ACUTE RIGHT VENTRICULAR FAILURE IN INTENSIVE CARE PATIENTS

Essay

Submitted for partial fulfillment of master degree

In intensive care medicine

By / Mohammed Saad Khalaf

M.B.B.CH

under supervision of

Prof. Dr. Mohammed Reda Abd El Gawad

Professor of Anesthesia and Intensive Care
Faculty of Medicine
Ain Shams University

Dr. Hala Ezzat Ali Eid

Assistant Professor of Anesthesia and Intensive Care
Faculty of Medicine
Ain Shams University

Dr. Tamer Nabil Ibrahim

Lecturer of of Anesthesia and Intensive Care
Faculty of Medicine
Ain Shams University

Ain Shams University

2011

فشل البطين الأيمن الحاد في مرضى الرعاية المركزة

رسالة
توطئة للحصول علي درجة الماجستير
في الرعاية المركزة

مقدمة من

طبيب/ محمد سعد خلف
بكالوريوس الطب و الجراحة

تحت إشراف

أ. د. محمد رضا عبدالجواد

أستاذ التخدير و الرعاية المركزة

كلية الطب
جامعة عين شمس

د. هالة عزت على عيد

أستاذ مساعد التخدير و الرعاية المركزة
كلية الطب
جامعة عين شمس

د. تامر نبيل ابراهيم

مدرس التخدير و الرعاية المركزة
كلية الطب
جامعة عين شمس

جامعة عين شمس
٢٠١٠

Acute right ventricular failure (RVF) is a frequent and serious clinical challenge in the intensive care unit. The presence of acute RVF not only carries substantial morbidity and mortality, but also complicates the use of commonly used treatment strategies in critically ill patients.

Acute RVF is a complex clinical syndrome that can result from any structural or functional cardiac and noncardiac disorder that impairs the ability of the right heart to fill or eject appropriately and is characterized by new onset or gradually or rapidly worsening RV signs and symptoms requiring urgent therapy irrespective of the underlying cause. It is estimated to account for 3% of all acute heart failure admissions and confers worse mortality rates than acutely decompensated LVF.

The most common etiologies of RVF in the ICU are left ventricular (LV) failure, RV ischemia, acute pulmonary embolism (PE), pulmonary hypertension (PH), sepsis, acute lung injury, cardiac tamponade, and post-cardiothoracic surgery states. Arrhythmias and pericardial, congenital, and/or valvular heart disease may also contribute

RV dysfunction occurs directly due to cardiodepressant effects of proinflammatory cytokines, cardiac microthrombi, and ischemia and/or arrhythmias or indirectly due to LV dysfunction, afterload increases from endothelial dysfunction, HPV, pulmonary emboli, and/or pulmonary microthrombi, as well as pre-load decreases (induced or aggravated by capillary leak syndrome). Mechanical ventilation contributes to RV dysfunction by negatively affecting pre-load and/or afterload.

LV dysfunction induces RV dysfunction via afterload increase, and/or displacement of the interventricular septum toward the RV with subsequent impairment of RV filling (known as ventricular

CONTENTS

List of Abbreviations

6 MWT	*6 minute walk time*
AC	*Anticoagulation*
ANA	*Antinuclear antibody test*
Ao	*Aorta*
ARDS	*Acute respiratory distress syndrome*
ARVD	*Arrhythmogenic right heart dysplasia*
AS	*Aortic sac*
ASD	*Atrial septal defect*
AV	*Atrioventricular*
AVV	*Atrioventricular valves*
ACC	*American College of Cardiology*
ACE	*Angiotensin-converting enzyme*
AHA	*American heart association*
A HA/ACC	*American Heart Association/ American College of Cardiology*
ATII	*angiotensin II*
BAS	*Balloon atrial septostomy*
BNP	*B-type natriuretic peptide*
CABG	*Coronayangio bypass graft*
cAMP	*Cyclic Adenosine monophosphate*
CCBs	*Calcium channel blockers*
cGMP	*Cyclic guanosine monophosphate*
CHD	*Congenital heart disease*
CHD/VHD	*Congenital/valvular heart disease;*
CHF	*Congestive heart failure*
CI	*Cardiac index*
CINC1	*cytokine induced neutrophil chemoattractant*
CO	*Cardiac output*
COPD	*Chronic obstructive pulmonary disease*
COX2	*cyclooxygenase 2*
CRT	*cardiac resynchronization therapy*
CT	***Computed tomography***
CTPA	*CT pulmonary angiography*
CTD	*Connective tissue disease*
CTEPH	*Chronic thromboembolic PH*
cTnI	*Cardiac troponin I*
CVP	*Centra venous pressure*

