Postoperative care after cardiac surgery

ESSAY
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LIST OF ABBREVIATIONS

ACTH: Adrenocorticotropic hormone.

AF: Atrial fibrillation.

AKICS: Acute kidney injury after cardiac surgery.

aPTT: Activated partial thromboplastin time.

ARDS: Adult Respiratory Distress Syndrome.

ATP: Adenosine tri-phosphate.

AV: Atrio-ventricular.

C: Complement.

CABG: Coronary artery bypass grafting.

CAD: Coronary artery diseases.

cAMP: Cyclic adenosine monophosphate.

CHF: Congestive heart failure.

CK: Creatine kinase.

cNO: Constitutive nitric oxide.

CNS: Central nervous system.

COPD: Chronic obstructed lung diseases.

CPB: Cardiopulmonary bypass.

CR1: Complement receptor Type 1.

CRT: Cardiac resynchronization therapy.

CT: Computed tomography.

CVAs: Cerebrovascular accidents.

DDDPM: Dual (A+V) (Dual-chamber pacemaker)

DSWI: Deep sternal wound infections.

EC: Endothelial cell.

ECF: Extracellular fluid.

ECG: Electrocardiogram.

ECLS: Extracorporeal life support.

ECMO: Extracorporeal Membrane Oxygenation.

EF: Ejection fraction.

EVH: Endoscopic vessel harvesting.

FDP: Fibrin degradation products.

GFR: Glomerular filtration rate.

GI: Gastro-intestinal.

GP: Glycoprotein.

HES: Hydroxyethyl starch.

HIT: Heparin-induced thrombocytopenia.

HPA: Hypothalamic pituitary- adrenal.

HS: Heparin sulphate.

HUVEC: Human umbilical vein-derived endothelial cell.

IABP: Intra-aortic balloon pump.

ICAM: Intercellular adhesion molecule.

ICD: Implantablecardioverter-defibrillator.

IFNg: Interferong.

IL-6: Interlukin-6.

IMA: Internal mammary artery.

IMV: Intermittent mandatory ventilation.

INR: International normalization ratio.

kDa: Kilodaltons.

LMWH: Low molecular weight heparin.

LPS: Lipopolysaccharide

LV: Left ventricle.

LVADs: Left ventricular assist devices.

LVEDP: Left ventricular end-diastolic pressure.

LVEDV: Left ventricular end-diastolic volume.

MOF: Multiple organ failure.

MRSA: Methicillin-resistant S. aureus.

MS: Molar substitution.

MW: Molecular weight.

NF: Nuclear factor.

NNE: Northern New England.

NO: Nitric oxide.

Nos: Nitric oxide synthase.

NS: Normal saline.

NSAIDs: Non-steroidal anti-inflammatory drug.

NSTEMI: Non ST segment myocardial infarction

OMT: Optimal medical therapy.

OPCABs: Off pump coronary artery bypass.

PAF: Platelet-activating factor.

PCI: Percutaneous coronary intervention.

PCWP: Pulmonary capillary wedge pressure.

PE: Pulmonary embolism.

PECAM: Platelet-endothelial cell adhesion molecule.

PEEP: Positive end expiratory pressure.

PF4: Heparin-platelet factor 4.

PPS: Postpericardiotomy syndrome.

PT: Prothrombin time.

PVR: Pulmonary vascular resistance:

RBC: Red blood cell.

RCT: Randomized controlled trial.

RT: Reptilase time.

RV: Right ventricle.

RVADs: Right ventricular assist devices.

sCR1: Soluble complement receptor 1.

SIRS: Systemic inflammatory response syndrome.

SNP: Sodium nitroprusside.

STS: Science and technology studies.

SVR: Systemic vascular resistance.

TAVI: Transcatheter aortic valve implantation.

TEE: Trans-esophageal echocardiography.

TIAs: Transint ischemic attakes.

TNF-a: Tumor necrosis factor-a.

TT: Thrombin time.

UFH: Unfractionated heparin.

VAC: Vacuum-assisted closure.

VCAM: Vascular cell adhesion molecule.

VTE: Venous thromboembolism.

