

Pulmonary Artery Pressure Changes in Neonates

Thesis

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قالوا

سببنا انك لا تعلم لنا
إلا ما علمتنا إنك أنت
العليم العظيم

صدق الله العظيم

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List of Contents

Title	Page No.
List of Tables	5
List of Figures	6
List of Abbreviations	9
Introduction	1
Aim of the Work	3
Review of Literature	
▪ The Fetal Circulation	4
▪ Pulmonary Artery Pressure in Neonates	18
▪ Congenital Heart Diseases in Neonates	41
Subjects and Methods	56
Results	67
Discussion	86
Summary	95
Conclusion	100
Recommendations	101
References	102
Arabic Summary	

List of Tables

Table No.	Title	Page No.
Table (1):	Essential components for a normal neonatal transition.....	13
Table (2):	Main causes of neonatal pulmonary hypertension.....	20
Table (3):	Treatment of neonatal pulmonary hypertension.....	36
Table (4):	Inotropic and vasopressor drugs commonly used in pulmonary hypertension.....	38
Table (5):	Demographic characteristics of studied groups.....	68
Table (6):	Maternal data of studied groups.....	69
Table (7):	Clinical data of studied groups.....	70
Table (8):	Echocardiographic findings of studied groups.....	74
Table (9):	Comparison between patients and control groups as regards RVESP values in each week.....	78
Table (10):	Comparison between patients with RVESP >34 mmHg and patients with RVESP ≤ 34mmHg by the end of the 4 th week as regards demographic and clinical data.....	80
Table (11):	Comparison between patients with RVESP >34 mmHg and patients with RVESP ≤ 34 mmHg by the end of the 4 th week as regards Echocardiographic parameters.....	82
Table (12):	RVESP percentage of change in each week.....	83
Table (13):	Comparison between patients with high and low RVESP percentage of change as regards demographic and clinical data.....	84
Table (14):	Comparison between patients with high and low RVESP percentage of change as regards Echocardiographic parameters.....	85

List of Figures

Fig. No.	Title	Page No.
Figure (1):	A simplified scheme of the fetal circulation	5
Figure (2):	Schematic representation of the human placenta	8
Figure (3):	Flow chart of the fetal circulation shunts.....	10
Figure (4):	Pulmonary vascular resistance during transition from fetal to postnatal life	12
Figure (5):	Scheme for neonatal pulmonary circuit showing passage of blood from the right ventricle to the lungs and from the lungs to the left atrium	16
Figure (6):	Endothelium-derived mediators.....	19
Figure (7):	An apical 4-chamber view of the heart. The chambers and valves are labelled	24
Figure (8):	Colour Doppler has been applied. The Tricuspid regurge jet is seen as the blue-yellow streak heading upwards away from the tricuspid valve	24
Figure (9):	Doppler interrogation has been applied to determine the velocity of the Tricuspid regurge jet.....	25
Figure (10):	Radiograph obtained shortly after birth shows bilateral, coarse pulmonary opacities due to meconium aspiration	25
Figure (11):	Pathophysiology of meconium aspiration syndrome (MAS)	27
Figure (12):	Chest X-ray appearances in neonate with MAS.....	28
Figure (13):	Comparison between chest X-ray and LUS for a neonate with MAS.....	30
Figure (14):	Example of neonatal pneumonia presenting as patchy, asymmetric opacities with a small right pleural effusion	33
Figure (15):	Classic respiratory distress syndrome (RDS).....	35
Figure (16):	Classification of congenital heart disease.....	42
Figure (17):	Anatomy of atrial septal defect	44

List of Figures (cont...)

Fig. No.	Title	Page No.
Figure (18):	Chest x- ray showing prominent main pulmonary artery segment and increased pulmonary vascular markings.....	44
Figure (19):	Atrial septal defect is seen as a break in the interatrial septum	45
Figure (20):	Schematic representation of the location of various types of ventricular septal defects (VSDs) from the right ventricular aspect	46
Figure (21):	Chest x-Ray of a neonate with VSD showing cardiomegaly and increased pulmonary vascular markings	47
Figure (22):	Ventricular Septal Defect	48
Figure (23):	Patent ductus arteriosus:.....	49
Figure (24):	Frontal chest radiograph in a neonate with patent ductus arteriosus	51
Figure (25):	Patent Ductus Arteriosus	52
Figure (26):	Atrio Ventricular Septal Defect.....	53
Figure (27):	Chest x- ray of a newborn with AVSD showing non specific cardiomegaly	54
Figure (28):	Echocardiogram of the apical 4-chamber view demonstrating a partial atrioventricular septal defect (AVSD)	55
Figure (29):	Neuromuscular maturity of Ballard score:.....	59
Figure (30):	Physical maturity of Ballard score	60
Figure (31):	Key of Ballard score	60
Figure (32):	Normal blood pressure in neonatal age group	62
Figure (33):	Normal heart rate centiles of neonatal age group	63
Figure (34):	Normal respiratory rate for neonatal age group	63
Figure (35):	Respiratory rate of studied groups.	71
Figure (36):	Respiratory rate of control group.....	71
Figure (37):	Respiratory rate of patients group.	72
Figure (38):	Body surface area of studied groups.	73

List of Figures (cont...)

