INTRODUCTION

The direct mortality associated with cardiac surgery operations has steadily declined as these operations became more familiar, and as improvement have been made in methods of preoperative preparations and in postoperative management ***(John el al., 2000).***

The complications secondary to cardiac operation which usually occur in I.C.U in early postoperative period still represent serious threats to patient survive ***(John et al., 2000).***

The natural of medical complications related to cardiac operations are extremely variable in their manifestations, and methods of management may be quite different when they occur in patients who have not been operated ***(Bojar, 2011).***

The medical complications following cardiac operations involve disturbance in cardiac rhythm and contractile slate, electrolyte balance, as well as in the functions of the renal, pulmonary, gastrointestinal, hematopoietic, or central nervous systems ***(Nashef et al., 1999).***

The early postoperative course for most patients after cardiac surgery is characterized by typical pattern of pathophysioiogical derangements that benefits from standardized and multimodal of care process which have been used to improve efficiency, quality and safety of patient ***(Mastrigl et al., 2005).***

AIM OF THE ESSAY

The aim of this essay is to discuss incidence and management of postoperative complications occurring in I.C.U among patient undergoing cardiac surgery.

POST-OPERATIVE CARDIOVASCULAR DYSFUNCTION

It is not surprising that cardiac dysfunction after cardiac surgery is a major cause of MOD. Postoperative cardiac complications may include bleeding, pericardial tamponade, arrhythmias, right and/or left ventricular failure, coronary vessel restenosis, valvulopathy, and the less frequently cardiac wall rupture and/or perforation. Both tachyarrhythmias and bradyarrhythmias represent a major cause of morbidity, increased LOS, economic burden, MOD, and mortality. Though atrial tachyarrhythmias (atrial fibrillation) is the most common arrhythmia after cardiac surgery, ventricular arrhythmias and conduction disturbances may also transpire ***(Peretto et al., 2014)***.

Upon arrival in the ICU, an efficient transfer of care from operation room staff to ICU staff is mandated, while at the same time vital signs are to be maintained stable. The initial goals in postoperative cardiac recovery are sufficient analgesia, normothermia, adequate oxygenation and ventilation, control of bleeding, restoration of intravascular volume, optimization of blood pressure and cardiac output to maintain organ perfusion and metabolic stabilization ***(Roekaerts and Heijmans, 2012)***.

Table (1): Expected hemodynamic variables in the immediate post-operative period ***(Roekaerts and Heijmans, 2012)***

Parameter	Expected value
Mean arterial pressure (MAP)	60 - 90 mmHg
Systolic blood pressure (SBP)	90 - 140 mmHg
Right atrial pressure (RAP)	5 - 15 mmHg
Pulmonary artery wedge pressure (PAWP)	10 - 15 mmHg
Cardiac index (CI)	2.2 - 4.4 L/min/M2
Systemic vascular resistance (SVR)	1400 - 2800 dyn-s-cm5

Pathophysiology during the early postoperative period

Hypothermia

Hypothermic cardiopulmonary bypass is usually terminated after the patient has rewarmed to a core body temperature of at least 36° C ***(Ho et al., 2009)***. However, patients usually arrive in the ICU with lower core temperatures. This drop in temperature from end of CPB until arrival in the ICU is due to the cool ambient temperatures in the operation room, poor peripheral perfusion and anesthesia-induced inhibition of normal themoregulation. Even patients operated under normothermic CPB, have a tendency to significantly cool down before conclusion of surgery. Hypothermia has many potential adverse effects ***(Sessler, 2001)***. It increases the systemic

vascular resistance (SVR) which increases myocardial afterload and myocardial oxygen demand. This compensatory mechanism to provide core warming may contribute to slow warming of peripheral tissues. Drugs that provide vasodilation may improve peripheral perfusion. To prevent hypotension, warmed infusions should be administered concomitantly. Peripheral vasodilation augments heat loss, and core hypothermia may therefore persist. Hypothermia also precipitates shivering, thereby increasing CO2 production and oxygen consumption, and predisposes to ventricular arrhythmias and coagulation cascade impairments ***(Rajagopalan et al., 2008)***.

Therefore, warming should be hastened by forced-air warming blankets, heated humidifiers in the ventilator circuit and warmed infusion fluids. The use of other types of warming blankets or radiant heading hoods can also be considered ***(Frank et al., 1997)***.

