LEFT VENTRICULAR ASSIST DEVICE MANAGEMENT IN THE INTENSIVE CARE UNIT

Thesis

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in General Intensive Care Medicine

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List of Abbreviations

ABG..... Arterial blood gases

ACE..... Angiotensin converting enzyme

ALT..... Alanine aminotransferase

AST..... Aspartate aminotransferase

AV..... Atrioventricular valves

B blockers..... Beta blockers

BiVAD ..... Biventricular assist device

BMI ..... Body mass index

BNP ..... Brain-Type Natriuretic Peptide

BUN..... Blood urea nitrogen

CABG ..... Coronary artery bypass grafting

CAD..... Coronary artery disease

CBC..... Complete blood picture

CHF..... Congestive heart failure

CK ..... Creatine kinase

CO ..... Cardiac output

CPB ..... Cardiopulmonary bypass

CS ..... Cardiogenic shock

CT..... Computed Tomography

CVP..... Central venous pressure

DT..... Destination therapy

List of Abbreviations (Cont...)

ECMO ..... Extracorporeal membrane oxygenation

EGD..... Esophagogastroduodenoscopy;

EGD..... Esophagogastroduodenoscopy;

GFR..... Glomerular filtration rate

GI..... Gastrointestinal

HCG..... Human chorionic gonadotropin;

HIT..... Heparin induced thrombocytopenia

Hrs..... Hours

IABP..... Intra aortic balloon pump

IM..... Intramuscular

INR..... International normalized ratio

IV..... Intravenous

L/min/m2..... Liter /minute/meter power two

LDH..... Lactate dehydrogenase

LV..... Left ventricle

LVAD..... Left ventricular assist device

MAP..... Mean arterial pressure

mcg/kg/min..... Microgram/ kilogram/minute

MCS..... Mechanical circulatory support

mg/d..... Milligram/day

mg/kg..... Milligram/kilogram

List of Abbreviations (Cont...)

MI ..... Myocardial infarction

mm Hg ..... Millimeter mercury

MR..... Mitral regurgitation

MRI ..... Magnetic resonance imaging

NPV ..... Negative predictive value

PA ..... Pulmonary artery

PAD ..... Peripheral artery disease

PCI..... Percutaneous coronary intervention

PCWP ..... Pulmonary capillary wedge pressure

PCWP ..... Pulmonary capillary wedge pressure

PEEP..... Peak end expiratory pressure

pg/mL..... Picogram per milliliter

PO..... Per mouse

PRA..... Plasma renin activity;

PT ..... Prothrombin time

PTCA..... Percutanous transluminal coronary angioplasty

PTT..... Partial thromboplastin time

PVAD..... Percutaneous ventricular assist device

PVR..... Peripheral vascular resistance

RBCs..... Red blood cells

rpm..... Revolutions per minute

List of Abbreviations (Cont...)

RV..... Right ventricle

RVAD ..... Right ventricular assist devices

RVEDV ..... Right ventricular end diastolic volume

RVESV ..... Right ventricular endsystolic volume

RVSWI ..... Right ventricular stroke work index

SA ..... Sinoatrial

SC ..... Subcutaneous

SIRS ..... Systemic inflammatory response syndrome

TEG..... Thromboelastography

US ..... Ultrasonography.

VADs..... Ventricular assist devices

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INTRODUCTION

The prevalence of heart failure is increasing and the prognosis of cardiogenic shock remains dismal. Cardiogenic shock is a common endpoint of multiple disease processes that is characterized by myocardial dysfunction, depressed cardiac output and end-organ hypoperfusion. Cardiogenic shock is associated with significant morbidity and mortality. The current gold-standard therapy in advanced cardiogenic shock remains cardiac transplantation, but the eligible candidates far outnumber the available donor organs (**Babaev et al., 2005).**

During the last 20 years, significant progress has been made in the treatment of cardiogenic shock, related not only to new pharmacotherapies [Angiotensin converting enzyme (ACE) inhibitors and beta-blockers], but also to device therapy and invasive treatment. These advances have resulted in an improved prognosis and also quality of life in patients with severe advanced heart failure (**Naples et al., 2008).**

Continuous-flow left ventricular assist systems are new generation devices with a number of major advantages over previous pulsatile technology (LVAD) have emerged as the standard of care for advanced heart failure patients requiring long-term mechanical circulatory support. Continuous-flow

LVAD have been developed during the past decade in response to the need for smaller and more durable device **(Rogers et al., 2010).**

Evidence-based management of LVAD-supported patients in the ICU is becoming increasingly important for optimizing outcomes and for enhancing survival. Topics that are generic to most continuous-flow devices include patient selection, pre-operative assessment and optimization, intraoperative considerations, post-operative patient management, patient education, and outpatient medical therapy **(Lahpor, 2009).**

