

Gastrointestinal Complications after Open Heart Surgery

An Essay

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Degree in General Surgery**

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Abbreviations

<i>ALT</i>	Alanine aminotransferase
<i>AP</i>	Alkaline phosphatase
<i>aPTT</i>	Activated partial thromboplastin time
<i>ARF</i>	Acute renal failure
<i>AST</i>	Aspartic aminotransferase
<i>BP</i>	Blood pressure
<i>C. difficile</i>	Clostridium difficile
<i>CABG</i>	Coronary artery bypass grafting
<i>cAMP</i>	Cyclic adenosine monophosphate
<i>CBC</i>	Complete blood count
<i>CCK</i>	Cholecystokinin
<i>CK-MB</i>	Myocardial muscle creatine kinase
<i>COP</i>	Cardiac output
<i>CPB</i>	Cardiopulmonary bypass
<i>CR1</i>	Complement receptor Type 1
<i>CRF</i>	Chronic renal failure
<i>CT</i>	Computerized tomography
<i>EC</i>	Endothelial cells
<i>EGD</i>	Esophagogastroduodenoscopy
<i>ERCP</i>	Endoscopic retrograde cholangiopancreatography
<i>GIT</i>	Gastrointestinal tract
<i>IABP</i>	Intra-aortic balloon pump
<i>ICAM</i>	Intercellular adhesion molecule
<i>ICU</i>	Intensive care unit
<i>IKB</i>	Inhibitory KB protein
<i>IL-</i>	Interlukin-
<i>iNO</i>	iNOS derived NO
<i>iNOS</i>	Inducible form of the enzyme NOS
<i>IV</i>	Intravenous
<i>Lab.</i>	Laboratory
<i>MOF</i>	Multiorgan failure
<i>MRI</i>	Magnetic resonance imaging
<i>NF-kB</i>	Nuclear factor-KB
<i>NO</i>	Nitric Oxide

<i>NOMI</i>	Nonocclusive mesenteric ischemia
<i>NOS</i>	Enzyme producing NO
<i>NPO</i>	Non per os
<i>NSAID</i>	Nonsteroidal anti-inflammatory drugs
<i>NYHA</i>	New York Heart Association
<i>PAF</i>	Platelet-activating factor
<i>PECAM</i>	Platelet-endothelial cell adhesion molecule
<i>PEEP</i>	Positive end-expiratory pressure
<i>PPIs</i>	Proton pump inhibitors
<i>PT</i>	Prothrombin time
<i>RUQ</i>	Right upper Quadrant
<i>SIRS</i>	Systemic inflammatory response syndrome
<i>SMA</i>	Superior mesenteric artery
<i>SNP</i>	Sodium nitroprusside
<i>TEE</i>	Trans-esophageal echo
<i>TNF-α</i>	Tumor necrosis factor- α
<i>TPN</i>	Total parenteral nutrition
<i>VCAM</i>	Vascular cell adhesion molecule

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Introduction

Introduction

The development of major surgeries including cardiac surgery was retarded for centuries by lack of knowledge and technology. Significantly, the general anesthetics, ether and chloroform, were not developed till the middle of the nineteenth century. These agents made major surgical operations possible, which created an interest in repairing wounds to the heart. In cardiac surgery, the development of cardio-pulmonary bypass (CPB) made repair of intracardiac lesions possible. Lillihei (a famous cardiac surgeon) wrote: “A physician at bedside of a child dying of an intracardiac malformation as recently as 1952 could only pray for a recovery. Today, with the heart-lung machine, correction is a routine”. **Stephenson, 2003.**

Many complications have been reported following cardiac surgeries involving all body systems including the gastrointestinal tract (GIT). **Hammon and Edmunds, 2003.**

These GIT complications have been reported involving the entire tract and liver as well following particularly open heart surgeries. The most common problems involve the upper GIT and liver. **Kelberman and Levine, 1992.**

These GIT complications are infrequent events, but a clinically important health care problem because they are often difficult to diagnose and associated with high morbidity and mortality rates. **Anderson et al, 2005.**

Incidence of all intra-abdominal side effects after CPB is about 1% to 2%, but mortality ranges from 12.5% to greater than 90% depending upon the specific complication and the surgical intervention needed. **Vijay and Gold, 2003.**

Aim of the work

To discuss the GIT complications following open-heart surgery and reach adequate strategies for avoidance and treatment.

