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LOW CARDIAC OUTPUT STATE AFTER OPEN HEART SURGERY

THESIS

Submitted in Partial Fulfilment
For The Degree of M.D.
In Cardiac Surgery

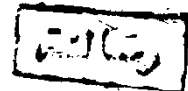
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Logic is not simple
If it was, it might be,
And if it were so, it would be,
But as it is n't, it ain't,
That's Logic.

lewis **Carrol** - ALICE IN WONDERLAND



DEDICATION :

To those Ever Lightened Candles in my life :

FIRST : To my LORD, who created me, supported me, and ever wait for me.

SECOND : To my Professors and Teachers, who gave me for nothing, the weapons in my hand to face my life and future.

THRIDLY : To my Dear Collaegue, who supported me, and ever stands beside me.

FOURTHLY : To my Mother, who always Prays, and GOD hear.

FIVTHLY : To my Wife, who patiently and heartly helps.

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I
INTRODUCTION
&
AIM OF WORK

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INTRODUCTION AND AIM OF WORK

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Low output syndrome (L.O.S.) is very common and serious complication, faced with after open heart surgery. It forms a challenging problem, and a major obstacle, for the success of cardiac operations, in all cardiac centres . Most, if not all postoperative complications, may end in L.O.S.

L.O.S. implies that the heart following operative interference under total cardiopulmonary bypass, is unable to deliver a satisfactory cardiac output. It may appear early rendering weaning from bypass difficult, or may set in a bit later in the early postoperative period.

A cardiac output unable to maintain body functions , results in serious disturbances of the internal environment (homeostasis), for example acidosis will result, which depress the myocardium further, and complete a vicious circle .

In 1967, Richard C. Lillihai et al, described under the term of low output syndrome following cardiopulmonary bypass, the postoperative picture of cardiac patients suffering from; hypotension, and signs of hypoperfusion of tissues as cerebral, visceral, and cutaneous. Other e.g. Grant (1967), regarded the syndrome as merely another name for shock, precipitated primarily by cardiac factors.

However, low output syndrome is usually cardiogenic, though multifactorial in origin. The factors may either be inherent in the patient himself preoperatively, and were regarded by the surgeon as an acceptable risk ,

or have been underestimated or even missed. Or mostly they occur in the peri-operative period, due to a multitude of precipitating causes, starting from anaesthesia, until transfer to the recovery room, or even later. This lead to the introduction of the many effective measures aiming at myocardial preservation & protection, before, during, and after surgery.

The syndrome once established is neither difficult to diagnose clinically, nor to confirm and differentiate by measurements of the cardiac output, with the other intracardiac pressures, and doing other investigations. The problem however lies in the multiplicity of the precipitating factors, and often their combination, which makes early detection and prophylaxis a hard, but never the less a gratifying task.

The study of the causes, prophylaxis and treatment of this syndrome, lead to the recent improvements and the new techniques used in open heart surgery (H.A. David et al 1982), to mention only a few such as :-

- The judicious use of deeper degrees of hypothermia, specially in infants, and the use of cold cardioplegia with external cooling of the heart (Shumway), during the prolonged aortic cross clamping .
- Good venting to avoid heart distention during surgery. Venting the left ventricle through the apex, could be replaced in presence of adhesions or weak myocardium, by venting through the left atrium, right superior pulmonary vein, pulmonary artery, or by using a wide bore needle transseptal via the right ventricle .
- Intentional opening of the right pleura and drain, in bleeding situations or long bypass , to avoid the occurrence of early cardiac tamponade.

- Electrical pacing of the heart, both atrial & ventricular, to combat both blocks, and resistant postoperative arrhythmias when necessary.
- The wiser application of pharmacological inotropic agents as regards; the time of initiation, dosage, and the choice of the drug.
- The use of mechanical support for the failing heart, as the intra-aortic balloon pump (I.A.B.P.), once the pharmacological support fails .
- The reduction of the peripheral vascular resistance during and after the operation specially if it was raised, by drugs such as nitroprusside drip to ease the work of the contracting ventricle & also in treatment of heart failure etc.

