NORMOTHERMIC HEART SURGERY CONTINUOUS CORONARY PERFUSION OF THE ELECTROCHEMICALLY ARRESTED HEART AT NORMOTHERMIA

ESSAY
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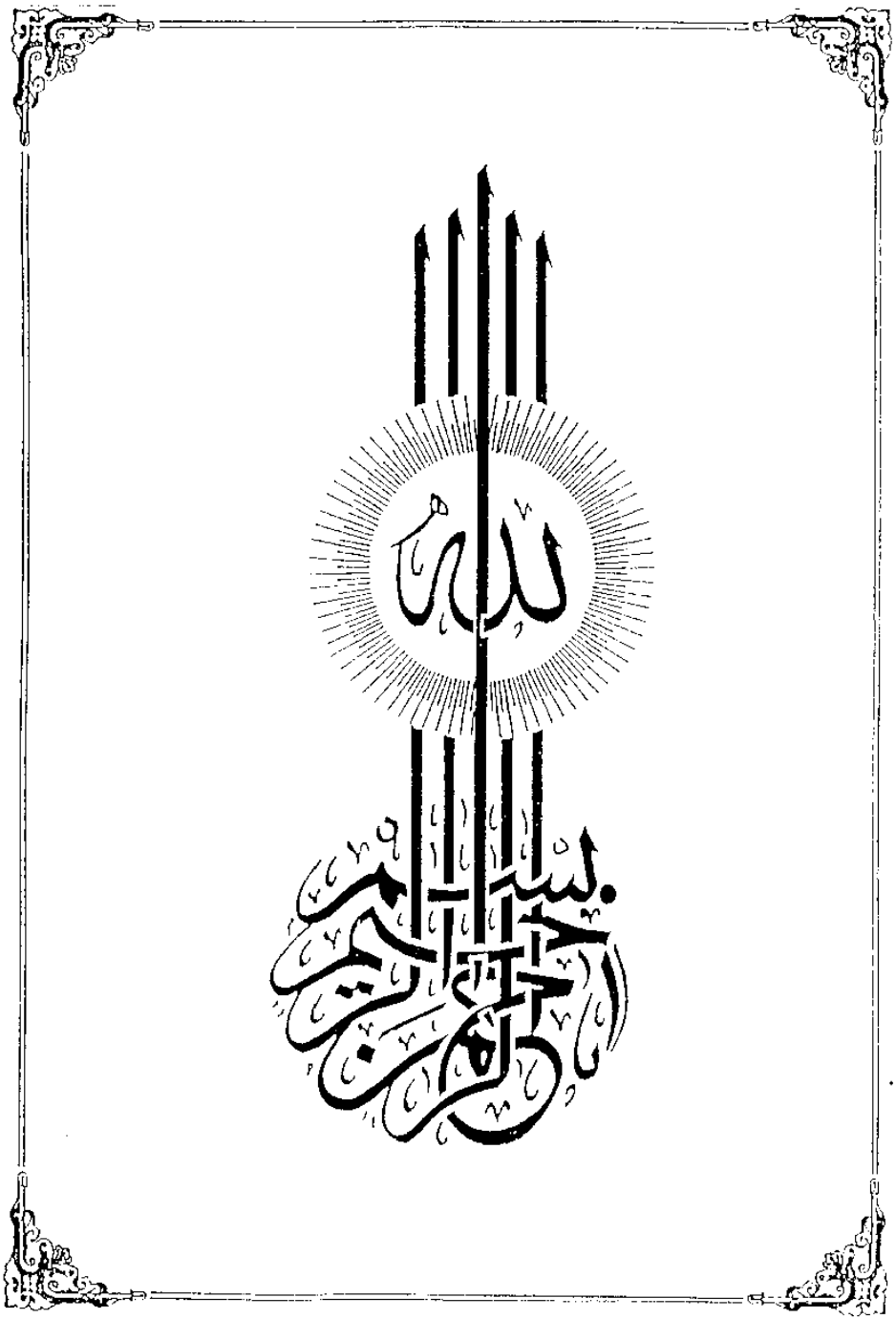

