MANAGEMENT OF LOW CARDIAC OUTPUT STATES DURING OPEN CARDIAC SURGERY

ESSAY

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BY

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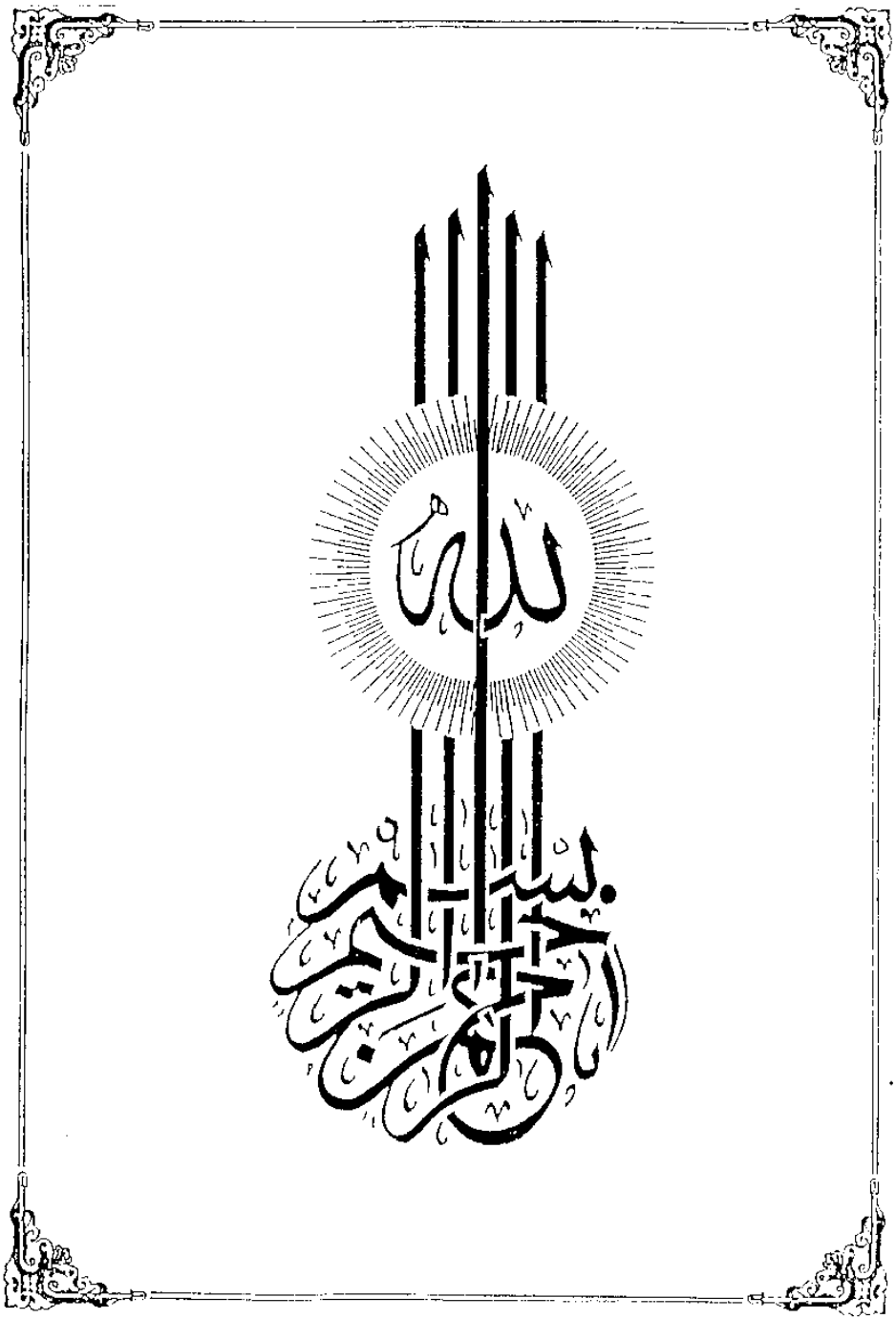

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INTRODUCTION

A small percentage of patients during open cardiac surgery develop low cardiac output syndrome, which is defined as a cardiac index less than 2 L/min/m^2, associated with a systolic arterial pressure less than 90 mmHg, and evidence of poor tissue perfusion.