CHAPTER 1

Pathophysiology of Cardiac Surgeries

Pathophysiology of Cardiac Surgeries

Introduction:

Cardiac surgery is unique because blood exposed to nonendothelial cell surfaces is collected and continuously recirculated through the entire body. This contact with synthetic surfaces in the perfusion circuit and multiple tissues within the wound triggers a massive defense reaction. This reaction, the inflammatory response to CPB, initiates a powerful thrombotic stimulus and the production, release and circulation of a host of microemboli and vasoactive and cytotoxic substances that affect every organ and tissue within the body. CPB and open heart surgery are simply not possible without heparin; thus the inflammatory response to CPB describes the consequences of exposing heparinised blood to non-endothelial cell covered surfaces. **Edmunds, 2003.**

Pathophysiology of cardiac surgeries comprises the effect of the produced microemboli, the associated inflammatory response during CPB and the low flow states either intraoperatively or postoperatively. **Mangi et al., 2005.**

To understand the pathophysiology of these surgeries, first there must be an overview on the CPB and its perfusion system and components.

Perfusion system

Components:

During CPB, blood is typically drained by gravity into the venous reservoir of the heart-lung machine via canulas placed in the superior and inferior venae cavae or a single canula placed in the right atrium. Blood from the reservoir is pumped through a membrane oxygenator into systemic arterial system, usually through a canula placed in the distal ascending aorta. The complete heart-lung machine includes many additional components. Most manufacturers consolidate a membrane oxygenator and venous reservoir into one unit. A micro filter-bubble trap is added to the arterial line. Depending on the operation, various suction systems are used to return blood from the surgical field, cardiac chambers and lower aorta to the reservoir. In addition to adjusting pump flow, partial and occluding clamps on venous and arterial lines are used to direct and regulate flow. Sites for obtaining blood samples and sensors for monitoring pressures, temperatures, oxygen saturation, blood gases and pH are included, as are various safety devices. Other components include heat exchanger, pumps, tubes, and connectors. **Hessel and Edmunds, 2003.**

A separate circuit for administering cardioplegic solutions (solutions that are used to arrest the heart during the CPB) at

controlled composition, rate, and temperature is usually included in the system. Figure (1-1) shows a typical CPB circuit.

Hessel and Edmunds, 2003.

- **Venous Reservoir:**

The venous reservoir serves as volume reservoir. This reservoir serves as a high-capacitance (i.e., low pressure) receiving chamber for venous return, facilitates gravity drainage, is a venous bubble trap, provides a convenient place to add drugs, fluids or blood. As much as 1–3 liters of blood may be translocated from the blood to the circuit when CPB is initiated. Reservoirs may be rigid plastic canisters (open type) or soft collapsible plastic bags (closed types). The rigid canisters facilitate volume measurement and venous air management, often have larger capacity, are easier to prime (priming fluid is the fluid added to the reservoir before initiation of the bypass). Disadvantages include the use of silicon antifoam compounds, which may produce micro-emboli, and increase activation of blood elements. Soft bag reservoirs eliminate the blood-gas interface and by collapsing reduce the risk of pumping massive air emboli. **Hessel and Edmunds, 2003.**

- **Oxygenators:**

Membrane oxygenators imitate the natural lung by interspersing a thin membrane of either microporous polypropylene (0.3-0.8 μ m pores) or silicon rubber between the gas and blood phases. Membrane oxygenators are safer, produce

less particulate of gaseous micro-emboli, are less reactive to blood elements and allow superior control of blood gases. With microporous membranes, plasma-filled pores prevent gas entering blood but facilitate transfer of both oxygen and CO₂. Because oxygen is poorly diffusible in plasma, blood must be spread as a thin film (approximately 100 µm) over a large area with high differential gas pressures between compartments to achieve oxygenation. Areas of turbulence and secondary flow enhance diffusion of oxygen within blood and thereby improve oxyhemoglobin saturation. CO₂ is highly diffusible in plasma and easily exits the blood compartment despite small differential pressures across the membrane. **Hessel and Edmunds, 2003.**

Conduct of CPB:

The surgeon is directly responsible to the patient for the outcome of operation, he needs a close working relationship with the anesthesiologist and perfusionist. CPB is started at the surgeon's request with concurrence of the anesthesiologist and the perfusionist. As the venous return enters the machine, the perfusionist progressively increases arterial flow while monitoring the patient's blood pressure and volume levels in all reservoirs. Once full stable CPB is established for at least two minutes, lung ventilation is discontinued, perfusion cooling may begin, and the aorta may be clamped for arresting the heart.

Hessel and Edmunds, 2003.

Life threatening complications during CPB:

Life-threatening incidents occur in 0.4% to 2.7% of operations with CPB and the incidence of serious injury or death is between 0.06% and 0.08%. Massive air embolism, aortic dissection, dislodgement of cannulas, and clotting within the circuit during perfusion are the principal causes of serious injury or death. Malfunctions of the heater-cooler, oxygenator, pumps, and electrical supply are the most common threatening incidents related to equipment. Other threatening incidents include premature takedown or clotting within the perfusion circuit.

Mejak et al., 2000.