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REVIEW OF CARDIOPLEGIC STRATEGIES DURING CARDIAC OPERATIONS

Review of cardioplegic strategies during cardiac operations

Introduction and historical background

Pharmacologic cardioplegia was not used widely until the past fifteen years because of previous reports of left ventricular damage following cold hypertonic potassium citrate blood as introduced by Melrose et al, in 1955 . Studies by Bretschneider et al., (1975), Kirsch et al., (1972), and Hearse et al., (1976) provided a solid framework for the renewed interest in cardioplegia which has resulted in the intraoperative use of pharmacologic cardioplegia by most surgeons throughout the world. it was showen that the problem with Melrose solution was inappropriate concentration of it's constituents, rather than an inappropriate composition. The new studies fully support the original cardioplegia constituents of Melrose solution and now safe concentration of alkaline, hypertonic potassium citrate, and cold blood is used to stop the heart whenever the aorta is clamped during clinical surgery (Fallette et al., 1978), . Cold cardioplegia solutions are

used almost universally to prevent intraoperative myocardial ischemic damage during aortic clamping as cardiac demands in the arrested hearts is directly proportionate to temperature in absence of rapid arrhythmias, e.g. O_2 demands decrease to third when temp. drop from 31°C to 22°C but in the presence of beats or fibrillation the cardiac demand increases eight to tenfold of energy consequently, despite the low temp. More recently, it was shown that the inclusion of oxygen in the cardioplegia solution expands the therapeutic scope for clinical cardioplegia. It was also shown that these solutions can be delivered warm to allow their use for active resuscitation before ischemia is imposed, and to avoid and reverse ischemic and reperfusion damage before and after aortic uncalmping (Buckberg et al., 1989).

Factors affecting the myocardial energy supply during aortic clamping :

There are two factors affecting myocardial energy supply : 1) oxygenated blood coming from noncoronary collateral blood flow and 2) intrinsic or extrinsic substrate stores.

Noncoronary collateral blood flow is present to a variable degree in all hearts. This flow is highest during arrest, especially in cases of hypertrophy and coronary artery disease. All cardiac surgeons have noted a) blood coming from the coronary ostia during aortic valve replacement ; b) blood coming from the coronary arteriotomy during coronary revascularization when the aorta is clamped and flaccid, and c) the frequent recurrence of electromechanical activity (beating or fibrillating) in pharmacologically arrested hearts signifying that the cardioplegic solution is no longer in contact with the myocardial cells and that sufficient energy stores are available for electromechanical coupling. Conversely, ischemically arrested hearts receive the same noncoronary collateral flow but can never resume electromechanical activity during aortic clamping as they have expanded their high-energy phosphate stores by fibrillating or beating themselves to a halt (Buckberg, 1989).

The second determinant of supply is myocardial glycogen or exogenous glucose. Oxygenated hearts undergo aerobic metabolism but the heart receiving little or no oxygen supply must undergo anaerobic metabolism to generate some energy to maintain cell

membranous viability. Anaerobic glycolysis requires the presence of substrate (i.e. glucose or glycogen), and a metabolic environment (i.e. buffering) to allow anaerobic energy production (Buckberg, 1989).

Factors affecting the myocardial energy demands during aortic clamping :

Myocardial oxygen demands are determined principally by electromechanical activity. The heart that is fibrillating or beating while ischemic has a much higher energy requirement than the heart that is arrested. Fig (1) The second determinant of demand is the wall tension within the myocardium, and the third is the myocardial temperature that governs metabolic rate directly (Buckberg, 1989) (Fig. 2).

