr>
<tr>
<td>- 1% or 2% lidocaine without epinephrine.</td>
<td>- Introducer sheath with haemostasis valve.</td>
</tr>
<tr>
<td>- Assorted needles and syringes.</td>
<td>- Introducer dilator.</td>
</tr>
<tr>
<td>- Three-way stopcock.</td>
<td>- Three-way stopcock.</td>
</tr>
<tr>
<td>- Heparinized saline in sterile bowl.</td>
<td>- 60-cc syringe.</td>
</tr>
<tr>
<td>- No. 11 scalpel.</td>
<td>- Male leur plug.</td>
</tr>
<tr>
<td>- 2-0 silk suture.</td>
<td>- Pressure tubing extension.</td>
</tr>
<tr>
<td>- ECG and pressure monitoring equipment.</td>
<td>- Balloon pump connector.</td>
</tr>
<tr>
<td>- Defibrillator and cardiac arrest cart.</td>
<td></td>
</tr>
<tr>
<td>- Portable fluoroscopy if insertion is at bedside.</td>
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</tr>
</tbody>
</table>

(Quaal, 1993)

2- The surgical cutdown insertion (open insertion technique):

The femoral artery is exposed and a longitudinal arteriotomy is created. A 5 cm segment of 8-10 mm diameter vascular graft is anastomosed in an end-to-side fashion to the femoral artery at a 45-degree angle. The IAB is passed through the vascular graft and into the artery and positioned as described previously. The IAB is fixed in position by tying umbilical tapes around the vascular graft. The central catheter is brought through the most caudal aspect of the skin incision and the wound is closed in layers. The vascular graft should be located deep to the skin closure (Richenbacher and Pierce, 2001).


*Hebeler (1989)* suggested direct placement of a percutaneous sheath in the femoral artery under direct vision through a purse-string suture after exposure of the artery, rather than placement through an end-to-side vascular graft.

3- **Sheathless balloon insertion:**

Sheathless percutaneous insertion of IAB catheters is a technique that reduces the effective catheter diameter by elimination of the arterial sheath and may decrease the rate of vascular complications associated with insertion using a sheath (*Diver, 1993*).

A sheathless insertion technique is employed in a non-anticoagulated patient. Haemostasis at the puncture site is not as good with sheathless insertion as it is with insertion using a sheath (*Richenbacher, 1999*).

**Advantages of sheathless balloon insertion:**

Elimination of the arterial sheath from the balloon counterpulsation insertion technique confers a significant, clinically relevant reduction ineffective device diameter (*Diver, 1993*). Reduction in balloon shaft diameter results in a decrease in the vascular complication rate associated with IAB counterpulsation. This technique minimizes the constriction to blood flow in the femoral artery facilitating IAB use in patients with known or suspected peripheral vascular disease (*Braunwald et al., 2001*).

**Contraindications to sheathless insertion:**

Sheathless IAB insertion should not be attempted in patients with severe scarring or fibrosis at the femoral access sites or with obesity. The
likelihood of successful sheathless insertion is decreased in such patients, therefore balloon catheter insertion should be attempted through a sheath by the standard percutaneous technique (Diver, 1993).

B- Balloon catheter insertion by the ascending aorta or aortic arch:

If an abdominal aortic aneurysm or peripheral vascular disease precludes femoral arterial insertion, the IAB may be inserted directly into the ascending aorta or transverse aortic arch (Santini and Mazzucco, 1997).

These techniques are usually reserved for intra or postoperative patients. Numerous technical maneuvers have been used successfully to allow safe and easy insertion as well as nonoperative removal. A simple technique that does not require reexploration for removal may be preferred. A 10 to 12-mm aortotomy is made over a partial occlusion clamp and woven Dacron graft is beveled and sewn to the aortotomy. The IAB catheter is threaded distally through the graft while the partial occlusion clamp is removed (Pae et al., 1996).

C- Other insertion sites:

In desperate situations, the IABP may be inserted through the indwelling suprainguinal bypass grafts (LaMuraglia et al., 1991), the ascending aorta, the axillary artery, the subclavian artery and the common iliac artery (Buchanan et al., 1994), but complications are more frequent. Insertion via axillary route has been advocated to allow patient mobility when an IAB is a bridge to transplantation (Pae et al., 1996)
Timing and trigger of IABP:

Timing and trigger are terms often confused when working with IABPs. It is important that the operator understands these terms.

Timing: is the relationship between the balloon inflation and deflation, and the heart systole and diastole.

Trigger: is the events or signals, whether from the ECG waveform, the arterial pressure waveform, or the intrinsic pump rate, which are used to institute inflation and deflation of the balloon (Halligan et al., 1997).

Timing:

To achieve optimal effect of counterpulsation, inflation and deflation need to be correctly timed to the patient’s cardiac cycle. Timing of balloon inflation and deflation is best adjusted with the pump frequency set to assist every other beat. The beat ratio of 1:2 facilitates comparison between the patient’s own ventricular beats and augmented beats to determine ideal IABP timing. It is important that, for proper timing, the inflation of the IAB occurs at the beginning of diastole just after closure of the aortic valve, which is represented on the arterial pressure waveform as the dicrotic notch. Deflation of the balloon should occur immediately prior to the arterial upstroke (Oberwalder 1999).

Use of the central aortic pressure tracing from the balloon lumen is preferable to peripheral arterial monitoring because of the delay and change in arterial contour of the pulse wave in peripheral vessels. So, IABP inflation and deflation must be set earlier in the arterial trace when this trace is taken from a site distant from the aortic root (Richenbacher, 1999).
- Effects of proper timing:

The effect of proper timing of the IAB counterpulsation is two fold. During inflation there is an increase in diastolic pressure due to the displacement of volume. This increased diastolic pressure leads to increased coronary perfusion. When deflation occurs, there is a sudden decrease of pressure within the aorta that causes a decreased aortic end diastolic pressure. This decreased end diastolic pressure equals decreased afterload. Afterload reduction causes a reduction in myocardial work, oxygen consumption and in cardiac output (Halligan et al., 1997).

Loss of optimal haemodynamic benefit will occur with inappropriate timing of balloon inflation and deflation, which is determined from the lack of significant haemodynamic response and from inspection of the central aortic pressure tracings (Brown, 198).

Methods of triggering:

1) The ECG waveform:

The most common method of triggering the IAB is from the R wave of the patient’s ECG signal. Input to the balloon console is provided from the skin leads or the bedside monitor. Mainly balloon inflation is set automatically to start in the middle of the T wave and the deflate, prior to the ending QRS complex. Tachydysrhythmias, electrocardiery, cardiac pacemaker function, and poor ECG signals may cause difficulties in obtaining synchronization when the ECG mode is used. In such cases the arterial pressure waveform for triggering may be used (Oberwelder, 1999).

Dysrhythmias:

Dysrhythmias timing the balloon for irregular rhythms is difficult and the circulatory support provided by the balloon is compromised; in
these patients attempts are made to convert the patient to a sinus or paced rhythm or to slow (80-90 beats/min) atrial fibrillation using the appropriate drugs or cardioversion. For tachycardias over 110-120 beats/min the balloon is timed to provide inflation on alternate beats if the machine is not able to reliably follow each beat (McCarthy and Golding, 1977).

Electrocautery:

Electrocautery interferes with the ECG and therefore interferes with the proper triggering the timing. Most balloon consoles incorporate electrosurgical interference suppression (ESIS) which theoretically allows the balloon to function in the ECG mode during the use of electrocautery. In practice, ESIS is not particularly effective, and it is simpler to change the trigger source to the arterial pressure trace when electrocautery is employed (Richenbacher, 1999).

Cardiac pacemaker:

The atrial pacemaker spike can make difficulties that interfere with triggering the timing. The atrial pacemaker spike may cause premature inflation of the balloon because it can be interpreted as the QRS complex. The solution for this problem is to use the atrial pressure wave as the triggering source or to pace the atrium in the bipolar mode. The later requires two atrial leads and results in much smaller pacemaker spike (Williams, 1993).

2) The arterial pressure waveform:

inflation should occur at the dicrotic notch with deflation just before the onset of the aortic upstroke. This method is used as an alternative for ECG signals and it is specially useful in operating room.
where electrocautery may interfere with the ECG signal: *(Shinn and Joseph, 1998).*

3) **The intrinsic pump rate:**

Some balloon pumps offer an internal trigger mode which provides continuous IAB counterpulsation at a preselected heart rate varying between 40 and 120 pulses per minutes and does not require an external trigger signal. The internal trigger is utilized during CPB to provide some degree of pulsatility to the systemic blood flow. The balloon console should never be placed in the internal trigger mode if the patient is generating cardiac output. Should the patient lose arterial blood pressure as in arrest situation, the IABP console is placed on an internal trigger mode *(Richenbacher, 1999).*

**Weaning from the IABP support:**

Weaning from IABP is defined as the process of bringing about a physiological transition to the condition in which cardiac action is no longer supported by mechanical circulatory assistance. Urgent situations that necessitate immediate interruption of removal of IAB as severe ischaemia of the leg through which the balloon was inserted or leak in the balloon, the IABP is removed and is often resumed using the other extremity for insertion of a new IAB catheter *(Kantrowitz et al, 1993).*

**Criteria of readiness for weaning:**

**Clinical parameters:**

1- Blood gas values, fluid volume, temperature, cardiac rhythm and haemoglobin, haematocrite, and electrolyte levels must all have been corrected.

2- Urinary output must be greater than 0.5 cc/kg/hr.
Haemodynamic parameters:
1- Systolic arterial pressure must be greater than 90 mm Hg.
2- Mean arterial pressure must be greater than 70 mmHg.
3- Cardiac index must be greater than 2.1 L/min/m².
4- Systemic vascular resistance must be less than 2100 dyne/sec/cm².
5- Pulmonary capillary wedge pressure must be greater than 18 mmHg.

Techniques of weaning:
Two methods of weaning are currently recommended, namely frequency ratio and volume weaning. However, allowance must be made for the need to stop heparin administration four hours before removal of the IABP from the patient and platelet count must be checked prior to removal of IAB.

1- Frequency ratio weaning:
In this method, the ratio of assisted to total numbers of heart beats is reduced in a stepwise fashion, from 1:1 to 1:2 to 1:3 and so on. If no haemodynamic deterioration occurs after 15 to 20 minutes at a given assist ratio, then weaning can continue until the balloon pumping is finally stopped. There are studies suggest this type of weaning is not giving the patient intermediate levels of balloon assistance due to the rapid changes in the afterload reduction between assisted and unassisted beats. This is considered by these studies to be the same as stopping the balloon. However, this method is commonly used today and recommended by some balloon manufactures.
2) Volume weaning:

with this method the balloon volume is decreased by a small portion (20-25% of the total volume). Assistance is continued for 15 to 30 minutes while the patient’s haemodynamic parameters are observed closely. If the patient tolerates this reduction, the volume is reduced by approximately 20% and so on. However, the balloon volume is not reduced below approximately 20% of its initial total volume.

