1

Intensive Care Management of Patients in the First 24 Hours After Cardiac Surgery

Villalobos J. A. Silva, Aguirre J. Sanchez, Martinez J. Sanchez,
Franco J. Granillo and Garcia T. Zenón
Universidad Nacional Autónoma de México (UNAM), Tamaulipas
México

1. Introduction

The cardiac surgery continues having a fundamental roll in the therapeutic arsenal of many heart deseases in spite of the spectacular advances that determined drugs or different forms of interventionist cardiology have experimented during the past few years. The present impact of the heart surgery is due to the constant increase of the cardiovascular risk factors, related to the increase in the life expectancy in last the three decades, the clinical approach of the ischemic cardiopathy towards the repair has taken to the creation and development of techniques and methods at the moment used in the miocardic revascularization surgery; the roll of the coronary surgery initiated by Sabiston in 1962 and popularized by Favaloro in 1967 has had an exponential development with the purpose of to exclude the ill part from the artery by placing a bypass to improve the perfusion of the ischemic area. Nevertheless the other side of the balance is the pharmacological treatment whose objective is to look for the balance between the supply and demands in the ischemic scope at the expense of a smaller consumption of oxygen (VO2), diminution of the inflammatory local metabolism, control of trombotics phenomena, etc. Now on the basis of the knowledge and acquired experience we establish a margin of durability of 90% to 10years in grafts of internal mammary (AMI) and 50-60% of venous grafts (HV), depending on the vascularized area and the type of vein, in relation to the arterial grafts the average life is of 90% to 5 years with sufficient information of early stenosis problems. Most of the post-operated patients recover in a fast and complete form, which depends on the quality of the performed surgery and an opportune and suitable handling as all the symptoms of the organism recover of the effects of: anesthesia, cardiopulmonary derivation (CEC) and surgical stress. Nevertheless some patients who present combinations of indicators of preoperative risk like: age outpost, antecedents of miocardic revascularization, recent and acute miocardic heart attack (IAM), ejection fraction (EF) low or diabetes, have a much greater surgical risk to the one of the habitual patient. At the moment there are certain characteristics that have determined a fast recovery as they are the early extubation, to avoid major sedation, the disconnection of the support devices and the suspension of drugs as rapidly as possible. A fast treatment before a: Low cardiac cost, alteration of the pulmonary function, hemorrhages, coagulopathy and fever is essential to be able to obtain a fast recovery of the patients; at the moment the surgical indications of the miocardiac revascularization (RVM) have been based in relation to the number and degree of affectation of the coronary arteries, decreased ventricular

function and more and more by the increase of percutaneous interventions there are patients with greater risk and worse diagnosis which has modified the results in the last years, as well as a reduction in the number of surgeries per year. Mortality in Europe and the United States is lower than 2,5% with a survival that oscillates respectively between 97-80% of 1 to15 years. Its reduction increased as of the eighth year, is in relation to the average life of the grafts, its occlusion, progression of the disease and development of its comorbidities. [1,2,3.]

2. Preoperative surgical risk

From the antiquity, man has tried to anticipate or predict the facts to come, that is to say the prediction of the results in cardiovascular surgery has been and continues being a goal. The stratification of the patients in different risk levels, previous to the accomplishment of a cardiovascular surgery has diverse intentions, among them the clinical decision making with respect to the accomplishment or not of the surgical procedure or the derivation of the patient for another type of treatment. It is necessary to establish preoperative conducts that can reduce the risk to cost-effectiveness, to establish the stratification of the risk sometimes is difficult, being implicit some factors of individual risk that can be interpreted of different way and has like objectives:

1. Identification of patients of high risk with critical allocation, to adapt the perioperatory problems.
2. To diminish the morbidity and mortality, establishing strategies of preoperative, intraoperating and post-operative treatment.
3. It is indispensable to know the heart disease suitably. (Physiopathology, diagnosis, treatment, and perioperatory complications).
4. A suitable evaluation and treatment of the cardiopathy surgical patient, requires a team work and communication between: patient-surgeon, cardiologist and **intensivist**.
5. Several factors are known that modify the individual risk as they are the age, the sort, previous the cardiovascular function, the renal diseases and you will tilt, respiratory function and other related factors.

John and collaborators established 7 fundamental criteria like and 13 important criteria which are an influence in the morbi-mortality of the post operated patient. (CHART 1)

Age

Every time it is more frequent than patients with age greater to 60 years, are admitted. The cardiovascular risk is increased by above of 65 years by the presence of his comorbidities, age outpost with cardiac symptoms and a key point is the selection of this type of patients, since with this secondary mortality to the surgery is determined. In these patients it is frequent the existence of important complications as they are the low cardiac cost, acute myocardial infarct of the perioperatory, surgical re-intervention by bleeding, acute renal insufficiency, pneumonia, prolonged ventilation, all these increase to the percentage of complications and mortality.

Gender

Many studies exist that have determined that the woman has a greater risk of mortality after the coronary revascularization; because they have minor corporal surface and the size of the coronary glasses is smaller. As far as the sort it does not seem to be predictive of mortality in valvular procedures, mitral and aortic.

Predictors of post-CABG mortality	
Age	Height
Sex	Weight
Urgency of operation	PCI during current admission
Prior heart surgery	Date of most recent MI
LVEF	History of angina
Percent stenosis of LM coronary artery	Ventricular arrhythmia
Number of major coronary arteries with >70% stenosis	CHF
	Mitral regurgitation
	DM
	CVD
	PVD
	COPD
	Creatinine level

Chart 1.

Cardiovascular state

Is the most important aspect of perioperative mortality, the important factors are: severe valvular disease, reoperation, left ventricular function, infarct to the myocardium previous, cardiac insufficiency, emergency surgery and upheavals of the rate.

Respiratory function

The pulmonary disease chronicle is a risk factor to prolong the mechanical ventilation, to have a more difficult weaning, and associated to the pulmonary arterial hypertension, its extubation requires major care.

Mortality

The explanation of the diminution in mortality has been the present methods of myocardic protection with retrograde cardioplegia, hypothermia since the ischemia diminishes triphosphate of adenosine (ATP), altered sanguineous flow, calcium overload, reduction of intracellular calcium sensitivity, sarcoplasmatic dysfunction and the presence of free oxygen radicals. The morbidity and mortality fall significantly in spite of being increased the risk factors, the tendency of the secondary complications of comorbid sufferings have increased until in 30-40%.

The preoperative cardiovascular evaluation provides recommendations for stratification with risk and handling of proposals by the American College of Physicians: Medical history, clinical exploration, ECG, X-ray of thorax, laboratories, tests ECG to the exercise, ambulatory monitoring (Holter), ventriculography to radio nuclear, heart ultrasound, coronary angiography, thallium scintiscanning. [4.5.6.7.8.9] (CHART 2)

In resistance to indices of multi-factor risk, the functional classification New York Heart Association (NYHA) and the American Society of Anesthesiology (HANDLE) are used of routine by the anesthesiologist. Nevertheless, these classifications do not designate a predicting result after the surgery reason why its predictive ability in operating room is limited.

ASA

Healthy patient, with a process located without systemic affection.

PREOPERATIVE EVALUATION OF CARDIAC SURGERY
History
1. History of bleeding: Use of antiplatelet and anticoagulant medication. 2. Smoking (COPD, bronchospasm). 3. Alcoholism (cirrhosis) 4. Diabetes (reactions to protamine, prior infection) 5. Neurological symptoms. 6. Venous insufficiency. 7. Distal vascular reconstruction. 8. Urologic symptoms. 9. Gastric ulcer or gastrointestinal bleeding. 10. Infections (Urol) 11. Allergies
Physical Examination.
1. Skin infection / rash. 2. Dental Caries (dental) 3. Vascular examination (carotid, abdominal aneurysm and peripheral pulses) 4. Heart / Lung (congestive heart failure, new murmur). 5. Varicose veins.
Laboratories
1. Hematologic: PT, PTT, platelets, Hb, Time Ivy. 2. Chemistry: Electrolytes, BUN, QS, PFHS. 3. Urinalysis. 4. Chest X-ray AP and lateral. 5. Electrocradiogram.

Chart 2.

Patient with slight systemic disease.
Patient with serious, but non incapacitated systemic disease.
Patient with serious and incapacitated systemic disease, that constitutes a constant threat for the life.
Dying patient, whose life expectancy does not exceed the 24 hours, is realized or not it to his surgery.

NYHA

Patient with heart disease, without limitations of physical activity.
Patient with disease, with slight limitation ofordinary physical activity, fatigue, palpitations, designs or angina pain.
Patient with heart disease, with noticeable limitation of physical activity, less than the ordinary physical activity, cause tires, palpitations design or pain angina.
Patient with heart disease, incapacity to walk and physical activity, symptoms of cardiac insufficiency, angina.
There are over 100 studies of perioperatory risk-prognosis stratification that have tried to identify adverse predictive factors, the greater limitation of these studies is that they were realized in a single institution, small groups, or the cardiac experience of the anesthesiologistwas the one taken into account. Between the preoperative risk-prognosis

classifying stratification, transoperatory and post-operative factor risks are the following scales: Parsonnet, Tumman, Higgins, Tu, Hannan, Connor, Cleveland Clinic, EuroSCORE, Ontario Provincial Risk (OPR) among others, which vary in predicting correlation of risk of morbidity and mortality in relation to distribution of patients by risk, the average one hoped, and the averageobservada.[10, 11.12.13] (**Chart 3**)

3. Stratification of risk using data base

The basic information of data is used to improve the clinical practice producing reports with validity and application, evaluating the results, optimizing and improving the cares of the patient, the high mortality is identified in a small and specific sub-group classified like of high risk. The obtained results are used to change institutional programs diminishing mortality in patients of high risk like of smaller risk, mainly in patient put under coronary revascularization diminishing the mortality from 4,5% to 1,5%.

The European for System Cardiac Operative Risk Evaluation (EuroSCORE) is a predicting logistic model of hospitable mortality in patients submissive cardiac intervention, starting off of 18 variables of risk and with a coefficient beta associated to each of them, it provides the probability of dying of each individual, this model was created and validated initially in across-sectional study of 19,030 European patients in 1999, and it has become, since then, in the used model more in the world in this type of patients. Most of the authors agree in raising that the Euroscore is a system simple additive that it provides to facultative a tool of easy handling to consider the death risk. A variant of the much more simple logistic model, denominated Euroscore additive, that awards a weight determined to each factor of risk that presents the patient, the sum of these weights exists provides the approximated probability to die.

The interesting exercise to compare logistic Euroscore with additive already has been realized, but in fact it contributes certain confusion, and the conclusions that are obtained are understood easily observing.

The preoperative surgical risk models are made on the basis of cardiac surgery using cardiopulmonary bypass (CPB). However, it can be applied to off-pump cardiac surgery, as evidenced by Vazquez Roque, in a study in 208 patients undergoing bypass surgery without cardiopulmonary bypass and found that the mean EuroSCORE was significantly higher in patients who died. When comparing patients with and without major complications can see that the mean EuroSCORE was also significantly higher in patients with major complications.

Without fatal perioperative morbidity results in an increase of stay in postoperative care unit and overall hospital stay, this increases resource consumption and costs per patient. The EuroSCORE proved to have a discriminating power and acceptable calibration in predicting these events, we can say that Euroscore, despite being designed a risk score based on patients who underwent cardiac surgery using CPB may be used to predict the risk of death and major complications in patients who are going to be revascularized without the use of cardiopulmonary bypass. This is a novel technique that still suffers from risk scores based on the preoperative characteristics of their own patients. The study uses databases Euroscore retrospective studies, to provide predictive models of morbidity, mortality and prolonged stay in the postoperative intensive care unit, which can be used to improve the quality of postoperative care in different institutions is a tool to categorize patients for cardiac surgery in several subgroups.

Risk factors	Punctuation
Female sex	1
Morbid obesity	3
Hypertension (p. s.> 140 mmHg)	3
EJECTION FRACTION	
Good> 50%	0
Moderate 30-49%	2
Poor <30%	4
age years	
70-74	7
75-79	12
>80	20
Reoperation	
Primary	5
Secundary	10
Preoperative BIAC	2
Left Ventricular Aneurysm	5
Emergency surgery and angioplasty	10
Dialysis	10
Catastrophic States	10-50
Mitral valve surgery	5
PAP > 60mmHg	8
Aórtic	5
Gradient > 120 mmHg	7
Revascularization + Valve Surgery	2

Chart 3.

Due to differences in adult cardiac surgery in the countries of Europe the EuroSCORE is responsible for assessing the quality of surgical care, the analysis is performed for each individual to individual and predicting mortality among the countries of the study is were: Germany, England, Spain, Finland, France and Italy. The EuroSCORE model was satisfactory in all countries with a $p < 0.05$, despite epidemiological differences between European countries the discriminative power of EuroSCORE was good in Spain and in other countries excellent. [14, 15,16,17, 18,19,20.]

4. Cardiac risk evaluation of anesthetic (CARE)

The scale used most recently is called Cardiac Anesthesia Risk Evaluation Score (CARE), prospective studies in cardiac surgery demonstrated a significant number of prognostic information was obtained from only a few clinical variables or clinical trial, were validated

and compared with 3.548 patients with Parsonet, Tuman, and Tu, the CARE is a simple risk classification predicts morbidity and mortality on a scale which means ordinary CARE1 low risk, high risk means CARE5 and Care 2-4 as intermediate risk, based on clinical trial recognized three variables: [21.22]

Scale of anesthetic Cardiac Risk Assessment (CARE).

- Heart disease stable without other medical problems, surgery scheduled for a low surgical risk.
- stable heart disease with one or more controlled medical problems, set to a low-risk surgery.
- uncontrolled medical problem, or patient scheduled for surgery high risk.
- uncontrolled medical problem, scheduled for surgery high risk.
- advanced or chronic heart disease, scheduled for cardiac surgery that delayed it can complicate or improve their lives.

Cardiopulmonary bypass (CBP)

On-pump bypass also known as cardiopulmonary bypass is a method used in coronary bypass surgery, this device has been used in cardiac surgery since 1960, due to the high incidence of perioperative mortality due to low spending, it is increasingly used in the last decade, increasing its survival up to 60%. Cardiac surgery and cardiopulmonary bypass activate the inflammatory response, characterized by cardiovascular and pulmonary disorders, this inflammatory response that occurs during cardiac surgery is presented by three processes:

- Contact of blood with the cardiopulmonary bypass machine.
- Development of ischemia and reperfusion injury.
- Release of endotoxins.

The extent and duration of the inflammatory response depend on many factors including the composition of the solution pump, the presence of pulsatile perfusion, pharmacological agents used to reduce the response, the use of mechanical filtration, the type of extracorporeal circuit and temperature during cardiopulmonary bypass. During CPB flow decreased splenic occurs, which induces the crossing of endotoxins by the lumen, activating the inflammatory response, endotoxins are potent initiators of the inflammatory cascade, which in turn causes production of cytokines and complement activation.