Low cardiac output remains a big complex and challenging problem in all cardiac centres.

Extensive hemodynamic monitoring is essential to be able to correctly determine the causes and the effects of the different therapeutic interventions.

Technological advances in pulmonary artery catheters e.g., thermodilution, oximetry, have provided more information about the patient's hemodynamics.The recent introduction of transesophageal echocardiography in the monitoring of cardiac surgical patients, has enabled the anesthesiologist to map the external and internal anatomy and function of the heart and great vessels.

The management of these patients is difficult and usually involves the use of inotropic agents and vasodilators to manipulate preload, cardiac contractility and afterload. When pharmacological support alone is inadequate to reverse the low cardiac output syndrome, mechanical circulatory support must be added. The intra-aortic balloon pump supports the failing left ventricle by augmenting diastolic pressure and unloading the aorta during systole.

In this study, the following items will be discussed: physiology of the cardiac output, assessment of the cardiac function, hemodynamic monitoring during open cardiac surgery, causes of low cardiac output during open cardiac surgery and management of low cardiac output during open cardiac surgery.

Chapter 1

PHYSIOLOGIC CONSIDERATION

The heart acts to pump the blood in the circulation in order to transport oxygen and other nutrients to the cells of the body, to remove metabolic waste products from the cells of the body, and to carry substances, such as hormones, from one part of the body to another.

With every beat, the performance of the heart may be considered the net result of three major determinants: preload, afterload and contractility. The heart rate then determines the performance of the heart relative to time. Cardiac performance is further influenced by many factors, including the synchrony of ventricular contraction, atrial function, neural control, drugs, hormones and metabolic products, and pericardial properties **(Hurst, 1990)**.

Myocardial Excitation-Contraction Coupling

All studies done for understanding of the mechanisms by which the action potential stimulus initiates the contractile process in heart muscle have emphasized the central role of calcium ion [Ca^{++}] in excitation-contraction coupling **(Hurst, 1990)**.

With the initiation of the action potential in ventricular myocardium (figure 1), there is a very rapid influx of Na^{+}, which produces the rapid electrical spike and overshoot during phase zero of the action potential. During the plateau phase of the action potential [phase 2], there is a slow inward influx of Ca^{++} through slow channels in the myocardial cell membrane, or sarcolemma, into the intracellular fluid, or sarcoplasm, and possibly into the sarcoplasmic reticulum **(McDonald, 1985)**. The action potential also spreads from the myocardial cell membrane down the extensive transverse [T] tubular system, which consists of sarcolemma invaginations especially near the Z bands, and are in direct continuity with the extracellular space.

The action potential descends the T-system into the triadic

junctions, in which a single T-system tubule is in extremely close proximity to two terminal cisternae or extensions (lateral sacs) of the sarcoplasmic reticulum.

The exact mechanism by which the action potential depolarization is transferred from the sarcolemma and the T-system to the intracellular sarcoplasmic reticulum is unknown, though it is probable that the relatively small, early, initial trans-sarcolemma calcium flux mediates this role **(Fabiato, 1985)**.

Once the sarcoplasmic reticulum is depolarized, the excitation spreads rapidly throughout the sarcoplasmic reticulum, and relatively large amounts of Ca^{++} are released from the sarcoplasmic reticulum into the sarcoplasm during the plateau phase of the action potential. The Ca^{++} binds to specific sites on the contractile proteins, permitting the fibril to contract.

Thus, a very low concentration of inward Ca^{++} movement, produced by the action potential, is the initiating stimulus for the calcium triggered release of Ca^{++} from the sarcoplasmic reticulum **(Sperelakis, 1985)**.