DA	*Ductus arteriosus*
DSA	*Digital subtraction angiography*
ECG	*Electrocardiogram*
ECMO	*Extracorporeal membrane oxygenation*
EDP	*End-diastolic pressure*
ERB	*Endothelin receptor blockers*
ESC	*European Society of Cardiology*
ET-1	*Endothelin-1*
FRC	*Functional residual capacity*
HF	*Heart failure*
HPV	*Hypoxic pulmonary vasoconstriction*
HTN	*Hypertension*
IABP	*Intra-aortic balloon pump*
ICU	*Intensive care unit*
ITP	*Intrathoracic pressure*
iNO	*Inhaled nitric oxide*
Iv	*Intravenous*
IVC	*Inferior vena cava*
LA	*Left atrium*
LAP	*Left atrial pressure*
LCA	*Left coronary artery*
LSCA	*Left subclavian artery*
LV	*Left ventricle*
LVAD	*Left Ventricular assist device*
LVAD/RVAD	*Left/right ventricular assist device*
LVF	*Left ventricular failure*
LVEF	*Left ventricular ejection fraction*
MAP	*Moan arterial pressure*
MI	*Myocardial infarction*
MRI	*Magnetic resonance imaging*
MPAP	*Mean pulmonary arterial pressure*
NO	*Nitric oxid*
NS	*Non-significant*
NYHA	*New york heart association class*
PA	*Pulmonary artery*
PACs	*Pulmonary artery catheters*
PAH	*Pulmonary arterial hypertension*

PAP	*Pulmonary arterial pressur*
PAWP	*P ulmonary artery wedge pressure*
PBW	*Predicted body weight*
PCI	*Percutaneous coronary intervention*
PCWP	*pulmonary artery capillary wedge pressure*
PDE5	*Phosphodiesterase 5*
PE	*Pulmonary embolism*
PEEP	*Positive end-expiratory pressur*
PGH2	*prostaglandin H2*
PH	*Pulmonary hypertension*
PIOPED II	*Prospective Investigation of Pulmonary Embolism Diagnosis II*
PLA	*Phospholipase A2*
PMN	*polymorphonuclear*
P_{plat}	*Plateau pressure*
PPHT	*Porto-pulmonary hypertension*
P_{TA}	*Trans- airway pressure*
PVR	*Pulmonary vascular Resistance*
RA	*Right atrium*
RAP	*Right atrial pressure*
RCA	*Right coronary artery*
RCT	*Randomized placebo controlled trial*
RHF	*Right heart failure*
RPA	*Right main pulmonary artery*
RRT	*Renal replacement therapy*
RSCA	*Right subclavian artery*
RT	*Randomized trial*
RV	*Right ventricle*
RVAD	*Right ventricular assist device*
RVD	*Right ventricular dysplasia*
RVEDV	*Right ventricularend-diastolic volume*
RVEF	*Right ventricular ejection fraction*
RVF	*Right ventricular failure*
RVMI	*Right ventricle myocardial infarction*
RVSP	*Right ventricular systolic pressure*
S1Q3T3	*Large S wave in lead I, a large Q wave in lead III and an inverted T wave in lead III*
SA	*Sinuatrial*
SVR	*Systemic vascular resistance*

TAPSE	*tricuspid annular plane systolic excursion.*
^{99m}Tc-DTPA	*Technetium-99m labelled diethylenetriamine pentaacetate*
TGA	*transposition of the great arteries*
TGF	*tumor growth factor*
TLC	*Total lung capacity*
TOF	*Tetralogy of Fallot*
TP	*Tracheal pressure*
TR	*Tricuspid regurgitation*
TSM	*Trabecula septum marginalis*
TTE	*Trancethiracic echocardiography*
TV	*Tricuspid valve*
VADs	*ventricular assist devices*
VO2	*Maximal oxygen consumption*
V/Q	*Ventilation-perfusion*
V_T	*Tidal volume*

List Of Figures

List Of Figures Cont…….

