# Prevalence of Undiagnosed Anemia in Patients with Congenital Heart Disease

#### **Thesis**

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by

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# **List of Abbreviations**

Abb.	Mean
AS	Aortic Stenosis.
<b>ASD</b>	Atrial Septal Defect.
AVSD	AtrioVentricular Septal Defect.
BAV	Bicuspid Aortic Valve.
BF	Breast Feeding.
CBC	Complete Blood Count.
CCT	Cardiac Computed Tomography.
CHD	Congenital Heart Disease.
COA	Coarctation Of Aorta.
CT	Computed Tomography.
<b>DMT</b>	Divalent Metal Transporter.
<b>D</b> NA	Deoxyribonucleic acid.
<b>DORV</b>	Double Outlet Right Ventricle.
FF	Formula Fed.
<b>FISH</b>	Fluorescence in situ hybridization.
<b>HCT</b>	Hematocrit Value.
Hgb	Hemoglobin.
HLHS	Hypoplastic Left Heart Syndrome.
<i>IDA</i>	Iron Deficiency Anemia.
LV	Left Ventricle.
LVOT	Left Ventricular Outflow Tract.
<i>MCHC</i>	Mean Corpuscular Hemoglobin Concentration.
<i>MCV</i>	Mean Corpuscular Volume.
<b>MDCT</b>	Multi Detector Computed Tomography.

Abb.	Mean
MRA	Magnetic Resonance Angiography.
MRI	Magnetic resonance Imaging.
<b>P</b> A	Pulmonary Artery.
<b>PD</b> A	Persistent Ductus Arteriosus.
PGE1	Prostaglandin E1.
PS	Pulmonary Stenosis.
<i>RA</i>	Right Atrium.
<b>RBCs</b>	Red Blood Cells.
RDW	Red cell distribution Width.
RHC	Reticulocyte Hemoglobin Content.
RV	Right Ventricle.
RVOT	Right Ventricular Outflow Tract
SV	Single Ventricle.
SVD	Single Ventricle Defect.
TA	Tricuspid Atresia.
<b>TAPVR</b>	Total Anomalous Pulmonary Venus Return.
TEE	Trans Eosophageal Echo.
TGA	Transposition of Great Arteries.
TIBC	Total Iron Binding Capacity.
<i>TOF</i>	Tetralogy Of Fallot.
<b>TOGV</b>	Transposition Of Great Vessels.
TTE	Trans Thoracic Echo.
<b>VSD</b>	Ventricular Septal Defect.



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#### INTRODUCTION

Congenital heart disease (CHD) is a complex problem which can affect the patient, the family, the society and the country. CHD describes a wide range of conditions resulting from abnormalities of the heart structure and/or function that is present from birth. The severities of conditions are classified as complex, moderate, and simple (Marelli et al., 2007). CHD is the most common birth defect, with approximately 9 in 1,000 new born being affected (Van der Linde et al., 2011). Although CHD is the most common survivable birth defect, the etiology of most CHD remains unclear (Yuan et al., 2013). It represents one of the most common causes of death of children at different ages starting from abortion of the fetus, stillbirth, death of the patient at birth, during infancy, during early childhood, during late childhood and may at adulthood (Ferencz et al., 2008).

Anemia is a widespread public health problem associated with an increased risk of morbidity and mortality, especially in pregnant women and young children. Among the numerous nutritional factors. both (such as vitamin and mineral and non-nutritional deficiencies) (such as infection hemoglobinopathies), that contribute to the onset of anemia, iron deficiency and malaria play an important role. Given the role of iron in oxygen transport and the low levels of available iron in the diets of a large proportion of the global population, it is assumed that iron deficiency is one of the biggest contributing factors to the global burden of anemia. Iron deficiency is considered one of the ten leading global risk factors in terms of its attributable disease burden (WHO, 2002).

Childhood anaemia poses a major public health issue leading to an increased risk of child mortality, as well as the negative consequences of iron-deficiency anaemia on cognitive and physical development. At its special session on children in 2003, the United Nations General Assembly set a goal to reduce the prevalence of anaemia by one third by 2010 (WHO, 2004). Despite this, the incidence of anaemia in children aged under 5 between 1990 and 2010 has actually increased (Kassebaum et al., 2013).