A frequent complication of systemic inflammatory response is the evolution to multiple organ failure (MOF) including respiratory failure, shock and renal failure, development of FOM is the most important determinant for the postoperative increases those patients who have risk factors such as prolonged mechanical ventilation (intubation ≥ 48 hours), increased volumes of lower urinary nitrogenous and persistence of vasopressors, resulting in an increase in mortality to 41%. The inflammatory response and also condition FOM phenomena of hemolysis, thrombocytopenia and leukopenia, in the first 24 hours of the end of cardiopulmonary bypass can be seen that the total count of leukocytes undergoes an increase with significant changes in the differential count, the leukocytosis persisted in 72 hours, in the differential count reports a significant increase in neutrophils and monocytes and decreased lymphocyte counts during the first days. Postoperative fever in the second and third day in patients undergoing cardiac surgery is accompanied by an increase in neutrophils, two times the initial value during cardiopulmonary bypass activation of neutrophils is manifested by leukocyte sequestration in the pulmonary circulation at the

time of reperfusion of the vascular bed can lead to endothelial and parenchymal injury, in immunocompromised patients and prolonged intubation favors the development of infections. Neutrophils represent the most significant source of oxygen free radicals, which is associated with myocardial dysfunction and pulmonar.[23, 24,25,26,27.]

The CBP decreased flow causes splenic bacterial translocation which conditions, these cross the intestinal lumen and activate the inflammatory response have different degrees of hemodynamic compromise, noting in addition sequential elevations of endotoxin followed by elevations in levels of cytokines and these correlate the degree of myocardial dysfunction. Endotoxins are potent initiators of the inflammatory cascade causing cytokine production, production and complement activation, their presence is associated with the development of lactic acidosis, decreased peripheral vascular resistance and left ventricular dysfunction. The cardiovascular effects of cytokines are mediated by nitric oxide which involves interaction between leukocytes and endothelium and the mechanisms that cause these effects are the presence of circulating endotoxin, lipopolysaccharide (LPS) of gram-negative bacterial cell wall that interact with host cells to promote the release of mediators, lipopolysaccharide increases because the immune response by binding to protein carriers of LPS forms a complex that is a thousand times more potent to induce the release of tumor necrosis factor (TNF) and the union lipopolysaccharide occurs between the CD14 receptor of macrophages is that the activation of kinases and TNF. The inflammatory response can be maintained by several factors including the production of cytokines such as TNF-alpha (α), interleukin 1 (IL-1), IL-1 (beta), interleukin 2 (IL-2), interleukin 6 (IL-6), interleukin 8 (IL-8), interleukin 10 (IL-10), interferon and colony stimulating factors, which may be related to postoperative complications. The release of cytokines produce clinical manifestations in patients with cardiopulmonary bypass, such as fever, altered level of consciousness that occurs microembolisms due to encephalopathy. The crystalloid solutions used to prime the pump bypass hemodilution while causing turbulence and osmotic pressure during cardiopulmonary bypass cause lesions in the cell membrane of erythrocytes and hemolysis eventually causing mainly postoperative bleeding and coagulopathy platelet dysfunction. It is possible that renal failure during cardiopulmonary bypass is due to changes in renal perfusion during periods of hypotension or, for low blood flow, vasoconstriction and microembolism, likewise, hemoglobinuria may also cause significant renal dysfunction as a result of hemolysis during CPB, cardiopulmonary bypass can also cause susceptibility to infections, Sabick et al found that deep sternal infection occurs in 2% in patients undergoing extracorporeal pump versus 0.2% in off-pump patients ($p < 0.04$).[28]

Intra aortic balloon counterpulsation (BIAC)

The intra-aortic balloon counterpulsation is the method used in the treatment of severe cardiac dysfunction and potentially reversible in the perioperative and postoperative cardiac surgery is indicated in the shock associated with myocardial infarction or other complications intractable cardiac ischemia, with or without infarction, ventricular failure post CPB, and so on.

There are two main effects of this device, the first is to increase coronary blood flow improved myocardial oxygen availability by increasing diastolic perfusion pressure and the second is the blood moves during balloon inflation reduces ventricular work by reducing afterload with rapid deflation in systole and thus decreasing myocardial oxygen demand, this increases the heart rate up to 20%, thus the signs associated with BIAC surgical procedure are:

Low cardiac output syndrome, perioperative
As a bridge to cardiac transplantation
Acute mitral insufficiency
Perioperative arrhythmias are difficult to control

Christeson, showed that the use of preoperative IABP reduced hospital costs and length of stay in revascularized and reoperation, Dietl, also reported a stay of 10 days vs. 12 days in those with BIAC those who did not have it at a cost average hospital stay of $ 4000 per paciente. Although minor complications associated with IABP placement has been estimated at 6.5% and higher (vascular surgery and requiring transfusion) of 2.1% is greater than the benefit conferred on all preoperative placement to reduce mortality significantly vs to who are placed in the postoperative period (mortality 8.8% vs 28.2%, p <0.0001) in conclusion, the use of BIAC has increased over the past 10 years, significantly as evidenced by the record made in 29, 961 patients England and Canada, where the increase in aortic counterpulsation in cardiac surgery in the last 6 years is 47%. [29.30.]

Initial assessment and aost-aurgical therapy (TiPQ)

There should be a systematic evaluation of the patient immediately on arrival to postoperative intensive care unit (TiPQ), communication with the surgical team and anesthesia should provide an overview of the intervention performed and the response of the cardiovascular system and perioperative hemodynamic treatment and handling of medication. Although initially it may focus attention on an aspect of patient status (eg, existing arrhythmia on arrival), it is essential to develop a systematic approach to evaluation. The patient is fully independent and dysfunction of the elements of support systems can be fatal quickly. Surgical dressings must be kept intact during the first 24 hours in order to control the infection. If handling is necessary for diagnostic purposes must follow a strict technique. Upon transfer, the patient should continue with the pharmacological management started in the operating room and continued monitoring that includes at least fan movement, Electrocardiography digital, non-invasive blood pressure (PBIN) and pulse oximetry (SO2). Upon arrival to the ICU should be corroborated electrocardiographic tracing displayed on the monitor and make an immediate transfer line 12-lead ECG or chest circle when necessary. Capnography (PECO2) and central venous pressure (CVP) are measures that must be reactivated immediately, it also calibrates the transducer invasive blood pressure monitor confirms his bed and his blood pressure is checked immediately after the pacemaker (MCP) epicardial and functionality, if the patient arrives with pulmonary catheter also must be calibrated immediately and make a hemodynamic profile to assess their cardiovascular status at the time and determine current therapeutic recommendations behaviors common pulmonary catheter placement are EF <40%, patients with combined valve implantation (aortic-mitral) or severe acute heart failure diagnostic doubt its hemodynamic profile. It is important to immediately verify the patency of chest tubes and immediately upon arrival quantify pleural drainage at 15, 45 minutes and hourly for the first 24 hours, and assess their macroscopic features and clot formation. The ventilatory parameters must be set in the next 5 minutes upon arrival and assess the degree of de-recruitment and the need for alveolar opening Pa02/Fi02 if their relation ship is less than 200 mm Hg and if their hemodynamic status is not compromised, the monitoring hypothermia and its management is immediate. During the first 15 minutes should be evaluated central venous saturation (SvO 2), arterial blood gases, acid-base status, serum electrolytes (ABG) and serum examinations required, such as hemoglobin, platelets, hematocrit, serum

electrolytes, coagulation, time Ivy, myocardial enzymes, prealbumin, liver function tests (LFT), nitrogenous, Cystatin C, urine sediment and start of urine collection for urinary urea nitrogen (NUU), subsequent tests are given in the next 6 hours or before be necesario.[31, 32,33,34,35]

5. Postoperatively not complicated

At present, the cardiovascular monitoring is noninvasive and invasive integral part of the intensive care critically ill cardiac patient.

Cardiovascular monitoring

The proper outcome after cardiac surgery depends on preoperative and postoperative status of the myocardium rather than coronary anatomy in the postoperative period is uniformly diminished contractility compared to the preoperative magnitude and duration of this depression depends on the severity of chronic dysfunction, the presence of recent ischemic events, efficacy and complications of operative procedures and the intraoperative course. All intraoperative events play a significant role in recovery processes are the most important anesthetic management, cardioplegia and cardiopulmonary bypass duration. Events and preoperative and intraoperative interventions vary in magnitude and duration but in characteristic result in reduced myocardial contractility and compliance, which affects the postoperative management and eventual evolution. Ejection fraction in the preoperative less than 35%, the presence of ischemia or infarction in the immediate preoperative have substantial postoperative management, patients with outflow tract obstruction by a disease associated with hypertrophic chronic hypertension or stenosis aortic present particular difficulties postoperatively. It is always important to optimize left ventricular preload in TiPQ evaluated through filling pressures but not always filling pressures adequately reflect preload, defined as ventricular end-diastolic volume, so that ideally should be monitored with end-diastolic volume index and cardiac index continued, ie in real time. Patients with chronic volume overload, as in those with mitral regurgitation are dependent on adequate volume resuscitation in these patients the response of blood pressure and heart rate are usually a better guide to the proper preload because pulmonary occlusion pressure and pulmonary artery pressure are insensitive, except at the ends of hypovolemia and fluid overload in this situation, blood pressure and cardiac index can significantly change before filling pressures. [36.37]

Ventricular work is a crucial element in the postoperative management of heart surgery, the hypertrophic ventricular pressure overload or are intolerant to significant changes in heart rate when the heart rate is high, the filling time can be shortened enough to compromise the volume of end-diastolic volume and thus cardiac output, in contrast, when heart rate is below the time needed to develop the maximum end-diastole, cardiac output may decline relative to the low frequency. Assuming that the preload is adequate, a heart rate between 90 and 100 beats per minute is optimal for a hypertrophic myocardium, where compliance is significantly diminished synchronized atrioventricular contraction plays a significant role in ensuring optimal preload, so these patients require sinus rhythm or a dual-chamber pacemaker. The ventricle with volume overload in contrast, is more tolerant of tachycardia and loss of atrioventricular synchrony. When ventricular compliance at the end of diastole decrease as a result of increased heart rate, systolic emptying may be better in these patients,

sinus rhythm below 75 beats per minute tends to be more deleterious to an abnormal rhythm frequencies above 90 per minute, with low heart rates, prolonging the diastolic filling time committed ventricular ejection fraction because the ventricle is more dilated. Ultimately in ventricles with volume overload tachycardia and loss of atrioventricular synchrony can be better tolerated than sinus bradycardia. Determining the degree of reduction in contractility admission to TiPQ is problematic, the main contributors to the decrease in postoperative contractility including ejection fraction before surgery less than 35%, CPB time, especially if the duration exceeds 120 minutes. In patients undergoing valve procedures, it is advisable to perform intraoperative transesophageal echocardiography at the end of CPB as this is useful to assess valve function and ventricular dynamics. If the preoperative ejection fraction is greater than 35% and the operative course was satisfactory, decrease myocardial compliance in the first 4-6 hours in the unit and then quickly returns TiPQ values similar to or better than the preoperative values. Patients with an ejection fraction before surgery less than 35%, presence of perioperative ischemia or complicated operative course may require a longer time to recover or make permanent dysfunction, myocardial depression may persist for an extended period of time. These factors may affect the withdrawal of ventilatory support and necessitate the use of oxygen in prolonged, the tachypnea may be a reflection of compromised perfusion rather than primary respiratory failure.

Maintaining normal blood pressure is critical in the early hours of postoperative necessarily invasive measurement must be continued for at least 24 hours for analysis beat to beat but if it is non-invasive measurement must be measured regularly every 5 minutes but the objectivity of plethysmographic measurements are reliable non-invasive automatic in the absence of intense vasoconstriction and a very high frequency. Class I recommendation, level of evidence C. The optimal MAP in the first 6 postoperative hours especially in revascularized patients should be 65 to 80mmHg, maintaining adequate tissue perfusion to all organs and prevent bleeding at the sites of anastomosis of the bypass. The goal of hemodynamic monitoring of critically ill patients is to assess the adequate perfusion and tissue oxygenation, using intermittent or continuous measurement of oxygen saturation both considered acceptable, although the measurement of lactate may be useful lacks precision as a measure of status tissue metabolism in patients with mechanical ventilation is recommended central venous pressure of 12-14 mm Hg to offset the increase in intrathoracic pressure especially those with PEEP> 5mmHg. A similar consideration is the elevation of intra-abdominal pressure (IAP is approximately normal. 5-7 mmHg in critically ill patients) as it is inversely proportional to tissue perfusion pressure (PPP) and dependent on the mean arterial pressure (MAP) ie: PPA = MAP - IAP.

The good use in the immediate postoperative period of Swan-Ganz implies a broad knowledge of hemodynamics by the doctor for a proper training and constant use of the device, much of the value of this catheter for monitoring the hemodynamic status is based on their ability adequate to measure pulmonary capillary pressure which we take as a measure of left ventricular preload. The subsequent interpretation of a good wedge pressure curve are not simple things, implies among other things, the tip of the catheter has been placed in a part of the lung that the condition of zone 3, the ball was not over-inflated or default, the catheter is floating in the right place and has not migrated back, there is no strong auto-PEEP and various other things. However there are many more and more data, which indicate that the use of postoperative pulmonary catheter in heart depends on many

factors that have little to do with their true indications or the severity of the patient also has serious complications observed in studies presented to the placement of a Swan-Ganz pulmonary artery in this group of patients where metabolic changes, hypothermia, cardioplegia, ischemia or myocardial stunning favor residual arrhythmias, thromboembolic events, infections, pulmonary infarction or to knotting of the catheter, not to mention does not improve cardiovascular survival in critically ill patients.

You must have a central venous line and periodically monitor the central venous saturation (SvO2) and good availability in the administration of appropriate fluids for resuscitation in the first six hours following the early goals of Rivers and colleagues, as therapy guided by objectives has been shown to improve survival in critically ill patients susceptible to revival under the supervision of personnel in these areas.

During the first hours must meet the physiological parameters already discussed, however those who do not meet these criteria should be reassessed therapeutic conduct, analyze ECG, diuresis, PVC, the need to correct up to 30% hematocrit and evaluate the need for inotropic or increase in dosage especially if the central venous saturation is <70%, or GC-invasive measurements have decreased their acceptable ranges. The SvO 2 is a determinant and with greater sensitivity in the postoperative monitoring of oxygenation, perfusion, oxygen consumption and microcirculatory level, now has shown that goal-directed resuscitation, in which this has been a decisive objectives in the first six hours significantly decreased cardiopulmonary morbidity and mortality in patients to assess critical to tissue oxygenation indirectly: this is defined as adequate oxygen supply to demand, the supply is always greater than consumption (VO2), in cases of circulatory shock and severe hypoxemia there is a significant fall in DO2 but the VO2 is maintained by the compensation determined by the EO2%, which explains a security mechanism that ensures the proper use until it descends DO2 a critical point where consumption becomes dependent on the contribution anaeróbico initiating cellular metabolism. [38,39]

The use of PiCCO (press contour cardiac output) can continuously monitor cardiac output, the variability of pulse pressure and stroke volume. Likewise, estimates of the severity of intermittent pulmonary edema, intravascular volume and intrathoracic cardiac chambers, two measures related to ventricular preload.

Electrocardiographic monitoring or driving is important in the first six hours after a stroke thoroughly evaluated on admission electrocardiogram to TiPQ, you need continuous monitoring of electrical activity and identify arrhythmias or morphological changes that warrant immediate management. You also have to periodically assess the proper functioning of epicardial pacemaker generator. Management should be initiated when the ventricular rate is above 110lat/min.

Respiratory monitoring

First of all clinical assessment remains the mainstay in respiratory monitoring, inspection of the chest is important from the patient's arrival to TiPQ is important to assess that there is synchrony in movements of both amplexación amplexión and thorax as well as observation of permeability each of chest drains and their exact position placed by the surgical team must verify proper endotracheal tube position, surely the next step is auscultation of lung regions in search of clinical syndromes or abnormal sounds that suggest some clinical suspicion is necessary to assess respiratory mechanics measurements in our patient by the ventilator as well as scheduling parameters. Sets the following startup parameters:

Mode: Assist-control (volume or pressure)
Respiratory rate = 10-15 L / min;
Tidal volume = 6-8 ml / kg;
FiO2 = to maintain SaO2> 90%.