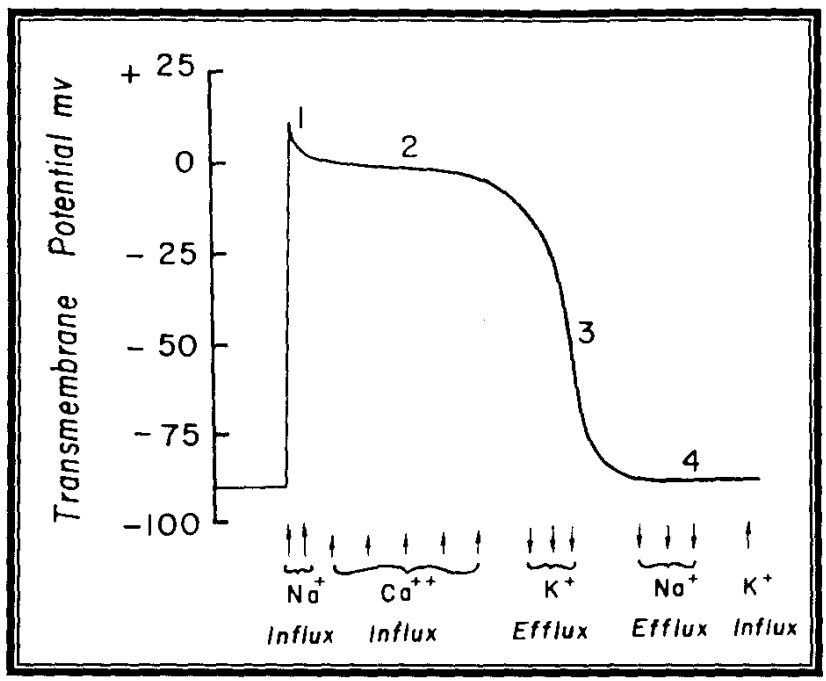

Figure (1): *Schematic action potential of human ventricular myocardium, together with probable electrolyte movements. The initial phase zero spike and overshoot is related to a sudden influx of Na^+. This is followed by a slower, maintained influx of Ca^{++} during the plateau phase 2.. The phase of Ca^{++} efflux is not well-defined in human ventricular myocardium, but presumably it occurs during phase 4* ***(Hurst, 1990)****.*

The increased sarcoplasmic free Ca^{++} diffuses to the myofibrils, where it binds to subunits of troponin, which are located periodically along thin actin filaments. In the absence of Ca^{++} , troponin works through tropomyosin, which course along the actin filament, to prevent actin from interacting with myosin. Once Ca^{++} attaches to troponin, the binding of troponin to actin is inhibited. This produces a conformational change in tropomyosin that enhances the formation of cross-bridges between myosin and actin, resulting in contraction **(Hurst, 1990)**.

The velocity and the amount of tension developed by the actin-myosin myofilaments are directly related to the amount of Ca^{++} available to inhibit troponin to induce contraction. So, it is likely that many drugs, such as digitalis, sympathomimetic amines, xanthines and phosphodiesterase inhibitors, have their influence upon myocardial contractility through their effect upon available intracellular Ca^{++} **(Colucci et al., 1986)**.

Nervous Control of the Heart

The nerve endings of sympathetic fibres synthesize norepinephrine and store it in granules. Both atria and ventricles contain β_1 and β_2 receptors. Other studies had indicated that β_2 receptors are located especially in vascular smooth muscle or in the SA or the AV nodes **(Longabaugh et al., 1986)**. Upon stimulation, sympathetic fibres cause the local release of norepinephrine, which acts locally upon the beta receptors, which are present on the fibre surface, to enhance the activity of adenylate cyclase, which in turn catalyzes the conversion of adenosine triphosphate [ATP] to cyclic adenosine 3',5'-monophosphate [AMP] **(Sutherland, 1970)**.

Cyclic AMP activates protein kinases that phosphorylate the slow calcium channel, increasing myocardial Ca^{++} and myocardial contractility (figure 2) **(Hurst, 1990)**.