Fig. No.	Title	Page No.
Figure (39):	Distribution of cardiac lesions causing left to right shunts in studied patients (group I).	75
Figure (40):	Distribution of tricuspid regurge degree in studied patients (group I).	75
Figure (41):	Left ventricular diastolic diameter values of studied patients (group I).	76
Figure (42):	Distribution of interventricular septal thickness values at diastole of studied patients (group I).	76
Figure (43):	Fractional shortening values of studied patients (group I).	77
Figure (44):	Birth weight values of the studied patients (group I).	81

List of Abbreviations

Abb.	Full term
<i>2D.....</i>	<i>2 dimensional</i>
<i>3D.....</i>	<i>3 dimensional</i>
<i>ASD.....</i>	<i>Atrial septal defect</i>
<i>AV.....</i>	<i>Aortic valve</i>
<i>AVSD.....</i>	<i>Atrioventricular septal defect</i>
<i>BSA.....</i>	<i>Body surface area</i>
<i>CHD.....</i>	<i>Congenital heart disease</i>
<i>ECMO.....</i>	<i>Extra corporeal membrane oxygenation</i>
<i>EPSPAP.....</i>	<i>Estimated peak systolic pulmonary artery pressure</i>
<i>HFOV.....</i>	<i>High frequency oscillatory ventilation</i>
<i>HFJV.....</i>	<i>High frequency jet ventilation</i>
<i>IVH.....</i>	<i>Intarventricular hemorrhage</i>
<i>LA.....</i>	<i>Left atrium</i>
<i>LV.....</i>	<i>Left ventricle</i>
<i>MAP.....</i>	<i>Main pulmonary artery</i>
<i>MAS.....</i>	<i>Meconium aspiration syndrome</i>
<i>NO.....</i>	<i>Nitric oxide</i>
<i>PAAT.....</i>	<i>Pulmonary artery acceleration time</i>
<i>PAP.....</i>	<i>Pulmonary artery pressure</i>
<i>PASP.....</i>	<i>Pulmonary artery systolic pressure</i>
<i>PDA.....</i>	<i>Patent ductus arteriosus</i>
<i>PFO.....</i>	<i>Patent foramen oval</i>
<i>PPHN.....</i>	<i>Perisitent pulmonary hypertension</i>
<i>PVR.....</i>	<i>Pulmonary vascular resistance</i>
<i>RA.....</i>	<i>Right atrium</i>
<i>RDS.....</i>	<i>Respiratory distress syndrome</i>
<i>RV.....</i>	<i>Right ventricle</i>
<i>RVEF.....</i>	<i>Right ventricular ejection fraction</i>
<i>RVESP.....</i>	<i>Right ventricular end systolic pressure</i>

List of Abbreviations (cont...)

Abb.	Full term
<i>RVSP</i>	<i>Right ventricular systolic pressure</i>
<i>SSRIs</i>	<i>Selective serotonin reuptake inhibitors</i>
<i>SVC</i>	<i>Superior vena cava</i>
<i>TR</i>	<i>Tricuspid regurgitation</i>
<i>TRJV</i>	<i>Tricuspid regurgitation jet velocity</i>
<i>TRV max</i>	<i>Tricuspid regurge maximum velocity</i>
<i>TT</i>	<i>Transient tachypnea</i>
<i>TTE</i>	<i>Transthorathic echocardiography</i>
<i>VSD</i>	<i>Ventricular septal defect</i>

INTRODUCTION

Directly after birth, major changes in the respiratory and cardiovascular systems are required for postnatal survival (*Van Vonderen et al., 2014*).

A dramatic reduction in pulmonary arterial pressure and resistance occurs with an increase in oxygen tension and blood flow (*Gao and Raj, 2010*).

Studies suggest a gradual drop in pulmonary artery systolic pressure (PASP) with increasing age, but the wide-range of variation and the small sample sizes cannot permit adequate appraisal of the rate of decline. Patent ductus arteriosus (PDA) and tricuspid regurgitation (TR) are frequently found in neonates, and echocardiographic measurements of the Doppler flow velocity across the PDA and the tricuspid regurgitant jet velocity have been shown to yield accurate values for PASP (*Hu et al., 2014*).