Over 90% of postoperative patients reach TiPQ Units with mechanical assistance in breathing, so the generally invasive respiratory monitoring, however, is always important clinical evaluation since joining our service to have an initial assessment and observe its evolution in the first 24 hours. Not established the need for routine use of PEEP in patients without complications due to decreased functional residual capacity (FRC) in these patients and atelectasis appear 5 minutes after the onset of anesthesia are not important and no impact on arterial oxygenation postoperatively. However, in the first hour is important to evaluate the relationship and consider PaO2/FiO2 lung history to give an appropriate value, it is also important to rule out existence of residual pleural effusion or pneumothorax because both entities are also involved in lowering the ratio PaO2/FiO 2, and evaluated these considerations and having a stable hemodynamic status can be performed alveolar recruitment maneuver (ARM) is a technique that uses a sustained increase in airway pressure with the aim of recruiting collapsed alveolar units, increasing lung area available for gas exchange and consequently arterial oxygenation. Recent studies concern the use of MRA after cardiac surgery, during which the authors believe that lung function and oxygenation are decreased> 20% with the use of cardiopulmonary bypass and the inflammatory response with the exponential growth atelectasis. Cardiac surgery with cardiopulmonary bypass includes the complete collapse of the lungs, thus, the move will improve oxygenation by opening collapsed lung regions. The results of current work suggest that alveolar recruitment maneuvers are safe procedure in patients with cardiovascular surgery and reduces the frequency of postoperative atelectasis.

The decline of ventilatory support should generally be within 24 hours, ideally Fast-track implementation of the ventilation is the goal in most patients (Fast-track cardiac anesthesia-FTCA-). The importance of predicting the timing of weaning is that both the early weaning as the unnecessary prolongation of mechanical ventilation is deleterious to the patient. In addition only with the clinical trial is difficult to predict how successful disconnection accurate (50% positive predictive value and 67% negative predictive value) because these results justify the implementation of objectives and accurate methods to identify patients who are able to adequately of extubated successfully.

Early extubation is associated with a significant reduction in costs associated with mechanical ventilation, there is now several modalities to a quick and safe extubation, spontaneous ventilation with pressure support (VPS) with 2 dual ventilation PEEP levels (BiLevel) or that pressure release ventilation (APRV).

Patients extubated within 24 hrs it has decreased PaO2/FiO2 ratio <200 is recommended the use of noninvasive mechanical ventilation for 24 hrs demonstrated by Yoshiyuki Takami et al, as it must be part of primary treatment strategy in patients with acute pulmonary edema in this group of patients and reduces the need for endotracheal reintubation and lower mortality when compared against conventional treatment with oxygen therapy in postoperative heart patient.

Pulse oximetry has been used to transfer the patient to the operating room TiPQ unit and required at all times to monitor the state of oxygenation does not replace the determination of arterial blood gases, however the devices currently are more sensitive and less margin for

error, which has gained considerable ground in noninvasive respiratory monitoring. The most common cause of inaccurate readings of SpO2 is movement, affects the ability of light to travel from the light-emitting diodes (LED) to the photodetector, parkinsonism, seizures, tremors, cause problems with detecting saturation with falsely high measurements in low perfusion states, such as low cardiac output, vasoconstriction, hypothermia, hypovolemia, severe hypotension, particularly in cardiac surgery, the oximeter reading is difficult, however, this noninvasive method provides reliable early the decrease in oxygen saturation before they show clinical signs of hypoxemia.

Capnometry capnography and monitoring is a noninvasive method useful in the postoperative cardiovascular since it is constantly evaluating the level of carbon dioxide (CO2) exhaled and its graphical representation is undoubtedly an important tool in the management of mechanical ventilation in the first hours after surgery. Carbon dioxide the patient is transported from the cells into the lungs through the venous blood, mostly in the form of bicarbonate (HCO3) and dissolved in small amounts in plasma and bound to hemoglobin, the amount of CO2 that comes the alveoli is determined by its production and flow of venous blood that is, its perfusion (Q), on the other hand, their removal is an almost direct function of alveolar ventilation (V). Therefore, the PaCO2 is the result of the relationship between ventilation and perfusion: the ratio = V / Q

1. In the case where the alveolar ventilation equals pulmonary blood perfusion, PaCO2 is very similar to PaCO2 in these cases changes in the PaCO2 almost exactly reflect those observed in PaCO2.
2. In cases where ventilation is inappropriately high with respect to the infusion, ie there is a high degree of "dead space" (VD), PaCO2 is considerably lower than PaCO2.
3. In cases where the ventilation is decreased in relation to infusion, PaCO2 is close to the values of PCO2 in venous blood, ie the PvCO2, and results in a V / Q low. This occurs in those clinical situations in which the airways or alveolar sick or increased pulmonary blood perfusion.

As evaluated in the above three points the lungs are not physiologically homogeneous and therefore carbon dioxide at the end of expiration (ETCO2) is the average of the mixture of all different types PaCO2 alveoli. It's called PaCO2-ETCO2 gradient to the difference between arterial CO2 pressure and the pressure of CO2 in the alveoli (ETCO2), which normally is 1-5 mmHg. This small difference is due to the small dead space that exists in normal conditions. But knowing the physiological concepts, translation in the immediate postoperative clinic may be:

- A ETCO2 of 0 usually means the patient is not breathing, however, can also be the result of a malfunctioning fan or a disconnect from it.
- The decrease in ETCO2 suggests a decreased production of CO2, hypothermia or a fall in transport, low cardiac output, excessive alveolar ventilation, hyperventilation or a malfunctioning fan. fig 6
- Increased ETCO2 may be the result of excessive production of CO2, hyperthermia or sepsis or a decrease in alveolar ventilation

Arterial blood gases are essential to making the postoperative management of blood gases were performed on all patients from their initial arrival and subsequent to any adjustment or correction fan electrolyte and acid-base status, interpretation of blood gases is sometimes difficult; laboratory results must always be studied in light of the clinical picture, by the systematic approach to each of the values. In the critically ill patient post-operative heart

surgery is also necessary to know the values of venous blood gases. The determination of arterial and venous blood gases provides three basic values through direct measurement of the respective electrodes:

1. Partial pressure of oxygen dissolved in plasma, PaO2.
2. Partial pressure of carbon dioxide dissolved in the plasma, PaCO2.
3. The degree of acidity or alkalinity of the plasma pH.

The PaO2 is the rate of oxygenation of the blood an indicator of the intensity of the presence of molecular oxygen dissolved in plasma, is the expression of the efficiency of alveolar ventilation-perfusion and alveolar capillary diffusion normal to achieve the transfer oxygen from inside the alveolus to pulmonary capillary blood. The PaCO2 is a ventilation parameter also reflects the respiratory component of acid-base and is a highly reliable method that reflects without confusion or error, unless you have a fan failure or bad programming environment with respect to individual clinical Patient postoperados.[40, 41,42,43]

Renal monitoring

The clinical value in the immediate postoperative period is important because the simple quantification of urine schedule can objectively evaluate renal function, however there are several circumstances where the patient usually attends with minimal deterioration and time indicated by decreased urine output, factors are multi-age, prior renal impairment, intravascular volume cash cytokines by CBP, controlled hypotension, bleeding, and so on. The incidence of acute renal failure (ARF) in cardiac surgery with cardiopulmonary bypass, between 1% and 45%, and in most schools in a 1% to 15%. The incidence of severe ARF that required renal replacement therapy techniques, the work varies between 1% and 11.1%, thanks to a study by Charujas and colleagues demonstrate and validate a "ARF score" in patients undergoing heart surgery and determine the risk of postoperative renal failure in taking a score: 1 to 17 than to predict the risk of acute renal failure expressed in percentage.

The CBP has also been involved for many years in the genesis of renal damage associated with cardiac surgery, initially manifested by microscopic or gross hematuria depending on the severity, though studies of the last fifteen years, using sensitive techniques to identify kidney damage have suggested greater importance of perioperative generator impairment and acute renal failure, as opposed to possible damage of CPB. Most of these studies have been performed in elective postoperative patients with normal preoperative renal function, recent studies comparing off-pump coronary surgery versus conventional coronary surgery, show no differences in postoperative renal damage, however the valve patients have been considered within the group at high risk of kidney damage. So today in the early hours 24 hours urine volume remains the most reliable parameter of renal impairment and prayers whatever their origin (hypovolemia, nephrotoxicity, ischemia, contrast, etc). [44,45,46,47]

Metabolic monitoring

Hyperglycemia in the perioperative period is associated with increased morbidity, decreased survival and increased costs. A number of observational studies have shown that improved control of glucose levels in diabetic patients undergoing coronary revascularization improves the outcome. Van den Berghe et al demonstrated in surgical ICU patients, 63% of whom were postoperative cardiac surgery, the control of glucose concentrations of 80-100 mg / dl was associated with a relative decrease in mortality over

40% when compared with controls. This study has been criticized for lack of blind control, administration of high doses of glucose control and high incidence of hypoglycemia. The current recommendation is to try to keep blood glucose below 150 mg / dl, this was secondary to a study called normoglycemia in Intensive Care Evaluation Survival Using Glucose Algorithm-Regulation (NICE-SUGAR) used to test the hypothesis that intensive of blood sugar reduces mortality 90 days in this study showed higher mortality from severe hypoglycemia. Because polyuria in the early hours, the release of antidiuretic hormone and hyperaldosteronism that characterizes the patient operated on with CPB, it is common the presence of hypokalaemia which must be corrected for values greater than 4.0 mEq / L, just as occurs hypomagnesemia should be corrected usually there is usually no changes in serum calcium or other ions that require correction.

The dilutional hyponatremia type being increased total body sodium, the use of mannitol and / or furosemide during CPB produces a polyuria in the first two to three hours postoperatively INSTANT that can reach 1000 ml / hour, with a tendency to normalize within hours, the usual consequence is the need to infuse fluids resulting hypovolemia. [48.49]

Neurological monitoring

It is essential in the first 24 hours because it is not uncommon to find deficits in different degrees and which are generally grouped into cognitive dysfunction, which is the most common disorder and unnoticed, and that their identification will be necessary to carry out mini-mental and demonstrate an early deficit and time of their higher mental functions. All patients coming to the unit with residual sedation TiPQ so within the next 6 hours there is a 95% elimination of sedatives, since coming patients should be evaluated clinically to assess the integrity of the stem bark and well-get a first impression to rule out diagnoses and cerebral ischemic event or bleeding. In patients who quickly integrates a focus fasciocorporal study should be completed image and a more detailed review to have an early management and prevent secondary damage. [50.51]

Moreover, patients with prolonged CPB tend to have greater involvement of cardiorespiratory function and hemodynamic instability preoperatively intraoperative surgery more complicated, hence the increased incidence of neurological disorders may be related to these factors rather than the CPB time, now happens with hypotension and cerebral hypoperfusion which is another postulated mechanism of neurological damage. The CBP is under hypothermia and anesthesia, both of which lower the cerebral metabolism and thus cerebral blood flow as there is less demand on the other hand, hemodilution decreases blood viscosity by decreasing its resistance to move, so lower blood pressure can keep the same cerebral blood flow. Thus, it alters the autoregulation curve of cerebral blood flow may keep it even with blood pressures of 50 to 60 mm Hg, studies measuring regional cerebral blood flow in patients during CPB have shown that blood pressure can reach 50 mm Hg without altered cerebral blood flow. Moreover, the flow can reach 19 cm 3 per 100 grams of tissue per minute without psychometric alterations detected between pre and post operative. Glasgow is interpreted evaluation at baseline and 6 hours by issuing a neurological assessment, monitoring with bispectral index (BIS) in patients who have to initiate a secondary sedation is necessary to identify a level of sedation adecuado.[52, 53 , 54]

Hematologic monitoring

Hematologic monitoring after surgery is associated with anemia hemodilution and blood loss, the minimum necessary use of blood products has shown improvement in morbidity

and mortality in recent years. This consideration has resulted from recent meta-analysis and evidence-based medicine, evidence shows the association of adverse effects such as increased costs, morbidity and mortality especially in the group <65 years so even 7.5gr hemoglobin can be well tolerated and no increased risk increase in elderly patients without ischemic but the minimum allowed is 8.5 grams, although a large number of scientific publications are those who prefer to have an algorithm in relation to individual patient characteristics as shown in the table below . The indications for transfusion in patients with coronary disease are valid when hypovolemia has been corrected, optimized hemodynamics and oxygenation after correction tachycardia. [55,56,57]

It is common in the postoperative cardiac surgery hours uncomplicated run smoothly. At approximately 36 hours the patient can be transferred to a general room with telemetric control.

6. Complicated postoperative

Cardiovascular

Hypotension and hypoperfusion injury may condition not directly related to the surgical procedure and include cardiac tamponade, a new myocardial ischemia, tension pneumothorax, hemothorax or significant bleeding related to arterial cannulation. Rarely produce acute thrombosis of a graft or coronary embolization. The electrocardiogram (ECG) may be of diagnostic aid because it is expected that the initial postoperative ECG changes does not show or reveal abnormalities preoperatively limited ST-T. If there are significant changes in ECG repeated, should be thought of an occlusive lesion of one of the grafts.

In the presence of suspected acute ischemia should indicate intravenous nitroglycerin, the risk of a perioperative myocardial infarction is present from the preoperative to the hospital and even after the diagnosis of acute myocardial infarction (AMI) presents difficulties in the perioperative You must have a combination of ECG, cardiac enzymes and echocardiography can occasionally make the diagnosis. Frequently observed nonspecific ECG changes a large percentage of patients have an increased enzyme and troponin I (TnI) generally exceeds the levels observed in AMI unrelated to cardiac surgery. The loss of graft thrombosis has been reported in up to 10% of grafts in the first week in the hours following the surgery, aspirin and possibly clopidogrel appears to reduce the prevalence of AMI diagnosis although postoperative postoperative AMI is difficult itself has a significant effect on morbidity and mortality in the long term.

Arrhythmias

Low cardiac output syndrome

The low cardiac output syndrome (LCOS), is characterized by decreased performance of cardiac function where the cause may damage myocardial and cardiogenic shock condition corresponds to a failure in the balance between central cardiac pump and control components peripherals, including: a) the tone of the peripheral circulation and b) neurohumoral regulators of vascular tone, with the arrival insufficient oxygenated blood to peripheral tissues to meet metabolic needs, their presence is associated with high mortality at that requires immediate diagnosis and treatment. The multiple causes can produce or aggravate this syndrome can be grouped, for descriptive purposes, the following pathogenic mechanisms:

1. Reduced preload. The major cause is the leakage of fluid into the interstitial space, excessive bleeding, polyuria, the use of high levels of positive end-expiratory warming excessive vasodilator drug use, cardiac tamponade, and so on.
2. Increase in afterload. It can affect both the left and right ventricle. in the cause of increased afterload are systemic hypertension, pulmonary hypertension, the replacement of the mitral valve in mitral regurgitation, etc..
3. Reduced contractility. The main causes of decreased contractility are perioperative AMI, drugs with negative inotropic effect, the phenomena exaggerated ischemia-reperfusion during aortic clamping, and so on. In relation to the phenomenon of ischemia reperfusion is important to note contractile deterioration often not immediately apparent to the patient's admission to the ICU. In these cases, there is a period of normo-or hyper ventricular early after reperfusion. This period is short (hours) and is followed by a gradual depression of systolic function, leading in many cases to a false sense of security in the early postoperative hours, when this phenomenon is unknown.
4. Changes in heart rate and heart rate. Are due to extreme bradycardia, supraventricular or ventricular tachyarrhythmias and impaired high-risk condition.
5. Metabolic and electrolyte. Acidosis, hypoxemia, hypo-or hypercapnia, hyperkalemia, hypocalcemia and may contribute to the development of this syndrome.
6. Inadequate surgical management. Sometimes not achieved the expected result from the technical point of view and this can generate a low output syndrome, such as poor condition can bridge aortocoronary junction in myocardial revascularization surgery, prolonged pump time with poor poor systemic perfusion and hypothermia induced, the presence of a residual stenosis in mitral commissurotomy, miss-match of prosthetic valve replacement or plasty, and so on.

