MYOCARDIAL PRESERVATION FOR OPEN HEART SURGERY

ESSAY

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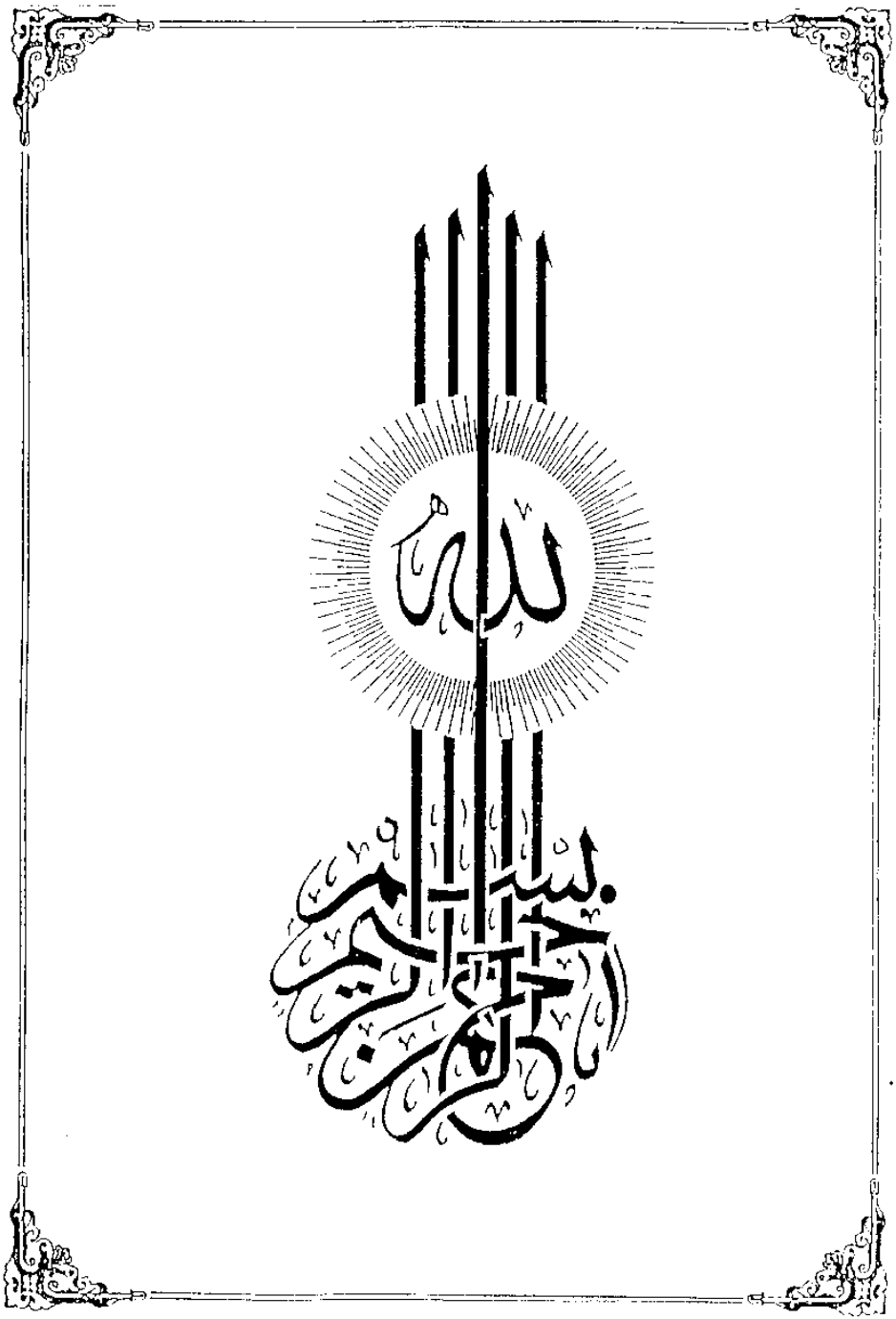

C O N T E N T S

INTRODUCTION

It became evident during the early years of open cardiac surgery that a major portion of the deaths occurring in the early post operative period were related to a form of acute cardiac failure manifested clinically as the low cardiac out put syndrome. Surgeons and cardiologists at first assumed that this syndrome was an unavoidable complication of operations that involved extensive direct cardiac manipulation and the radical physiological changes of cardiopulmonary bypass in patients with already serious heart disease. In two reviews of the medical complications of open heart surgery published in 1965 (Williams et al.) and 1966 (Rosky and Rodman), the problem of low cardiac output in the early post operative period was discussed in detail, but myocardial necrosis was not identified as a possible etiology. But in 1967 Taber and colleagues implicated this necrosis as the cause of low cardiac output in the early postoperative period. Also in 1969, Najafi and colleagues described acute diffuse hemorrhagic subendocardial necrosis in patients dying soon after valve replacement and related it to techniques of intraoperative management, particularly the use of continuous ventricular fibrillation during coronary perfusion for aortic valve replacement.