PATHOPHYSIOLOGY AND CAUSES OF CARDIOGENIC SHOCK

The heart is a muscular organ, located in the thoracic cavity in between the lungs and posterior to the sternum, 60% of it lying to the left of the median plane. The heart's lateral projection extends from rib 3 to 6. Caudally the heart extends as far as the diaphragm. It functions as a circulatory pump. (**longo et al., 2011)**

Cardiogenic Shock

(CS) is a state of end–organ hypoperfusion due to cardiac dysfunction resulting in inability of the heart to maintain adequate cardiac output **(Babaev et al., 2005).**

Diagnosis

Cardiogenic shock is a physiologic state in which inadequate tissue perfusion result from low cardiac out put with Persistent hypotension (systolic blood pressure < 80 to 90 mmHg or mean arterial pressure 30 mmHg lower than base line), tachycardia, narrow pulse pressure, peripheral pulses are rapid and faint and may be irregular, severe reduction in cardiac index (<1.8 L/min/m2 without support or < 2.0 to 2.2 L/min/m2 with support) and adequate or elevated filling pressure (left ventricular end diastolic pressure >18 mmHg or right ventricular

end diastolic pressure > 10 to 15 mmHg). Hypoperfusion may be manifest clinically by cool clammy extremities, poor capillary refill, decreased urine out put, cyanosis and/or alteration in mental state **(Reynolds et al., 2008).**

Causes

Acute or chronic left ventricular failure is the classical scenario in cardiogenic shock.

1. Systolic dysfunction: The primary abnormality in systolic dysfunction is decreased myocardial contractility. Acute MI or ischemia is the most common cause; cardiogenic shock is more likely to be associated with anterior MI. The other causes are severe myocarditis, end stage cardiomyopathy (including valvular causes), myocardial contusion and prolonged cardiopulmonary bypass.

2. Diastolic dysfunction: increased left ventricular diastolic chamber stiffness contributes to CS commonly during myocardial ischemia, ventricular hypertrophy, restrective cardiomyopathy, ventricular interdependence, external compression by pericardial tamponade but also in the late stages of hypovolemic shock and septic shock.

3. Valvular dysfunction: valvular dysfunction may lead to cardiogenic shock acutely or may aggravate other

etiologies of shock. Acute mitral regurgitation secondary to papillary muscle rupture or dysfunction is caused by ischemic injury. Rarely, acute obstruction of mitral valve by left atrial thrombus or myxoma also may result in CS by means of severely decreased cardiac output. Aortic and mitral regurgitation reduce forward flow, raised end-diastolic pressure and aggravate shock associated with other etiologies.

4. Cardiac arrhythmia: Ventricular tachyarrhythmias often are associated with CS. furthermore, bradyarrhythmias may cause or aggravate shock due to another etiology. Sinus tachycardia and atrial tachyarrhythmia contribute to hypoperfusion and aggravate shock**.**

5. Coronary artery disease: Cardiogenic shock is associated with the loss of more than 40% of the left ventricular myocardium, right ventricular infarction or mechanical complication of MI (e.g, acute mitral regurgitation, ventricular septal rupture, free wall rupture) also may lead to cardiogenic shock. in patient with previously compromised left ventricular function, even a small infarction may precipitate shock. Cardiogenic shock is more likely to develop in people who are elderly or diabetic or in those who have a previous inferior infarction **(Dzavik**

et al., 2007).

6. Other causes:

- Large RV infarction occurs in up to 30% of patient with inferior MI and becomes hemodynamically unstable in 10% of those patient.
- Global hypoxemia and respiratory acidosis
- Endocarditis
- Papillary muscle dysfunction or rupture
- Myocardial depressant drugs (e.g, beta-blockers, calcium channel blockers, antiarrhythmics)
- Greatly increased afterload
  - Aortic stenosis
  - Hypertrophic cardiomyopathy
  - Dynamic aortic outflow tract obstruction
  - Coarctation of the aorta
  - Malignant hypertension
- Metabolic derangement (eg, acidosis, hypophosphatemia, hypocalcemia)
- Right ventricular failure
- Greatly increased afterload

- Pulmonary embolism
- Pulmonary vascular disease (e.g, pulmonary arterial hypertension, veno-occlusive disease)
- Hypoxic pulmonary vasoconstriction
- Peak end expiratory pressure(PEEP)
- High alveolar pressure
- Acute respiratory distress syndrome
- Pulmonary fibrosis
- Sleep disordered breathing
- Chronic obstructive pulmonary disease

(Jeger et al., 2008)

Pathophysiology

(CS) as a temporary or permanent derangement in the entire circulatory system.

LV pump failure is the primary insult in most forms of CS. The degree of myocardial dysfunction that initiates CS is often severe with the loss of more than 40% of the myocardial muscle. A decrease in coronary perfusion lowers cardiac output. A decrease in cardiac output leads to a further decrease in systemic and coronary perfusion.This exacerbates ischemia and cause cell death in the infarct border zone and the remote zone of