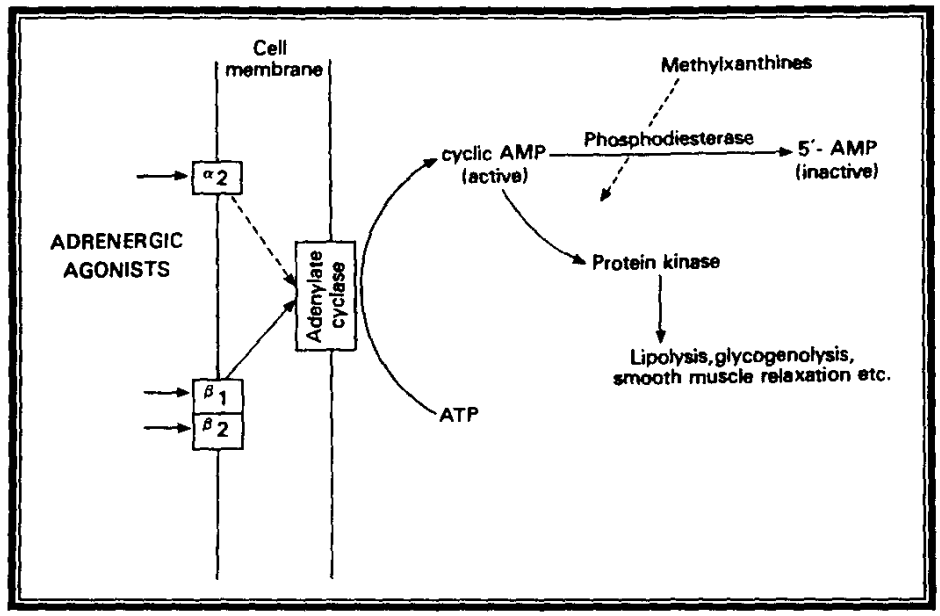

Figure (2): *Relationship between adrenergic agonists and the production of cyclic AMP. Binding of agonist to the adrenoceptors on the cell surface membrane activates either stimulation ($\rightarrow \beta_1, \beta_2$) or inhibition ($\rightarrow \alpha_2$) of the enzyme adenylate cyclase, which catalyzes the conversion of ATP to cyclic AMP and is, in turn, inactivated by the intracellular enzyme, phosphodiesterase. Cyclic AMP interacts with cytoplasmic protein kinase to initiate various cell functions (Hurst, 1990).*

Sympathetic nerve fibres reach the entire atria and ventricles as well as the SA and AV nodes, while vagal fibres, which cause local release of acetylcholine, influence predominantly the atrial musculature and SA and AV nodes. Some vagal innervation, however, has also been shown to reach the ventricles, and vagal stimulation can decrease ventricular contractility modestly. Vagal stimulation generally has opposite effects to sympathetic stimulation on the SA node. At any given instant, the effect of the nervous system on the heart is the net balance of these two opposing controls, which usually vary reciprocally. Vagal stimulation, which is generally inhibitory, normally pre-

dominates, and maintains the usual resting heart rate **(De Geest et al., 1965)**.

The Cardiac Output

The cardiac output is the amount of blood pumped by the heart to the peripheral circulation per minute. It equals the stroke volume [SV] per beat times the heart rate [HR] per minute:

Cardiac output = Stroke volume x Heart rate [CO = SV x HR]

Normal average values are a cardiac output of 5-6 L/minute in a 70 kg man, with a stroke volume of 60-90 ml/beat, and a heart rate of 80 beats/minute.

To compare patients with different body sizes, cardiac output may be corrected in relation to body surface area, and then called the cardiac index [CI], which equals the cardiac output divided by the body surface area [BSA]:

$$CI = \frac{CO}{BSA}$$

The normal value for a 70 kg man is 2.5-3.5 L/min/m^2 **(Guyton, 1986)**.

The two main determinants of cardiac output are heart rate and stroke volume, which is affected by four factors: preload,