This method does not cause the rapid change in the after load reduction. So, it is more physiological way of weaning. Although, some balloon manufacturers do not recommend this method due to concerns of clot formation because of decreased inflation, this method was not considered a problem as the balloon volume is not reduced below 20% of its total volume (Halligan et al., 1997).

Criteria of successful weaning:

1- Haemodynamic parameters within normal range with clinical evidence of adequate perfusion.
2- Satisfactory clinical condition for several hours after the IABP is turned off (Kantrowitz et al. 1993).

A patient who can not be weaned is balloon dependent. For this patient in whom definitive surgical treatment has been excluded, the other possibilities include cardiac transplantation and a permanently implanted left ventricular assist device (Kantrowitz et al., 1993).
IABP removal:

For removal of IABP the following points should be considered:

1- Discontinuation of the anticoagulant (Richenbcher, 1999). If the patient is fully anticoagulated and balloon removal is required immediately due to urgent situation, the balloon catheter and sheath are removed over a guidewire and a new femoral sheath is inserted to maintain haemostasis (Brown, 1998).

2- Criteria of successful weaning should be fulfilled before IBP removal.

3- The insertion site is prepped and in a sterile fashion.

4- The individual with drawing the balloon should wear a gown, gloves and protective eye wear due to the potential for gushing of blood during IABP removal (Richenbcher, 1999).

5- Conscious patients should receive a narcotic agent (e.g., meperidine 25 mg intravenously).

6- The securing sutures are cut.

7- IAB pumping is stopped (Mahaffey et al., 1998).

According to the routes and techniques of insertion and the condition of the patient, the removal of IABP may be done through one of the following techniques:

A) Percutaneous balloon removal:

This technique is used for removal of IABP inserted by standard percutaneous technique and not complicated by limb ischaemia. In this technique the balloon is disconnected from the console and completely deflated using a 50 ml syringe. The IABP catheter is withdrawn through the introducer sheath. Then, the balloon and sheath are withdrawn as a unit (Richenbcher, 1999).
B) Open IABP removal:

The indications for open IABP removal are:

1- Open IABP insertion.
2- Proximal percutaneous insertion in a morbid obese patient.
3- IAB counterpulsation complicated by limb ischaemia.
4- Transthoracic balloon insertion (Richenbacher, 1999).

Removal of an IABP inserted surgically through a femoral artery graft necessitates reopening of the surgical wound. The ligature securing the graft to balloon is cut. The balloon is disconnected from the driving console and is evacuated using a syringe. After balloon removal, the vascular graft is removed in its entirety and a Fogarty catheter is sometimes passed into the distal femoral artery to remove any suspected thrombi. The femoral arteriotomy is usually closed by a saphenous vein patch (Richenbacher, 1999).
Indication of intraaortic balloon pump:

Table (17): IABP counterpulsation indications

Left ventricular failure or cardiogenic shock:
- Myocardial infarction.
- Cardiomyopathy.
- Severe myocardial contusion.
- Septic shock.
- Drug induced.

Mechanical complications of acute myocardial infarction.
- Mitral regurgitation.
- Ventriaclar septal defect.

Post myocardial infarction ventricular irritability.
Unstable angina refractory to medical therapy.
Support for high risk PTCA patients.
Failed PTCA.
Thrombolytic therapy of acute myocardial infarction.
Failure to wean from CPB.
Low cardiac output syndrome.
Stabilization of high risk patients undergoing general anaesthesia.
Bridge to transplant.
Stunned myocardium.

(Oberwald, 1999)

contraindications to IABP:

Table (18): Relative and absolute contraindications to the insertion of IABP

<table>
<thead>
<tr>
<th>Absolute contraindications</th>
<th>Relative contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>- severe aortic insufficiency.</td>
<td>Aortic aneurysm.</td>
</tr>
<tr>
<td>- Dissecting aortic aneurysm.</td>
<td>Sepsis.</td>
</tr>
<tr>
<td>- Malignant dysrhythmias:</td>
<td>Severe vascular disease.</td>
</tr>
<tr>
<td>• Asystole.</td>
<td>Mild aortic insufficiency.</td>
</tr>
<tr>
<td>• Ventricular fibrillation.</td>
<td></td>
</tr>
<tr>
<td>• Pulseless electrical activity.</td>
<td></td>
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</tbody>
</table>

(Smith et al., 2000)
Complications of IABP:

IABP is an effective mean of supporting the circulation. Balloon support has proven to be an effective adjuvant in patients with circulatory instability, allowing time for subsequent diagnosis and selection of appropriate therapy.

Balloon support is not without complications. This risk must be weighed against possible benefits for any particular patient (Pae et al., 1996).

Table (19) intraaortic balloon pump complications:

<table>
<thead>
<tr>
<th>Vascular</th>
<th>Miscellaneous</th>
<th>Balloon</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Arterial injury</td>
<td>- Haemolysis</td>
<td>- Perforation</td>
</tr>
<tr>
<td>(perforation dissection)</td>
<td>- Thrombocytopenia</td>
<td>(puncture)</td>
</tr>
<tr>
<td>- Aortic perforation</td>
<td>- Infection</td>
<td>- Tear (during insertion)</td>
</tr>
<tr>
<td>- Aortic dissection</td>
<td>- Claudication</td>
<td>- Incorrect positioning</td>
</tr>
<tr>
<td>- Femoral artery</td>
<td>- (postremoval)</td>
<td>- Gas embolization</td>
</tr>
<tr>
<td>thrombosis</td>
<td>- Haemorrhage</td>
<td>- Inadvertent removal</td>
</tr>
<tr>
<td>- Peripheral</td>
<td>- Paraplegia</td>
<td></td>
</tr>
<tr>
<td>embolization</td>
<td>- Entrapment</td>
<td></td>
</tr>
<tr>
<td>- Femoral vein cannulation.</td>
<td>- Spinal cord necrosis.</td>
<td></td>
</tr>
<tr>
<td>- Pseudoaneurysm of femoral vessels.</td>
<td>- Left internal mammary artery occlusion</td>
<td></td>
</tr>
<tr>
<td>- Lower extremity ischaemia</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Compartment syndrome</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- Visceral ischaemia</td>
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<td></td>
</tr>
</tbody>
</table>

(Galle et al., 1999)

Predisposing factors:

Female gender, peripheral vascular disease, body surface area < 1.65 m², and age ≥ 75 years are the four prominent, independent predictors of a serious IABP complications (Ferguson et al., 2001). Other risk factors include diabetes, smoking cardiogenic shock, hypertension, and prolonged IABP therapy (Lange, 1993 and Franco, 1999). Percutaneous insertion, sheathless insertion, and insertion using smaller catheter size have less risk for complications when compared with surgical insertion, sheathed insertion, and insertion using larger catheter size (Aroesty, 2000).
Anesthetic Management of Patients With Intrathoracic Gadgets
ANESTHETIC MANAGEMENT OF PATIENTS WITH INTRATHORACIC GADGETS

Primary management of anesthesia in patients with intrathoracic gadgets, pacemakers or ICDs requires attention to their medical and psychological problems, as well as understanding of these pulse generators and their likely idiosyncrasies in the operating or procedure room.

Preoperative evaluation:
Preoperative assessment of the patient

The patient with an implanted pacemaker should receive the same attention afforded to any other patient. Baseline history and physical examination and laboratory evaluation are important. Patients with implantable devices are mostly of ASA grade III and above (Reid and Appleton, 1999).

History is important to evaluate the underlying disease that led to the implantation. The patient should be ischaemic or valvular heart disease, diabetes mellitus, hypertension, and other medical illnesses. Patients with known coronary artery disease (previous infarction or classic angina with a positive stress test), have an increased incidence of re-infarction if the myocardial infarction occurred within 6 months of the proposed surgical procedure (Shah et al., 1990).

Patients with a prior revascularization (CABG) are presumed to be at lower risk. With regard to the optimal length of time between CABG and noncardiac surgery, Cruchley and colleagues (Cruchley et al., 1983).
Anesthetic Management of Patients with Intrathoracic Gadgets

Reported that the rate of morbidity decreased significantly after 3 months. Diabetic patients have a higher probability of CAD than nondiabetic patients. The duration of the disease and other associated endorgan dysfunction should be taken in to account. Diabetic patients are at very high risk for silent angina and myocardial infarction (Borgos et al., 1989).

Hypertension has also been associated with an increased incidence of silent myocardial ischaemia and infarction.

General evaluation of patients with valvular heart disease should be primarily concerned with determining the severity of the lesion and its haemodynamic significance, residual ventricular function, and the presence of secondary effects of pulmonary, renal and hepatic function. Questions should concern exercise tolerance, fatigability and pedal oedema and shortness of breath in general (dyspnea), when lying flat (orthopnea), or at night (paroxysmal nocturnal dyspnea) Morgan GE and Mikail MS, 1996).

History of Drug Intake:

It is important that the patients with intrathoracic gadgets are receiving optimal medical therapy. Knowledge of preoperative antidysrhythmic medications for each individual patient would be useful if intraoperative VT or VF occurs. ICD implantation may obviate the need for long-term antidysrhythmic drug therapy in a large number of patients. However, frequent shocks due to atrial tachyysrhythmias and/or ventricular dysrhythmias will require the initiation of drug therapy in the management of certain patients. In addition to the possibility of decreasing VT rates out of programmed detection zones, therapy with
antidysrhythmic drugs also may cause prodysrhythmia. An increase in ICD therapies correlating with recent initiation of an antidysrhythmic agent should raise this suspicion (Gollob MH and Seger JJ, 2001).

The effect of antidysrhythmic drugs on the minimum energy requirement for successful defibrillation, or defibrillation threshold (DET), requires special attention. At ICD implantation, the DFT is determined and a 10-Joules safety margin typically is added to ensure defibrillation efficacy. Some antidysrhythmic agents may alter the DFT as a result of their electrophysiologic properties.

In general, class IA antidysrhythmic drugs, such as quinidine and procainamide, appear to have little effect on the DFT at therapeutic doses. The short term administration of class IB agents, including lidocaine, phenytoin, and mexiletine, has been shown to increase the DFT. The class III agents (amiodarone and sotalol) are commonly used for the maintenance of sinus rhythm in patients with paroxysmal atrial fibrillation. Their effects on the DFT are disparate. Amiodarone has been shown in several studies to elevate the DFT while sotalol consistently has no effect or lowers the DFT (Brode SE, et al., 1997).

The pharmacologic characteristics of the various antiarrhythmic drugs can affect anesthetic management. Disopyramide is similar to quinidine and procainamide in its antiarrhythmic effectiveness. Disopyramide is mainly excreted by the kidneys, but hepatic disease increases its half life. This drug often produces anticholinergic effects, including tachycardia, urinary retention, and psychosis. Hepatitis has also been reported to have occurred after its use (Roizen MF, 2009).
Little is known of the interaction of bretylium with anesthetic agents. Because bretylium blocks the release of catecholamines, chronic therapy with this drug has been associated with hypersensitivity to vasopressors. Quinidine is dependent on the kidneys for excretion, can have vagolytic effects that can decrease AV block, and is associated with blood dyscrasias and gastrointestinal disturbances (Jones RI, 1996).