Blood loss after cardiac surgery

Careful hemostasis in the operation room is the cornerstone in reducing postoperative blood loss. However, bleeding can also be medical and determing the cause of bleeding is often difficult. Although the clinical situation must be individualized for each patient, bleeding in general should not exceed 400 mL/hr during the first hour, 200 mL/hr for each of the first 2 hours, or 100 mL/hr over the first four hours ***(Bojar, 2011)***.

There are numerous medical causes for bleeding following cardiac surgery. Residual heparinization is common post cardiac surgery and usually occurs when insufficient protamine is used or heparinized pump blood is transfused following CPB. Platelet dysfunction is also common following cardiac surgery. The CPB circuit itself leads to contact activation and degranulation of platelets, resulting in their dysfunction. Fibrinolysis frequently occurs after CPB, caused by activation of inflammatory or coagulation pathways. Coagulation factors may decrease from activation and dilution in the CPB circuit. There has been a dramatic increase in the iatrogenic use of heparin and newer antiplatelet, antithrombotic and thrombolytic drugs during (interventional) treatment of acute coronary syndromes. If revascularization surgery is warranted immediately after these treatments, the anticoagulant effect of these drugs is notable in the postoperative period. Conventional coagulation tests are helpful to identify the coagulation abnormality contributing to the bleeding. Common laboratory testing includes Hb, platelet count, aPTT, INR, and fibrinogen level. Thromboelastography is also commonly used and has been demonstrated to reduce transfusion requirements ***(Levi et al., 2010)***.

Blood transfusion management

Transfusion burden may in the future be interpreted as a quality indicator in cardiac surgery that must balance risks and

benefits to achieve cost-effective optimal clinical outcomes ***(Shander and Goodnough, 2010)***.

Fluid resuscitation

Cardiac surgery and CPB elicit a systemic inflammatory response which produces a capillary leak. Therefore, fluid resuscitation with crystalloids and/or colloids is necessary to offset the hemodynamic consequences of the capillary leak and the vasodilation that occurs from rewarming and vasodilating drugs. However, the maintenance of intravascular volume in the leakage phase occurs at the expense of expansion of the interstitial space ***(Bundgaard-Nielsen et al., 2009)***.

After the capillairy leak has ceased and hemodynamics has stabilized, diuretics are often used to eliminate the excessive salt and water administered during surgey and the early postoperative phase. This forced diuresis may beneficially affect pulmonary function and early successful extubation. Several intraoperative measures that have been implemented throughout the years caused a reduction in the inflammatory response and may have contributed to the faster recovery times currently observed after cardiac surgery. The measures include the use of membrane oxygenation, centrifugal pumps, anti-fibrinolytic drugs and steroids, leukocyte filters and coated CPB tubings ***(Henry et al., 2011)***.

Monitoring and assessing volume status

Heart failure cannot be ascertained unless the volume status is optimal. However, it is difficult to ascertain volume loading using single haemodynamic measures. Pressure estimates such as pulmonary capillary wedge pressure and central venous pressure are generally unreliable indicators of LV and RV preload. Uncoupling between PCWP and LVEDP frequently occurs as a consequence of elevated pulmonary vascular resistance, pulmonary venoconstriction, mitral stenosis and reduction in transmural cardiac compliance. Volumetric estimates by echocardiography or transpulmonary thermal dilution techniques are more predictive of preload. In predicting fluid responsiveness in ICU patients, it is preferable to use more reliable dynamic indicators reflecting hypovolaemia, such as stroke volume variation, than static parameters ***(Renner et al., 2009)***.

Several devices are now being used to assess cardiac function based on pulse contour analysis of an arterial waveform. Routine monitoring typically includes continuous telemetry, measurement of the arterial blood pressure via an arterial catheter, measurement of the cardiac filling pressures via a pulmonary artery catheter (ie, right heart catheter, Swan Ganz catheter), continuous assessment of the arterial oxygen saturation via pulse oximetry, and continuous measurement of the mixed venous oxygen saturation via an oximetric pulmonary artery catheter. Such monitoring allows instantaneous assessment of cardiopulmonary physiology ***(Heijmans et al., 2010)***.

