Clinical Implications of Cardiac Assisted Devices in Low Cardiac Output Syndrome

ESSAY
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بسم الله الرحمن الرحيم
قَالُوا سُبْحَانَكَ لاَ عِلْمَ لَنَا إِلاَّ مَا
عَلَّمْتَنَا إِنَّكَ أَنتَ الْعَلِيمُ الْحَكِيمُ
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Abdel Moniem El-Saied Abdel Moniem Allam

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List of Abbreviations

AMI	: Acute myocardial infarction.
ATP	: Adenosine triphpsphate.
ATT	: Alternative-To-Transplant.
A-V fistula	: Arterio Venous fistula.
A-V O_2	: Arterial-Venous oxygen gradient.
A-V valves	: Atrioventricular Valves.
BiVAD	: Biventricular assist device.
BSA	: Body surface area.
BTB	: Bridge to bridging.
BTC	: Bridge to candidacy.
BTR	: Bridge to recovery.
BTT	: Bridge to transplantation.
CABG	: Coronary artery bypass grafting.
$CaCO_2$	: Arterial CO_2 concentration.
CO	: Cardiac output.
CPB	: Cardiopulmonary bypass.
$CvCO_2$	: venous CO_2 concentration.
DT	: Destination therapy.
ECMO	: Extracorporeal membrane oxygenation.
EDP	: End diastolic pressure.
EDV	: End diastolic volume.
EF	: Ejection fraction.
ESV	: End systolic volume.
EVLW	: ExtraVascular Lung Water.
FDA	: Food and drugs administration.
GEDV	: Global End-Diastolic Volume.
Hb	: Hemoglobin.
HR	: Heart rate.
IABP	: Intra-aortic ballon counterPulsation.
INTERMACS	: Interagency registry for mechanical circulatory support.
ITBV	: IntraThoracic Blood Volume.
LCOS	: Low Cardiac Output Syndrome.
LiCl	: lithium chloride.

LV : Left Ventricule.

LVEDV : Left Ventricular End-Diastolic Volume.

LVESV : Left Ventricular End-Systolic Volume.

LVH : left ventricular hypertrophy.

MAP : Mean Arterial Pressure.

MR : Mitral Regurge.

MPA : Mean Pulmonary Artery Pressure.

NASA : National Aeronautical and Space Administration.

PA : Pulmonary Artery.

PaO_2 : Arterial oxygen Tension.

PAWP : Pulmonary Artery Wedge Pressure.

PCI : Percutaneous Coronary Intervention.

PEEP : Positive End Expiratory Pressure.

PLVAD : Percutaneous Left Ventricular Assisted Devices.

PPV : Pulse Pressure Variation.

PVI : plethysmographic variability index.

PVR : Pulmonary Vascular Resistance.

RAP : Right Atrial Pressure.

RV : Right ventricule.

RVAD : Right Ventricular Assist Device.

SaO_2 : Arterial Oxygen Saturation.

SBP : Systolic blood pressure.

SPV : Systolic Pressure Variation.

SV : Stroke Volume.

SvO_2 : Mixed Venous Oxygen Saturation.

SVR : Systemic Vascular Resistance.

SVV : Stroke Volume Variation.

TAHs : Total Artificial Hearts.

VCO2 : CO_2 consumption.

VADs : Ventricular Assist Devices.

VA-ECMO : Veno-Arterial ECMO.

VSD : Ventricular Septal Defect.

VV-ECMO : Veno-Venous ECMO.

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Introduction

Low cardiac output syndrome is defined as the pathophysiological state in which the cardiac output is not sufficient to maintain blood flow to meet metabolic needs of the body. Low cardiac output states are more common in patients with heart failure (low ejection fraction, cardiomegaly, and elevated left ventricular end diastolic pressure), longer durations of cardiopulmonary bypass, and in women ***(Lund* et al., *2010)***.

Heart failure is the final common pathway for many chronic heart diseases. With the aging of population and advances in the treatment of cardiac diseases, the number of patients with heart failure continues to increase. Although the majority of patients will remain stable for several years with standard medicines and surgery, a growing number will develop symptoms of advanced heart failure and may be referred for evaluation for heart transplant. For selected patients who are too ill to wait for a heart donor or who are not eligible for a heart transplant because of age or other medical problems, ventricular assist devices offer lifesaving therapy ***(Givertz, 2011)***.

A ventricular assist device (VAD) is a mechanical pump used for temporary blood circulation support. It decreases the workload of the heart while maintaining adequate flow and blood pressure. VADs can replace the left ventricle (LVAD), the right ventricle (RVAD), or both ventricles (BIVAD) ***(Wilson et al., 2009)***.