The diagnosis of low cardiac output syndrome after surgery can be established through the clinic or by hemodynamic monitoring. Hypotension is the warning sign and used more widely, however, patients with moderate decrease in cardiac index may retain acceptable levels of low systemic blood pressure which minute volume is high or normal. Oliguria is the most common signs of urinary volume monitoring is time and calculate the minute volume through the renal plasma flow and rhythm of diuresis, but lacks specificity. No doubt the hypothermia of the extremities and the temperature difference between central and extremity: these signs are not very useful in the immediate postoperative period because patients usually come to body temperature, cardiovascular recovery with low and sometimes remain so for several hour, despite attempts to overheat. In the first hours after surgery are patients with marked vasomotor instability (vasoconstriction - vasodilation) for which no specific capillary filling. On the other hand in the first hours after surgery can be found lactic acidosis, which in many cases does not reflect the present situation, but situations of decreased perfusion in the operating room occurred body, markedly decreasing the diagnostic and prognostic value with other medical and Finally, the decrease in mixed venous saturation, this parameter depends on cardiac output and oxygen consumption level of the tissue, so in the first 60 minutes is more specific cardiac output. Due to the low sensitivity and specificity of symptoms from the first sign of consensus definitions in cardiovascular recovery, recently published, is required to make the clinical diagnosis of low cardiac output syndrome, patients present simultaneously at least 2 of the following criteria:

Hypotension (systolic blood pressure below 90 mmHg).

Oliguria (urine output less than 0.5 ml / kg / hr).

The hemodynamic evaluation allows continuous monitoring of cardiac function postoperatively, being indispensable implementation in this particular group of patients. This is done by direct measurement of cardiac output by thermodilution technique. Authors such as J. Kirklin consider being in the presence of low cardiac output when cardiac index less than 2.2 L/min/m2 in the early hours after surgery and less than 2.4 L/min/m2 in the first postoperative day, other authors consider the value 2.0 L/min/m2 index of heart as the limit for the diagnosis of low cardiac output syndrome, with values between 2 and 2.5 L/min/m2 cardiac index usually require therapeutic intervention, whatever its value must be accompanied by systemic vascular resistance values normal or elevated, for differentiation vasoplegic syndrome that presents with decreased systemic vascular resistance. The radionuclide ventriculography with Technetium 99 is an excellent diagnostic tool, with it you can obtain the ejection fraction as much of the left ventricle of the right ventricle, and allows cardiac tamponade diagnosed by the presence of pericardial blood or clots in relation to Echocardiography its main drawback is to obtain an acceptable acoustic window in this group of patients, however these problems have been solved with transesophageal echocardiography. LCOS mortality is very high, the study CONAREC III mortality of patients suffering from low cardiac output syndrome was 44.7%, compared to those patients who did not suffered and whose mortality was only 4.9% in the same study when considering all the excuses of death, this syndrome was the most frequent (28.9%) in patients undergoing coronary bypass surgery. Another study observed the ESMUCICA un12% mortality in CABG and valve surgery in 25-45%) and ESMUCICA II (26% mortality in valvular).

The therapeutic management should follow a similar pattern of the pathogenesis stating:

a. Optimize preload
b. Optimizing afterload
c. Optimizing the pace and heart rate.
d. Increase inotropy.

The optimal preload for each patient is different and depends on the heart for each patient and how they estimated. When you need to optimize the preload is used intravascular volume expansion with either colloids, crystalloids or both, while, as if what is required is a decrease in preload is done with diuretics, vasodilators, with predominant effect in the venous bed as nitroglycerin, mechanical ventilation with PEEP or hemofiltration if the patient is oliguric renal failure.

Afterload also depends on heart disease for each patient and if you have any other special situation. The most commonly used drugs to reduce vascular resistance and therefore afterload are vasodilators with predominant effect on the arterioles, such as sodium nitroprusside. Another important therapeutic elements are warming persist in hypothermic patients, sedation and analgesia in patients who have pain or anxiety they generate strong isometric muscle contraction, and oxygen in varying concentrations in those with hypoxemia and pulmonary vasoconstriction bed with increased pulmonary vascular resistance and consequently the right ventricular afterload. To get the rhythm and heart rate can be used atrial pacing in case of a sub-optimal heart rate in sinus rhythm and normal atrioventricular conduction, ventricular pacing in cases of atrial fibrillation with low ventricular response, and sequential pacing, atrioventricular case of complete atrioventricular block. The presence of tachyarrhythmias can be managed with drug therapy, over-stimulation or cardioversion shock as appropriate.

Commonly used catecholamines such as dopamine, dobutamine, isoproterenol, epinephrine, norepinephrine and inotropic catecholamines not milrinone and levosimendan, being necessary in many cases the combination of more than one. Catecholamines, particularly dopamine are the drugs most often used and indiscriminately without taking into account many times the preload. The most important are the catecholamines and phosphodiesterase inhibitors, there are few data concerning the use of levosimendan. these agents have proven effective in improving myocardial contractility or heart rate or both catecholamines are more potent chronotropic and inotropic agents determine side effects such as increased myocardial oxygen consumption of the myocardium, tachycardia, arrhythmias, and increased in afterload can make your job difficult. B-adrenergic receptors may also be downregulated in patients with previous heart failure. This has increased the interest in the use of inhibitors of phosphodiesterase III and more recently, the calcium sensitizer levosimendan. In a study by Labriola et al. Nijhawan et al.) Drug compared to placebo in patients with low cardiac output syndrome after surgery, in which documented an increase in cardiac output and ejection fraction and a decrease systemic vascular resistance in patients treated with levosimendan. Gillies et al. Conducted a systematic review of the literature on the use of inotropic agents in patients with cardiac surgery, in which certain recommendations were documented, each with a particular level of evidence.

Respiratory

Respiratory dysfunction in postoperative heart surgery patients is a common problem that results in a significant increase to 25% mortality and significant morbidity with impact on cost and hospital stay, atelectasis is a frequent occurrence in the immediate postoperative period The incidence of atelectasis in the postoperative period of 40 to cardiac 70%, the term is derived from Greek: Atel and ektasis mean incomplete expansion of a segment or lobe is characterized by volume loss and collapse of alveolar region manifested radiographically as an area opacified. The severity of atelectasis increases with more time to pump, more bridges and prolonged ischemia, the opening of the pleura, phrenic nerve injury, intraoperative and very low temperatures. Thoracotomy alters lung function by shallow breathing (restrictive functional pattern), and vital capacity may be reduced by up to 45 to 70%, the pain diminishes deep breath and a cough can lead to ineffective with the consequences in lung mechanics and bronchial hygiene. Another important factor is the presence of atelectasis, diaphragmatic paralysis caused possibly by phrenic nerve injury caused by surgery or by the use of topical agents or cold cardioplegia. Decubitus position maintained, leads to changes in regional distribution of ventilation and perfusion of the lung, lung inflation decreases along a vertical axis from ventral to dorsal supine and when spontaneous breathing begins immediately after surgery and in the supine position ventilation is distributed mainly dependent areas of the lung. In contrast, during mechanical ventilation, this pattern changes and the distribution of ventilation is primarily aimed at non-dependent areas in both positions, therefore, the subsidiaries tend to collapse. Studies have shown that prone position ventilation becomes more homogeneous. Mechanical restraint of ventilation is produced by several factors explain such as bronchial secretions accumulating in dependent areas, pleural effusion or dysfunction of chest drains in the first hours after surgery. Alveolar recruitment maneuver is a technique that uses a sustained increase in airway pressure with the aim of reducing atelectasis by recruiting collapsed alveolar units, increasing the

lung area available for gas exchange and consequently arterial oxygenation. Different methods are used alveolar recruitment in the postoperative patient, it is important to know that before the maneuver is to have an adequate intravascular volume and residual sedation after surgery. Ventilatory strategies proposed to achieve alveolar recruitment in surgical patients are based on the use of pressure (PEEP or CPAP) ranging between 20 and 40 cm H2O for varying periods of time. The effects of positive end-expiratory should be monitored continuously, as some of the side effects include decreased venous return by increasing the average pressure of the airways, impaired lung perfusion overdistended areas (increase dead space), increased pulmonary vascular resistance and right heart dysfunction, barotrauma and impaired renal blood flow, which are frequent causes of hemodynamic compromise in critically ill patients with cardiovascular disease and those with intravascular volume deficit. Pleural effusion is frequently observed in the immediate postoperative period, but a considerable percentage persist for more than 30 days, the incidence is 41-87% in postoperative patients, although most are not significant pleural effusions, a study of 602 heart postoperative patients showed pleural effusion in 63%, more than 30 days but less than 5% need thoracentesis for resolution in those patients who had more than 5 days chest drains pleural related to infections associated with atelectasis and ipsilateral lung infection, said box by fever, productive cough and an alveolar infiltrate on chest radiograph. It is important to differentiate whether it is indeed a spill transudate or if there is a pulmonary complication due to an infection with a pleural early. To make this distinction using the criteria of Light, which are more sensitive at identifying exudates, they meet at least one of the following criteria: (transudates none)

1. Relationship between pleural fluid protein and serum 0.5,
2. Relationship between pleural fluid LDH and serum 0.6,
3. In pleural fluid LDH greater than the 2 / 3 parts of the upper limit of normal for serum LDH.

Other proposed criteria for an exudative pleural effusion are:

4. Cholesterol> 43 mg / dl,
5. Gradient-pleural serum albumin less than 1.2 g / dl.

The thoracic duct that enters the thorax through the right diaphragm and flows into the left subclavian vein, has collateral lymphatic sometimes can be injured during surgery and result in a chylothorax. This pleural fluid milky-white at times and some colored (yellow or red) in other, has high content of lymph (chylomicrons), with a triglyceride level above 100 mg / dl and cholesterol below 200 mg / dl , treatment includes not remove the chest tubes because the fistula may close spontaneously in the thoracic duct short time, starting with parenteral nutrition for 10 to 14 days to reduce the production of intestinal lymph and thereby reduce the flow through the thoracic duct. If despite these measures fail to control the chylothorax, pleurodesis can be performed. Injury or acute progressive respiratory insufficiency (ALI / ARDS) is a multifactorial process of respiratory damage from pulmonary or extrapulmonary origin and is defined by the European-American consensus.

In patients with postoperative heart surgery the incidence is 5 to 20% depending on the type of surgery, severity, time in surgery, bleeding, age, EuroSCORE, comorbidities, and so on.

In elderly patients shows the highest incidence due to low physiological reserve and are more likely to have postoperative complications, the impact of gender remains controversial, although female gender was not shown to be an independent predictor of LPA in a large study recent cohort, two small studies identified a strong association between

female gender and the incidence of ALI after cardiac surgery. Vascular risk factors are independent predictors of LPA include diabetes, kidney failure, hypertension. These markers of systemic atherosclerotic disease are associated with an increased risk of major complications. Preexisting renal insufficiency is a strong predictor of ALI (OR, 2.3), which confirms the findings of several studies, increased atherosclerotic burden associated with renal failure. Chronic obstructive pulmonary disease (COPD) identified preoperative was also an independent risk factor, confirming that patients with COPD who are undergoing valve surgery or surgery RVM have two or three times the chance of LPA in severe COPD has been associated with excess postoperative mortality in patients with MVR. Pathophysiological observed that lung damage is mixed but the most important finding in the lungs during the early stages of ALI / ARDS is the presence of severe pulmonary edema secondary to increased permeability of capillary endothelium and alveolar epithelial barrier of . Simultaneously, increased pulmonary vascular resistance as a result of thromboembolic events and reflex vasoconstriction, these morphological characteristics are a complex reaction of the lung to different nosological agents and processes and not related to the nature of the causal process. During mechanical ventilation in the immediate postoperative behavior is rest to follow by pulmonary alveolar protection strategy as ALI / ARDS is a syndrome characterized by loss of functional residual capacity, increased lung and short circuits refractory hypoxemia FiO2. The standard or optimal tidal volume is difficult to determine, because in the inflamed lung or ALI / ARDS alveolar pressures each area has specific and requires its own level of PEEP to keep open during expiration. The alveolar pressures and volumes that can reach areas not dependent overdistended lung, are often insufficient to ensure the recruitment of regions dependent edema and atelectasis by maintaining recruitable lung areas open, there is distension of the healthy areas of the lung, it is explained that the regional compliance of the lung are different as well as mentioned Gattinoni.

Postoperative bleeding

One of the most frequent complications encountered in the management of patients, approximately 20% of patients present with significant bleeding and only 5% required reintervention. Predictive factors for bleeding include age, renal failure, cardiopulmonary bypass time, liver failure, hypothermia, secondary fibrinolysis, NSAIDs, etc; bleeding contributes to more days of ventilation hospital stay and mortality.

The definition of excessive bleeding in postoperative patients occurs in 5% -10% approximately and only 3% required reoperation, bleeding, and reoperation are 2 independent predictors of poor prognosis. In those patients with postoperative bleeding can be divided into two categories: surgical bleeding (bleeding venous layer anastomosis, sternum, anywhere stitches), non-surgical bleeding (caused by coagulopathy).

Risk factors for bleeding are preoperative such as pharmacologic agents (thrombolytic PTCA, antiplatelet drugs, anticoagulation) in the case of ASA should be discontinued 5-7 days before surgery and anticoagulation is recommended to have INR <1.5, five days prior to surgery, vitamin K malabsorption, liver disease due to decreased synthesis of clotting factors, SLE, amyloidosis, prior chemotherapy, and so on.

Intraoperative risk factors are: pump bypass, hypothermia, use of heparin during cardiopulmonary bypass generation of fibrinolytic activity and postoperative risk factors known are octogenarians, non-elective surgery, low BMI, CPB> 150 min, grafts ≥ 5, surgical reintervention.

During the first 6 hrs of PO should immediately obtained objective results of platelet count and coagulation, for early medical or surgical management, clinical criteria is in relation to the following table.

In the management of patients with heavy bleeding PO should commence administration of blood products such as red cell concentrates without doubt the goal is to maintain the optimum level in arterial blood content [CaO2 = CaO2 = Hb (g/100ml) x 1.34 (ml O2 / g) x% SaO2 + (PaO2 x 0.0031) = ml blood O2/100ml)]. They are prepared with 300 ml volume with low WBC (<5x106 cells) to reduce alloimmunization and avoid possible TRALI or lung damage. Cell salvage, the process by which collects a patient's own blood during surgery for later transfusion in the same patient is a reliable alternative to donor blood transfusion when needed. We found 23 studies investigating the effectiveness of cell salvage in cardiac surgery, conclude that apparently there is insufficient evidence to support the use of cell salvage in cardiac surgery but the methodology in the studies were flawed and may be biased.

