

**EVALUATION OF THE
CARDIOVASCULAR
SYSTEM IN CHILDREN WITH
ANEMIA OF VARIOUS CAUSES**

**THESIS
SUBMITTED FOR PARTIAL FULFILMENT OF
MASTER DEGREE
IN CARDIOLOGY**

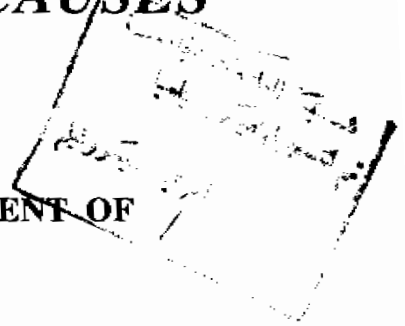
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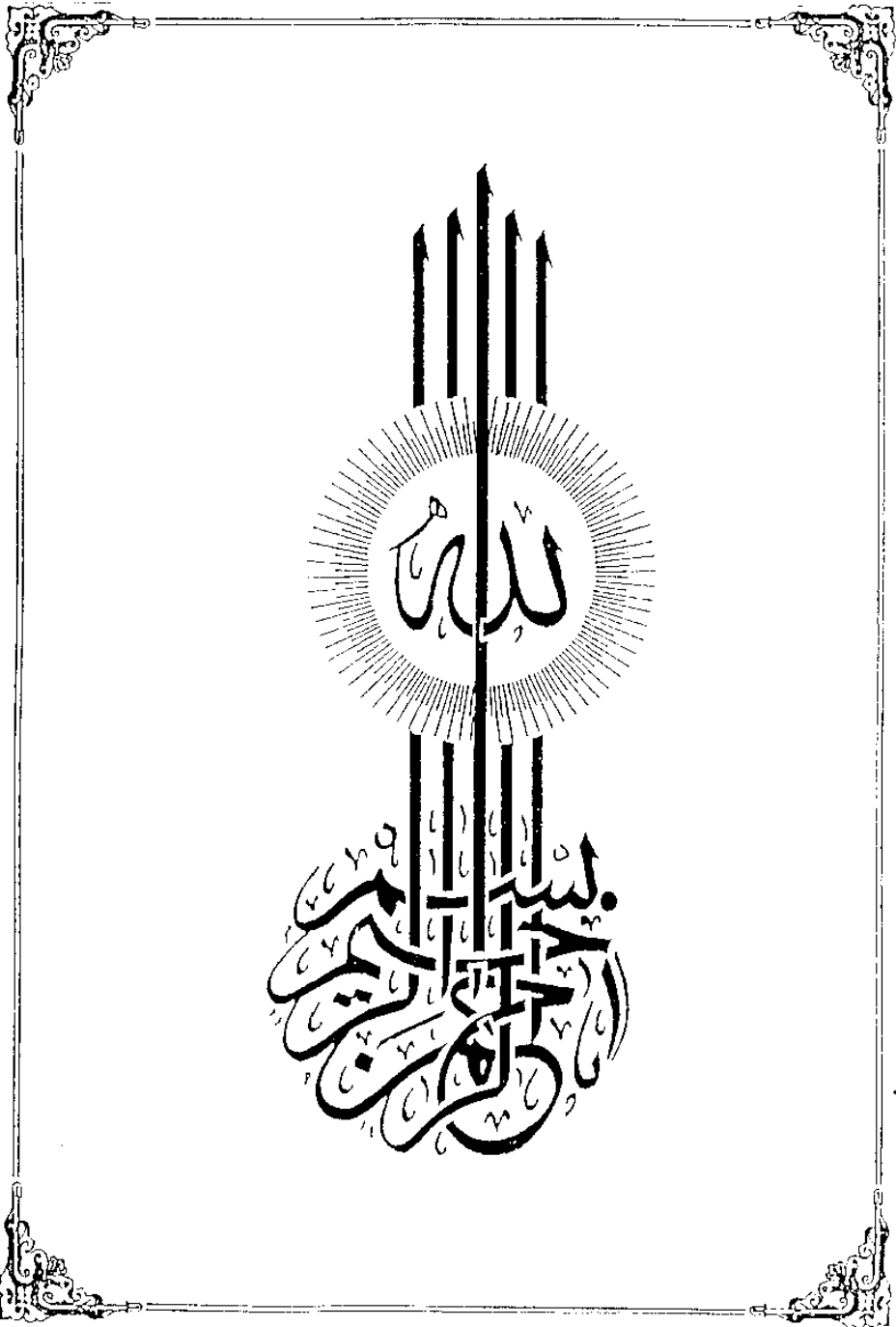
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***TO
MY PARENTS***



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Abbreviations

G.6.P.D	=	Glucose - 6 - phosphate dehydrogenase.
Hb	=	Hemoglobin .
M.C.V	=	Mean corpuscular volume .
M.C.H.C	=	Mean corpuscular hemoglobin concentration .
mg	=	Milligram .
mRNA	=	Massenger ribonuclic acid .
dl	=	Deciliter .
Y	=	Year .
hs	=	Hours .
ng	=	Nanogran .
ug	=	Microgram .
FEP	=	Free erythrocyte protoporphyrin .
Kg	=	Kilogram .
m	=	Month .
α	=	Alpha .
β	=	Beta .
δ	=	Delta .
γ	=	Gamma .
Hb _F	=	Fetal gemoglobin .
Hb _A	=	Adult hemoglobin .

