CARDIOPROTECTIVE ACTION OF THE TERMINAL WARM BLOOD CARDIOPLEGIA IN ACYANOTIC PEDIATRIC CARDIAC SURGERY

Thesis

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Abstract

Background

The surgical management of children with CHD has been advanced significantly in the past decade. Advances in unders tanding of the mechanisms of myocardial injury so, many strategies for myocardial preservation are based on decreasing myocardial metabolic rate by temperature manipulation and arrest of electromechanical activity.

Recent studies suggest the effect of Terminal warm blood cardioplegic in decreasing oxidative stress and free radical release with reperfusion.

Material and Methods

Forty patients undergoing congenital heart surgery disease were randomly divided into 2 groups TWBC and control group and each subdivided into two groups infants and children haemodynamics parameters were measured lactate and arterial blood gases are measured before and after by pass, Also Troponin I.

Results:

There was a significant less increase in lactate level and Troponin level of in children receiving TWBC than children of control group. There was a significant less increase in lactate level in infants receiving TWBC than infants of control group but a significant high increase in Troponin level in infants in TWBC group than infants in control group .

conclusion

TWBC has protective effects in children and adolescents as it does in adults. But still, there are some precautions in using TWBC in infants.

Key words: Terminal warm blood cardioplegia, congenital cardiac surgery , pediatric

List of abbreviations

CHD	**Congenital heart disease**
CNS	**Central nervous system**
CVS	**Cardiovascular system**
ASD	**atrial septal defect**
VSD	**Ventricular septal defect**
AVC	**artrioventricular canal**
PVR	**Pulmonary Vascular resistance**
PBF	**Pulmonary blood flow**
PGE_1	**Prostaglandin E_1**
PDA	**Patent ductus Arteriosus**
RVOT	**Right Ventricular outflow tract**
DORV	**double outlet right Ventricle**
UVH	**Univententricular heart**
RV	**Right Ventricle**
CHF	**Congenital heart failure**
FFA	**Free Fatty Acid**
ATP	**Adenosine Triphosphate**
LV	**Left ventricle**
PAP	**Pulmonary artery Pressure**
LAP	**Left atrial pressure**
SVR	**Systemic Vascular resistance**
CO	**Cardiac output**
HR	**heart rate**
CVP	**Central Venous Pressure**
CAD	**Coronary artery Disease**
CPB	**Cardiopulmonary bypass**

TCA	**Total circulatory arrest**
DHCA	**deep hypothermic circulatory arrest**
CMR	**Cerebral metabolic rate**
MHCPB	**Moderate hypothermic cardio pulmonary bypass**
CVR	**Cerebral vascular resistance**
βARS	**β adrenergic receptors**
ECMO	**Extracarporeal membrane oxygenator**
CPO	**Citrate phosphate dextrose**
KCl	**Potassium chloride**
N.S	**Normal Saline**
Tham	**tromethamine**
IV	**intravenous**
MAC	**minimal alveolar concentration**
ACT	**Activating Clotting time**
R^+	**Potassium**
My	**Magnesium**
NaHcoz	**Borluim bicarbonate**
TWBC	**terminal warm blood cardiopegia**
ICV	**intensive care unit**
BP	**blood pressure**
TWBCP	**terminal warm blood cardioprotection**
TNI	**troponin I**

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Introduction

The surgical management of children with congenital heart disease (CHD) has been advanced significantly in the past decade.

Congenital heart defects in children are repaired at younger ages and with more definitive operative strategies rather than palliations are pursued. Improved outcome of surgery for (CHD) depends on identifying patients vulnerable to organ failure, modifying the preoperative and intraoperative risk factors, and effective interventions for cardiac preservation during surgery for (CHD). ***(Brown et al., 2003).***

Advances in the understanding of the mechanisms of myocardial injury and the composition, delivery and the conduct of myocardial preservation are integral parts of improved outcome.

Strategies for myocardial preservation are based on methods that decrease myocardial metabolic rate by temperature manipulation and arrest of electromechanical activity using various modification of cardioplegic solution ***(Doenst et al., 2003) and (Cecer, et al., 2002).***

Cold blood carioplegia (6-8C^{o}) decreases lactate release and preserves adenine nucleotides ***(ATP and ADP)***, ***(Ascione et al., 2002).***

Blood has become the preferred vehicle for cardioplegia in adults and children ***(Inhnken et al., 1995).*** ***I***t is assumed that oxygen carried by hemoglobin provides additional protection during ischemia.