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INTRODUCTION

Over time, the spectrum of surgical procedures available to patients with cardiac and great vessel disease continues to diversify and to mature. In parallel, our ability to match a widening range of surgical procedures with the individual needs of the patient has evolved as well. The relationships among preoperative patient-related risk factors and procedure-related perioperative care form the basis of the ongoing process of surgical care. Through a precise understanding of the long- and short-term benefits of a given surgical procedure, the risks for early and late complications can be evaluated to ensure that responsible clinical decision making occurs ***(Kirklin et al, 2007).***

Until 1953, cardiac surgery was in its infancy and was more of a curiosity, except for treatment of rheumatic mitral stenosis, beginning in 1923 with Cutler's successful case of a closed mitral commissurotomy with a tenotomy knife at the Peter Bent Brigham Hospital in Boston. The only successful heart operations done before 1953 were closed techniques for mitral stenosis, a few clinical experiments in 1952 with "open" heart by deep hypothermic arrest by John Lewis at the University of Minnesota. On May 6th, 2003, we celebrate the 50th anniversary of the first successful open-heart operation performed with the use of the heart-lung machine, one of the most important forms of therapy in the history of cardiac disease. The world owes John H. Gibbon the man who invent the heart lung machine, an enormous debt of gratitude for pioneering the technology of cardiopulmonary bypass and persisting for 23 years in its development until he got it just right, on the morning of May 6th, 1953***(Aris, 1997).***

In 1947 Thomas Holmes Sellors (1902–1987) of the Middlesex Hospital operated on a Fallot's Tetralogy patient with pulmonary stenosis and successfully divided the stenosedpulmonary valve***(Lawrence and Cohn.2007) .***

Surgery in great vessels (aortic coarctation repair, Blalock-Taussig shunt creation, closure of patent ductusarteriosus), became common after the turn of the century and falls in the domain of cardiac surgery, but technically cannot be considered heart surgery ***(Johnson, 1999).***

Since the 1990s, surgeons have begun to perform "off-pump bypass surgery" coronary artery bypass surgery without cardiopulmonary bypass. In these operations, the heart is beating during surgery, but is stabilized to provide an almost still work area in which to connect the conduit vessel that bypasses the blockage; in the U.S., most conduit vessels are harvested endoscopically, using a technique known as endoscopic vessel harvesting (EVH),some researchers believe that the off-pump approach results in fewer post-operative complications, such as postperfusion syndrome, and better overall results. Study results are controversial as of 2007. The surgeon's preference and hospital results still play a major role***(Lawrence and Cohn, 2007).***

CHAPTER(1)

PATHOPHYSIOLOGY DURING THE POSTOPERATIVE CARDIAC SURGERY

Cardiac surgery with cardiopulmonary bypass (CPB)provokes a systemic inflammatory response syndrome (SIRS). Contact of the blood components with the artificial surface of the bypass circuit, ischemia–reperfusion injury, endotoxemia and operative trauma are all possible causes of SIRS. This inflammatory reaction may contribute to the development of postoperative complications, including myocardial dysfunction, respiratory failure, renal and neurologic dysfunction, bleeding disorders, altered liver function, and ultimately, multiple organ failure (MOF). A number of different strategies, including new pharmacologic agents, CPB circuits and components, and surgical techniques, have been employed during the last few years in attempts to minimize the impact of SIRS on the outcome ofcardiac surgical patients. However, the complex pathophysiology of this problem has not allowed, until now, the use of a single strategy.***(Paparella et al, 2002)***

1- Acute phase reaction — stimuli and mediators:

The activation of the acute phase reaction during CPB isan extremely complex process. It occurs at differenttimes and has various triggers: the surgical trauma itself,blood contact with the non-physiological surfaces of theextracorporeal circuit, endotoxemia and ischemia. Severalmediators which are involved, exert synergistic effects, andthereby amplify this process.***(Laffeyet al, 2002)***

I. *Contact and complement systems:*

Exposure of blood to the extracorporeal circuit activatesthe contact system. The active form of factor XII convertsprekallikrein to kallikrein and initiates the intrinsic coagulationcascade that leads to the formation of thrombin. Thecomplement system is also activated, mainly through itsalternative pathway. The CPB circuit lacks the endothelialcell (EC) surface inhibitors that normally limit cofactor C3 activation, and this contact activation, along with the stimulus of kallikrein, provokes theformation of anaphylotoxins C3a and C5a with anaphylactic and chemotactic activity.The activation of the classic pathway factors C4 and