Echocardiography is of great value in the perioperative cardiac surgical setting. It not only is helpfull in assessing the optimal volume status, but may also immediately identify causes of cardiovascular failure, including valvular problems, cardiac tamponade, systolic anterior motion of the anterior mitral valve leaflet and pulmonary embolism. Echocardiography may differentiate between acute right, left and global heart failure as well as between systolic and diastolic dysfunction. If there are echocardiographic signs of RV failure, a pulmonary artery catheter (PAC) preferably with continuous SvO2 measurement should be introduced. PACs can differentiate between pulmonary hypertension and RV ischaemia, which necessitates a reduction of RV afterload. PAC and TEE are complementary to each other for diagnosis and treatment of the cardiac surgical patient. Indications for the use of a PAC are, high risk and/or complex cardiac surgery, hemodynamic instability, low cardiac output syndrome, pulmonary hypertension, differentiating between severe right and left ventriclular dysfunction, vasodilation/vasoconstriction, hypovolemia. SvO2 in combination with lactate concentration was used postoperative as a goal-oriented hemodynamic therapy to improve outcome ***(Carl et al., 2010)***.

Although pulmonary artery catheters are used routinely in most centers, there is a paucity of empirical evidence to support this practice ***(Sandham et al., 2003)***. Most pulmonary artery catheters are removed within 12 to 24 hours of surgery if

significant vasopressor, vasodilator, or inotropic therapy is no longer required. Fluid shifts should be closely monitored by frequent assessment of the central venous pressure and/or pulmonary artery occlusion pressure (ie, pulmonary capillary wedge pressure), chest and mediastinal tube drainage, urine output, and patient weight. Measurement of arterial blood gases, hemoglobin concentration, platelet count, coagulation parameters, serum electrolytes, and serum creatinine is routinely performed on a daily basis. Most experts maintain serum glucose between 140 and 180 mg/dL (7.8 and 10 mmol/L) during the period following cardiac surgery, since hyperglycemia is associated with worse outcomes in this population ***(Ma et al., 2015)***.

Risk stratification

Risk stratification is increasingly used in open-heart surgery to help adjust resources to predicted outcome. According to all scoring systems major clinical risks include heart failure, unstable coronary syndromes, significant arrhythmias and severe valvular disease. The euro SCORE is mostly used to calculate operative risk, although updating its sensitivity is warranted ***(Heijmans et al., 2003)***.

In addition to scoring systems, levels at hospital admission of B-type natriuretic peptide (BNP) and the amino-terminal fragment of pro-BNP (NT pro-BNP) are powerful predictors of outcome with regard to in-hospital mortality and rehospitalisation in heart failure patients ***(Lainchbury et al., 2010)***.

Cardiac dysfunction

Poor cardiac function during the early postoperative period is associated with an increased risk of death ***(Lazar et al., 2009)***. It is usually suspected when there is unexplained postoperative hypotension, tachycardia, or pulmonary edema. Evaluation of suspected cardiac dysfunction consists of the following ***(Aps, 1997)***:

A- Mechanical complications

Not all mechanical complications of cardiac surgery involve the heart. Non-cardiac mechanical complications include pneumothorax, hemothorax, and endotracheal tube malposition. These complications can be readily identified on a chest radiograph. Pneumothorax and hemothorax often require tube thoracostomy, while endotracheal tube malposition requires repositioning. Apart from the complications strictly related to the surgical technique, such as cardiac tamponade following suture leakage, occlusion of an artery graft or paravalvular regurgitation, one of the most common and severe complications that can occur immediately after cardiac surgery procedures is heart pump failure ***(Siribaddana, 2012)***.

Mechanical complications of cardiac surgery are usually detected by echocardiography or invasive hemodynamic assessment. Examples include spasm or occlusion of a coronary artery graft, prosthetic valve paravalvular regurgitation, cardiac tamponade, hematoma, and systolic anterior motion of the mitral valve with

left ventricular outflow tract obstruction. Treatment of the mechanical complications of cardiac surgery is usually surgical ***(Carl et al., 2010)***.

B- Physiologic complications

Once mechanical complications have been excluded, an initial decision should be made about whether the patient's cardiac dysfunction (ie, low cardiac output) is most likely due to an insufficient stroke volume or heart rate. If it is likely due to diminished stroke volume, then it should be determined whether this is likely due to inadequate left ventricular preload (ie, intravascular hypovolemia), excessive left ventricular afterload (ie, hypertension), and/or poor inotropy (ie, cardiomyopathy). Data from the echocardiogram and/or invasive hemodynamic assessment can be used to inform these judgments. Treatment should be directed at the presumed abnormality, while the underlying cause is sought.