Amiodarone causes a peripheral neuropathy and has been associated with hypertension, bradydysrhythmias and reduced cardiac output during anesthesia. No data document such a effect for depolarizing muscle relaxants.

Lack of adverse reports does not indicate that all anti-dysrhythmic drugs should be continued through the time of surgery. Pharamacokinetic studies have not yet determined whether anesthesia (or anesthesia with specific agents) alters the volume of distribution or clearance of these drugs to extent sufficient to warrant changing the dosage or dosage schedule in the perioperative period (Rotzen MF, 2000).

Diuretics can be discontinued to reduce problems associated with hypovolaemia and haemodynamic instability (Kam PCA, 197).

Patients with ICD frequently suffer from depression as a result of stresses associated with frequent discharges of the ICD and may be receiving antidepressant medications.

Antidepressant medications include monoamine oxidase (MAO) inhibitors, and tricyclic antidepressant drugs. MAOIs bind irreversibly to the enzyme MAO, thereby increasing intraneuronal level of amine
neurotransmitters (serotonin, norepinephrine, dopamine, epinephrine, octopamine). Interactions between MAOIs and a variety of foods and drugs containing indirect acting sympathomimetic substances such as ephedrine or tyramine can occur if MAOI is given. The most serious effects of this interaction are convulsions and hyperpyrexic coma (particularly after narcotics). Anesthetic management of a patient given an MAOI can be confusion. For this reason it is widely accepted practice to discontinue MAOIs at least 2 to 3 weeks before any planned operation. Emergency surgery on patients given MAOIs can be punctuated by hemodynamic instability. Regional block can be attempted as treatment for postoperative pain to avoid having to give narcotics (Stack CG, et al., 1988).

Tricyclic antidepressant drugs also block the reuptake of neurotransmitters and cause their acute release. Given chronically these drugs decrease the stores of noradrenergic catecholamines. Tricyclic antidepressant drugs also produce side effects similar to those of atropine (dry mouth, tachycardia, delirium, urinary retention) and can cause changes on ECG (changes in T wave, prolongation of the QRS complex bundle branch block or other conduction abnormalities, premature ventricular contractions). Although dysrhythmias induced by tricyclic antidepressants have been treated successfully with physostigmine, bradycardia has sometimes occurred. Drug interactions with tricyclic antidepressants include fatal dysrhythmias after halothane and pancuronium (Roizen MF, 2000).

The newer antidepressants (the SSRIs) also have serious side effects. Switching between drugs for depression can cause hyperpyrexia and coma. Thus, switching prior to surgery should not be requested casually (Miller AS, 1991).
Clinical assessment:

Performing simple maneuvers such as determining pulse rate and blood pressure are important. A pulse rate that is slower than the pacemaker rate and blood pressure are important. A pulse rate that is slower than the pacemaker rate could indicate a failure of pacing capture, or oversensing. The chest and heart should be auscultated for signs of left ventricular failure and murmurs. Knowing that the patient has carotid bruits and hypertension becomes important, because even a short loss of pacing could lead to decreased cerebral perfusion. Listening for bruits also over the subclavian and femoral arteries and abdominal aorta will give an estimate of the extent of arterial disease (Zaidan JR, 2000).

Investigations:

An extensive laboratory examination usually is unwarranted. Since these patients likely will be taking diuretics and digoxin, consider ordering serum electrolytes. Incidence of pacemaker failure in hyperkalaemia, hypokalaemia, and acidosis have been reported (Anderson C and Madsen GM, 1990).

A chest x-ray distinguishes between a unipolar and bipolar lead system, reveals the course of the lead, and pinpoint the location and possibly the identity of the generator. Comparing past and recent chest x-rays will help determine if the electrode has changed positions within the heart. A change in the position of the electrode could warn of changes in voltage threshold or sensing capabilities. An ECG discloses the patient’s dependence on the pacemaker in addition to reveal pacemaker dysfunction (Zaidan JR, 2000).
Aside from these suggestions, all other laboratory tests should be based on the individual patient’s requirements. They include e.g. stress ECG, echocardiography for assessment of ventricular and valvular function, and coronary angiography (Fleisher LA, 2000).

Clinical evaluation of the pacemaker:

The main reason to evaluate the pacemaker system is to determine if the system will continue to function normally throughout the perioperative period. Anesthetics can perform this evaluation by noting the history and physical examination, by analyzing the ECG, and by placing the magnet over the pacemaker to determine if capture occurs.

While taking the history, note the recurrence of symptoms that lead to implantation of the pacemaker. These symptoms, which include syncope, light headedness, and dizziness, might be secondary to cause other than arrhythmias. The anesthetics should ask the patient about the location of his pulse generator and he should have a look to patient’s ID card with full information about his device parameters.

The physical examination can offer some idea of pacemaker function. A heart rate lower than the pacemaker’s programmed rate has several interpretations.

- The pacemaker could be abnormally functioning by oversensing ECG events. this is solved by reprogramming to a less-sensitive R-wave setting.
- The battery could be depleted.
- The pacemaker could be normally functioning during programmed hysteresis. Hysteresis allows the patient’s intrinsic heart rate to
decrease to a rate lower than the pacing rate and therefore maintaining sinus rhythm for longer periods of time.

(Zaic in JR, 2000)

- During the physical examination the patient is asked to exercise the muscles in the area of the generator to determine if muscle potentials will inhibit the pacemaker. If inhibition occurs, it is possible that the pacemaker also could be inhibited during shivering.
- The ECG gives important information.
- The paced rate should be equal to the programmed rate.
- If the pacemaker is properly sensing, then pacemaker impulses and paced beats will not occur in the ST segment or on the T wave.
- Pacing impulses should be followed by a paced beat. If they are not, then the pacemaker might have lost pacing. In these instances, call the cardiologist.
- The intrinsic heart rate is faster than the programmed rate, and there are no pacing impulses on the ECG, then the pacemaker is probably functioning normally. To determine that the pacemaker can emit impulses a magnet is placed over the pacemaker or the patient is asked to perform Valsalva maneuver to lower the intrinsic heart rate below the paced rate to determine if pacing impulses appear (Zaidan JR., 2000).

For programmable devices, interrogation with a programmer remains the most reliable method for evaluating lead performance and obtaining current program information. Appropriate reprogramming is the safest way to avoid intra-operative problems. Increasing pacing amplitude
to 5 volts (minimum) prior to any procedure in which electro-surgical cautery will be used is required.

Clinical evaluation of ICD:

In general, all ICD should be deactivated during surgery as they can inappropriately discharge because of extraneous signals, such as myopotentials caused by shivering, or fasciculations induced by suxamethonium, diathermy, orthopaedic instrumentation, and by false sensing as a result of sinus tachycardia or rapid atria fibrillation. However, with the third- and fourth-generation ICD, selective deactivation of the anti-tachycardia, low energy cardioversion and defibrillation functions can be achieved with the anti-brady ardia pacing function remaining activated. This is useful if the patient develops severe bradycardia during operation (Kam PCA, 1997).

Most devices will suspend tachydyssrhythmia detection (and therefore therapy) when a magnet is appropriately placed over the device. Antitachycardia therapy in some devices can be permanently disabled by magnet placement for 30 seconds. In all cases a cardiophysiolohist who is experienced in the management of intra-thoracic should be consulted and attending the operative procedure (Tung RT and Bajaj AK, 1995).

Preoperative visit:

Preoperative anaesthetic visit is very important for patient reassurance, good explanation for the patient and his close relatives about expected anesthetic techniques that might be used and their risks. Informed high-risk consent should be taken (Reid M and Appleton PJ, 1999).
Premedication:

Satisfactory premedication prevents sympathetic activation, which adversely affects the myocardial oxygen supply-demand balance. Overmedication is equally detrimental, however, and to be avoided because it may result in hypoxaemia, respiratory acidosis and hypotension. Because most patients with pacemakers are elderly and have cardiac disease, heavy sedation should be avoided to prevent cardiopulmonary depression (Yao FSF, 1998).

Intraoperative management:

General and regional anesthetic techniques can be used, provided that they take into account the patient’s underlying medical illnesses, the patient’s desires, and the planned surgical procedure. Management of anesthesia in patient with an implantable device includes monitoring to confirm continued function of the generator, ready availability of equipment, and drugs to maintain and acceptable intrinsic heart rate and function.

Patient positioning:

When positioning the patient for a particular procedure, we should avoid any pressure over the subcutaneous device and we should not hyperextend the limb on the side of the implantable device to avoid lead dislodgement (Seifert PC, 1999).

An increase in heart rate may result from changing the patient’s position. This occurs in rate-responsive pacemakers that sense body movement to adjust heart rate (Anderson C and Madsen GL, 1990).
Monitoring:

For pacemaker, ECG monitoring must include the ability to detect pacemaker discharge, and the ability to insure that myocardial electric activity is converted to mechanical systoles. Mechanical systoles can be evaluated by palpation of pulse, auscultation, pulse oximetry, plethysmogram, or arterial waveform. Some patients might need to have their pacing rate increased to meet an increased oxygen demand.

ECG monitors occasionally count the current impulses used to measure impedance (in rate adaptive pacemakers that depend on respiratory rate and minute ventilation) and therefore falsely elevate the monitored heart rate. On the other hand, respiratory rate-sensing ECG monitors use small currents in microamperes to measure the respiratory rate. These currents may be interpreted by the pacemaker as if there is hyperventilation and lead to increased pacing rate (Zaidan et al., 2000).

Pulmonary arterial catheterization requires consideration of the possible complications of inserting the catheter versus the benefits derived from the data. Complications include infection from a break in sterile technique and dislodgement of the pacemaker leads from the endocardial surface. The endocardial electrode's design suggests that it likely will not become dislodged while the catheter is being inserted. This is true if the electrodes have been in place for more than 4 weeks. There are no data to determine if the catheter's withdrawal can dislodge the electrode (Cohen Am and Hoxby EJ, 1997).

A central venous pressure (CVP) catheter can be inserted without difficulty; however, it is extremely important to maintain strict sterile technique.
For ICD, ECG monitoring is very important and the ability to deliver external cardioversion or defibrillation must be present during the time of ICD disablement (Gomez – Bordas et al, 1998).

**Anesthetic technique:**

There is no evidence that anesthetic drugs or events likely to be associated with the preoperative period alter the stimulation threshold of an artificial cardiac pacemaker or ICD. No special anesthetic technique is required for patients with intrathoracic gadgets but some interactions can occur and should be taken into account (Fleisher LA, 2000).

**Induction of general anesthesia:**

The choice of drugs to produce anesthesia is not altered by the presence of a properly functioning pacemaker or ICD. There is a theoretical risk that marked muscle fasciculation induced by succinylcholine may cause inhibition of the pacemaker if this activity is sensed by the pacemaker and interpreted as myocardial activity. A defasciculating dose of a nondepolarizing neuromuscular blocking drug can be used before administering succinylcholine (Sidhu VS 1991).