Alterations in the number or function of platelets may have effects ranging from a clinically insignificant prolongation of bleeding time to large defects of hemostasis, platelet transfusion is usually required when it decreases the count: <50,000, is individualized 50-100 the case and over 100,000 were transfused if the time of Ivy is more than 10 minutes with continued bleeding. Can be obtained by platelet concentrates (40-70ml) or platelet apheresis (200-300ml).

The use of fresh frozen plasma in postoperative patients offers all the clotting factors and plasma proteins needed to improve the prothrombin time and clotting better ensure hemodilution coagulopathy, caution should be exercised in bleeding secondary to heparin, as a source ATIII natural and should not be used prophylactically. To replace clotting factors to be used a dose of 10 to 20 mL / kg, which could increase the concentration factor by 20% immediately after infusion. [58,59,60]

The cryoprecipitate is a concentrate of plasma proteins of high molecular weight cold rush its volume is approximately 15 to 20 mL after removing the supernatant plasma containing concentrations of factor VIII: C (procoagulant activity), 80 to 120 U; factor VIII: vWF (von Willebrand factor), 40 to 70%, fibrinogen, 100 to 250 mg, and Factor XIII, 20 to 30%.

Most of the work with hemostatic agents were designed to assess the therapeutic efficacy and to assess potential toxic effects, so that there are still definite data on the safety of hemostatic agents. Many studies on these agents have used perioperative blood loss and other parameters with endpoints of little clinical importance, whereas other studies did not have enough power to evaluate the clinical outcome of importance, such as mortality or need for reoperation.

Pharmacological agents that decrease postoperative bleeding are desmopressin is a synthetic analogue of natural vasopressin, with the advantage of having less vasoconstriction, is recommended for use in the immediate postoperative hemostatic, unlike aprotinin has fewer side effects such as anaphylaxis, thrombosis and renal failure. Aprotinin has been used in recent times but because it is a bovine protein, there is an increased risk of anaphylaxis, especially if you already had previous exposure, and its cost is higher. When used at low doses acts as antidiuretic hormone and is 10-20 times the dose that increases hemostatic function and plasma levels of factor VIII, von Willebrand factor (vWF) and tissue plasminogen activator (tPA), releasing these factors endothelium and liver. Also observed increased platelet aggregation, the result is the shortening of bleeding time. Administered

IV (0.3ug/Kg), SC (0.3ug/Kg) but for obvious reasons dministration IV is recommended in postoperative patients. The best response is expected between 30-60min after parenteral administration.

Inside are antifibrinolytic drugs: aprotinin (a direct inhibitor of the fibrinolytic enzyme plasmin) is the only drug approved by published and the Food and Drug Administration (FDA) to minimize transfusion requirements in coronary bypass surgery, directly inhibits the fibrinolytic enzyme plasmin, plasma kallikrein, tissue trypsin and activated coagulation factor XII, the highest recommended dose is> 700mg.

Are also used tranexamic acid and aminocaproic acid, but have not been approved by the FDA for this indication, their mechanisms of action are the first to inhibit the binding of plasmin to fibrin occupying the binding sites of lysine of the proenzyme plasminogen and the second is the same mechanism of action, but 10 times more potent. Doses are 10-30g and maintenance 1-3gr/hr and the second 3-10gr with 20-250mg/hr maintenance.

There are reviews of meta-analysis on the effectiveness of antifibrinolytic agents compared with placebo, aprotinin or tranexamic acid, but not aminocaproic acid, reduced the need for blood transfusion by 30% and saved about 1 unit of blood per operation. There was no difference in efficacy between regimens with high or low doses of aprotinin, while varying doses of tranexamic acid and aminocaproic's not possible to assess the relationship between dose and efficacy. As for the most relevant clinical events, the relative risk of reoperation for excessive bleeding was significantly reduced in patients receiving aprotinin, compared with those receiving placebo, although the mortality rate remained unchanged. Both tranexamic acid and aminocaproic acid significantly decreased these events. Therefore, the results of the work checked and reviews indicate that antifibrinolytic drugs are effective hemostatic agents in cardiac surgery. Reductions in transfusion requirements and reoperation for bleeding seem to be confirmed by the narrow confidence intervals of likelihood ratios, indicators of relative risks. There were no sufficient data on the effectiveness that allow definitive conclusions regarding the use of antifibrinolytic agents in other situations. [61,62,63,64,65,66,67]

While other review reports that aminocaproic acid and tranexamic acid are safe, it is noteworthy that the works included were smaller than the jobs studied aprotinin. Therefore, the authors say, the safety data are not reliable, especially with regard to thrombosis. Currently, the Blood Conservation using Antifibrinolytics: a randomized work in a population submitted to cardiac surgery (Randomized Trial in a Cardiac Surgery Population) or BART, which is still enrolling patients, is designed to enroll 2970 patients with indications for cardiac surgery high risk, to determine whether aprotinin is superior to tranexamic acid or aminocaproic acid to reduce the risk of massive postoperative bleeding. Secondary endpoints were overall mortality and adverse effects such as cardiovascular disease and kidney failure. For all available data, the authors argue that the evidence that aprotinin reduces perioperative bleeding and immediate postoperative transfusion requirement is sound. However, note that despite the large number of clinical trials that have addressed the drug, its effectiveness in reducing the need for reoperation has just emerged from reviews and lack of evidence about its effect on mortality. [68,69,70]

Recombinant activated factor VII (rFVIIa) acts locally at the site of tissue injury and alterations of the vascular wall by binding to exposed tissue factor, generating small amounts of thrombin sufficient to activate platelets. The activated platelet surface can then form a template on which rFVIIa half the direct or indirect activation of coagulation to

generate thrombin in the end much more and convert fibrinogen to fibrin. The clot is stabilized by inhibition of fibrinolysis, secondary to activation of the inhibitor of thrombin-activatable fibrinolysis mediated by rFVIIa. The availability of rFVIIa has expanded treatment options for acute bleeding in hemophilia patients. This drug is not a panacea, but it has efficacy in patients with trauma and excessive bleeding resistant to other treatments. However, the encouraging results obtained so far must be confirmed by other studies, are also necessary cost-effectiveness studies, as it is an expensive drug. The authors recommend to take with caution the results of studies recognized even before considering the evidence as a guideline. We have tried to increase the power and efficacy of rFVIIa by molecular engineering acting on DNA, but no studies. [71,72,73,74,75]

Perioperative myocardial

Although virtually all patients have some degree of increase in cardiac enzymes after surgery. The perioperative myocardial infarction is one of the most serious complications after CABG (RVM), an incidence of 5-20%, and is associated with significant morbidity and mortality in the post-surgical high. The pathogenesis of IPO vasa in the various mechanisms by which the placement of coronary artery bypass bridges leading to myocardial necrosis: The most common is acute occlusion of the hemoducto, twist it, subtotal graft stenosis or spasm, saying recent articles The presence of collateral arterioles protects perioperative stroke patients.

The perioperative myocardial infarction (IPO) type 5 belongs to heart and is defined according to the latest consensus established in 2007 by the AHA / ACC as an increase of at least 5 times the baseline or reference biomarkers, along with the emergence of new q waves left bundle branch block on electrocardiogram, or coronary angiography showing acute occlusion of hemoductos and imaging evidence of recent loss of viable myocardial tissue.

According to Thielmann, the increase in markers of myocardial damage, can be used to discriminate between perioperative stroke related to the placement of coronary artery bypass bridge, or another cause. So analyzing 3308 patients with MVR, I conclude that the 94 who underwent coronary angiography, 56 had stroke related to the placement of coronary artery bypass bridges, 38 was not related to the procedure. Levels of troponin I, rather than CK / CK-MB rose significantly in the first group with respect to the second, considering the troponin I as the best marker to discriminate between IM surgery associated with those who are not associate the procedure with a cutoff of 10.5 ng / ml, and those in which MI was directly associated with the placement of non-hemoductos with a cutoff of 35.5 ng / ml.

In peri-operative myocardial infarction not associated with coronary bypass grafts, is due to mechanisms such as inadequate cardioplegic perfusion, incomplete revascularization, distal coronary microembolization caused by surgical manipulation, recent unstable angina, poor left ventricular function. This early detection of perioperative myocardial infarction plays an important role in treating either early coronary angiography and angioplasty, trying to preserve left ventricular function as a predictor of long-term survival. Obviously the presence of perioperative myocardial infarction is associated with a high rate of heart failure and long-term mediately, as evidenced by Steuer, analyzing patients with MVR 7.493 assessing the number of readmissions for heart failure. Found that 7.7% (576) were readmitted for heart failure. Of these, 20% (114) had perioperative myocardial infarction. [76,77,78,79,80,81]

Tamponade

It is characterized by symptoms and signs of hemodynamic instability due to the restrictive effect on cardiac contraction, usually observed within the first 6hrs associated with poor permeability of their drains. The prevalence of this complication varies between different publications, ranging from 0.8% -8.5%, there are reports in which said one of the most frequent causes, to the use of internal mammary artery for coronary artery bypass bridge, on the other hand, the use of a single anterior mediastinal tube, instead of 2 tubes (front and back) has been associated with a high rate of pericardial fluid and as a result of these patients tamponade.La course evaluation should be carried out by echocardiography, and not just those with a radiological image suggestive of this entity (heart carafe), since by this method because of cardiac surgery postoperative patients identified only 50% of patients. This is important because several studies have shown that those patients who develop pericardial effusion, even without hemodynamic compromise, increased risk of supraventricular arrhythmias, sternal dehiscence, prolonged hospital stay and a significant reduction in exercise tolerance. [82,83,84]

Finally, the treatment will be those with hemodynamic compromise, is permeated drains, by performing emergency echocardiography and reoperation. [85,86,87,88,89]

Vasoplegic Syndrome

Vasoplegic syndrome (SV) is a severe form of systemic inflammatory response syndrome (SIRS), which ranks its expression on the cardiovascular system. A number of reports considered the vasoplegia as a recognized complication of cardiac surgery, the main clinical manifestation is the presence of hypotension, usually severe, which features the distinctive clinical feature of responding with little or no input from volume.

In cardiac surgery, the reported incidence is 8 to 10%, even up to 40%, these differences often depend on characteristics of the study population (ventricular function), the type of intervention assessed (use or not of CPB, type cardioplegia) and mainly from the diagnostic criteria used. As mentioned previously, the key point is the presence of hypotension, usually with a systolic blood pressure (SBP) <85 mm Hg, and / or mean arterial pressure (MAP) <50 mm Hg.Un clinical data necessary to consider the diagnosis of Vasoplegia is the lack of response to volume expansion. A hypotensive patient in the postoperative period of cardiac surgery, central venous pressure (CVP) reduced elevation of the same after the infusion of 500 or 1000 ml of solutions (even at higher volumes) should lead to the posing of vasoplegia. [90,91,92,93]

Strict diagnostic confirmation will require the use of hemodynamic monitoring, the presence of Swan Ganz catheter will allow a broader determination descended filling pressures, by providing values of pulmonary capillary pressure (PCP) reduced.

Diagnostic criteria

1. Clinical (only allow them to suspicion)
   Low blood pressure response with little or no volume expansion aapropiada
2. Hemodynamic
   Hypotension (SBP <85 mm Hg / TAM <50 mm Hg)
   Reduced filling pressures (CVP <5 mm Hg / Wedge <10 mm Hg)
   Minute volume and normal or elevated cardiac index (CI equal to or greater than 2.5 L/min/m2)
   Reduced systemic vascular resistance (SVR <800 dinas/seg/cm-5)

Vasopressor requirement (> 0.5 mcg / kg / min noradrenaline or> 10 mcg / kg / min dopamine)

3. Other
   a. In operating room, with open chest
      PVC hypotension with low (<5 mm Hg) refractory avolumen associated with excellent observation of left ventricular contractility.
   b. postoperative
      PVC hypotension with low (<5 mm Hg) refractory avolumen associated with echocardiography (bidimensionalcon good window or transesophageal) with apreciaciónde good left ventricular contractility.

The main therapeutic goal is sustain perfusion to vital organs like the kidney, brain, liver and heart. This also implies the initial use of volume, the use of two types of drugs, drugs with pressor effect, linked to its exclusive or non-selective action on alpha adrenergic receptors, such as metaraminol or phenylephrine among the first, and epinephrine, norepinephrine or dopamine among the latter. The use of drugs associated with beta-adrenergic effect, will result in some measure, an increase of myocardial oxygen consumption in the same direction, their association with postoperative arrhythmias, has also been reported. The second drawback associated with, and probably the most important clinical refractoriness to vasopressors is that certain forms of vasoplegia postoperative manifest. This refractoriness drugs raises the utility of antagonists or inhibitors of NO and the enzyme guanylate cyclase, we consider a rational therapeutic approach more physiological. Two drugs are the most studied, methylene blue, and vasopressin [94,95,96,97,98]

Methylene Blue (AM): its therapeutic action is based on the inhibitory effect of NO or blocking of the enzyme guanylate cyclase. This drug has been considered in several isolated reports in a series without a control group and essentially in a randomized control group. Leyh et al. reported 54 patients with refractory postoperative vasoplegia the use of norepinephrine, treated with 2 mg / kg AM. Fifty-one patients showed favorable hemodynamic changes in the course of one hour post-treatment. Three patients died in the hospital course of the picture (5.6%). The study lacked a control group. Another key finding is the shorter of the table between those treated with AM. In these, vasoplegia resolved completely within two hours after the start of infusion, whereas in those managed conventionally, the box is extended in time, such extension of time associating with a higher incidence of complications and late onset sepsis and multiorgan dysfunction . Several authors have agreed with this finding, giving unfavorable prognostic value of the persistence over time of the SV, accepting that a breakpoint located between 36 and 48 hours is a marker of poor prognosis.

Vasopressin: Vasopressin (antidiuretic hormone arginine vasopressin), Argenziano et al. described the association between the shock with vasodilatation after bypass surgery and deficiency of vasopressin. Which is secreted by the neurohypophysis regulates tubular permeability to water, typically having limited participation in the control of BP. Under conditions of hypotension, such as bleeding or vasoplegia itself is a rapid depletion of endogenous. It allows a rapid reversal of hypotension, especially in patients refractory to vasopressors. In addition, the hormone increases vascular sensitivity to catecholamines and increases urine output, based on its direct action on glomerular efferent arteriole, unlike catecholamines, whose site is located on the therapeutic afferent arteriole. The proposed dose is 0.05 to 0.1 unit / minute.