Only in recent years, it has been realized that death from low cardiac output soon after cardiac surgery is usually caused by perioperative myocardial necrosis. This necrosis

may not only cause moderate cardiac impairment in the early post operative period, but the long term result of the operation may also be affected. (Vincent R. Conti & William A, Lell, 1982).

Prevention of myocardial injury during the ischemic interval is critical to survival of patient and has been the subject of investigation during the past decade. Improvements in myocardial protection resulting from this work have contributed significantly to reductions in mortality and morbidity in most patients undergoing cardiac surgery.

History of Myocardial Protection

Hypothermia was the first method of myocardial protection to be applied to clinical intracardiac procedures (Lewis and Taufic 1953 and Bigelow et al., 1954). When the viability of the brain and myocardium was sustained with moderate systemic hypothermia (30°C), vena caval inflow was occluded and the heart opened for brief periods. Although this approach provided limited operative time, simple anomalies such as atrial septal defects could be repaired with a high degree of success (Brock and Ross, 1955).

After the development of mechanical cardiopulmonary bypass, extracorporeal circulation was combined with profound systemic hypothermia in an effort to improve the protection of body organs during periods of ischemic cardiac arrest (Sealy et al., 1957, 1960 and Drew and Anderson, 1959). By lowering the body temperature to 15°C, the circulation could be reduced or stopped for periods of up to 1 hour, providing optimal technical conditions for cardiac surgical procedures. The patient was then rewarmed using the extracorporeal circuit, and the operation was concluded. As time progressed, most surgeons largely abandoned this technique, but interstingly, it has reemerged in recent years as a highly acceptable method for repair of several categories of cardiovascular disorders (Barratt-Boyes et al., 1971 and Crawford and Saleh, 1981).

Chemical arrest of the heart was introduced by Melrose and associates in 1955.

After occlusion of the ascending aorta, a 2.5% solution of potassium citrate in blood was injected into the aortic root, achieving a flaccid arrest of the myocardium. The intracardiac repair was then performed with a quiet, motionless operative field. With re-establishment of coronary blood flow and washout of the potassium, normal myocardial contraction resumed. Although a moderate clinical experience with this method was obtained, subsequent reports of direct myocardial injury by the potassium citrate led to abandonment of this practice (Helmsworth et al. 1959 and McFarland et al. 1960).

As with hypothermia, however, the principle of chemical arrest was to be modified over the next decade and reintroduced into clinical practice in the 1970s.

After the experience with potassium citrate, most surgeons adopted moderate systemic hypothermia and continuous or intermittent coronary perfusion as the routine method of myocardial protection. Although this method reduced operative mortality rates, it soon became evident that myocardial injury occurred in a significant number of patients. Subendocardial necrosis was observed frequently in hypertrophied ventricles, even with relatively short periods of ischemia (Najafi et al., 1971). In addition, problems associated with coronary

perfusion cannulas, continued myocardial tone, and coronary venous effluent impaired operative exposure and complicated the technical conduct of the procedure.

An important addition to the technique of intermittent ischemic arrest was topical cardiac hypothermia (Shumway et al. 1959). Reduction of myocardial temperature during the ischemic period slowed metabolic processes and improved tolerance to anoxia. As early as 1964, Shumway and associates achieved excellent results with this method and reported a marked reduction in operative mortality (Hurley et al. 1964). The addition of topical endocardial cooling provided better transmural myocardial protection, and ischemic periods of over 2 hours were tolerated with good recovery. The principles developed by the Stanford University group are widely accepted today and constitute a major contribution to cardiac surgery.

Although chemical arrest of the heart generally had been abandoned in the early 1960, Bretschneider (1964) continued to pursue the concept of induced cardioplegia. Using a solution with a low sodium concentration to depolarize the myocardial cells, mannitol to maintain osmolarity, and procaine to stabilize cell membranes, Bretschneider could achieve rapid arrest and preservation of tissue ATP levels for prolonged periods (Bretschneider et al., 1975). Kirsch and associates 1972 developed a similar solution containing magnesium, procaine, and aspartate, which was injected into

the ascending aorta as a bolus to arrest the heart. Like Breischneider, Kirsh believed that elimination of sodium, potassium, and calcium prevented the utilization of ATP for membrane transport and slowed the decay of organic phosphate. Excellent clinical results with both of these solutions were later reported (Sondergoard et al. 1975 and Bleese et al. 1978).

Hearse and associates (1976) took a slightly different approach to chemical cardioplegia. Rather than using an intracellular solution, these investigators developed a predominantly extracellularformula based on sodium as the primary cation (Hearse et al. 1976 a). Emphasis was placed on making the solution as physiologic as possible. Specific additives were included to arrest the heart and maintain membrane integerity. The St-Thomas solution has been used clinically since 1975 (Braimbridge et al., 1977) and has gained wide spread acceptance and application.