Succinylcholine could also increase the stimulation threshold of the pacemaker or ICD by an acute increase in the plasma potassium concentration. Clinical experience suggests that succinylcholine is usually a safe drug in the pacemaker patient. If myocardial inhibition does occur, it is generally transient and asymptomatic (Coen AM and Hoxby EJ, 1997).

The myoclonus resulting from some intravenous anesthetic drugs is unlikely to induce changes heart rate (Bloomfield P and Bowles GM, 1989).
Beside producing unconsciousness, the barbiturates can cause mild muscular excitatory movements such as hypertonus, tremors or twitching and respiratory excitatory effects including cough and hiccups. The dose-dependent incidence and severity of these effects are greater after methohexital than after thiopentone. Although these excitatory effects are not disturbing enough to limit the use of the barbiturates, atropine or opioids given just prior to anesthetic induction will minimize the excitatory effects, whereas premedication with phenothiazines or scopolamine exaggerates them. Inadequate induction doses can also evoke excitatory responses because inhibitory areas of the brain are the first to be depressed.

Heart rate increases more after methohexital than after equivalent doses of thiopentone. No arrhythmias occur after induction of anesthesia by the barbiturates as long as hypoxaemia and hypercarbia are avoided. The barbiturates also decrease sympathetic output from the CN and do not sensitize the heart to catecholamines (Frangen RJ and Avram MJ, 2000).

The minimal effect of etomidate on cardiovascular function sets it apart from other fast acting induction agents. The usual induction dose and a relatively large dose of etomidate produce minimal changes in cardiovascular parameters. However, etomidate, lacking analgesic efficacy, may not totally ablate the sympathetic response to laryngoscopy and intubation. For the smoothest haemodynamic induction/intubation sequence, a low dose (1.5-5.0 ug/kg) of fentanyl is often combined with etomidate (Reves et al, 2000).
Maintenance of anesthesia:

The main objective in anesthesia maintenance is to void factors and drugs that induce VT or VF and to maintain haemodynamic stability.

In general volatile and current thresholds when added to a narcotic-relaxant anesthetic technique (Kam PCA, 1997).

Volatile anesthetics: volatile anesthetics cause direct negative chronotropic actions in vitro by depressing sinoatrial node activity. However, alterations in heart rate in vivo are primarily determined by the interaction of volatile agents and baroreceptor reflex activity. Halothane does not appreciably change heart rate in humans because this agent attenuates baroreceptor reflex responses. Heart rate increases to variable degrees with enflurane; however, these increases in heart rate may be insufficient to preserve cardiac output (Ebert JJ, et al., 198:).

Isoflurane increases heart rate in response to simultaneous decreases in arterial pressure. These findings occur with this volatile agent because baroreceptor reflexes are relatively preserved when compared with those of equi-MAC concentrations of halothane and enflurane. Desflurane also causes dose-related increases in heart rate in humans. Desflurane- and isoflurane–induced tachycardia may be more pronounced in pediatric patient or in the presence of vagolytic agents and conversely may be attenuated in neonates and geriatric patients or by the concomitant administration of opioids. Rapid increases in the inspired desflurane concentration above 1 MAC may be associated with further transient increases in heart rate and arterial pressure resulting from sympathetic nervous system activation. Interestingly, similar increases in
heart rate are also observed when the inspired isoflurane concentration is rapidly increased (Murat et al., 1989).

The cardiovascul car stimulation induced by rapid increases in desflurane or isoflurane concentration in humans results from activation of tracheopulmonary and systemic receptors and is attenuated by pretreatment with beta-adrenoceptor antagonists, alpha-adrenoceptor agonists, or opioids. In contrast to the findings with isoflurane and desflurane, sevoflurane neither alters heart rate nor causes cardiovascular stimulation during rapid increases in anesthetic concentration in humans (Pagel et al., 2000).

Volatile anesthetics slow the rate of sinoatrial node discharge by direct and indirect effects on sinoatrial node automaticity. These actions may be altered in vivo by vasoactive drugs or autonomic nervous system activity. Halothane, enflurane and to some extent, isoflurane shorten cardiac action potential and effective refractory period duration in normal Purkinje fibers. However, these agents prolong His-Purkinje and ventricular conduction times (Bosnjak ZJ and Kampine JP, 1983).

Halothane, enflurane and isoflurane also prolong atrioventricular conduction time and refractoriness. When combined with the direct actions of volatile anesthetics on sinoatrial node discharge, these data suggest that volatile anesthetics have the potential to produce bradycardia and atrioventricular conduction abnormalities. However, primary disturbances in atrioventricular conduction leading to second or third degree atrioventricular block in humans probably do not occur with volatile anesthetics in the absence of conduction disease or drugs that directly prolong the atrioventricular conduction time (Pagel et al., 2001).
Volatile agents may have antiarrhythogenic actions against abnormal cardiac electrophysiologic mechanisms produced by myocardial ischaemia or infarction. Halothane, enflurane and isoflurane have been shown to be cardioprotective against ventricular fibrillation produced by coronary artery occlusion and reperfusion. Volatile anesthetics may also exert antiarrhythmic effects by opposing subsidiary pacemaker activity in infarcted myocardium (Turner et al, 1987).

Volatile agents have been shown to prolong the QT interval in humans. These data suggest that patients with idiopathic or acquired long QT syndrome may be at greater risk of developing torsades de pointes tachycardia during anesthesia with these drugs.

Halothane and, to a lesser extent, other volatile anesthetics sensitize myocardium to the dysrhythmogenic effects of epinephrine. Sensitization is the interaction between volatile anesthetics and catecholamines that leads to reduction in the threshold for both atrial and ventricular arrhythmias. Halothane-epinephrine induced arrhythmias are attenuated by pretreatment with thiopentone. Halothane-catecholamine sensitization also promotes abnormal automaticity of dominant and latent atrial pacemakers. These effects may produce premature ventricular contractions and arrhythmias originating from the his bundle. Intact sinoatrial node function reduces the incidence of epinephrine-induced ventricular escape during halothane anesthesia and is protective against his bundle arrhythmias (Woehlck et al, 1995).

**Nitrous oxide:** reversible atrioventricular dissociation has been reported in humans anesthetized with nitrous oxide and volatile or opioid-based anesthetics. Addition of nitrous oxide to halothane anesthesia
lowers the threshold at which arrhythmias occur. This observation may result from a combination of sympathetic nervous system stimulation by halothane. The incidence of arrhythmias has been shown to be reduced during combined nitrous oxide-opioid anesthesia when compared with halothane anesthesia alone (Pagel et al., 2000).

Subcutaneous emphysema occurs during implantation and is aggravated by nitrous oxide. Nitrous oxide used shortly after placement of a pacemaker and subcutaneous emphysema both have been reported as inhibiting unipolar pacemaker function. Apparently, the ground electrode in the generator's casing did not have good contact with expanded nitrous oxide. In these cases, applying pressure over the pacemaker reestablished pacing (Lamas et al., 1986).

**Xenon:** has been shown to cause minimal systemic and pulmonary haemodynamic effects in vivo, preserve myocardial contractility in humans, and attenuate increases in plasma epinephrine and cortical concentrations associated with surgical stimulation. Xenon also produces relatively minor alterations in the determinants of LV afterload. Taken together, these recent data indicate that xenon produces very subtle cardiovascular effects during isoflurane anesthesia (Hettrick, 1998).

**Total intravenous anesthesia (TIVA):**

**Effect of propofol:** the pharmacokinetics of propofol following a wide range of doses as well as following continuous infusion has been evaluated by numerous investigators. Following a single bolus injection, whole blood propofol levels decrease rapidly as a result of both redistribution and elimination. Because the required decrease in
concentration for awakening following anesthesia or sedation with propofol is generally less than 50 percent, recovery from propofol remains rapid even following prolonged infusion. This is the base for its use for total intravenous anesthesia.

Heart rate does not change significantly after an induction dose of propofol. It has been suggested that propofol either resets or inhibits the baroreflex, thus reducing the tachycardic response to hypotension. Propofol has no direct effect on senatorial node function or on normal atrioventricular and accessory pathway conduction. Heart rate may increase, decrease, or remain unchanged when anesthesia is maintained with propofol. An infusion of propofol results in a significant reduction in both myocardial blood flow and myocardial oxygen consumption, a finding that suggests that the global myocardial oxygen supply/demand ratio is preserved (Ebert et al, 1992).

**Effect of intravenous opioids:** Central neurally mediated mechanisms are the primary mechanism of opioid-induced bradycardia. Morphine also has a direct effect on the sinoatria node and atrioventricular conduction. Asystole may follow opioid-induced bradycardia. Premedication with, or concomitant administration of, beta adrenergic or Ca^{2+} entry blockers can exacerbate bradycardias and may result in asystole. Severe bradycardia and asystole often appear prior to or during laryngoscopy and intubation. Periods of asystole may resolve spontaneously, but they usually respond to atropine (0.4-0.8 mg IV). On occasion, much larger doses of atropine (>1.0 mg), isoproterenol, or a precordial thump may be required to treat severe bradycardia (asystole) (Bailey et al., 2000).
Opioids may depress cardiac conduction. Fer anyl slows atioventricular node conduction and prolongs the RR interval, the atioventricular node refractory period, and Purkinje fiber action potential duration. Sufentanil also prolongs action potential duration in Purkinje (Royster et al., 1988).

Opioids can also prolong the QT interval. the risk of opioid use in patients with QT abnormalities or taking medications such as quinidine is unknown. However, both sufentanil and alfentanil have been demonstrated to be devoid of electrophysiologic effects on the normal or accessory pathways in patients with Wolff-Parkinson-white syndrome (Sharpe, 1994).

Clinically, cardiac conduction disturbances due to opioids are very rare, but they may be more likely to occur in the presence of Ca2+ entry or beta adrenergic blockers. Although the effects of opioids on cardiac conduction could theoretically lead to the development of reentry-type dysrhythmias, in practice this rarely occurs. Indeed, the overall effect of opioid anesthesia is antiarrhythmic. Antifibrillatory effects have been suggested to be produced by fentanyl. Some of the electrophysiologic actions of opioids resemble those of class III antiarrhythmic compounds and may underlie the antiarrhythmogenic potential of opioids (Bailey et al., 2000).

Effect of muscle relaxants:

Pancuronium causes a moderate increase in heart rate and, to a lesser extent, in cardiac output, with little or no change in systemic vascular resistance. Although pancuronium-induced tachycardia has been
attributed to a vagolytic action, numerous investigators have implicated the sympathetic nervous system as well. Succinylcholine and gallamine actually reduce the incidence of epinephrine-induced arrhythmias. Possible because of enhanced atrioventricular conduction, the incidence of arrhythmias from pancuronium appears to increase during halothane anesthesia. The drug interaction among tricyclic antidepressants, halothane, and pancuronium must be avoided. Use of another nonvagolytic muscle relaxant, such as vecuronium or cisatracurium, is probably the most convenient approach in a patient receiving tricyclic antidepressants who must have halothane anesthesia.