The use of off-pump surgery was associated with less inflammatory response, with lower incidence of postoperative SV. However, the picture is commonly seen and may present a favorable course. Vasoplegic syndrome is associated with a poor prognosis, when it is resistant norepinerina poorer prognosis with increased morbidity and mortality. The reported mortality after cardiac surgery is 24% in series reported by Levin and colleagues, and 25% in series reported by Gómez et al, in which case the syndrome persisted for up to 48hr vasoplegic. [99,100,101,102,103]

7. Drug therapy

The support with vasopressor, vasodilator and inotropic therapeutic behavior is common in the first 24 hours secondary to hemodynamic effects induced hypothermia, myocardial stunning, extracorporeal circulation, hypovolemia, sedation, and so on. Despite the frequency of use of catecholamines are well known adverse effects such as increased myocardial consumption, arrhythmogenic, favor delirium, and so on. [104,105,106]

Dobutamine

Amine as the structure similar to dopamine is primarily a beta-adrenergic agonist relatively selective beta-1. Is much more effective and positive inotropic and positive chronotropic capacity less arrhythmogenic than dopamine, has no affinity to dopamine receptors and therefore lacks the renal effect. A standard dose (2-15μg/kg/min) positive inotropic responses observed with a slight increase in heart rate and decreased peripheral resistance. [107,108]

In the perioperative setting is used primarily as inotropic, often combined with vasopressor either to maintain adequate cardiac output and blood pressure and to achieve a combined effect of cardiac output and perfusion. On the other hand as shown by Susana Lobo et. al; randomizing 50 patients over 65 high-risk cardiac surgery, receiving IV fluids liquid + vs Dobutamine, the largest number of perioperative complications observed was present in those who do not use vs those who had dobutamine infusion: 52 % vs 16%, and mortality at 60 days was 28% vs 8% in the dobutamine group. [109,110,111,112,113,114]

The drug's half life is 3-5 min, one of several effects is the progressive decrease in blood pressure and pulmonary wedge has an advantage over the effect of dopamine beta for a smaller effect observed tachycardic and arrhythmogenic action and dilation in the pulmonary circulation has been confirmed in the peripheral circulation. As observed Romson et al; administered dobutamine in varying doses and patients undergoing cardiopulmonary bypass the heart rate changes depending on the dose and this is lower in individuals over 65 years, there were minimal changes in blood pressure, instead a decrease in pulmonary capillary wedge pressure and central venous pressure increased systemic vascular resistance remained in a mild and constant left ventricular performance also increase due to increased heart rate [115,116]

Phosphodiesterase inhibitors

They are a family of enzymes involved in cellular physiology by regulating the concentration of intracellular second messengers are known at present eight of these isoforms of phosphodiesterases, which interests us is the number III. Cyclic AMP, produced from the stimulation of beta-adrenergic receptors may have two destinations: the culminating with an increase in cardiac contractility, and the other consisting of the degradation of cAMP to 5-AMP, produced by phosphodiesterase III . Inhibition of this

enzyme protects cAMP, promoting their destination to the increase in contractility. It now has a group of inotropic drugs whose mechanism of action is precisely in the inhibition of phosphodiesterase-III. Of this group stand amrinone and milrinone for the extensive clinical experience has accumulated with its use. These substances belong to the bipyridines, this is a positive inotropic effect supplemented by a peripheral vasodilator, which contributes to a better ability to emptying of the heart. The hemodynamic effects of milrinone, administered as a loading dose of 50 micrograms / kg followed by continuous infusion of 0.35 to 0.75 micrograms / kg / min is significant reductions in diastolic pressure in the aorta, the mean aortic pressure and systemic vascular resistance by about 11% ejection fraction of left ventricle is increased by about 14%, these effects are closely related to plasma concentrations. [117,118,119,120]

In the postoperative period especially in patients receiving milrinone pump has several effects on pulmonary circulation and inotropism as evidenced Mitsunori et al, which randomized 30 patients undergoing cardiac surgery treated with milrinone was reported, reduced the mean pressure of right atrial pressure in the pulmonary artery wedge, mean pulmonary pressure and systemic vascular resistance without making a significant change in mean arterial pressure or heart rate.

On the other hand the use of Milrinone has been shown to be beneficial in patients undergoing CSRC bomb and right ventricular dysfunction prior. Jong H. et al analyzed the effect of infusion of milrinone in patients undergoing CSRC and right ventricular dysfunction (VD) found no increase in cardiac index, heart rate, and decreased systemic vascular resistance. Changes in right ventricular ejection fraction were not significant, whereas in cardiac output and RV afterload if they were, finally improves graft flow in the breast and in the middle cerebral artery during surgery of the CSRC.

Dopamine

Dopamine (D) precursor of norepinephrine in the biological synthesis, there are specific receptors for this substance, especially in the renal circulation, where it produces a vasodilatory effect which favors renal tubular function (Hiberman et.al 1984). At the heart there are dopamine receptors, but its function on contractility is weak and little known, this effect is not accompanied by an increase in resistance as pronounced as with peripheral epinephrine and norepinephrine under in vessels predominantly to dopamine receptor stimulation.

The mechanism of action is dose dependent at relatively low doses (1-5µg/kg/min) stimulates dopamine receptors predominantly with subsequent renal and mesenteric vasodilation (Szerlip, et. Al 1991). A moderate dose of 5-10 mg / kg / min stimulates beta adrenergic receptors leading to positive inotropic effects and high-dose alpha-adrenergic stimulation 10-15µg/kg/min carries peripheral vasoconstriction.

By perioperative is used for its effects on the renal circulation as well as its positive inotropic effect can be used in improving the ICC states inotropism significantly, the clinical effect is seen immediately as the drug's half life is 3 -4 min. Among its side effects can cause or exacerbate tachyarrhythmias, because its effect is mediated by increased levels of norepinephrine. At present medical evidence did not show benefit of using low doses of dopamine effect of splanchnic vasodilatation and renal function, however, this drug increases oxygen consumption at promoting tubular tubular ischemia, in addition there is poor correlation between blood levels with dose infused.

Norepinephrine

Its structure is similar to that of epinerfrina is the endogenous neurotransmitter for postganglionic sympathetic nervous system, its basic function is to stimulate alpha-1 receptors and less beta-1 receptors and beta-2. Intravenous administration of norepinephrine increases blood pressure by increasing peripheral vascular resistance due to this increase, heart rate tends to decrease due to a vagal reflex which overrides the stimulation of myocardial beta-1 receptors, their After stimulation of these receptors causes a recent positive inotropic effect especially at low doses.
Under normal conditions this amine decreases renal blood flow (with minimal changes in glomerular filtration rate) and mesenteric, splanchnic and liver. The administration of norepinephrine should be through a central line to avoid tissue necrosis. Is much more convenient administration via infusion and the usual dose is 0.01 to 0.1 mg / kg / min or 2-15 mcg / min. So perioperative norepinephrine may be used at low doses for its chronotropic effect and vasoconstrictive properties (intermediate dose). Especially for its effect on the peripheral circulation is indicated in cases where failure is demonstrated in the ability of vasoregulation, because it increases blood flow by increasing systemic blood pressure especially in shock. It should be used so cautious in patients with MAO inhibitors. The tx hypovolemic shock with norepinephrine leads to severe multiorgan hypoperfusion.
The use of norepinephrine in patients undergoing cardiac surgery is controversial because of fear that has regard to the commitment in the function mediated renal vasoconstriction. Hiroshi Morimatsu, et. randomized 100 patients to post-operative heart to norepinephrine infusion in line and this study was carried out monitoring of renal function with infusion of norepinephrine in postsurgical hypotension TAM <70 mmHg. The results was an increase in central venous pressure, decreased systemic vascular resistance index with increased heart rate will eventually change in serum creatinine of treaties. Kwak Y, showed that one of the applications of norepinephrine after surgery is the treatment of hypotension in patients with chronic pulmonary hypertension as they are benefiting from the control of blood pressure without increasing the PSAP but rather decreases many of them.

Nitroglycerin

Its mechanism of action is through biotransformation in vascular smooth muscle by activating guanylyl cyclase thereby resulting in an increase of cyclic GMP and thus vasodilation. The effectiveness of nitroglycerin decreases after 18-24 hr by a phenomenon of tolerance due to decreased formation of nitric oxide in this way. The NTG has the ability to vasodilate both beds (arterial and venous) at low doses, dominated by its vasodilatory effect and increases venous capacitance and thus decreasing venous pressure and diastolic filling. However high doses of nitroglycerin significantly increases venous capacitance and systemic arteriolar resistance, thereby decreasing the systolic blood pressure and cardiac output. As a mechanism the body responds by reflex sympathetic tachycardia and peripheral arteriolar vasoconstriction, despite this effect on the coronary circulation is vasodilation of both healthy and affected artery atherosclerosis, and also increase collateral circulation areas although its main effect is on the arteries coronary larger caliber and low on resistance of lesser caliber. On myocardial oxygen requirements, mainly affects the ventricular wall tension by increasing venous capacitance, which in turn decreases venous return to the heart leading to a decrease in ventricular wall tension and myocardial oxygen consumption. Another beneficial effect is that the decrease in pre-produced increase in LV perfusion and favoring the subendocardium.

Their metabolism is primarily via hepatic glutathione by organic nitrate reductase. Its effect is dissipated in 30-60 min intravenous Its effect is achieved after 90 seconds and is dose dependent. The usual dose of 0.5-3 mg is / kg / min infusion or 5-200 g / min and 0.5 mg bolus. In post-qx therapy use in peri-and post-qx:

- Myocardial ischemia associated with ventricular arrhythmias, especially when this is caused by halogenated anesthetics.
- Myocardial ischemia with an increase in pulmonary capillary pressure associated with a persistence in the inhalation anesthetics.
- Coronary Spasm
- Intravenous administration has been proposed as prophylactic coronary bypass surgery to prevent episodes of ischemia by vasospasm
- Useful for the treatment of hypertension during surgery RVM.

Sodium nitroprusside

One of the most commonly used vasodilators in the perioperative period, penetrating the endothelium acts to form nitric oxide, this results in the production of guanine monophosphate to guanine triphosphate. Thus cyclic GMP is the second messenger that triggers calcium binding. Its effect occurs seconds after the start of infusion. The commonly used dose is 1-40 mcg / min.

At low doses predominantly dilated arteries and arterioles, and how the dose increases also becomes a venodilators. As with nitroglycerin may occur reflexively tachycardia. And increased venous capacitance and thereby reducing cardiac output. On the other hand it is important to consider that as an important arteriodilatador can produce the phenomenon of coronary steal, mostly because it does not vasodilation in arteries affected by atherosclerosis, reducing the flow in the latter.

Among other effects has the ability to produce dilation of the pulmonary vascular bed arriving to produce hypoxia. Another effect is less desirable thiocyanate intoxication, which prevent tissue oxygen delivery by blocking the final stages of the respiratory chain.

Its administration should be in glucose solution covering both the drug and the line connecting the infusion pump. The usual dose is 40 - 300 micrograms / min. Going to be titrated according to a patient's response.

Vasopressin

Vasopressin also known as antidiuretic hormone is a peptide product of the hypothalamus and stored in the posterior lobe of the pituitary. Feedback effects in several organs including the brain where it acts as a neurotransmitter regulating body temperature, nociception and adenocorticotropica hormone release. In the pulmonary vasculature, moderate dose of vasopressin causes vasodilatation while high doses produce vasoconstriction. Vasopressin also has other effects on thrombosis and hemostasis, including platelet aggregation and release of factor VIII and von Willebrand.

Plasma levels of vasopressin in patients after undergoing bypass surgery ranges from 100 -200 pg / ml, while the hemorrhagic shock promotes the release of plasma concentrations of 1000 pg / ml. Several publications indicate that the usual dose of this drug is between 0.01 to 0.1 U / min, and is effective in patients with shock vasodilation without adverse effects.

Hypotension refractory to high doses of alpha-adrenergic agonists after cardiac surgery, after the use of cardiopulmonary bypass, has been referred to as Sx vasoplegic. This

vasopressin has been used for this treatment with encouraging results. Masseti and colleagues studied 16 patients with intravenous vasopressin (0.1-1 IU / min) for the treatment of hypotension refractory to maximum dose of norepinephrine (> 30 mg / kg / min). Preoperative ejection fraction was 40.5% and NYHA functional class 3.5. Getting an increase in blood pressure of 89 mmHg baseline to 116, increase in SVR from 688 to 1043, decreased cardiac index from 2.6 to 2.9 L/min/m2, urine volume increased from 36.8 to 72.8 ml / h.

Whereas high doses of vasopressin and effective in the treatment of Sx vasoplegic after cardiac surgery with cardiopulmonary bypass.

In a recent study, Argenziano et al found that about 10% of patients undergoing cardiac surgery experienced hypotension by vasodilation after bypass surgery, which not necessarily is associated with cardiogenic or septic shock. Interestingly, in situations in which hypotension persists after surgery RVM, smooth muscle cells become less sensitive to circulating catecholamines. This phenomenon is due to decreased function of adrenergic receptors, the study of 50 patients conducted at Columbia Presbyterian Medical Center undergoing cardiac surgery were treated with vasopressin in the operating room or intensive therapy in the first 24 hr surgery (6). All patients had less than 60 mmHg TAM and decreased systemic resistance, despite support with catecholamines. This administration of vasopressin infusion of 0.09 U / min increased the TAM from 58 to 75 mmHg, the SVR increased from 920 to 1200 dyne s cm and achieving a reduction in the administration of norepinephrine in 32%.

Nesiritide

Brain natriuretic peptide also known as BNP is a neurohormone secreted by the left ventricle in response to increased stress (both pressure and volume in the varga) in the ventricular wall. Physiological actions of BNP include natriuresis, vasodilation and neurohormonal modulation. So the tx with BNP has emerged as a viable option in the tx of acute CHF. Moreover, its determination of serum is currently used to differentiate cardiac dyspnea pulmonary dela type. In general, levels of BNP 100pg/ml excludes minors 1 decompensated CHF, whereas values greater than 500pg/ml indicates decompensation.

Neseritide is the recombinant form of endogenous human BNP. Has been shown to decrease filling pressures, increase cardiac output and improve the clinical condition of patients with decompensated CHF. In August 2001 was approved by the FDA for tx of CHF in those with decompensated dyspnea at rest or with minimal effort. The recommended dose is 2µg/kg initial bolus followed by infusion of 0.01µg/kg/min extended to a maximum of 48 hr.

Several studies have examined the possible application of perioperative neseritide so, in patients with left ventricular dysfunction who will undergo heart surgery. In a study prospectivco, open, randomized controlled, Brackbill et al examined the use of perioperative infusion of neriritide and showed improvement compared with milrinone. We included 40 hemodynamically stable patients with LVEF 35% or less that were undergoing bypass surgery. And they were randomized to a bolus of nesiritide or milrinone intraoperatively followed by an infusion of any of them for 24 hr. The time spent in post-qx therapy was the primary outcome measure. The incidence of post-qx ICC, the rate of readmission within 30 days, mortality and other clinical parameters were compared. Patients receiving nesiritide had a hospital stay of 50.6 + / - 46.8 hours

compared with 44.1 + / - 23.5 hours of receiving milrinone (p = 0.57). The incidence of post-qx ICC also showed no significant results in both groups (p = 0.25). On the third day of follow up, no significant differences in SBP readmission between the two drugs and there was no impact on mortality, the authors concluded that nesiritide does not reduce hospital stay post-qx like not modify other parameters of disease compared with nesiritide in MVR and stable ventricular function.
In a prospective double-blind (NAPA) Mentzer et al; consider the role nesiritide might play in patients with left ventricular dysfunction with MVR with cardiopulmonary bypass. Patients with ejection fraction less than or equal to 40% who underwent MVR and DCP were randomized to receive nesiritide or placebo for 24-96 hr after induction of anesthesia. The post-qx renal function, hemodynamic parameters and drug use (primary endpoints) were evaluated in patients with MVR with DCP, mortality and safety (secondary endpoints) were evaluated in all patients who received the drug, 303 patients randomized, 279 received the drug and 272 underwent MVR with DCP. Compared with placebo, nesiritide was associated with a slight increase in serum creatinine (0.15 + / - 0.29 mg / dl versus. 0.34 + / - 0.48 mg / dl, p <0.001) and a fall in glomerular filtration rate (- 10.8 + / - 19.3 mL/min/1.73 m (2) versus -17.2 + / - 21.9 mL/min/1.73 m (2), P = 0.001) during hospital stay or stay on 14. On the other hand, patients treated with nesiritide had a shorter hospital stay (p = 0.043) and lower mortality at 180 days. (P = 0.046). The authors concluded that nesiritide in the context of RVM with CPB is associated with improved renal function post-qx and possibly increased survival.