In 1973, Gay and Ebert reintroduced the concept of potassium cardioplegia into clinical practice. Using a solution containing 25 mM/L of potassium chloride, they hypothesized that direct myocardial injury with the Melrose solution had been caused by the high potassium concentration and hypertonicity. By reducing the potassium level, rapid diastolic arrest could be achieved and the tolerance of ischemia safely prolonged. The combination of profound myocardial hypothermia and the rapid potassium arrest has

emerged as the most widely used method today. A recent modification, introduced by Buckberg and associates, is the use of cold hyperkalemic blood to induce and maintain cardiac arrest. (Follette et al. 1978, 1978b). The physiologic nature of blood as well as the improved buffering and oxygen transport capacity makes this technique an attractive option.

Blood cardioplegia is rapidly gaining clinical application (Cunningham et al. 1979 and Olim et al. 1981) and excellent results have been recently published.

PHYSIOLOGY OF MYOCARDIUM

The heart comprises four anatomical and physiological systems:

1. The muscle of the heart.
2. The heart valves.
3. The pacemaking and conducting tissue of the heart.
4. The coronary vessels.

1. The muscle of the heart:

The cardiac muscle is striated but the cells have their cell membranes in continuity so that depolarization of the membrane of one cell spreads eventually to all other cells. The atrial muscle is thin and separated from the ventricular muscle by the fibrous atrioventricular ring.

The normal right ventricular wall is about 3 mm in thickness and the left ventricle (LV) 12 mm.

Structure of the myocardium: By electron microscopic studies, the myocardium consists of columns of striated muscle fibres. Each fibre consists of an outer membrane, the sarcolemma, which surrounds numerous striated myofibrils arranged longitudinally. The myofibrils composed of myosin and actin molecules. Each myofibril is interrupted at intervals by dark lines known as Z lines. Two Z lines limit longitudinally the sarcomere which is the functional unit of the myocardium. Mitochondria, inclusions of fat and glycogen are also present in the sarcoplasm close to the myofibrils. The sarcolemma is

invaginated by a system of transverse or T tubules which penetrate the sarcomere at the Z lines. This ensures activation of all myofibrils at the same time. The lumen of these tubules is continuous with the extracellular fluid. Also in the myocardium, where the end of one muscle fibre abuts one another, intercalated discs are found in the membrane of both fibres and always occurring at Z lines. Intercalated discs provide low resistance bridges for the spread of excitation from one fibre to another and they permit cardiac muscles to function as if it were a syncytium.

2. The heart valves:

The aortic and pulmonary valves are simple three cusped (semilunar) which rely on their commissural attachments to the aorta and pulmonary artery for their stability. The tricuspid and the mitral valves, since they are attached to thin fibrous rings "the mitral and tricuspid annuli" require the chordae tendineas and the papillary muscles to maintain their competence. As each ventricle contracts, the distance from the apex to the annulus shortens but contraction of the papillary muscles maintains the valve cusps in the correct position within the ventricular cavity.

3. The pacemaking and conducting tissues of the heart:

The normal pacemaker is the sino-atrial (SA) node, found at the junction of the superior vena cava and right atrium (RA).

Its tissue has the property of rhythmic electrical depolarizaton which spreads to the atrial muscle causing it to depolarize and contract, when freed from the influences of sympathetic and parasympathetic (vagus) nerves, it has an intrinsic rate of 80-90 per min. in adults, up to 130 in infants. The AV node is in the lower part of the RA, there is no specialized conducting tissue connecting SA and AV nodes.

The AV node, although normally paced by the SA node, can also depolarize spotaneously but at a slower rate of 45-50 per min. From the AV node the AV bundle (bundle of His) passes through the fibrous AV ring, into the membranous part of the interventricular septum and divides into right and left branches. The right branch runs below the endocardium of the septum, to the apex of the RV. The left branch gives off a small branch to the interventricular septum and then divides into anterior and posterior (or inferior) divisions. Apart from the AV bundle there is normally no other electrical link between the atria and ventricles. (Asynopsis of cardiology, 1979).

4. The coronary vessels:

The two main coronary arteries are the left and right:

The right coronary artery arises from the anterior aortic sinus, it runs in AV groove and supplies branches to the RA, SA node, most of the anterior surface of the RV and

usually ends as the posterior descending branch supplying the inferior surface of the right and left ventricles, the posterior part of the interventricular septum and the AV node.

The left coronary artery arises from the left posterior aortic cusp and has two main branches, the anterior descending branch and the circumflex branch. The anterior descending runs in the anterior interventricular sulcus and supplies a small part of the RV, the anterior part of the interventricular septum and the anterior and apical parts of the LV. The circumflex branch runs in AV groove giving one or more marginal branches supplying the lateral wall of the LV, a branch to the LA and sometimes the posterior interventricular branch.

The dominant coronary artery is defined as that which gives rise to the branch supplying the AV node and posterior descending arteries at the inferior surface of the heart. The right coronary artery is dominant in 90% of cases. (Fig. 1).

The heart has two venous systems: The majority accompany the corresponding arteries to join the coronary sinus which drain into the RA near the tricuspid valve, and the other veins drain directly into the atria and RV.