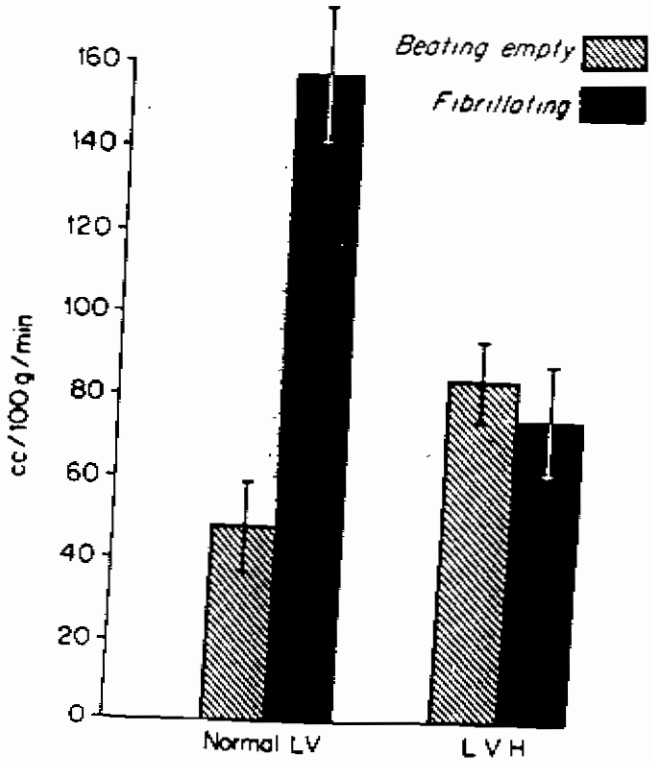

Fig.(1) Left ventricular subendocardial blood flow in beating empty and fibrilating normal and hypertrophied hearts. Note the marked increase in subendocardial blood flow during fibrillation in normal left ventricle, and the failure to augment flow in hypertrophied ventricle that is allowed to fibrillate

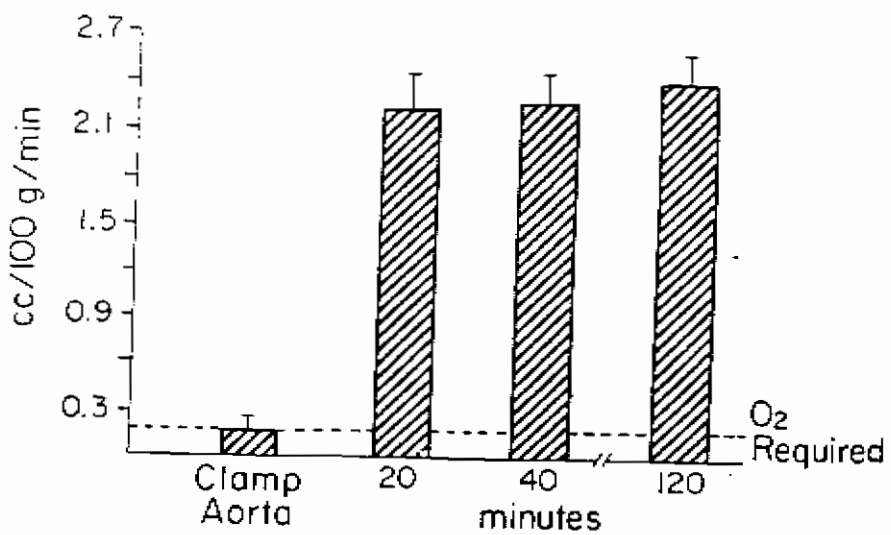

Fig.(2) Myocardial O_2 UPTAKE DURING 4° blood cardioplegia infusions. Note: [1] very low mycardial O_2 demands (O_2 requirement), indicated by hateched line during cardioplegic induction (clamped aorta0; [2] O_2 uptake 10 x in excess of basal demands with cardioplegic replenishments at 20-min intervals.

Effect of ischemia on ionic pumps :

1- Ischemia is known to inhibit Na^+ & K^+ pump leading to increase intracellular Na^+ which draws water leading to cell swelling, loss of K^+ to outside causes further depolarization & ensures arrest .

2- Another effect of ischemia is the failure of Ca++ sequestration which leads to increase free intracellular Ca++ myocardial contracture which can occur during aortic clamping & worsen by reperfusion.