Blood cardioplegia provides catalase in red blood cells increasing the free radical scavenging capacity and providing buffering capacity by histidine and other blood proteins. ***(Allen et al., 2001)***

In the neonate exposed to hypoxic stress and reoxygenation injury, blood cardioplegia facilitates the repair of injured myocardium, replenishes

depleted energy stores and protects against further damage. ***(Allen et al., 2001)***

Infants undergoing repair of ventricular septal defects were strudied using blood or crystalloid cardioplegia. Blood cardioplegia maintained myocardial adenine nucleotides, decreased lactate production and lowered troponin I by 42% compared to crystalloid cardioplegia. ***(Caputo et al., 2002)***

The use of terminal warm blood cardioplegia before removing the aortic crossclamp after cold blood cardioplegia may decreases the oxidative stress and free radical release with reperfusion. The effect of terminal warm cardioplegia on improvement spontaneous defibrillation rate, increased lactate, decreased troponin I release comparing to cold blood cardioplegia. ***(Yodya et al., 2003)***

Aim of the work

This study will compare the protective effect of cold blood cardioplegia with cold blood cardioplegia followed by terminal warm blood cardioplegia in pediatric patients younger than 4 years for surgical repair of acyanotic congenital defects.

Pathophysiological Classification of Congenital Heart Diseases

A decade ago the preferred approach to the infant with congenital heart diseases was palliation in infancy and repair later in childhood.

Today the tendency is to repair congenital cardiac lesions as early as possible, preferably in the neonatal period. ***(Maureen et al.,1999)***

The important philosophical change in the operative management of neonate with CHD stems from the recognition that:

(1) Early repair is safe and feasible.

(2) Considerable portion of the myocardial, pulmonary and CNS damage related to CHD is specifically due to prolonged exposure to abnormal blood flow patterns, hypoxia and cyanosis.

(3) An increase in morbidity and mortality is associated with long-term medical management of congenital cardiac patients.

Therefore, the goal of early surgical intervention is to restore normal blood flow patterns where possible and reduce long term morbidity. ***(Maureen et al., 1999)***

The marked spectrum of intra-cardiac shunts complicates the anesthetic approach to patients with CHD. Moreover, myocardial changes result from the hemodynamic stress and increased cardiac work incurred by these defects. ***(Rothstein et al., 1999)***

The anesthesiologist must not only understand the anatomy but also its hemodynamic and functional consequences on the CVS. Although an isolated heart defect may be identified, the entire cardiopulmonary system is usually affected ***(Rothstein et al., 1999).***

The developmental biology of the heart is a large field of study. Formation of the heart tube, looping, septation, and resultant systemic and pulmonary circulations is a complex process. Disruption at any point during primary morphogenesis results in the large spectrum of congenital heart defects being treated today. Genetic disorders responsible for these alterations can be classified into 3 types: chromosomal disorders, single-gene disorders, and polygenic disorders. ***(Clark et al.,2004).***

Common congenital heart defects

I- simple left to right shunts (acyanotic lesions with increased pulmonary blood flow)

1- Patent ductus arteriosus.
2- Atrial septal defect (ASD).
3- Venricular septal defects (VSD).
4- Endocardial cushion defect (AV canal).
5- Aorto-pulmonary window.

II- Pure obstructive lesions

1- Pulmonary stenosis.
2- Mitral stenosis.
3- Aortic stenosis.
4- Coarctation of the aorta.

III- Right to left shunts (cyanotic defects with decreased Pulmonary blood flow.

A- Teratology of Fallot

B- Pulmonary Atresia with Ventricular Septal Defect

C- Pulmonary Atresia with Intact Ventricular Septum

D- Tricuspid Atresia

IV- Complex Cyanotic Defects (Mixing Lesion)

A- Double Outlet Right Ventricle (DORV)

B- Univentricular Heart (Single Ventricle)

C- Transposition of the Great Arteries

D- Total Anomalous Pulmonary Venous Drainage

E- Truncus Arteriosus

F- Hypoplastic Left Heart Syndrome

I- simple left to right shunts (acyanotic lesions with increased pulmonary blood flow)

Left to right shunts result from a communication between the right and the left sided circulations. Flow across these defects is usually from the high resistance circuit (left side) to the low resistance circuit (right side). The amount of PBF is determined by the size of the defect and the ratio of the resistances down-stream the defect. ***(Hickey et al, 1989)***

Restrictive shunts are characterized by a significant pressure gradient across the defect. Flow across these small shunts is dictated to the size of the defect.

Non restrictive shunts are larger in size and therefore exhibit minimal pressure gradient across the defect. Flow is determined mainly by the ratio of the pulmonary vascular resistance (PVR) to the systemic vascular resistance (PVR). For example a high PVR would lessen the degree of left to right shunting. Nonrestrictive shunts generally produce excessive PBF, leading to pulmonary vascular congestion. ***(Hickey et al, 1989).***

Surgery is generally indicated for symptoms, pulmonary hypertension, cardiomegaly, left ventricular volume overload or strain or a calculated left to right shunt (the ratio of pulmonary to systemic blood