List Of Tables

FETAL AND NEONATAL DEVELOPMENT OF RIGHT VENTRICLE

A basic understanding of embryology is helpful in the study of cardiovascular and congenital heart disease. The Right Ventricle (RV) and RV outflow tract are derived from the anterior heart field, whereas the left ventricle (LV) and the atrial chambers are derived from the primary heart field ***(Zaffran et al., 2004).***

By the 3rd week of human gestation, passive diffusion of oxygen into the developing embryo becomes insufficient to support metabolism, blood has formed, and the primitive heart tube has begun beating; by the end of the 4th week, active circulation begins. Distinct components of the pulmonary and systemic circulation emerge from folding and twisting of the heart tube between the 3rd and 5th weeks of gestation (fig. 1), under control of a complex signaling network that includes the retinoic acid and neuregulin pathways. Soon after, the RV and pulmonary circulation begin to separate from the LV and systemic circulation by formation of the interventricular septum from the endocardial cushion, and the valves develop. At birth, full septation of the interatrial septum is normally complete, with only the foramen ovale remaining as a potential shunt between the right and left atria ***(Moorman et al., 2003).***

In the embryo and fetus, the RV is the dominant chamber, accounting for about 60% of total cardiac output. Because the embryo receives oxygen and nutrients from the placenta, only 15%-25% of total cardiac output enters the lungs. The remainder of right sided cardiac output is diverted to the systemic circulation via the foramen ovale to the left atrium and via the ductus arteriosus from the pulmonary artery to the

aorta. Between 40%-60% of descending aortic flow enters the placenta via the umbilical artery, and then returns via the umbilical vein to the liver or through the ductus venosus to the inferior vena cava ***(Kiserud et al., 2004).***

At birth, pulmonary vascular resistance (PVR) falls rapidly after expansion and oxygenation of the lungs, and RV cardiac output begins to flow predominantly through the pulmonary artery into the lungs. At that point, rising left atrial pressure seals off the one way "flap valve" of the foramen ovale. At birth, RV pressures still exceed systemic pressures, but these begin to fall over the next few hours to days. Shortly thereafter, the ductus arteriosus, under control of prostaglandin, begins to close, the LV hypertrophies as it takes over the systemic circulation, and the RV atrophies. By 3 weeks of age, pulmonary pressure has normally fallen below systemic pressure, and by adulthood the normal RV is incapable of generating more than 40-60 mmHg acutely ***(Greyson, 2010).***