Ventricular assist devices can be categorized in a number of ways: centrifugal, volume-displacement (pneumatic, pulsatile), or axial-flow. Pumps can be placed extracorporeally (pump outside the body) or implanted. To date, implanted devices still require a percutaneous lead of some sort to provide power and an information interface to a driver device and monitor ***(Naidu, 2011)***.

VADs are typically used as a bridge to recovery (temporary support), bridge to transplantation (for heart transplantation) destination therapy (permanent support), or bridge to candidacy (eligible for heart transplantation but need of a period of VAD support) ***(Givertz, 2011)***.

Physiological Review of The Cardiovascular System

The heart is composed of three major types of cardiac muscle: atrial muscle, ventricular muscle, and specialized excitatory and conductive muscle fibers. The atrial and ventricular types of muscle contract in much the same way as skeletal muscle, except that the duration of contraction is much longer. Conversely, the specialized excitatory and conductive fibers contract only feebly because they contain few contractile fibrils; instead, they exhibit either automatic rhythmical electrical discharge in the form of action potentials or conduction of the action potentials through the heart, providing an excitatory system that controls the rhythmical beating of the heart ***(Guyton and Hall, 2006)***.

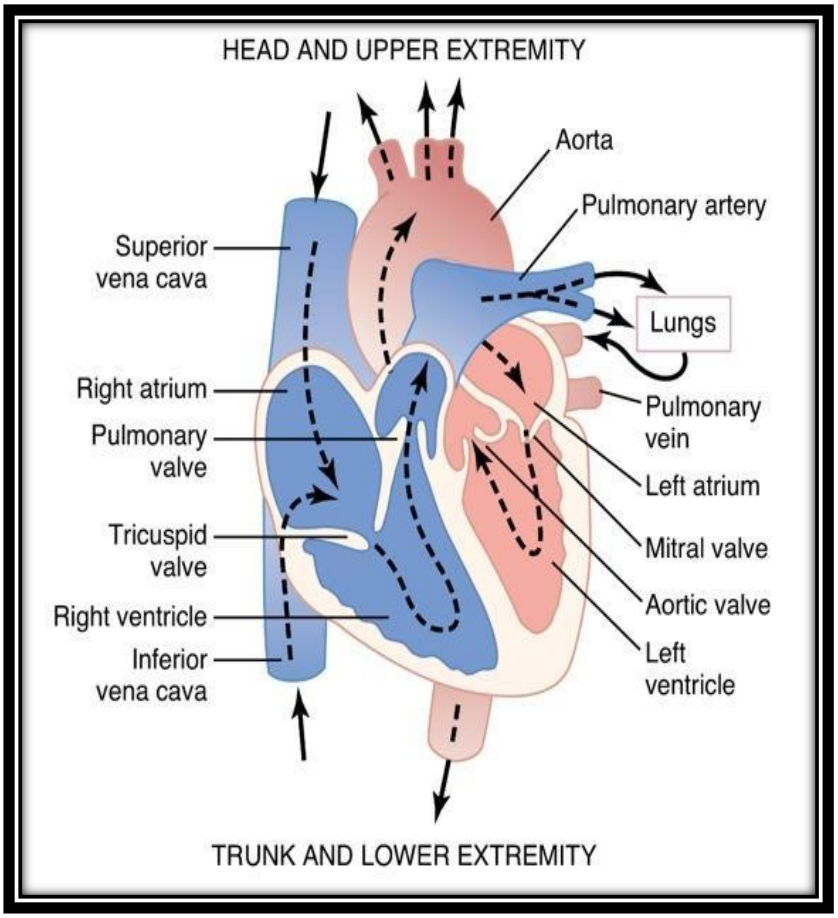

Figure 1: Structure of the heart and course of blood flow through the heart chambers and heart valves ***(Guyton and Hall, 2006).***

Atrioventricular Valves

Atrioventricular Valves (A-V valves) (the tricuspid and mitral valves) prevent backflow of blood from the ventricles to the atria during systole, and the semilunar valves (the aortic and pulmonary artery valves) prevent backflow from the aorta and pulmonary arteries into the ventricles during diastole. These valves, shown in (Figure 2) for the left ventricle, close and open passively. That is, they close when a backward pressure gradient pushes blood backward, and they open when a forward pressure gradient forces blood in the forward direction. For anatomical reasons, the thin, filmy A-V valves require almost no backflow to cause closure, whereas the much heavier semilunar valves require rather rapid backflow for a few milliseconds ***(Guyton and Hall, 2006)***.