Different degrees of depression of the myocardial function can be observed in most of the patients, whereas heart failure occurs in around 20% of the patients in the postoperative period, contributes to early mortality, and is caused by several factors: insufficient ventricular preload, inefficient ventricular contraction and increased ventricular after-load ***(Mebazaa et al., 2010)***.

Indeed, cardiac surgery often results in an impairment of myocardial function during the intervention: this can lead to

reperfusion damage at the end of surgery, when normoxia is restored ***(Corral-Velez et al., 2015)***.

Inadequate preload

The best measure of left ventricular preload is the left ventricular end diastolic volume (LVEDV) that can be evaluated by echocardiography or certain invasive hemodynamic measures: the pulmonary artery occlusion pressure (if a pulmonary artery catheter is in place) or the left atrial pressure (if a left atrial catheter has been placed). These hemodynamic measurements approximate the left ventricular end diastolic pressure (LVEDP), from which assumptions about the LVEDV can be made. Left ventricular preload may be inadequate during the immediate postoperative period because of loss of vasomotor tone, increased capillary permeability, intraoperative and postoperative blood loss, or high urine output due to hypothermia. In addition, left ventricular compliance is frequently reduced following cardiac surgery, producing diastolic dysfunction; a higher LVEDP is required to maintain a given preload in affected patients ***(Slama et al., 1996)***.

Hypovolemia induced by fluid loss, and left ventricular diastolic dysfunction (LVDD) are common causes of inadequate preload volume. The reduced compliance is the result of postischemic injury and "myocardial stunning," which results in inadequate myocardial diastolic relaxation. As an example, patients with significant left ventricular hypertrophy

due to aortic stenosis or hypertrophic cardiomyopathy often require pulmonary artery occlusion pressures of 18 to 22 mmHg to maintain an adequate preload to support the cardiac output ***(Ferreira et al., 2015)***.

Excessive afterload

Excessive ventricular after-load is usually related to postoperative hypertension, a frequent postsurgery reaction that can lead to a myocardial ischemia because of increased oxygen demand and ejection fraction decrease ***(Lonjaret et al., 2014)***.

Postoperative hypertension is common; it can cause decreased stroke volume and increased myocardial oxygen demand ***(Imren et al., 2008)***. The presumed etiology is systemic vasoconstriction, likely related to hypothermia induced during bypass ***(Fremes et al., 1984)***. The observation that rewarming does not closely correlate with resolution of the hypertension suggests that other factors may also be involved, such as humoral factors provoked by cardiopulmonary bypass ***(Estafanous and Tarazi, 1980)***.

Patients judged to have excessive afterload should be treated with a vasodilator. Sodium nitroprusside is the vasodilator of choice to reduce excessive vasoconstriction in the immediate postoperative period. The blood pressure must be continuously monitored during therapy because vasoconstriction may improve quickly during rewarming from the hypothermia, leading to hypotension that requires immediate discontinuation

of the sodium nitroprusside. Hypotension that persists despite the discontinuation of sodium nitroprusside should be initially treated with intravenous fluids, but may require vasopressors. Such hypotension is more common with normothermic bypass and longer cardiopulmonary bypass times, and less common in patients with diabetes, peripheral vascular disease, or a left ventricular ejection fraction of less than 40 percent ***(Downing and Edmunds, 1994)***.

Poor inotropy

Inadequate preload should be corrected by administering intravascular volume. Such patients should be monitored closely because many will continue to exhibit signs of cardiac dysfunction even after their preload has been corrected. This suggests that there is coexisting poor inotropy and therapy should be redirected toward the poor inotropy. Impaired ventricular function is suggested if the echocardiogram shows poor ventricular contraction and a low ejection fraction, or if the invasive hemodynamic measurements include a low cardiac output accompanied by a normal or high pulmonary artery occlusion pressure and normal or high systemic vascular resistance ***(Carl et al., 2010)***.

Postoperative ventricular function may be decreased due to intraoperative events, postoperative events, and/or hibernating myocardium. Examples include inadequate myocardial protection during cross-clamping of the aorta, ischemic