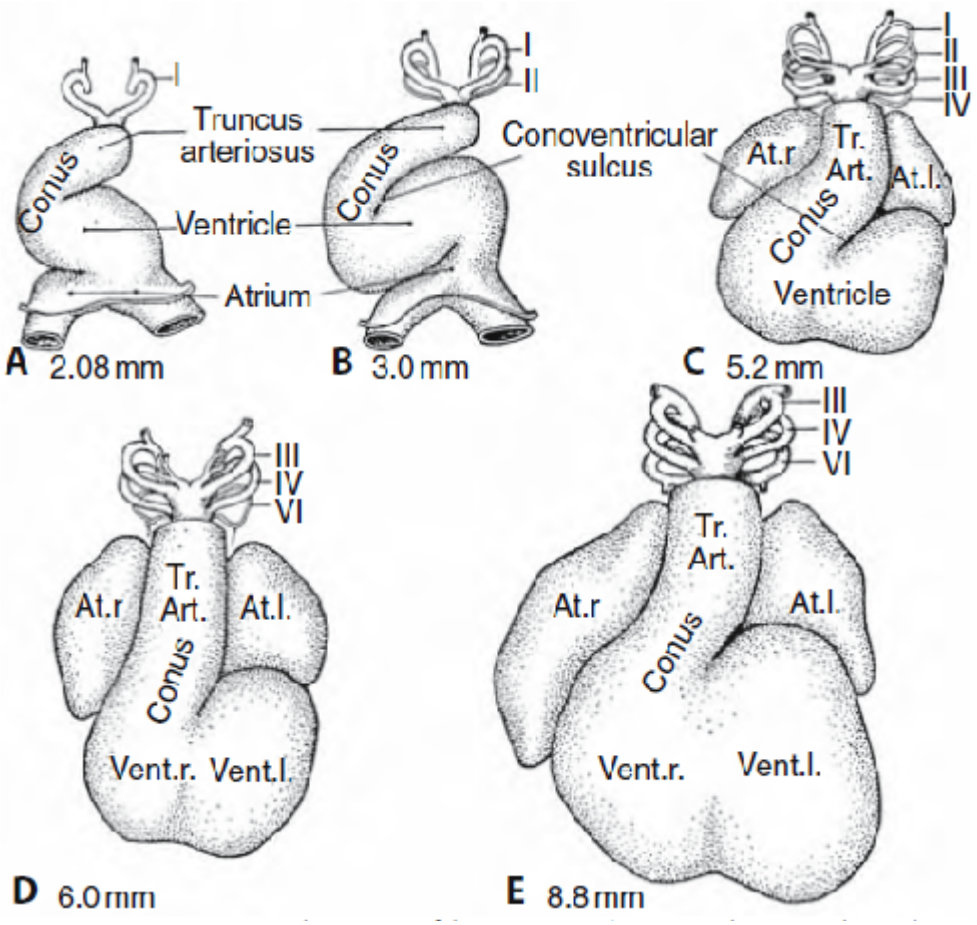


Fig.(1): Ventral views of human embryonic hearts that show bending of cardiac tube and establishment of major anatomic components.
At., atrium; l., left; r., right; Tr. Art., truncus arteriosus.
*(Adapted from **Srivastava, 2006**).*

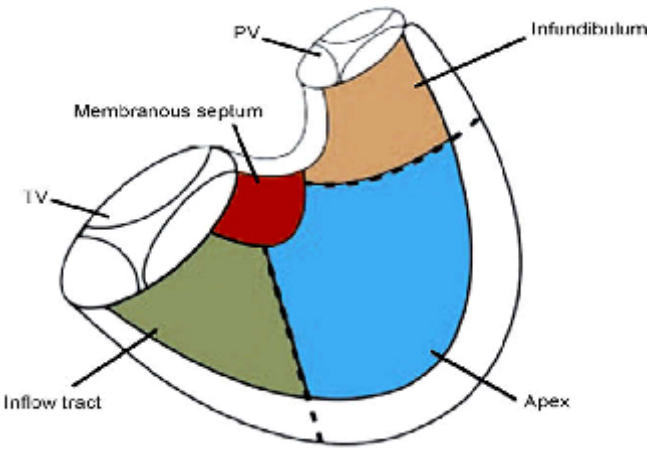
RIGHT VENTRICLE ANATOMY

The anatomy of the RV is both unique and complex. The RV appears triangular when viewed laterally, whereas in cross-section, it appears crescent shaped. Although the RV appears smaller than the LV in the four-chamber view, RV volume is, in fact, larger than the LV volume. Based on magnetic resonance imaging, the normal range of RV end-diastolic volume (RVEDV) is 49–101 mL/m2 (males, 55–105 mL/m2; females, 48–87 mL/m2), whereas the normal range of LV end-diastolic

volume is 44–89 mL/m2 (males,47–92 mL/m2; females, 41–81 mL/m2) ***(Haddad et al., 2009)***.

RV muscle mass is approximately one-sixth that of the LV explained by different loading conditions, as it pumps against approximately one-sixth the resistance of the LV. In childhood, there is a progressive regression of RV hypertrophy as PVR decreases ***(Per Lindqvist et al., 2008)***

The RV can be described in terms of 3 components (fig 2): (1) the inlet, which consists of the tricuspid valve, chordae tendineae, and papillary muscles; (2) the trabeculated apical myocardium; and (3) the infundibulum, or conus, which corresponds to the smooth myocardial outflow region **.** In the study of congenital heart disease (CHD), this division seems to be more practical than the traditional division of the RV into sinus and conus components**.** Additionally, the RV can also be divided into anterior, lateral, and inferior walls, as well as basal, mid, and apical sections ***(Anderson et al., 2005)***.



(Fig.2): Diagram of the right ventricle demonstrating its 3 major chamber components; inflow tract, infundibulum (outflow tract), and apex. *(Adapted from* ***Horton et al., 2009)***