3- A third effect is the loss of Mg^{++} leading to inhibition of respiratory enzymes & this can be the cause of decreased postischemic availability of energy.

Cardioplegia prerequisites AND COMPOSITION

Cardioplegia prerequisites and composition

Cardioplegia prerequisites:

The purpose of pharmacologic cardioplegia is to offset the aforementioned consequences of ischemia by producing an environment in which myocardial energy demands are reduced while energy is produced to meet those demands. The principal prerequisites for the clinical use of a cardioplegia solution are (1) protection of the heart from ischemic damage rather than producing damage by virtue of its composition (Fig. 3); (2) distribution to all mycocardial regions in amounts sufficient to produce the desired effects; (3) provisions for the solution's continued exertion of its protective effect for as long a period of aortic clamping as necessary; and (4) adequate laboratory testing of the solution under conditions which bear some resemblance to those encountered in clinical practice. Failure to adhere to the fourth principle has led to (a) most of the confusion existing today about current cardioplegic techniques; (b) persistent reluctance to accept cardioplegia because of prior experiences with inadequately tested solutions; and (c) the reason why clinical result are neither as perfect nor as bad as these

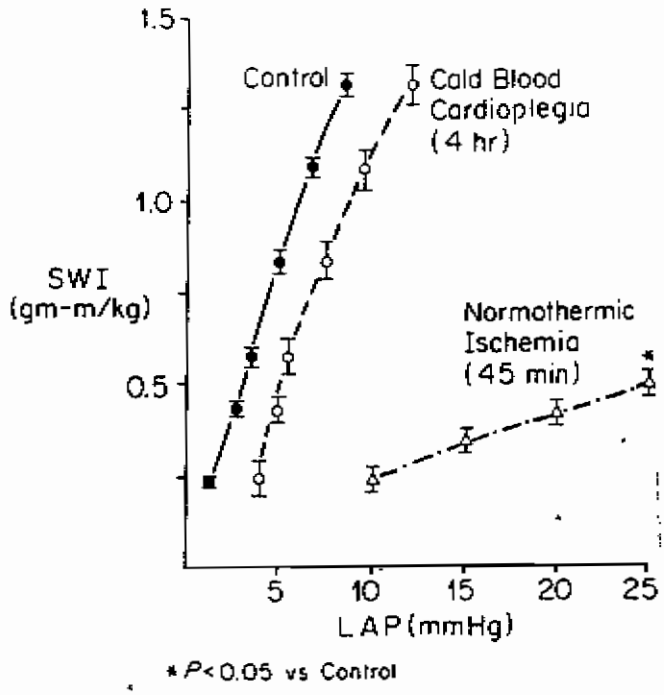

FIG. 3 Postischemic myocardial performance during inscription of left ventricular functioncurves 30 min after unclamping the aorta. Note: [1] marked depression in function following 45 min of normothermic ischemia; and [2] normal function after 4 h of multidose cold blood cradioplegia. LAP, left atrial pressure; SWI, stroke work index.

produced in the laboratory setting (Buckberg et al., 1989).

Cardioplegic composition:

The objectives of chemical cardioplegia are to stop the heart safely, create an environment for continued energy production and counteract deleterious effects of ischemia. Immediate arrest should be produced to lower energy demand and avoid depletion by ischemic electromechanical work. This is especially true with nonoxygenated cardioplegic solutions. Conversely high energy stores may be enhanced when cardioplegia is induced with oxygenated solution and delay in asystole is left problematic. Studies by Peyton et al, in 1982 showed that a substantial reduction in ATP stores had occurred during the brief period of electromechanical activity preceding normal pharmacologic cardioplegia with asanguinous solutions.

First, arrest can be achieved either by use of potassium, magnesium, procaine, or perhaps some hypocalcemic solution. Second, myocardial temperature should be lowered to reduce metabolic rate. This can be achieved usually with perfusion hypothermia with a cold