Several case reports have described severe bradycardia and even asystole following vecuronium or atracurium administration. All of these cases were associated with opioid administration. Subsequent studies indicate that vecuronium or atracurium alone does not cause bradycardia, but when combined with other drugs (e.g., fentanyl) that do cause bradycardia. The nonvagolytic relaxants such as vecuronium, cisatracurium, and atracurium simply allow this mechanism to occur unopposed. The moderate vagolytic effect of pancuronium is often used to counteract opioid-induced bradycardia (Savarese et al., 2000).

Some important points to be observed:

1- Oxygen saturation: Rate-adaptive pacemakers that use oxygen saturation in mixed venous blood as a sensor can be affected by the changes in oxygen saturation. Oxygen saturation in the mixed venous blood during general anesthesia may be affected: metabolism is reduced and oxygen extraction diminished, so oxygen saturation in mixed venous blood will usually be higher than normal provided that there are no problems with arterial oxygenation. The pacemaker will
pace at a preset basic heart rate at high oxygen saturations. However, patients with acute respiratory insufficiency or with drug-induced respiratory depression may have a low saturation that will stimulate the pacemaker to increase the heart rate. This may further impair the function of a failing heart (Andersen C and Madsen GM, 1990).

2- **Right ventricular pressure:** Myocardial contractility sensing pacemakers use right ventricular pressure as an index to indicate changes in the level of physical activity and thus change the heart rate accordingly. Several factors may contribute to changes in right ventricular pressure during general anesthesia, such as the patient’s position, e.g. head down, positive pressure ventilation, inferior vena cava compression, rapid infusion via a central venous catheter, or administration of sympathomimetic drugs. These changes could induce the pacemaker to stimulate the heart with an unphysiological rate (Savarese JJ et al., 2000).

3- **Body temperature:** temperature-sensing pacemakers use changes in body temperature to change heart rate. Increase in body temperature, especially if preceded by slight decrease in temperature, results in increased pacing rate. The anesthetist may predict problems in relation to warming of hypothermic patients. There is usually a temperature decrease of 0.5 to 1.0°C during general anesthesia which will make the pacemaker more sensitive to any subsequent increase in temperature. The increased sensitivity reduces the pacemaker’s reaction time to a change of activity (Fearnot NE and Evans ME, 1988).

Small increases in temperature will not raise the pace heart rate if the patient is normothermic at induction of anesthesia but later becomes slightly hypothermic. However, if the patient is already slightly hyperthermic, induction of anesthesia may lower the temperature to
normal and then even small increases will induce a rise in paced heart rate. Problem may also appear during rapid infusion of warmed fluids via a central venous catheter, since this will stimulate the pacemaker’s sensor to increase the pacing rate (Andersen C and Madsen GM, 1990).

4- **Minute ventilation**: respiration-sensing pacemakers use changes in thoracic impedance to detect respiratory rate and tidal volume. An increase in minute ventilation will lead to a matched increase in pacing rate. There may be problems with general anesthesia if the anesthetist is not familiar with the function of a respiration sensing pacemaker.

There are two case reports relating perioperative experiences with minute ventilation sensing pacemakers. In one case, during a cesarean section, the paced rate increased during hyperventilation to reoxygenate the patient. The paced rate remained at the lower rate limit during surgery, but increased again as the patient was awakening. In another case, a patient with a minute ventilation-regulated pacemaker had a transurethral resection of the prostate and his lungs were manually ventilated. Accidental hyperventilation led to a paced tachycardia and a decrease in arterial blood pressure, which was interpreted as a sign of hypovolaemia. However, when the anesthesiologist was occupied with setting up a blood transfusion, ventilation was reduced and this led to the pacing rate returning to normal (Madsen and Andersen, 1989).

A rare occurrence is a transvenous pacemaker failure associated with positive-pressure ventilation, which may reflect an abrupt volume change in the heart and cardiac septal deviation causing loss of electrode contact with the endocardial surface (Tung and Bajaj, 1995).
5- **Effect of drugs used as adjuvant to anesthesia:** Q-T sensing pacemaker measure the Q-T interval and change the pacing rate according to the variation in the measured interval. Q-T interval varies according to blood catecholamine level. Beta adrenoceptor blockers are reported to decrease the Q-T interval which leads to an attenuated response to physical activity in patients with a Q-T pacemaker (*Monk and Weldon, 1996*).

In addition to factors that affect the general threshold (PH, potassium level, local anesthetics), any medication which changes the T-wave configuration may be important since this may interfere with the rate-responsive function (*Andersen and Madsen, 1990*).

6- **Effect of acute changes in potassium concentration:** If potassium concentration acutely changes, the pacemaker impulse remains visible on the ECG; however, pacing capture fails. Severe hypokalaemia causes the resting membrane potential to be more negative. The pacemaker’s output would have to be increased to reestablish the charge density at the electrode-tissue interface and to stimulate the cells to threshold. Hypokalaemia could occur if the patient is suddenly hyperventilated and potassium moved into the cell or if the patient suddenly received large quantities of potassium-free solutions that might be administered during massive volume resuscitation.

Severe hyperkalaemia renders the resting membrane potential less negative so less current would be required by the myocardial cells to reach threshold. Hyperkalaemia could be caused by leakage of potassium from ischaemic myocardial cells, by the bolus administration of potassium, or possibly by potassium release secondary to the
administration of succinylcholine. Changes in potassium concentration can also cause variations in pacemaker sensing by changing the contour of the local R wave in the myocardium that surrounds the electrode. The clinical result is a reduction in or loss of sensing with all of its attendant complications (Zaidan, 2000).

**Emergence from anesthesia:**

It should be smooth and shivering is to be avoided. Inadvertent hypothermia is a common occurrence following major surgical procedures the major adverse effects are patient discomfort, vasoconstriction, and shivering. Full recovery sometimes takes many hours. Shivering increases metabolic rate and hence the need to increase cardiac output and minute ventilation. Not all patients who shiver postoperatively are hypothermic, a finding suggesting that the mechanism of this event may be related to inadequate descending control of spinal reflexes following inhalation of anesthesia. Longer anesthetic regimens are associated with a higher frequency of shivering. Prevention is important, and the use of a heated humidifier results in higher patient temperature. Patients who have had propofol have a lower incidence of shivering than those receiving thiopentone.

Hypothermic patients should have supplemental oxygen, warm intravenous fluids and blood, and external warming. External warming can be accomplished with thermal blankets or thermal ceilings which lower oxygen consumption. Patients who develop shivering should receive supplemental oxygen. Although many drugs have been used to treat postanaesthetic shivering, pethidine (meperidine) (25-30 mg IV) is very effective in both stopping the shivering and decreasing oxygen
consumption. In some patients, a second dose is necessary. Fentanyl is also effective, but for a shorter interval (Feeley and Macario, 2001).

Procedure-related effects:

There are many hospital sources of electromagnetic interference (EMI) that could be encountered in the operating room. There are few sources only that are truly a threat to the patient with pacemaker or ICD. Special precautions are taken for these procedures.

Special procedures in patients with implantable generators (Pacemaker or ICD):

1) Electrocautery: electro-surgical cautery use remains the principal intra-operative issue for the patient with a pacemaker or ICD. Between 1984 and 1997, Monk and Weldon notified 465 adverse events with pulse generator, 225 were from electrocautery and significant number of device failure occurred. Monopolar cautery is more likely to cause problems than bipolar cautery (Monk and Weldon, 1996).

Guidelines for electro-cautery use in the patients with pacemakers:

- Never position the ground plate of the electrocautery such that the pacemaker generator is between the ground plate and the active electrode of the electrocautery.
- Position the ground plate so that a line drawn between the two electrodes of the electrocautery are perpendicular to a line drawn between the pacemaker’s electrodes.
• Request that the surgeon not activate the electrocautery until ready to use it. The activated electrode does not have to touch the patient to affect the generator.

• Use the smallest possible current.

• Use the bipolar electrocautery.

• Do not use the electrocautery within 5 inches of the pacemaker. Consider programming to VOO or DOO activity to eliminate inhibition of the pacemaker by the electrocautery. Remember that reprogramming out of VOO or DOO activity still can occur.

• Consider programming to bipolar sensing if the pacemaker is using unipolar sensing. Although reprogramming can occur, bipolar sensing might reduce the possibility.

• Apply the magnet if pacing stops (Zaidan, 1995).

Guidelines for electro-cautery use in the patients with ICDs:

• If the patient has a separately implanted pacemaker, consider programming the pacemaker to asynchronous activity (VOO or DOO) to prevent inhibition while the electrocautery is being used. Pacemaker spikes that occur when a patient’s heart rate is greater than an asynchronously programmed pacemaker rate will be counted by the ICD as R waves. The ICD, therefore, could interpret a sinus tachycardia plus pacemaker spikes as VT and shock the patient.

• The electrocautery could reprogram the VVI and DDD pacing parameters of the ICD; however, the chance of reprogramming is extremely small. A magnet will not change pacing parameters in patients who require chronic pacing.

• Do not use electrocautery when the device is actively sensing and capable of delivering therapy. Program the ICD to “off” there are
no reports of an ICD reprogramming to “monitor plus therapy” once it has been programmed off. Some cardiologists reprogram the ICD to “monitor only” during surgery to record arrhythmias, but the device will then monitor electrocautery signal.

- If a programming device is not available, use the magnet to change the mode of activity. Most devices suspend monitoring and therapy for as long as the magnet is held over the generator; therefore, if the magnet is on the generator, they will neither sense nor discharge.

- Duration criteria must be met before charging takes place. Additionally, if the patient is in VT, the ICD reconfirms the arrhythmia before it delivers rapid pacing or shock therapy. These events give the device time to stop the process if an electrocautery signal is sensed as an arrhythmia. Using the electrocautery in short bursts might cause inappropriate sensing; however, sensing will not fulfill the duration criteria. Therefore the device will not charge. The ICD could interpret the signal from the electrocautery as VF even when the activated cautery is not in contact with the patient.

- A bipolar electrocautery has a smaller chance of causing sensing and discharge. A battery-powered electrocautery offers extra safety.

- Do not use the electrocautery within 6 inches of the device or leads.

- If the ICD begins to charge during the operative procedure, place the magnet over the ICD and keep it in that location. The ICD will discharge into the generator and will stop sensing. It will not stop pacing activity.
• When positioning the external defibrillator patches, ensure they are positioned perpendicular to a plane described by the generator and electrodes (Zaidan JR, 1999).

2) **Magnetic resonance imaging**: Implanted programmable pacemakers can revert to VOO activity, increase the ventricular rate above the high rate limit, or cease activity when they are subjected to strong magnetic fields such as that found in magnetic resonance imaging (MRI). Normal activity should resume after the magnetic field is eliminated. Magnetic resonance imaging is absolutely contraindicated by most generator manufacturers, and deaths have been reported (Kangralu et al., 1999).

3) **Electroconvulsive therapy**: electro-convulsive therapy (ECT) might affect cardiac muscle contractility and may cause dysfunction of the pacemaker (Drach et al., 1990).

4) **Extra-corporeal shock wave lithotripsy (ESWL)**: Extra-corporeal shock wave lithotripsy (ESWL) was initially contraindicated for the patient with pacemaker, because of the possibility that electrical interference from the spark gap generating the shock wave might inhibit, reprogram, or damage the pacemaker (Drach et al., 1990).