8. Medical treatment

Aspirin

There are currently a total of 8 studies with more than 2500 patients using aspirin CSRC. The doses used 325-1200 mg daily. Two of these studies showed significant benefit of aspirin a day after heart surgery. In contrast to the other 6 that saw no difference vs placebo with regard to occlusion of the bridges managed belatedly. In conclusion, the evidence so far suggests that the use of aspirin to reduce occlusion of coronary artery bypass bridges to 12 months after CSRC when given the 1st day after surgery, on the other hand is a medicine economic which is associated with few adverse effects and is of great benefit for patients with coronary artery disease peripheral with that aspirin should be given the most quickly as possible after cardiac surgery and continued indefinitely.

HIipolipemiantes

There are three studies involving 1900 patients to evaluate the use of these agents on the occlusion of coronary artery bypass grafts and the risk of cardiovascular events): The Post-CABG trial, LOCATE (Lopid Coronary Angiography Trial) and Cholesterol Lowering Atherosclerotic Study (CLAS). All three showed a significant reduction in the progression of atherosclerosis in coronary artery bypass bridges. Thus the long-term use of lipid-lowering drugs prevent the progression of atherosclerosis in both native arteries and in coronary bridges and reduces cardiovascular events, it was shown that the use of these agents reduces the progression of atherosclerosis after 2 years RVM.
Pan et al, found that after adjusting the demographic and clinical differences, the preoperative use of statins was associated with a 50% reduction in mortality, but showed no benefit in the occurrence of AF or IM. Dotan et al, found that statins were associated

with a significant decrease in cardiac mortality, unstable angina and arrhythmias 60 days to 1 year.

Beta blocker

Its use has been assessed by many studies, but in a perioperative cardiac surgery, Sjölander et.al, conducted a controlled double-blind study of 967 patients with MVR. Patients were randomized 4 to 21 days after RVM receiving 50 mg of metoprolol 2 times per day x 2 weeks and 100 mg of metoprolol per day vs placebo 2 x 2 years. There was no significant difference between the 2 study arms with respect to exercise capacity, however cn patients placebo had a higher rate of chest pain compared with the metoprolol group. On the other hand no significant difference in both groups with regard to revascularization, unstable angina, nonfatal MI or death at 2 years of follow-up.
Finally, Ferguson et al, in a cohort study investigated the use of preoperative B-blocker in 629 877 patients undergoing CSRC and showed a reduction to 30 days of drug-related mortality (OR 0.90, 95% CI0.87-0.93). This decrease was consistent with all groups of patients except those with LVEF <30% where there was no demonstrated benefit.

Calcium antagonist

Only one study has examined the effect of calcium antagonists initiated after surgery RVM, Gaudino et al; evaluating the benefits of calcium antagonists after the first year of revascularization. A total of 120 patients with normal perfusion function were randomized after 1 year of tx with 120 mg diatiazem to continue with or stop. No significant differences after 4 years of follow-up among the group of calcium antagonists and those who discontinued tx with respect to recurrence of angina (10% vs 12%), residual ischemia, 17% vs 18%) and cardiac death 2% vs 0%). In short there is little evidence to support the routine use of calcium antagonists or nitrates after cardiac surgery.

ACEI

Despite the known benefits of ACE inhibitors, only 4 studies examined perioperative prophylactic use of ACE inhibitors in patients with MVR. QUO VADIS In the study 149 patients were randomized to quinapril 40mg/día or placebo for 4 weeks before elective surgery RVM, treatment was continued for 1 year. The researchers found that quinapril significantly reduced 1-year clinical events, such as death from cardiovascular causes.

9. Anticoagulants and antithrombotics management

Thromboembolism and bleeding associated with anticoagulation comprise about 75% of the complications associated with prosthetic valves. antithrombotic therapy can reduce but not eliminate the possibility of this catrastofe. It is reported that the incidence of perioperative arterial thromboembolism is approximately 0.4 to 1.5% and the annual risk of stroke in high risk patients without anticoagulation is> 5.6% and <2.0% of major bleeding.
The risk for thrombus formation with prosthetic heart valves is seven times higher in the first month after valve replacement during the following months, years intracardiac position independent. The underlying pathophysiology of activation are factors in the systems of intrinsic and extrinsic coagulation of synthetic surface extracorporeal circulation of blood or from contact with surfaces or tissue devoid of collagen, a significant number of surgeons in favor of delaying the anticoagulant because the risk of bleeding, the incidence of pericardial tamponade and reoperation is eight times higher in

patients treated with high doses of heparin than in those treated with low-dose heparin for prevention of venous thrombosis.

Anticoagulation is recommended in the following cases:

- Lifetime on all patients with mechanical valves
- Lifetime in patients with biological valves who have other indications for anticoagulation Vgr: atrial fibrillation, heart failure, ventricular ejection fraction less than 30% left.
- For the first three months in patients with bioprostheses after insertion, with a target INR of 2.5. No emabrgo strategy with aspirin (low dose-100 mg DE75) is an alternative, but there have been no randomized studies supporting the safety of this strategy.

There is no consensus at the time of initiation of anticoagulation after surgery, but should begin during the first days postoperatively. (5 + -2)

10. References

[1] Lyons AS, Petruccelli RJ II. Medicine and illustrated history. New York: HN Abrams Inc, 1978.

[2] Vesalius A. De humani corporis fabrica. Budapest: Corvina / Magyar Helikon, 1972.

[3] Rutkow IM. Surgery. An illustrated history. St. Louis, Baltimore, Boston, Chicago, London, Madrid, Philadelphia, Sydney, Toronto: Mosby, 1993.

[4] Casey LC. Roles of cytokines in the pathogenesis of multisystem organ failure induced cardiopulmonary. Ann Thorac Surg 1993; S6: S92-S96.

[5] Huddy SP, Joyce WP. Pepper JR. Gastrointestinal Complications in Patients Who underwent cardiopulmonary 4.473 bypass surgery. Br J Surg 1991; 78:293-296.

[6] CHV Thakar, Jared JP, Worley S, Cotman K, Paganini EP. Renal dysfunction and Serious Infections After open-heart surgery. Kidney Int 2003; 64:239-246.

[7] Laffey J, Boylan J, Cheng D. The systemic inflammatory response to cardiac surgery. Anesthesiology 2002; 97:215-252.

[8] Kollef MH, Wragge T, Pasque Ch Determinants of Mortality and multiorgan dysfunction in cardiac surgery Patients Requiring Prolonged mechanical ventilation. Chest 1995; 107 (5) :1395-1401.

[9] Kalfin RE, Engelmann RM, Rousseau JA, Flack JE III, Deaton DW, Kreutzer DL, DK Dash. Induction of interleukin-8 expression cardiopulmonary bypass DURING. Circulation 1993; 88 [Part 2]: 401 - 406.

[10] Ascione R, Lloyd CT, Underwood MJ, Lotto A, Pitsis AA, Angelini GD. After coronary revascularization Inflammatory response With or Without cardiopulmonary bypass prospective randomized study. Ann Thorac Surg 2000; 69: 1198-1204.

[11] Nilsson L, Kulander L, Sven-Olov N, Eriksson O. Endotoxins in cardiopulmonary bypass. J Thorac Cardiovasc Surg 1990; 100:777-780.

[12] Pizzo PA. Empirical therapy and prevention of infections in the immunocompromised host. In: Mandell GL, Bennett JE. Dolin R, editors. Principles and practice of infectious diseases. 5th edition. New York: Churchill-Livingstone, 2000. pp. 3102-3112.

[13] Rossi F. The O2-forming NADPH oxidase of the phagocyte: nature, Mechanisms of activating and function. Biochim Biophysics Acta 1984, 853:65-71.

[14] Clermont G, Vergely C, Girard C, Rochette L. Cellular injury associated with extracorporeal circulation (in French) Ann Cardiol Angiol 2002; 51 (1) :38-43.

[15] Richard H, Marc S, Graeme R. The systemic inflammatory response to cardiopulmonary bypass: Pathological, Therapeutic, and pharmacological considerations. Anesth Analg 1997; 85, 766-782.

[16] Sabik JF, Gillinov AM, Blackstone EH, Vacha C, Houghtaling PL, Navia J. Does off pump coronary surgery reduce morbility and Mortality? J Thorac Cardiovasc Surg 2002; 124 (4) :698-706.

[17] Butler J, Rocker GM, Westaby S. Inflammatory response to cardiopulmonary bypass. Ann Thorac Surg 1993; 55:552-559. Nashef SA, Roques F, Hammill BG et al. Validation of European

[18] System for Cardiac Operative Risk Evaluation (EuroSCORE) in North American cardiac surgery. Eur J Cardiothorac Surg 2002; 22 (1) :101-5. Bridgewater B. Mortality data in adult cardiac surgery for named surgeons: retrospective Examination of Collected data prospectively on coronary artery surgery and aortic valve replacement. BMJ. 2005; 330:506-10.

[19] Nilsson J, Algotsson L, Hoglund P, Luhrs C, Brandt J. Comparison of 19 pre-operative Risk stratification models in open-heart surgery. Eur Heart J. 2006; 27:867-74.

[20] Nashef SA. Editorial comment EuroSCORE and the Japanese aorta. Eur J Cardiothorac Surg. 2006; 30:528-3.

[21] Pitkanen O: Intra-institutional predictor of outcome after-cardiac surgery: comparison entre a locally derived model and the EuroSCORE. Eur J Cardiothorac Surg 2000; 18: 703 - 710.

[22] Geissler HJ: Risk stratification in heart surgery: comparison of six score systems. Eur J Cardiothorac Surg 2000; 17: 400-406.

[23] Van Dijk D, Nierich AP, Jansen EW, Nathoo MH Suyker WJ, Diephuis JC, van Boven WJ, Borst C, Buskens E, Grobbee DE, Robles De Medina EO, of Jaegere PP. Early outcome after-versus off-pump coronary bypass on-pump surgery: results from a randomized study. Circulation. 2001, 104:1761-1766.

[24] Ranucci M, Soro G, Frigiola A, Menicanti L, Ditta A, Candido G, et al. Normothermic perfusion lung function and cardiopulmonary bypass After: effects in pulmonary Risk Patients. Perfusion 1997; 12:309-15.

[25] Diegel A, Doll N, Rauch T, Haberer D, Walther T, Falk V, et al. Humoral immune response coronary artery bypass grafting DURING: a comparison of limited approach, "off-pump" technique, cardiopulmonary bypass and Conventional. Circulation 2000; 102 (Suppl III) :95-100. Wan S, Izzat MB, Lee TW, Wan IY, Tang NL, Yim AP.

[26] Avoiding cardiopulmonary bypass in multivessel CABG you reduce myocardial injury and cytokine response. Ann Thorac Surg 1999; 68:52-7.

[27] Ascione R, Lloyd CT, Underwood MJ, Lotto AA, Pitsis AA, Angelini GD. After coronary revascularization Inflammatory response With or Without cardiopulmonary bypass. Ann Thorac Surg 2000; 69:1198-204.

[28] C. Weissman Pulmonary Complications After Cardiac Surgery. Semin Cardiothorac Vasc Anesth 2004, 8:185-213.

[29] Michaux I, Filipovic M, Skarven K, Schneiter S, Schumann R, Zerkowski HR, Bernet F, Seeberger MD. Effects of on-pump versus off-pump coronary artery bypass graft surgery on right ventricular function. J Thorac Cardiovasc Surg. 2006, 131:1281-1288.

[30] Pegg TJ, Selvanayagam JB, Karamitsos TD, Arnold RJ, Francis JM, Neubauer S, Taggart DP. Effects of off-pump versus on-pump coronary artery bypass grafting on early and late right ventricular function. Circulation. 2008:117:2202-2210. Malouf PJ, Madani M, Gurudeva.

[31] Stucchi R, Poli G, Fumagalli R. Hemodynamic monitoring in ICU. Minerva Anestesiol 2006, 72 (6) :483-7.

[32] Bigatello LM, George E. Hemodynamic monitoring. Minerva Anestesiol 2002, 68 (4): 219-25.

[33] Umana E, Ahmed W, Fraley MA, et al. Comparison of Oscillometric and intra-arterial systolic and diastolic Blood Pressure in lean, overweight, and obese Patients. Angiology 2006; 57 (1) :41-5.

[34] Araghi A, Bander JJ Guzman JA. Arterial blood pressure monitoring in overweight Critically ill Patients: invasive or noninvasive? Crit Care 2006, 10 (2): R64.

[35] Pinsky MR, Payen D. Functional hemodynamic monitoring. Crit Care 2005, 9 (6) :566-72.

[36] Hayes MA, Timmins AC, Yau EH, et al. Elevation of systemic oxygen delivery in the Treatment of Critically Ill Patients. N Engl J Med 1994, 330 (24) :1717-22.

[37] Pinsky MR. At the threshold of noninvasive functional hemodynamic monitoring. Anesthesiology 2007; 106 (6) :1084-5.

[38] Michard F, Teboul JL. Predicting fluid responsiveness in ICU Patients: a critical analysis of the evidence. Chest 2002; 121 (6) :2000-8.

[39] Arthur C. St. Andre, MD, FCCM; Anthony DelRossi, MD. Hemodynamic management of Patients in the first 24 hours after-cardiac surgery. Crit Care Med 2005, 33:2082-2093

[40] Matte P, Jacquet L, Van Dyck M, Goen M. Effects of Conventional physiotherapy, continuous positive airway pressure and non-invasive Ventilatory support with bilevel positive airway pressure coronary artery bypass grafting After. Acta Anaesthesiol Scand 2000; 44: 75-81.

[41] Estes RJ, Meduri GU. The pathogenesis of ventilator-associated pneumonia. Intensive Care Med 1995, 21:365-83.

[42] Montner PK, Greene ER, Murata GH, Stark DM, Timms M, Chick TW. Hemodynamic effects of nasal and face mask continuous positive airway pressure. Am J Respir Crit Care Med 1994, 149:1614-8.

[43] Yoshiyuki Takami and Hiroshi Ina. Beneficial effects of bilevel positive airway pressure under cardiopulmonary bypass surgery After. Interactive Cardiovascular and Thoracic Surgery 2 (2003) 156-159.

[44] Anavekar NS, MacMurray JJ, Velazquez EJ, et al. Relation Between Renal dysfunction and cardiovascular outcomes after-myocardial infarction. N Engl J Med 2004; 351:1285-95.

[45] Shamagian LG, Varela A, Pedreira M, Gomez I, Virgo A, Gonzalez-Juanatey JR. Kidney failure is an independent predictor of mortality in patients hospitalized for heart

failure and is associated with a worse cardiovascular risk profile. Rev Esp Cardiol. 2006; 59:99-108.

[46] Easy L, Núñez J, Bodí V, et al. Prognostic value of serum creatinine in acute coronary syndrome without ST-segment elevation. Rev Esp Cardiol. 2006; 59:209-16.

[47] Hillis GS, Croal BL, Buchan KG, et al. Renal function and outcome from coronary artery bypass grafting. Circulation. 2006; 113:1056-62.