The incidence of low output state has markedly diminished in our Institute, since the use of cold cardioplegia and other modern techniques of myocardial preservation & protection . It is diminishing further by the progress of years .

THE AIM OF THIS WORK ; is :-

1. To verify the incidence and mortality of low output syndrome, in our cases of open heart surgery-operated between 1977 & 1982 .
2. To delineate the possible causes and responsible factors-isolated or combined-, accused for precipitating L.O.S., in our patients and own environment .

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3. To evaluate the various methods used for their early diagnosis and differentiation, as well as their prompt management, with their end results.

As well as trying to compare this, to what is mentioned in the medical literature or done abroad.

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II
REVIEW
OF
LITERATURE

BASIC PHYSIOLOGY OF C.O.

- ** The heart, Cardiac cycle & Haemodynamics.
- ** The Cardiac output, its measuring & controll.
- ** The Cardiac reserve.
- ** Myocardial contractility & Role of calcium.
- ** Positive & negative inotropics.

THE HEART, CARDIAC CYCLE & HAEMODYNAMICS

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The heart acts as a biperfusional muscular pump , which propels the blood into two main closed circulations.(Green J.H. 1972, Ganong A.C. 1975, and Weber K.T. 1978) . They state that the right side pumps unoxygenated venous blood to the lungs to be oxygenated, and the left side pumps oxygenated blood reaching it from the lungs to all tissues of the body, in accordance to their local needs and metabolic requirements.

Normally, each cycle goes hand in hand at the same time with the other, with nearly the same cardiac output for each ventricle per minute. The blood is separated from the tissues by the capillary walls, and only tissue-fluid is filtered at the arterial ends of the capillaries, to be reabsorbed at their venous ends. Blood itself only gets in contact with the tissue cells in the sinuses, of the liver, spleen, and bone marrow. The pre-capillary resistant vessels(arteries), do not contain much of the blood volume, as do the post-capillary capacitant veins, which contain about 70 % of the total blood volume, so acting as a blood reservoir for emergencies, e.g. in **haemorrhage**.

The cardiovascular system is under humoro-nervous control through the autonomic nervous system (sympathetic & parasympathetic), which is dominant in stress situations .

The circulation is carried out by the heart contraction & relaxation i.e. the cardiac cycle, which is the events (electrical, mechanical, and other changes), that occur in the heart and blood vessels,

during one heart beat, systole and diastole.
(Braunwald E. et al. 1960, and Guyton A.C. 1975).
They state that, in the normal heart, which beats about 75 beat/minute, the cardiac cycle (one heart beat) lasts 0.8/second. It is shortened in tachycardia, and prolonged in brady-cardia. This change in duration of the cardiac cycle, is mainly due to change in its diastolic time period rather than its systolic time period. This would affect the ventricular filling & the diastolic length of the muscle fibres, and so would affect the cardiac output .

The cardiac cycle ; It equals 0.8 second, and is formed of atrial and ventricular components, both contraction & relaxation, which occur in succession, the atria preceding the ventricles.

- * Atrial systole = 0.1 sec., Diastole = 0.7 sec.
- * Ventricular systole = 0.3 sec., Diastole = 0.5 sec.

The ventricular component of the cardiac cycle (systole & diastole), is the most important event responsible for the cardiac output, and is subdivided for study into 8-phases (Fig. 1).

Ventricular systole (0.3 sec) :-

- (1) - Isometric (isovolumetric) contraction phase = 0.05 sec.
At the beginning of this phase closure of the atrio-ventricular valves occur , and the first heart sound is heard. At the end of this phase, opening of the semilunar valves occur .
- (2) - Maximum ejection phase = 0.15 sec., during which maximal ejection of blood occurs .