However, a review of 142 ESWL treatments in pacemaker dependent patients found a low (<1%) incidence of major pacemaker complication in these patients with only one pacemaker deprogrammed during the treatment period. Based on these findings, patients with pacemakers are considered acceptable candidates for ESWL provided that certain precautions are taken, including careful preoperative cardiac evaluation, a magnet or programming device available, an alternative pacing device available, and the patient positioned so that the pacemaker is not in the shock path (Monk and Weldon, 1996).
5) **Defibrillation and cardioversion:** Cardioversion or defibrillation if needed should be given promptly; one should remember that the patient. Not the pacemaker or ICD is being treated. The presence of ICD should not discourage standard cardiopulmonary resuscitation. Certain precautions are taken:

- Ideally the paddles should be placed in the anterior/posterior position.
- Try to keep the paddles at least 4 inches from the pulse generator.
- A pacemaker programmer should be available.
- The pacemaker should be integrated following the procedure.
- A pacemaker magnet must be ready to convert to synchronous mode (*Ievy et al., 1997*).

6) Nerve stimulator testing and therapy: Nerve stimulators applied directly over pacemaker generators inhibit the pacemakers. This inhibition is possible even if the stimulator was used on the arm. The pacemaker would remain in the inhibited mode until the nerve stimulator is turned off even if the heart rate was unacceptably low. Inappropriate detection of neuromuscular stimulators as VT or VF has been reported (*Tung and Bajaj, 1995*).

**Regional anesthesia:**

The local anaesthetics commonly used do not seem to affect pacing threshold or defibrillation energy (*Tung and Bajaj, 1995*).

Regional anesthesia for these patients is very much accepted providing that the same precautions should be taken. The primary cardiac electrophysiologic effect of local anesthetics is a decrease in the rate of depolarization in the fast conducting tissues of Purkinje fibers and ventricular muscle. This reduction in rate is believed to be due to a decrease in the availability of fast Na⁺ channels in cardiac membranes.
Action potential duration and the effective refractory period are also decreased by local anaesthetics.

Qualitative differences may exist among the electrophysiologic effects of various agents. Bupivacaine depresses the rapid phase of depolarization (V_{max}) in Purkinje fibers and ventricular muscle to a greater extent than does lidocaine. In addition, the rate of recovery from a use-dependent block is slower in bupivacaine treated papillary muscles than in lidocaine-treated muscles. This slow rate of recovery results in an incomplete restoration of Na^{+} channel availability between action potentials, particularly at high heart rates. In contrast, recovery from lidocaine is complete, even at rapid heart rates. These differential effects of lidocaine and bupivacaine have been advanced as explanations of the antiarrhythmic properties of lidocaine and the arrhythmogenic potential of bupivacaine (Berde CB. and Strichartz GR., 2000).

Extremely high concentrations of local anaesthetics depress spontaneous pacemaker activity in the sinus node, resulting in sinus bradycardia and sinus arrest.

Local anaesthetic drugs also exert profound effects on the mechanical activity of cardiac muscle. All local anaesthetics exert a dosedependent negative inotropic action on isolated cardiac tissue. This depression of cardiac contractility is proportional to the conduction blocking potency of the various agents. Thus, bupivacaine is more potent cardio-depressant than lidocaine (Lynch C., 1986).

Bupivacaine may produce severe cardiac arrhythmias, including ventricular fibrillation, in various animal species. Ventricular arrhythmias
were rarely seen with lidocaine, mepivacaine, or tetracaine. These electrophysiologic effects of bupivacaine may result in conduction abnormalities, leading to a reentrant type of arrhythmia similar to torsade de pointes arrhythmias (Reiz and Nath, 1986).

**Postoperative management**

Patient should preferably be managed in ICU for the first 24 hours postoperatively, for proper monitoring and cardiac reassessment, with availability of cardiologist and a device programmer (Tuz and Bajaj, 1995).

Postoperative pain should be avoided. Pain causes stimulation of sympathetic neurons and subsequent tachycardia, increased stroke volume, cardiac work, and myocardial oxygen consumption. The risk of myocardial ischaemia or infarction may be increased some of these sequelae may be reduced by using effective perioperative analgesic therapy (Ready, 2000).

**Postoperative pacemaker failure:** Failure of pacemaker lead-pulse generator system may occur with:

1- Myocardial ischaemia at the site of electrode attachment or in the conduction pathways around the electrode (increasing generator output may correct this kind of failure).

2- Hypokalaemia or hyperkalaemia and failure to capture due to increased threshold for pacing. Hyperkalaemia occurs with hypoventilation, acidosis, potassium overload, and potassium shift to the extracellular compartment and hypokalaemia occurs with the reverse conditions.
3- Postoperative shivering that causes myopotential interference with pacemaker output generation.

4- In suspected pacemaker failure, heart rate should be determined from palpation of the pulse, arterial waveform analysis, or oximetry, not through the ECG. Most pacemakers can be converted from synchronous to a fixed or asynchronous mode by placing a magnet over them. However, a programmable pacemaker require a programming transceiver, and magnet application may cause unpredictable resetting (Sidi and Gravenstien, 2000)

**Postoperative device check:**

1- The ICD must be re-integrated and re-enabled.

2- A pacemaker that was reprogrammed for the perioperative period should be reset appropriately.

3- For non-reprogrammed devices, the manufacturer recommend interrogation to ensure proper functioning and remaining battery life if electro-cautery was used (Watson, 1997).
ANESTHETIC MANAGEMENT OF PATIENTS WITH VENTRICULAR ASSIST DEVCIE AND OF PATIENT WITH INTRAOPHATIC BALLON PUMP

Pathophysiology before heart transplantation:

The pathophysiology of heart transplant candidates is predominantly end-stage cardiomyopathy. Such patients will normally have both systolic dysfunction (characterized by decreased stroke volume and increased end-diastolic volume) and diastolic dysfunction, characterized by an elevated intracardiac diastolic pressure. As compensatory mechanisms to maintain cardiac output fail, the elevated left ventricular pressures lead to increases in pulmonary venous pressures and development of pulmonary vascular congestion and edema. A similar process occurs if right ventricular failure also occurs. Autonomic sympathetic tone is increased in patients with heart failure, leading to generalized vasoconstriction as well as salt and water retention. Vasoconstriction and ventricular dilation combine to substantially increase myocardial wall tension. Over time, the high levels of catecholamines lead to a decrease in the sensitivity of the heart and vasculature to these agents via a decrease in receptor density (i.e., “down-regulation”) and a decrease in myocardial norepinephrine stores (Bristow et al., 1982).

Therapy of heart failure seeks to reverse or antagonize these processes. Patients refractory to even these measures may be supported with intra-aortic balloon counterpulsation, but is use if fraught with significant vascular complications and essentially immobilizes the patient. Many patients with low cardiac output are maintained on anticoagulants.
such as warfarin to prevent pulmonary or systemic embolization, especially if they have atrial fibrillation (Kaplan et al., 1999).

**Pathophysiology after heart transplantation:**

The physiology of patients after heart transplantation is of interest not only to anesthesiologists in cardiac transplant centers but also to the anesthesiology community at large because a substantial portion of these patients return for subsequent surgical procedures (Isono et al., 1987).

Cardiac denervation is an unavoidable consequence of heart transplantation. Many long-term studies indicate that reinnervation is absent (Rowan and Billingham, 1988) or at best partial or incomplete (Wilson et al., 1991) in humans. Denervation does not significantly change baseline cardiac function (Verani et al., 1988).

But it does substantially alter the cardiac response to demands for increased cardiac output. Normally, increases in heart rate can rapidly increase cardiac output, but this mechanism is not available to the transplanted heart. Heart rate increases only gradually with exercise, and this effect is mediated by circulating catecholamines. Increases in cardiac output in response to exercise are instead mostly mediated via an increase in stroke volume. Therefore, maintenance of adequate preload in cardiac transplant recipients is crucial. Lack of parasympathetic reinnervation is probably responsible for the gradual decrease in heart rate after exercise seen in transplant recipients, rather than the usual sharp drop (Kaplan et al., 1999).

Denervation has important implications in the choice of pharmacologic agents used after cardiac transplantation. Drugs that act
indirectly on the heart via either the sympathetic (epinephrine) or parasympathetic (atropine, pancuronium, edrophonium) nervous systems will generally be ineffective. Drugs with a mixture of direct and indirect effects will exhibit only their direct effect (leading to the absence of the normal increase in refractory period of the atrioventricular node with digoxin, tachycardia with norepinephrine infusion, and bradycardia with neostigmine (Backman et al., 1993).

Thus, agents with direct cardiac effects (such as epinephrine or isoproterenol) are the drugs of choice for altering cardiac physiology after transplantation. However, the chronically high catecholamine levels found in cardiac transplant recipients may blunt the effect of α-adrenergic agents, as opposed to normal responses to β-adrenergic agents (Borow et al., 1989).

Allograft coronary vasculopathy remains the greatest threat to long-term survival after heart transplantation. Allografts are prone to the accelerated development of an unusual form of coronary atherosclerosis that is characterized by circumferential, diffuse involvement of entire coronary arterial segments, as opposed to the conventional form of coronary atherosclerosis with focal plaques often found in eccentric positions in proximal coronary arteries (Tuzcu et al., 1995).

Mechanical ventricular assist devices have been successfully used to “bridge” patients who would otherwise die of acute heart failure awaiting transplantation (Mehta et al., 1994).
Anesthetic management:
Preoperative evaluation and preparation:

The preoperative period is often marked by severe time constraints due to the impending arrival of the donor heart. Nevertheless, a rapid history should screen for last oral intake, recent antiagulant use, intercurrent deterioration of ventricular function, or change in anginal pattern; a physical examination should evaluate present volume status, and a laboratory review (if available) and a chest radiograph should detect the presence of renal, hepatic, or pulmonary dysfunction. Many hospitalized patients will be supported with inotropic infusions and/or an intra-aortic balloon pump, and the infusion rates and timing of the latter should be reviewed (Kaplan et al., 1999).

Equipment and drugs similar to those usually used for routine cases requiring cardiopulmonary bypass should be prepared. A β-agonist such as epinephrine should be readily available both in bolus form and as an infusion to rapidly treat ventricular failure; and an α-agonist such as phenylephrine or norepinephrine is useful to compensate for the vasodilatory effects of anesthetics, because even small decreases in preload and afterload can lead to catastrophic changes in cardiac output and coronary perfusion in these patients.

Placement of invasive monitoring prior to induction will facilitate rapid and accurate response to hemodynamic events during induction. In addition to standard noninvasive monitoring, an arterial catheter and a PA catheter (with a long sterile sheath to allow partial removal during graft implantation) are placed after judicious use of sedation and, local anesthetics. Placing the arterial catheter in a central site rather than the radial artery will avoid the discrepancy between radial and central arterial
pressure often seen after cardiopulmonary bypass, but it may also be necessary to cannulate a femoral artery for arterial inflow for cardiopulmonary bypass if there has been a prior sternotomy. Floating the PA catheter into correct position may be difficult due to cardiac chamber dilation and severe tricuspid regurgitation. Large-bore intravenous access is mandatory, especially if a sternotomy has been previously performed, in which case external defibrillator/pacing paths may also be useful. The overall hemodynamic “picture” should be evaluated and optimized insofar as possible just prior to induction. If the hemodynamics seem tenuous, then starting or increasing an inotrope infusion may be advisable (Kaplan et al., 1999).