[48] Van den Berghe G, Bouillon, R., Mesotten, D., Braithwaite, S. S., Pei, J., Yi, D., Khoo, T. K., Olsen, K. A., Mohammedi, K., Roussel, R., Marre, M., Hall, P., Finfer, S., Chittock, D., the NICE-SUGAR Study Investigators, (2009). Glucose control in Critically ill Patients .. NEJM 361: 89-90 2009.

[49] Griesdale, D. G.S., de Souza, R. J., van Dam, R. M., Heyland, D. K., Cook, D. J., Malhotra, A., Dhaliwal, R., Henderson, W. R., Chittock, D. R., Finfer, S., Talmor, D. (2009). Intensive insulin therapy and Mortality Among Critically Ill Patients: a meta-analysis study Including NICE-SUGAR data. CMAJ 180: 821-827.

[50] Stamou SC, Hill PC, Dangas G, Pfister AJ, Boyce SW, Dullum MK, et al. After coronary artery bypass Stroke: Incidence, predictors, and clinical outcome. Stroke. 2001; 32:1508-13.

[51] Hogue CW Jr, Barzilai B, Pieper KS, Coombs LP, DeLong ER, Kouchoukos NT, et al. Sex Differences in Mortality and neurological outcomes after-cardiac surgery: a Society of Thoracic Surgery National Database Report. Circulation. 2001; 103:2133-7.

[52] CHW Hogue, Murphy SF, Schechtman KB, Davila-Roman VC. Risk factors for early or delayed stroke after-cardiac surgery. Circulation. 1999, 100:642-7.

[53] Libman RB, Wirkowski E Neystat M, Barr W, Gelb S, Graver M. Stroke associated with cardiac surgery. Determinants, timing, and stroke subtypes. Arch Neurol. 1997; 54:83-7.

[54] Perez-Vela JL, Ramos-González A, López-Almodóvar LF, Renes-Carreño E, type-Bárcena A, Rubio-Regidor M, et al. Neurological complications in the immediate postoperative period of cardiac surgery. Contribution of brain MRI. Rev Esp Cardiol. 2005; 58:1014-21.

[55] Williams GD, Bratton SL, Riley EC, et al. Coagulation tests cardiopulmonary bypass correlate with DURING blood loss in children undergoing cardiac surgery. J Cardiothorac Vasc Anesth 1999, 13:398-404.

[56] Despotism GJ, Filos KS, TN Zoysa, et al. Factors associated with Excessive Postoperative Blood Loss and hemostatic transfusion requirements: multivariate analysis in cardiac surgical Patients. Anesth Analg 1996; 82:13-21.

[57] Kessler C, Szurlej D, Von Heymann C. Management of refractory cardiac post-op bleeding with rFVIIa: Cases Reported to hemostasis.com registry [abstract]. J Thromb Haemost 2003:1131.

[58] Gomes WJ, Carvalho AC, Honorio Palma J, Concalvez I, Buffolo E. Vasoplegic Syndrome: A new dilemma. J Thorac Cardiovasc Surg 1994; 107: 942-3

[59] Ricardo Levin , Marcela Degrange. Síndrome vasopléjico en postoperatorio de cirugía cardíaca. Rev CONAREC Mayo-Junio 2006; (22), 84:78-81

[60] Czerny M, Baumer H, Kilo J. Inflammatory response and myocardial injury following coronary artery bypass grafting with or without cardiopulmonary bypass. Eur J Cardiothorac Surg 2000; 17: 737-42.

[61] Argenziano M, Choudhri AF, Mozami N, Levin H, Landry DH, Oz MC. A prospective randomized trial of arginine vasopressine in the treatment of vasodilatadory shock after left ventricular assist device placement. Circulation 1997; 96(suppl 2): II 286-290.

[62] Leyh, RG. Kofidis T, Strüber M, Fischer S, Knobloch K, Wachsmann B et al. Methylene blue: the drug of choice for catecholamine-refractory vasoplegia after cardiopulmonary bypass. J Thorac Cardiovasc Surg2003; 125: 1426-31

[63] Yiu P, Robin J, Pattison W. Reversal of refractory hypotension with single-dose methylene blue after coronary artery bypass surgery. JThorac Cardiovasc Surg 1999; 118: 195-6.

[64] Evora PRB. Should methylene blue be the drug of choice to treat vasoplegias caused by cardiopulmonary bypass and anaphylactic shock ?.J Thorac Cardiovasc Surg 2000; 119: 632-3.

[65] Pagni S, Austin E. Use of intravenous methylene blue for the treatment of refractory hypotension after cardiopulmonary bypass. J Thorac Cardiovasc Surg 2000; 119: 1297-8.

[66] Kofidis T, Struber M, Wilhelmi M et al. Reversal of severe vasoplegia with single-dose methylene blue after heart transplantation. J Thorac Cardiovasc Surg 2001;122: 823-4.

[67] Levin RL, Degrange MA, Bruno GF, Del Mazo CD, Griotti JJ and F.J. Boullon, Methylene blue reduces mortality and morbidity in vasoplegic patients after cardiac surgery. Ann Thorac Surg 2004; 77: 496-9.

[68] Gomes W, Carvalho AC, Palma JH, Teles CA, Branco JN, Silas MG et al. Vasoplegic syndrome after open heart surgery. J Cardiovasc Surg(Torino) 1998; 39: 619-23.

[69] Talbot MP, Temblay I, Denault AY, and Belisle S. Vasopressin for refractory hypotension during cardiopulmonary bypass. J Thorac Cardiovasc Surg 2000; 120: 401-2.

[70] Landry DW and Oliver JA. The pathogenesis of vasodilatory shock. N Engl J Med 2001; 345: 588-95.

[71] Mekontso-Dessap A, Houel R, Soustelle C, Kirsch M, Thebert D and Loisance DY, Risk factors for post-cardiopulmonary bypass vasoplegia in patients with preserved left ventricular function. Ann Thorac Surg 2001; 71:1428-32.

[72] Ozal E, Kuralay E, Yildirim V, Kilic S, Bolcal C, Kucukarslan N et al. Preoperative methylene blue administration in patients at high risk for vasoplegic syndrome during cardiac surgery. Ann Thorac Surg 2005; 79: 1615-19.

[73] shock after cardiac surgery: identification of predisposing factors and use of a novel pressor agent. J Thorac Cardiovasc Surg 1998;116(6):973—80.

[74] Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism Registry (ICOPER). Lancet 1999;353:1386-9.

[75] Georghiou GP, Brauner R, Berman M, Stamler A, Glanz L,Vidne BA, Erez E. Successful resuscitation of a patient with acute massive pulmonary embolism using emergent embolectomy. Ann Thorac Surg 2004;77:697-9.

[76] Yalamanchili K, Fleisher AG, Lehrman SG, Axelrod HI, LafaroRJ, Sarabu MR, et al. Open pulmonary embolectomyfor treatment of major pulmonary embolism. Ann Thorac Surg 2004;77:819-23.

[77] Tankut Hakki Akay, MDAtilla Sezgin, MD,Suleyman Ozkan, MD et. al Successful Surgical Treatment of Massive Pulmonary Embolism after Coronary Bypass Surgery. *Tex Heart Inst J 2006;33:498-500*

[78] Douketis JD, Kearon C, Bates S, Duku EK, Ginsberg JS. Risk of fatal pulmonary embolism in patients with treated venous thromboembolism. JAMA 1998;279:458-62.

[79] Roque Ramos, MD; Bakr I. Saiem, MD, FCCP;Maria P. De Pawlikowski, The Efficacy of Pneumatic Compression Stockings in the Prevention of Pulmonary Embolism After Cardiac Surgery CHEST 1996; 109:82-85

[80] John V. Booth, MBChB, FRCA*, Erin E. Ward, BS*, Kelly C. Colgan. Metoprolol and Coronary Artery Bypass Grafting Surgery: Does Intraoperative Metoprolol Attenuate Acute _-AdrenergicReceptor Desensitization During Cardiac Surgery? Anesth Analg 2004;98:1224 –31

[81] Dimitri Kalavrouziotis, *MD; Karen J. Buth, MSc; and Imtiaz S. Ali, MD.* The Impact of New-Onset Atrial Fibrillation on In-hospital Mortality Following Cardiac Surgery*CHEST 2007; 131:833–839*

[82] Roach GW, Kanchuger M, Mangano CM et al, for the multicenter study of perioperative ischemia research group and the ischemia research and education foundation investigators (1996) Adverse cerebral outcomes after coronary bypass surgery. N Engl J Med 335:1857–1864

[83] Hakala T, Hedman A (2003) Predicting the risk of atrial fibrillation after coronary artery bypass surgery ScandCardiovasc J 37:309–315

[84] Maisel WH, Rawn JD, Stevenson WG (2001) Atrial fibrillation after cardiac surgery. Ann Intern Med 135:1061–1073

[85] Siebert J, Rogowski J, Jagielak D, Anisimowich L, Lango R, Narkiewich M(2000) Atrial fibrillation after coronary artery bypass grafting without cardiopulmonary bypass. Eur J Cardiothorac Surg 17:520–523

[86] Tamis-Holland JE, Homel P, DuraniM, Iqbal M, Sutandar A, Mindich BP, Steinberg JS (2000) Atrial fibrillation after minimally invasive direct coronary artery bypass surgery. J Am CollCardiol 36:1884–1888

[87] W. JungU. MeyerfeldtR. BirkemeyerAtrial arrhythmias after cardiac surgeryin patients with diabetes mellitusClin Res Cardiol 95:Suppl 1, I/88–I/97 (2006

[88] Echt DS, Liebson PR, Mitchell LB et al (1991) Mortality and morbidity in patients receiving encainide, flecainide, or placebo: the cardiac arrhythmia suppression trial. N Engl J Med324:781–788

[89] Crystal E, Connolly SJ, Sleik K, Ginger TJ, Yusuf S (2002) Interventions on prevention of postoperative atrial fibrillation in patients undergoing heart surgery. A meta-analysis. Circulation106:75–80

[90] Daoud EG, Snow R, Hummel JD, Kalbfleisch SJ, Weiss R, Augostini R (2003) Temporary atrial epicardial pacing as prophylaxis against atrial fibrillation after heart surgery. J Cardiovasc Electrophysiol 14:127–132

[91] Vijayaraman P, Ellenbogen KA (2003) Postoperative atrial fibrillation: some more answers, some new questions. J Cardiovasc Electrophysiol 14:133–134

[92] Omran H, Jung W, Lüderitz B (1998) Dysfunction of the left atrium after cardioversion of atrial fibrillation. Am J Cardiol 81:837–838

[93] Roger J.F. Baskett, William A. Ghali, Andrew Maitland and Gregory M. HirschThe intraaortic balloon pump in cardiac surgery*Ann Thorac Surg* 2002;74:1276-1287

[94] Kusiak V, Goldberg S. Percutaneous intra-aortic balloon counterpulsion. Cardiovasc Clin 1985;15:281–302.

[95] McCarthy P, Golding L. Temporary mechanical circulatory support. In: Edmunds L, ed. Cardiac surgery in the adult. New York: McGraw-Hill, 1997:319–38.

[96] Dietl C, Berkheimer M, Woods E, Gilbert C, Pharr W, Benoit C. Efficacy and cost-effectiveness of preoperative IABP in patients with ejection fraction of 0.25 or less. Ann Thorac Surg 1996;62:401–9.

[97] Weintraub R, in discussion of Christenson J, Badel P, Simonet F, Schmuziger M. Preoperative intraaortic balloon pump enhances cardiac performance and improves the outcome of redo CABG. Ann Thorac Surg 1997;64:1237–44

[98] Aksnes J, Abdelnoor M, Bere V, Fjeld N. Risk factors of septicemia and perioperative myocardial infarction in a cohort of patients supported with intra-aortic balloon pump (IABP) in the course of open heart surgery. Eur J Cardiothorac Surg 1993;7:153–7.

[99] Creswell L, Rosenbloom M, Cox J, et al. Intraaortic balloon counterpulsation: patterns of usage and outcome in cardiac surgery patients. Ann Thorac Surg 1992;54:11–20.

[100] Naunheim K, Swartz M, Pennington D, et al. Intraaortic balloon pumping in patients requiring cardiac operations. J Thorac Cardiovasc Surg 1992;104:1654–61.

[101] Downing T, Miller D, Stinson E, et al. Therapeutic efficacy of intraaortic balloon pump counterpulsation: analysis with concurrent "control" subjects. Circulation 1981;64(suppl II):108–13.

[102] Tedoriya T, Kawasuji M, Sakakibara N, Takemura H, Watanabe Y, Hetzer R. Coronary bypass flow during use of intraaortic balloon pumping and left ventricular assist device. Ann Thorac Surg 1998;66: 477–81.

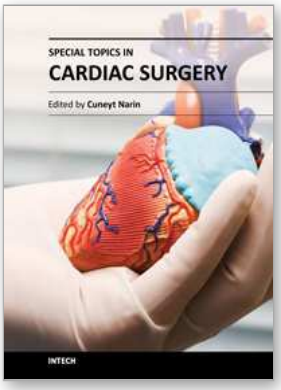
[103] Antiplatelet Trialists' Collaboration. Collaborative overview of randomised trials of antiplatelet therapy—II: maintenance of vascular graft or arterial patency by antiplatelet therapy. BMJ 1994;308:159-68.

[104] Burger W, Chemnitius JM, Kneissl GD, et al. Low-dose aspirin for secondary prevention—cardiovascular risks after its perioperative withdrawal versus bleeding risks with its continuation—review and meta-analysis. J Intern Med 2005;257:399-414.

[105] Ferraris VA, Ferraris SP, Moliterno DJ, et al. The Society of Thoracic Surgeons practice guideline series: aspirin and other antiplatelet agents during operative coronary revascularization (executive summary). Ann Thorac Surg 2005;79:1454-61.

[106] Sadony V, Korber M, Albes G, et al. Cardiac troponin I plasma levels for diagnosis and quantitation of perioperative myocardial damage in patients undergoing coronary artery bypass surgery. Eur J Cardiothorac Surg 1998;13:57-65.

[107] Thielmann M, Massoudy P, Schmermund A, et al. Diagnostic discrimination between graft-related and non–graft-related perioperative myocardial infarction with cardiac troponin I after coronary artery bypass surgery. Eur Heart J 2005;26:2440-7.



Special Topics in Cardiac Surgery
Edited by Prof. Cuneyt Narin

ISBN 978-953-51-0148-2
Hard cover, 308 pages
Publisher InTech
Published online 29, February, 2012
Published in print edition February, 2012

This book considers mainly the current perioperative care, as well as progresses in new cardiac surgery technologies. Perioperative strategies and new technologies in the field of cardiac surgery will continue to contribute to improvements in postoperative outcomes and enable the cardiac surgical society to optimize surgical procedures. This book should prove to be a useful reference for trainees, senior surgeons and nurses in cardiac surgery, as well as anesthesiologists, perfusionists, and all the related health care workers who are involved in taking care of patients with heart disease which require surgical therapy. I hope these internationally cumulative and diligent efforts will provide patients undergoing cardiac surgery with meticulous perioperative care methods.

INTECH
open science | open minds

InTech Europe
University Campus STeP Ri
Slavka Krautzeka 83/A
51000 Rijeka, Croatia
Phone: +385 (51) 770 447
Fax: +385 (51) 686 166
www.intechopen.com

InTech China
Unit 405, Office Block, Hotel Equatorial Shanghai
No.65, Yan An Road (West), Shanghai, 200040, China
中国上海市延安西路65号上海国际贵都大饭店办公楼405单元
Phone: +86-21-62489820
Fax: +86-21-62489821