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SGOT	=	Serum glutamic oxaloacetic transaminase .
pg	=	Picogram .
MCH	=	Mean corpuscular hemoglobin .
HPFH	=	Hereditary persistence of fetal hemoglobin .
HbS	=	Sickled hemoglobin .
L.V	=	Left ventricle .
S.C.A	=	Sickle cell anemia .
DNA	=	Deoxyribonucleic acid .
ATP	=	Adenosine triphosphate .
S.C.D	=	Sickle cell disease .
CO	=	Carbon monoxide .
Ig	=	Immunoglobulin .
S.C.T	=	Sickle cell trait .
CMV	=	Cytomegalo virus .
HLA	=	Human leucocytic antigen .
HS	=	Hereditary spherocytosis .
Na-K-ATPase	=	Sodium-Potassium adenosine triphosphatase .
C.O.P.	=	Cardiac output .
R.B.C.	=	Red blood cell .
S ₃	=	Third heart sound .
S ₄	=	Fourth heart sound .
TM	=	Thalassemia major .
E.C.G.	=	Electrocardiogram .

- III -

ES	=	End systole .
Pes	=	End systolic pressure .
E.D.D.	=	End diastolic diameter .
E.S.D.	=	End systolic diameter .
L.V.E.D.D.	=	Left ventricular end diastolic diameter .
L.V.E.S.D.	=	Left ventricular end systolic diameter .
L.V.E.T.	=	Left ventricular ejection time .
V.C.F.	=	Velocity of circumfrential fiber shortening .
cm	=	Centimeter .
E.velocity	=	Peak early filling velocity .
A.velocity	=	Peak atrial filling velocity .
I.D.A.	=	Iron deficiency anemia .
St. Dev.	=	Standard deviation .
Sec.	=	Second .
L.V.E.F.	=	Left ventricular ejection fraction .
P.E.T.	=	Pre ejection time .
E.T.	=	Ejection time .

INTRODUCTION

Chronic anemia of any cause leads to hemodynaemic changes to maintain tissue perfusion . Several mechanisms may compensate for reduced tissue oxygenation .

These compensatory mechanisms result in increased left ventricular preload and volume overload resulting in chamber dilatation . This dilatation results in thinning of the chamber walls as they are stretched causing left ventricular free wall thickness to be initially decreased . The increase in L.V. preload results in augmentation of fiber shortening by the Frank Starling mechanism .

The compensatory mechanisms result also in decreased systemic vascular resistance and impedance to left ventricular emptying . A low afterload will augment left ventricular ejection .

Left ventricular mass increases early when chamber dilation alone could not compensate for the increasing demands placed on the myocardium . These compensatory mechanisms can help maintaining normal L.V. ejection fraction despite progressive deterioration of contractile reserve .

Eventually the contractile abnormalities become so severe that even these optimally functioning compensatory mechanisms can not maintain normal left ventricular shortening characteristics and covert left ventricular dysfunctions become overt left ventricular decompensation .

There are no specific E.C.G. changes although E.C.Gs are commonly abnormal . In general it appears that the E.C.G. correlated poorly with clinical state and is of limited help in determining :

- a - The state of the myocardium .
- b - The presence of ventricular hypertrophy, so echocardiogr is better than E.C.G. because it can :
 - Evaluate the systolic and diastolic functions of the heart .
 - Evaluate the cardiac dimensions during systole and diastole .
 - It is useful in following up the patient and estimating the effect of treatment of anemia on the cardiac functions and dimensions .

AIM OF THE WORK

The purpose of this study is to evaluate the cardiac performance and cardiac abnormalities in a group of children with different types of anemia using E.C.G. and echocardiography .

This study is undertaken in an attempt to document the effects of anemia on the heart to enable more accurate prediction of the course and outcome of these patients .

Review of literature

Classification of anemia

Introduction

Anemia means a deficiency of red blood cells or it is a state of chronic oxygen lack due to quantitative and qualitative reduction of red blood cells below normal (**Guyton, 1991**) .

Classification of anemia

I- Anemia in the neonatal period :

Roloff and lukens, 1979 classified neonatal anemias as follow :

- Anemia at birth

- a - Erythroblastosis fetalis .
- b - Blood loss .

- **Anemia after first day of life :**

- a - Blood loss .
- b - Anemia with infection .
- c - Hereditary spherocytosis .
- d - Congenital nonspherocytic anemias .

- **Late anemia (1 - 3 months) :**

- a - Physiologic anemia .
- b - Vitamin E - deficiency .
- c - Folate deficiency .
- d - Congenital hypoplastic anemia .

According to **For Far and Arneil, 1984** causes of anemia in new born infants are the following :