Induction:

Most patients presenting for heart transplantation will not be in a fasting state, and should be considered to have a “full stomach”. Therefore the induction technique should aim to rapidly achieve control of the airway to prevent aspiration while avoiding myocardial depression. A regimen combining a short-acting hypnotic with minimal myocardial depression (etomidate, 0.3 mg/kg), a moderate dose of narcotic to blunt the tachycardic response to laryngoscopy and intubation (fentanyl, 10 μg/kg), and succinylcholine (1.5mg/kg) is popular (Weterman and Bjerke, 1988).

High-dose narcotic techniques with or without benzodiazepines have also been advocated (Hensley et al., 1987).

Vasodilatation should be countered with an α-agonist. Anesthesia can be maintained with additional narcotic and sedatives (benzodiazepines or scopolamine) (Berberich and Fabian, 1987).
Intraoperative management:

Following induction, the stomach can be decompressed with an orogastric tube and a transesophageal echocardiographic probe introduced while the bladder is catheterized. A complete transesophageal echo examination will often reveal useful information not immediately available from other sources, such as the presence of cardiac thrombi, ventricular volume and contractility, and atherosclerosis of the ascending aorta and aortic arch. Cross-matched blood should be immediately available once surgery commences, especially if the patient has had a previous sternotomy; patients not previously exposed to cytomegalovirus should receive blood from donors who are likewise cytomegalovirus negative. The period prior to cardiopulmonary bypass is often uneventful, apart from arrhythmias and slow recovery of coronary perfusion due to manipulation of the heart during dissection and cannulation. The PA catheter should be withdrawn from the right heart prior to completion of bicaval cannulation (Kasplan et al., 1999).

Maintenance of anesthesia:

The main objective in anesthesia maintenance is to avoid factors and drugs that induce VT or VF and to maintain haemodynamic stability.

In general volatile and current thresholds when added to a narcotic-relaxant anesthetic technique (Kam PCA, 1997).

Once cardiopulmonary bypass is initiated, ventilation is discontinued and the absence of a thrill in the carotid arteries is documented. Most patients will have an excess of intravascular volume, and administration of a diuretic and/or the use of hemofiltration via the pump may be beneficial by increasing the hemoglobin concentration.
dose of glucocorticoid (methylprednisolone, 500 mg) is administered as
the last anastomosis is being completed prior to release of the aortic
cross-clamp to attenuate any hyperacute immune response. During the
period of reperfusion an infusion of an inotrope is begun for both
inotropy and chronotropy. Transesophageal echocardiography is used to
monitor whether the cardiac chambers are adequately de- 
ired prior to
weaning from cardiopulmonary bypass (Kaplan et al., 1999).

Weaning from bypass begins after ventilation is resumed and the
cannula in the superior vena cava is removed. The donor heart should be
paced if bradycardia is present despite the inotropic infusion. Once the
patient is separated from bypass, the PA catheter can be advanced into
position. Patients with elevated PVR are at risk for acute right ventricular
failure, and may benefit from a pulmonary vasodilator such as
prostaglandin E1 (0.05 – 0.15 µg/kg/min) (Armitage et al., 1987).

Rarely, such patients will require support with a right ventricular
assist device (Fonger et al., 1986).

Transesophageal echocardiography will often provide additional
useful information about right and left heart function and volume, and
document normal flow dynamics through the anastomosis.

Protamine is then given to reverse heparin’s effect after
satisfactorily weaning from cardiopulmonary bypass. Continued
coagulaopathy despite adequate protamine is common after heart
transplantation, especially if there has been a prior sternotomy. Treatment
is similar to that employed for other post-bypass coagulopathies:
meticulous attention to surgical hemostasis, empiric ad
inistration of
platelets, and subsequent addition of fresh frozen plasma and cryoprecipitate guided by subsequent coagulation studie... High-dose aprotinin infusion decreases blood loss, transfusion of red blood cells and clotting factors such as platelets, fresh frozen plasma, and cryoprecipitate, and blood donor exposure after heart transplant via “redo” sternotomy (**Propst et al., 1994**).

After adequate hemostasis is achieved, the wound is closed in standard fashion and the patient transported to the intensive care unit (ICU).

**Postoperative management and complications:**

Management in the intensive care unit after the conclusion of the procedure is essentially a continuation of the anesthetic management after cardiopulmonary bypass (**Stein et al., 1995**).

The electrocardiogram; arterial, central venous and/or PA pressures; and arterial oxygen saturation are monitored continuously. Cardiac recipients will continue to require β-adrenergic infusion for chronotropy and inotropy for up to 3 to 4 days. Vasodilators may be necessary to control arterial hypertension and decrease impedance to left ventricular ejection. Patients can be weaned from ventilatory support and extubated when the hemodynamics are stable and hemorrhage has ceased. The immunosuppressive regimen of choice (typically consisting of cyclosporine, azathioprine, and prednisone, or tacrolimus and prednisone) should be started after arrival in the ICU. Invasive monitoring can be withdrawn as the inotropic support is weaned, and me iastinal tubes removed after drainage subsides (usually after 24 hours). Patients can usually be discharged from the ICU after 2 or 3 days (**Kapla et al., 1999**).
Early complications after heart transplantation include acute and hyperacute rejection, cardiac failure, systemic and pulmonary hypertension, cardiac arrhythmias, renal failure, and infection. Hyperacute rejection is an extremely rare but devastating syndrome mediated by preformed recipient cytotoxic antibodies against donor heart antigens. The donor heart immediately becomes cyanotic from microvascular thrombosis and ultimately ceases to contract (Weil et al., 1981).

This syndrome is lethal unless the patient can be supported mechanically until a suitable heart is found. Acute rejection is a constant threat in the early postoperative period and may present in many forms (e.g., low cardiac output, arrhythmias). Acute rejection occurs most frequently during the initial 6 months after transplantation, so its presence is monitored by serial endomyocardial biopsies, with additional biopsies to evaluate any acute changes in clinical status. Detection of rejection mandates an aggressive increase in the level of immunosuppression, usually including pulses of glucocorticoid or a change from cyclosporine to tacrolimus. Low cardiac output after transplantation may reflect a number of etiologies: hypovolemia, inadequate adrenergic stimulation, myocardial injury during harvesting, acute rejection, tamponade, or sepsis (Kaplan et al., 1999).

Therapy should be guided by invasive monitoring, transesophageal echo and endomyocardial biopsy. Systemic hypertension may be due to pain, so adequate analgesia should be obtained before bleeding blood pressure with a vasodilator. Because fixed pulmonary hypertension will have been excluded during the recipient evaluation, pulmonary hypertension after heart transplantation will usually be transient and
responsive to vasodilators such as prostaglandin E₁, nitrates, or hydralazine after either orthotopic or heterotopic placement (*Villanueva et al.*, 1989).

Atrial and ventricular tachyarrhythmias are common after heart transplantation (*Jacquet et al.*, 1990).

Once rejection has been ruled out as a cause, antiarrhythmics are used for conversion or control (except those acting via indirect mechanisms such as digoxin, or those with negative inotropic properties such as β-blockers and calcium channel blockers). Almost all recipients will require either β-adrenergic agonists or pacing to increase heart rate in the immediate perioperative period, but 10 to 25 percent of recipients will also require permanent pacing (*Scott et al.*, 1991).

Renal function often improves immediately after transplantation, but immunosuppressives such as cyclosporine and tacrolimus may impair renal function (*Platz et al.*, 1994).

Finally, infection is a constant threat to immunosuppressed recipients. Bacterial pneumonia is frequent early in the postoperative period, with opportunistic viral and fungal infections becoming more common after the first several weeks.
Summary
Summary

As the need for mechanical circulatory assistance is increasing, advances and development in its structure, function, and control continue to influence the outcome and the patient benefit. However, the devices available today are the result of long and hard work by many investigators, researchers, and clinicians.

Despite substantial improvement in myocardial protection and other technical advances, postoperative ventricular dysfunction persists as a complication in 2-6% of all patients undergoing cardiac or thoracic aortic surgery. Despite maximal inotropic therapy, 0.5-3% of these patients cannot be weaned readily from CPB and would require some form of mechanical cardiac assistance to achieve adequate systemic pressure and perfusion.

The currently available circulatory assist devices are the artificial cardiac pacemaker, the intraaortic balloon pump, the ventricular assist devices, and the implantable cardioverter defibrillator. Different haemodynamic and physiopathologic changes occur with the use of these mechanical circulatory assist devices and understanding of these changes is very important to help the surgeon, anesthetist, and perfusionists to decrease the morbidity of these changes on the patient.

A) Artificial cardiac pacemaker:

Electronic cardiac pacemakers are temporary or permanent (implanted) devices that electrically stimulate the heart.
Pacemakers consist of a power source (battery) that supplies energy for stimulation and other pacemaker functions, circuits for sensing and regulation of stimulation, and leads that connect the power source and electronic circuitry to electrodes.

Artificial pacing is indicated for treatment of persistent bradycardia of any origin if it compromises hemodynamics or predisposes to ventricular irritability manifested by premature beats or ventricular tachycardia (VT). The two major indications for permanent pacing are failure of impulse formation and failure of cardiac conduction. Clinically, sick sinus syndrome and complete heart block are the most common indications for pacemakers.

### Complications following pacemaker or PCD implantation:

<table>
<thead>
<tr>
<th>Early</th>
<th>Late</th>
<th>Early or late</th>
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<tbody>
<tr>
<td>Pneumo (hemo)- thorax</td>
<td>Thromboembolism, pulse generator erosion,</td>
<td>Lead dislodgement</td>
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<tr>
<td>subcutaneous emphysema</td>
<td>lead defects ↑ pacing thresholds</td>
<td>pacemaker arrhythmias</td>
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<tr>
<td>myocardial perforation</td>
<td>battery depletion</td>
<td>pacemaker infection</td>
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<tr>
<td>arterial lead placement</td>
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<td>pacemaker syndrome</td>
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<td>brachial plexus injury</td>
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<td>generator malfunction</td>
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<td></td>
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<td>extracardiac stimulation</td>
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### B) The intraaortic balloon pump:

The IABP is a catheter mounted intravascular device designed to improve the balance between myocardial oxygen supply and demand while increasing systemic perfusion to a modest degree. Other components of IABP include a pump, a gas source, and a microprocessor console.

The primary indications for IABP in cardiac surgical patients are inability to separate from CPB, poor haemodynamic function, and ongoing ischaemia following CPB despite increasing drug support.
Myocardial function often improves with the use of the IABP, and systemic perfusion and vital organ function are preserved. It is crucial to control heart rate and suppress atrial and ventricular dysrythmias to ensure proper balloon timing. As cardiac function returns, the assist ratio is gradually weaned from every beat to every other beat and so on assuming no further cardiac deterioration, then removed.