Anemia is defined as a hemoglobin level of less than the 5th percentile for age. Causes vary by age. Most children with anemia are asymptomatic, and the condition is detected on screening laboratory evaluation. Screening is recommended only for high-risk children. Anemia is classified as microcytic, normocytic, or macrocytic, based on the mean corpuscular volume. Mild microcytic anemia may be treated presumptively with oral iron therapy in children six to 36 months of age who

have risk factors for iron deficiency anemia. If the anemia is severe or is unresponsive to iron therapy, the patient should be evaluated for gastrointestinal blood loss. Other tests used in the evaluation of microcytic anemia include serum iron studies, lead levels, and hemoglobin electrophoresis. Normocytic anemia may be caused by chronic disease, hemolysis, or bone marrow disorders. Workup of normocytic anemia is based on bone marrow function as determined by the reticulocyte count. If the reticulocyte count is elevated, the patient should be evaluated for blood loss or hemolysis. A low reticulocyte count suggests aplasia or a bone marrow disorder. Common tests used in the evaluation of macrocytic anemias include vitamin B<sub>12</sub> and folate levels, and thyroid function testing. A peripheral smear can provide additional information in patients with anemia of any morphology (*Irwin and Kirchner, 2001*).

Anemia should not be considered a diagnosis, but a finding that warrants further investigation (Olhs and Christensen, 2004). In children, it is usually caused by decreased RBC production or increased RBC turnover (Oski et al., 2004). Iron deficiency commonly causes decreased RBC production. Risk factors include prematurity, poor diet, consumption of more than 24 oz of cow's milk per day, and chronic blood loss. Other causes of decreased RBC production include inflammation from chronic

infection or other inflammatory conditions, renal failure, medication use, viral illnesses, and bone marrow disorders (Wright et al., 2003 and Oski et al., 2003).

# Aim of the work

To detect the prevalence of undiagnosed anemia and its type among children with congenital heart diseases.

#### **CONGENITAL HEART DISEASES**

Congenital heart disease (CHD) is the most common cause of major congenital anomalies, representing a major global health problem. Twenty-eight percent of all major congenital anomalies consist of heart defects (*Dolk et al., 2011*). Reported birth prevalence of CHD varies widely among studies worldwide. The estimate of 8 per 1,000 live births is generally accepted as the best approximation (*Bernier et al., 2010*).

Congenital heart defect (CHD) may be defined as an anatomic malformation of the heart or great vessels which occurs during intrauterine development, irrespective of the age at presentation. Congenital heart defects may be classified into acyanotic and cyanotic depending upon whether the patients clinically exhibit Cyanosis (*Rao PS*, 2005).

Malnutrition is a constant phenomenon among children with congenital heart disease, irrespective of the nature of the cardiac defect and the presence or absence of cyanosis. The principal factor behind this is the inadequate biological use of the nutrients available, due to the elevated energy expenditure caused by the clinical conditions inherent to cardiac alterations (Sarni et al., 2005).

For this reason, children born with heart disease are considered part of a nutritional high-risk group (*Staebel*, 2000). In

this group, there is loss of body mass which affects the patient as a whole, including the heart and respiratory muscles, this compromises the myocardial and ventilatory functions, healing capacity and immunological competency, with the consequent increase in risk of infection (*Oba*, 2000).

Patients with increased pulmonary blood flow and pulmonary hypertension are more prone to develop malnutrition and growth retardation. Associated hypoxia in patient with cyanosis and pulmonary hypertension further increases the problem (*Vaidyanathan et al., 2008*).

#### **\* RISK FACTORS AND ETIOLOGY OF CHD**

In the majority of instances when a baby is born with CHD, there is no known reason for the heart to have formed improperly. Some types of CHD can be related to an abnormality of the chromosomes (5-6%), single gene defect (3-5%), environmental factors (2%), maternal diseases, maternal medications, maternal use of alcohol, cocaine or other recreational drugs during pregnancy (*Correa-Villansenor et al.*, 2008).

# A. Family History, Maternal and Environment risk factor:

• The risk of CHD increases when either parent has a CHD or when another sibling was born with CHD (Becker et al.,