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Pharmacological improvement of tissue perfusion during CPB in pediatric cardiac surgery

Essay

Submitted for Fulfillment of Master Degree in Anesthesiology

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DEDICATION

I dedicate this work to my family

For their help and assistance

INTRODUCTION

Cardiopulmonary bypass (CPB) is established during various cardiac operations to allow adequate systemic oxygenation and perfusion during the surgical procedure[1].

CPB being a non physiological condition, affects almost every body system secondary to hypoperfusion, even subclinically, and also produces a systemic inflammatory response due to contact of blood with mechanical surfaces. Much attention has been focused in recent years on the adverse effects of CPB, particularly in pediatric patients undergoing surgical repair. Evidence presented has shown significant activation of various inflammatory mediators, including components of the complement cascade, activation of neutrophils and platelets resulting in local and systemic production of circulating proinflammatory cytokines adhesion molecules [2].

Tissue perfusion is at risk during cardiac surgery and also in the immediate postoperative period [3].

The development of predictors of death involves evaluating multiple different cardio-respiratory physiologic indices. This approach is often difficult in infants with congenital heart disease (CHD) because of their small size, which limits invasive monitoring capabilities and reliable diagnostic options. Despite these obstacles, the search for predictors to help direct aggressive interventions in this patient population remains an important goal. It is a well known fact that tissue hypoperfusiojn is associated with lactic acidosis due to anaerobic

metabolism. Measurement of blood lactate levels can hence be used as a marker to assess the adequacy of tissue perfusion [4].

Patients undergoing cardiac surgery are often cooled core temperature of 25-28^{o}C during cardiopulmonary bypass (CPB) and rewarmed before its termination. During the rewarming period, the aim of the anesthesiologist is to achieve a uniform rewarming of the whole body. Despite rewarming core temperature often drops after discontinuation of CPB have been documented by many authors [5].

This drop in core temperature after termination of CPB is referred to as “afterdrop” and is said to be a sign of inadequate total body rewarming on CPB [6].

The surgeon’s decision about when to rewarm is critical; adequate rewarming requires time, but rewarming too soon removes the protective effects of hypothermia. Rapid rewarming often results in large temperature gradients between well-perfused organs and peripheral vasconstricted tissues; subsequent equilibration following separation from CBP decreases core temperature again [7].

Infusion of a vasodilator drug by allowing higher pump flows often speeds the rewarming process and decreases large temperature gradients [8].

The aim of this work is to discuss whether the negative effects of CBP on pediatric patients’ arterial tone and microcirculation can be overcome pharmacologically by different pharmacological agents that can be used during CPB.

Keywords:

Cardiopulmonary bypass (CPB)

Congenital heart disease (CHD)

After drop temperature, rewarming

Tissue perfusion

Monitoring of tissue perfusion

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List of abbreviations

ACTH	adreno-cortico-trophic hormone
AMP	adenosine mono-phosphate
ASA	American society of anesthesiologists
ASD	atrial septal defect
ATP	adenosine tri-phosphate
ATPase	adenosine tri-phosphatase
BIS	bi-spectral index
BSA	body surface area
BV	Blood volume
C3a	complement 3a
Ca^{++}	calcium
CBF	cerebral blood flow
CHD	congenital heart disease
CI	cardiac index
CMR	cerebral metabolic rate
$CMRO_2$	cerebral metabolic rate of oxygen
CNS	central nervous system
CO	cardiac output
CO_2	carbon dioxide
CPB	cardiopulmonary bypass
DHCA	deep hypothermic circulatory arrest
DNA	deoxyribonucleic acid
DNase	deoxyribonuclease
DO2	oxygen delivery
ECG	electrocardiogram
EEG	electroencephalogram
Fa	arterial gas concentration
FA	alveolar gas concentration
Fe^{+++}	ferric ion
FI	inspired gas concentration
FRC	functional residual capacity
H_2 blockers	histamine-2 receptor blockers
Hct	hematocrite
Hgb	hemoglobin.
HR	heart rate
ICP	intra-cranial pressure
ICU	intensive care unit
IL	interleukin
IVC	inferior vena cava

K^+	potassium
LV	left ventricle
MAC	minimum alveolar concentration
MAP	mean arterial pressure
MUF	modified ultra-filtration
Na^+	sodium
Na^+/K^+ ATPase	sodium potassium adenosine tri-phosphatase
NSR	normal sinus rhythm
NTG	nitroglycerine
º C	degree Celsius
O_2	oxygen
OR	operation room
$PaCO_2$	arterial carbon dioxide tension
PACU	post-anesthetic care unit
PaO_2	arterial oxygen tension
PDA	patent ductus arteriosus
PPB	plasma protein binding
PVR	pulmonary vascular resistance
RBC	red blood corpuscles
SNP	sodium nitroprusside
SpO_2	arterial oxygen saturation
SVC	superior vena cava
SVI	stroke volume index
SVR	systemic vascular resistance
TPV	Total priming volume
VSD	ventricular septal defect
WBC	white blood corpuscles
α	alpha

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