Complications associated with the IAP are primarily related to ischaemia distal to the site of balloon insertion. Direct trauma to the vessel, arterial obstruction, and thrombosis are the most common complications, although aortic perforation and balloon rupture occur rarely. Platelet destruction and thrombocytopenia may also occur.

C) Automatic implantable
Cardioverter defibrillator

Recurrent ventricular tachycardia or ventricular fibrillation that can result in sudden death in the survivor of cardiac arrest may be treated with an automatic implantable cardioverter defibrillator (AICD) that senses the onset of these ventricular dysrhythmias and delivers a synchronized 25-joule electrical discharge.

Table (11) Potential complications of ICD surgery:

<table>
<thead>
<tr>
<th>I) Complications resulting from the subclavian stick technique</th>
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<tr>
<td>Pneumothorax</td>
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<th>II) Surgical complications related to the pulse generator</th>
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<tr>
<td>Pocket erosion</td>
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<tr>
<th>III) Surgical complications related to the ICD leads</th>
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<tbody>
<tr>
<td>Lead dislodgement</td>
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</table>
Contraindications:

Implantation of an ICD is contraindicated in any patient who has a remedial cause of ventricular arrhythmias such as acute myocardial infarction, myocardial ischemia, electrolyte imbalance, drug toxicity, hypoxia, or sepsis.

D) The ventricular assist device:

The VAD is a blood pump that is designed to assist or replace the function of either the right or left ventricle. In the absence of right or left ventricular ejection, the RVAD supports the pulmonary circulation, while a LVAD provides systemic perfusion respectively. Implantable VADs are positioned intracorporeally in the anterior abdominal wall or within a body cavity other than the pericardium.

Extracorporeal VADs may be located in a paracorporeal position, along the patient’s anterior abdominal wall, or externally, at the patient’s bedside.

Infrequently, the heart is unable to meet systemic metabolic demands despite maximal pharmacologic therapy and insertion of the IABP. Under these circumstances, devices that actually pump blood and bypass either the left or right ventricle are required. These devices are effective because the injury producing myocardial dysfunction takes place intraoperatively and, more important, is often reversible. A second group of patients who have shown benefit from assist devices are those with chronic heart failure. These devices allow for hemodynamic support as a temporary measure prior to heart transplantation.

Complications of VADs are inadequate LVAD flow, right ventricular failure, hemorrhage, thromboembolism, infection, multisystem organ failure, device malfunction, and pump dependency.
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Arabic Summary
مع ازدياد الحاجة إلى المساعدة الميكانيكية للدورة الدموية تطورت الأجهزة المستخدمة في ذلك من حيث الترتيب والوظيفة والتحكم، والأجهزة المتاحة حالياً هي نتاج لعمل وجهد طويل وشاق قام به العديد من الباحثين والأطباء.

هذا وبالرغم من تحسن الأساليب المستخدمة فحماية القلب أثناء إجراء عمليات القلب المفتوح فقد يواجه الأطباء عدم قدرة القلب على الأداء بالرغم من استخدام الجرعات القصوى من العقاقير المضادة والداخنة للقلب وللدورة الدموية، ووجد أن نسبة تتراوح بين 50% إلى 1% من مرضى عمليات القلب المفتوح لا يمكن فصولهم من ماكينة القلب والرئة الصناعية وبالتالي فهم يحتجون إلى استخدام أحد الأجهزة الميكانيكية لمساعدة الدورة الدموية، ونتيجة لاستخدام مثل هذه الأجهزة الميكانيكية لمساعدة الدورة الدموية، تحدث تغيرات فسيولوجية في أعضاء الجسم المختلفة، ولتقليل مخاطر ومشاكل هذه التغييرات على المريض يجب على الجراح وطبيب التخدير والمسؤول عن تشغيل الأجهزة المغيرة للدورة الدموية أن يكونوا على دراية شاملة لك التغييرات.

من أهم الأجهزة المستخدمة لدعم الدورة الدموية سواء داخل حجرة العمليات أو خارجها ما يلي:

- منظم ضربات القلب الصناعي.
- المضخة الورودية في داخل الأورطي.
- الأجهزة المساعدة للبطين.
- جهاز الصمامات الكهربائية المنزوع.

(1) منظم ضربات القلب الصناعي:
إن منظم ضربات القلب الإلكتروني منه ما هو مؤقت ودائم والتي تحت القلب على البض كهربياً.

يتكون منظم ضربات القلب من مصدر طاقة "بطارية" ودائرة كهربائية وللمولد بما فيها الوصلات والتي تتم وتنظم عملية إثارة القلب.
ويستخدُم هذا الجهاز لعلاج البطيء الدائم لضربات القلب إذا كان ذلك مُؤثراً على آليات الدورة الدموية، وعلى ذلك فإن أهم دواعي الاستخدام هو فشل القلب في تكوين النبضة أو في توصيلها للأنابيب إلى الأذن.

إِن لاستخدام هذه الأجهزة مضايعات عديدة منها ما هو:

• مضاعفات مبكرة:
  - تجمع الدموع حول الرئة أو تجمع الدموع حول الرئة تُقرب عضلة القلب، وضع الوصلات في الشريان بدلاً من الوريد، وأخيراً إصابة الأعصاب المغنية للذراع.

• مضاعفات متأخرة:
  - ومنها حدوث جلطات نتيجة التهابات أو مضاعفات في الأوعية الدموية ناتجة وجود الوصلات داخلها.

وهكذا أي مضاعفات تحدث مبكرة أو متأخرة الحدوث ومنها حدوث عدم انتظام في ضربات القلب نتيجة الجهاز أو أن الجهاز المولد نفسه لا يعمل بصورة غير سليمة.

٢) جهاز الصدات الكهربائية الأوتوماتيكي المنزوع:

هو جهاز منزوع في المرضى الذين تعرضوا إلى حادث توقف في ضربات القلب، وذلك أن هذا الجهاز يستغرق بداية دخول المريض أو القلب في عملية توقف أخرى أو عدم انتظام في ضربات ووظيفة البطين فيقوم الجهاز بعملية كهربائية لإيقاف ارتعاش البطين وجعله يعمل بصورة جيدة في توليد نبضات أو لإعادة عدم انتظام نبضات القلب إلى انتظامها.

ولاستخدام هذا الجهاز بضايعات كثيرة منها:

• يوجد موارد على الرئة أو تجمع دموع على الرئة أو وجودهما معاً.
• إصابة الشريان وتكوين دموع، ووجود السداسية الهيائية، إصابة الأعصاب المغنية للذراع.
• حدوث تجمع دموع وعاصفة بكتيري في مكان زرع الجهاز.
• تزحلق وصلات وتغيير مكانها مع حدوث جلطات دموية في الوريد الذي به الوصلات مع حدوث سطحة رؤية.

لذلك لا يجب أبداً زرع هذا الجهاز في المريض الذي يعاني من جلطة حادة بالقلب، عدم انزعج في ملامح الدم، تسمم بالعقاقير، نقص نسبة الأكسجين بالدم أو يعاني من تلوث والتهابات بكتيرية.
المفصّلة البالونية في داخل الأورطى:

المضخة البالونية في داخل الأورطى عبارة عن بالونة في نهاية قسطرة تم تصميمها لكي تحسّن التوازن بين إعداد عضلة القلب بالأكسجين وبين حاجتها إلى عملها، تحسّن تناغم سعة الجسم المختلفة، ومن المكونات الأخرى للمضخة البالونية في داخل الأورطى: المضخة ومصدر الغاز وجهاز تحكم.

وتستخدَّم المضخة البالونية في داخل الأورطى في مرضى جراحات القلب عندما يصعب فهم ماهية القلب والرئة الصناعية، وعندما يحدث خلل في سريان الدم وضغطه، وعند حدوث نقص الدم المغذي لعضلة القلب لفترة طويلة مستمرة بعد استخدام مكينة القلب والرئة الصناعية على الرغم من استخدام العقاير وزيادة جرعتها.

وتتحسن غالباً وظيفة عضلة القلب مع استخدام المضخة البالونية في داخل الأورطى بالمحافظة على مستوى مناسب للدم المغذي لأساسة الجسم وعلى وظائف الأعضاء الحيوية، ولتحقيق أقصى استفادة من عمل المضخة البالونية في داخل الأورطى يجب أن تتوافق مع ضربات القلب ومن المهم أيضًا التحكم في معدل ضربات القلب والعمل على استنفادها. وعندما تعود وظيفة القلب إلى طبيعتها يتم تقليص عدد مرات نفخ البالونة وتريفيها تدريجياً بشرط عدم حدوث تدهور في أداء عضلة القلب ويتم إزالة المضخة البالونية في الشريان المغذي: له من أهم مضاعفات المضخة البالونية في داخل الأورطى نتيجة لحدود حمله، وقد يحدث أيضًا تشبع جدار الأورطى أو انفجار المضخة البالونية بما أن الصافات الدموية قد تدمر مما يؤدي إلى نقص عددها بالدم.

الأجهزة الممّعدة للبطن:

الجهاز المساعد للبطن عبرة عن مضخة للدم تم تصميمها لتساعد البطين أو تحل محله سواء البطين الأيمن أو الأيسر، ويسبن الجهاز المساعد للبطن الأول الدورة الدموية الرئوية بينما يبعد الجهاز المساعد للبطن الأيسر الدورة الدموية النظمية، ويمكن تقسيم الأجهزة المساعدة البطين إلى:

أ) أجهزة يتم وضعها داخل جسم المريض:
   - في جدار الأنبام الأمامي.
   - أو في نجد النبطن.
ب) أجهزة توضع خارج جسم المريض:
- إما تعلق إلى جدار البطن.
- وإما توضع بجانب سرير المريض.

وقد لا يستطيع القلب بعد إجراء عمليات القلب المفتوح أن يؤدي وظيفته على الرغم من استخدام الأجهزة القصوى من عناصر استخدام المضخة البارودية في داخل الأورطى. وفي هذه الحالة تم استخدام الأجهزة المساعدة للبطن حيث تقوم بمساعدة القلب حتى يتعافى من الإصابة التي لحقت به، وقد يستفيد مرضى هبوط القلب المزمن من الأجهزة المساعدة للبطن حيث يتم استخدامها لمساعدة الدورة الدموية حتى يمكن إجراء عملية زرع قلب بشرى.

ولالأجهزة المساعدة للبطن مضاعفات منها نقص كمية الدم التي يتم ضخها من خلالها، وحدوث هبوط في البطن الأيمن، وحدوث نزيف، وكون حلقات وسادات شربانية، وحدوث التهاب بذرة، وفشل في أجهزة الجسم، وجود عيوب في أداء المضخة الصناعية، واعتماد المريض على المضخة الصناعية وعدم القدرة على فصلها عنها.
التدخل التخديرى في مريض
يحمل أجهزة بالقلب

تمهيد للحصول على درجة الماجستير في التخدير وال căngة المركزية

رسالة مقدمة من
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