Half of congenital heart defects permit blood flow from a left sided chamber or blood vessel through a communication to the right side of the heart. These conditions are called left-to-right shunts. The patients are acyanotic but have increased pulmonary blood flow. The four most common left-to-right shunts are ventricular septal defect (VSD), patent ductus arteriosus (PDA), atrial septal defect (ASD), and atrio-ventricular septal defect (AVSD) (*Johnson and Moller, 2014*).

Echocardiography has revolutionized the practice of pediatric cardiology. The addition of Doppler and color flow mapping also gives physiological information about flow and pressures and enables the pediatric cardiologist to refer patients for surgical treatment without cardiac catheterization, especially in neonate and infants (*Awasthy and Radhakrishnan, 2013*).

AIM OF THE WORK

The aim of this study is to evaluate changes in pulmonary artery pressure during the neonatal period in full term newborns with congenital heart diseases with left to right shunts (VSD, ASD, PDA, AVSD) in comparison to healthy newborns.

Chapter 1

THE FETAL CIRCULATION

Because the fetal lungs are not inflated, and the fetus obtains oxygen and carbon dioxide gas exchange through the placenta, there must be some level of mixing and redirection of blood flow involving the ductus arteriosus, the ductus venosus, foramen ovale, the umbilical vessels and placenta (*Hines, 2013*).

Inspite that blood flow in the placental vasculature is governed by the same physiological forces of shear, pressure and resistance as in other organs, it is also uniquely specialized on the maternal and fetal sides. At the materno-fetal interface, the independent utero-placental and umbilico-placental circulations must coordinate sufficiently to supply the fetus with the nutrients and substrates it needs to grow and develop (*Thornburg and Samantha, 2013*).

The fetal heart differs from the extra-uterine heart in both structure and function. The fetal heart represents two circulations, which effectively run in parallel, with two ‘shunts’ connecting them, i.e. the ductus arteriosus and foramen ovale. In ex-utero life, the two circulations are referred to as pulmonary and systemic; however, in the fetus this distinction is somewhat euphemistic, and it may be more accurate to discuss right and left circulations (*Godfrey et al., 2012*).

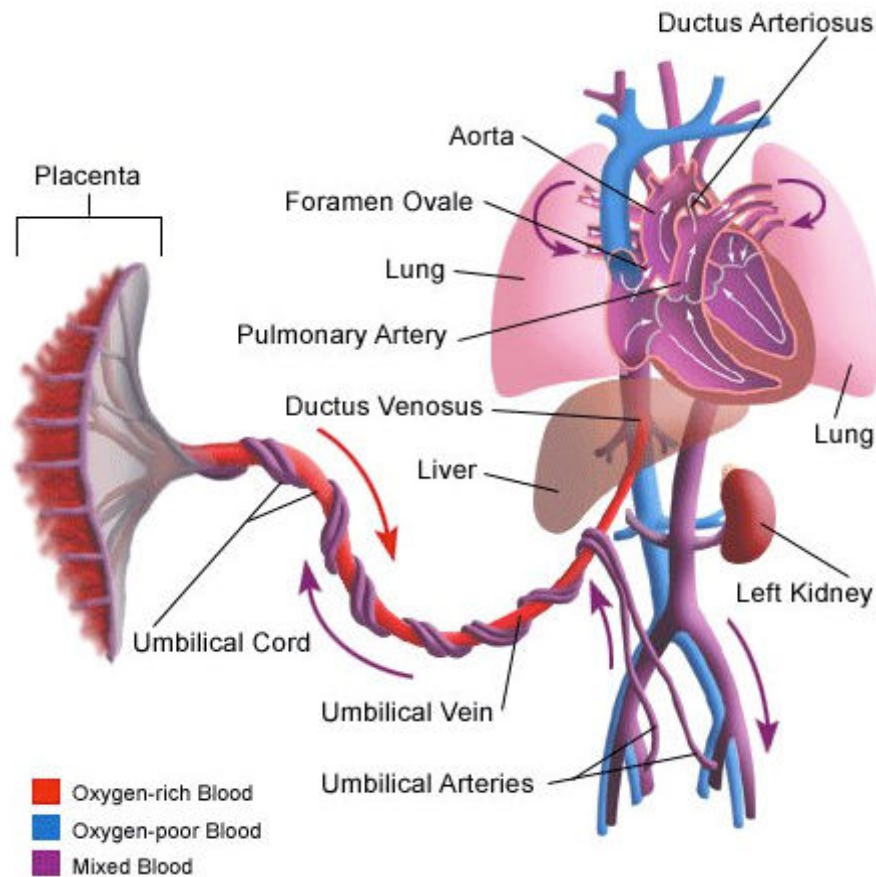


Figure (1): A simplified scheme of the fetal circulation. The shading indicates the oxygen saturation of the blood:

<http://study.com/academy/lesson/fetal-blood-circulation-diagram-lesson-quiz.html>).

Embryology:

By the third week of 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