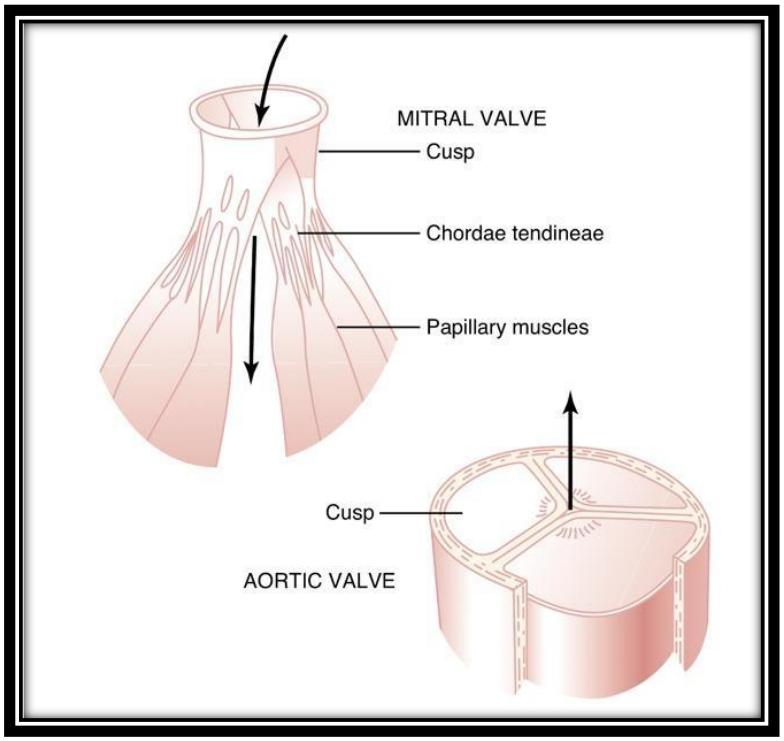

Figure 2: Mitral and aortic valves (the left ventricular valves) ***(Guyton and Hall, 2006)***.

Aortic and Pulmonary Artery Valves

The aortic and pulmonary artery semilunar valves function quite differently from the A-V valves. First, the high pressures in the arteries at the end of systole cause the semilunar valves to snap to the closed position, in contrast to the much softer closure of the A-V valves. Second, because of smaller openings, the velocity of blood ejection through the aortic and pulmonary valves is far greater than that through the much larger A-V valves. Also, because of the rapid closure and rapid ejection, the edges of the aortic and pulmonary valves are subjected to much greater mechanical abrasion than are the A-V valves. Finally, the A-V valves are supported by the chordae tendineae, which is not true for the semilunar valves. It is obvious from the anatomy of the aortic and pulmonary valves [as shown for the aortic valve at the bottom of (Figure 2)] that they must be constructed with an especially strong , yet very pliable fibrous tissue base to withstand the extra physical Stresses ***(Guyton and Hall, 2006)***.

Papillary Muscles

The papillary muscles that attach to the vanes of the A-V valves by the chordae tendineae are shown in (Figure 2). These papillary muscles contract when the ventricular walls contract, but contrary to what might be expected, they do not help the valves to close. Instead, they pull the vanes of the valves inward toward the ventricles to prevent their bulging too far backward toward the atria during ventricular contraction. If a chorda tendinea becomes ruptured or if one of the papillary muscles becomes paralyzed, the valve bulges far backward during ventricular contraction, sometimes so far that it leaks severely and results in severe or even lethal cardiac incapacity ***(Guyton and Hall, 2006)***.

Function of the Atria as Primer Pumps

Blood normally flows continually from the great veins into the atria; about 80 percent of the blood flows directly through the atria into the ventricles even before the atria contract. Then, atrial contraction usually causes an additional 20 percent filling of the ventricles. Therefore, the atria simply function as primer pumps that increase the ventricular pumping effectiveness as much as 20 per cent. However, the heart can continue to operate under most conditions even without this extra 20 percent effectiveness because it normally has the capability of pumping 300 to 400 per cent more blood than is required by the resting body. Therefore, when the atria fail to function, the difference is unlikely to be noticed unless a person exercises; then acute signs of heart failure occasionally develop, especially shortness of breath ***(Guyton and Hall, 2006)***.

Function of the Ventricles as Pumps

Filling of the Ventricles, during ventricular systole, large amounts of blood accumulate in the right and left atria because of the closed A-V valves. Therefore, as soon as systole is over and the ventricular pressures fall again to their low diastolic values, the moderately increased pressures that have developed in the atria during ventricular systole immediately push the A-V valves open and allow blood to flow rapidly into the ventricles ***(Guyton and Hall, 2006)***.

Emptying of the Ventricles during Systole, immediately after ventricular contraction begins, the ventricular pressure rises abruptly (Figure 3) causing the A-V valves to close. Then an additional 0.02 to 0.03 second is required for the ventricle to build up sufficient pressure to push the semilunar (aortic and pulmonary) valves open against the pressures in the aorta and pulmonary artery, blood begins to pour out of the ventricles ***(Guyton and Hall, 2006)***.