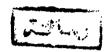
### Incidence of Heart diseases in school children



**Thesis** 

Submitted for partial fulfilment of the master degree in Internal Medicine

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### Dedication

To my wife and children Sara, Asmaa, Ahmed and Anas for their love.

### Acknowledgment

I would like to express my sincere feelings of gratitude to Professor Dr. Mohamed Abd El-Rahman Mousa. Professor of Internal Medicine, Faculty of Medicine, Ain Shams University, for his valuable advice, true encouragement, and continous guidance in the progress of this work.

I wish to express my deepest gratitude and appreciation to Professor Dr. Medhat S.El.Shafey. Professor of Internal Medicine, Faculty of Medicine, Ain Shams University for his unlimited cooperation.

My sincere thanks are to Dr. Mohsen Abd el Hamed Assistant Professor of Public Health. Ain Shams University, for his remarkably meticulous marshalling of the manuscripts.

I would like also to thank Dr. Ahmed Mabrouk Cardiologist from the Cardiac National institute for his valuable remarks and for his help throughout the practical part of this work.

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# **INTRODUCTION**

### Introduction

Rheumatic fever and Rheumatic heart diseases still form a major health problem in developing countries including Egypt, where many socioeconomic factors predispose to streptococcal affection which precedes their occurrence.

In Egypt, a prevalence rate of 4.3 per 2000 amoung school children in Cairo was reported (El Zawahry et al., 1970).

Abdin et al. (1972) concluded that fatalities due to Rheumatic fever and Rheumatic heart disease in children over 4 years were 2-3 times as much as fatalities due to all other chronic diseases of children of the same age.

A field study was conned out by strasser in 1973 under WHO supervision an Egyptian school children of age group 6-12 years. They forward the prevalence of Rheumatic heart disease to be 10 per 1000.

They reported a high incidence of group A Streptococcal infection affecting abort 30% of school children per year.

Discisso and taranta, in 1980, stated that the disease in Egypt has the highest reported mortality rate in the world of Rheumatic heart disease 27.s/10,000 and one of the highest for a cute rheumatic fever.

# AIM OF THE WORK

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This study was carried out with the aim of determining the incidence of rheumatic heart disease among school children in different Educational zones in Cairo governorate in 1987-1988.

Statistical comparison between the incidence of Rheumatic heart disease in different educational stages in the last 30 years.

Statistical comparison between the incidence of Rheumatic heart disease in different countries of the world.

To evaluate the health care provided to school children suffering from Rheumatic fever and Rheumatic heart disease by the school health services.

### (1) Introduction

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### **REVIEW OF LITERATURE**

### (2) AETIOLOGY OF RHEUMATIC FEVER

Lancefield (1962) differentiated streptococci into distinct groups and demonstrated that group A was the chief cause of streptococcal infection in man.

Rheumatic fever is considered a sequelae of a preceding infection with group A beta haemolytic streptococci.

The salient features of the evidence linking group A streptococcal infections aetiologically to acute rheumatic fever are; epidemiologic, bacteriologic, immunologic, experimental and clinical (Henny, 1972).

### 1) Epidemiological Evidence:

Epidemics of exudative pharyngitis due to group A streptococci in closed populations are followed by an approximate 3% attack rate of acute rheumatic fever (Rammel et al., 1952). Group A streptococci and rheumatic fever are similar in their geographic, climatic and seasonal incidence (Paul, 1952).

### 2) Immunologic Evidence:

Rheumatic fever does not occur without a streptococcal antibody response. Group A streptococci produces a number of extracellular antigens such as streptolysin O, streptokinase, hyaluronidase, desoxyribonuclease and nicotinamide adenine dinucleotidase; each of these antigens evokes specific antibodies in human sera but not all patients respond

uniformly to each antigen for example 15-20% of patients with acute rheumatic fever do not have elevated antistreptolysin antibodies. Evidence of recent streptococcal infection is obtained in 95% (stollerman, 1956).

### 3) Experimental Evidence:

The heart disease produced in experimental animals originally by repeated infections of streptococci (Murfy, 1960), and more recently by antiheart antigen (Halbert et al., 1973) resembles closely that of rheumatic fever.

The long persistence of undegradable streptococcal cell wall mucopeptide antigen in the heart may give rise to rheumatic fever like lesions in mice and rabbits and the joints in rats (Ohanian et al., 1969).

### 4) Clinical Evidence:

The association of acute rheumatic fever with sore throat and the concept a latent period were recognized during the 19th century particularly by Haygarth and Haig Brown (Taranata, 1978).

### 5) Prophylactic Evidence:

Both primary and secondary attacks of rheumatic fever can be prevented by promote treatment or prevention of streptococcal infections with antimicrobial therapy (Henny, 1972).

### (3) PATHOGENESIS OF RHEUMATIC FEVER

The striking reduction of the first attack of rheumatic fever when streptococcal infections are treated with penicillin and of secondary attacks in patients who are receiving continuous antimicrobial prophylaxis provides additional support for the role of streptococcal infection in the pathogenesis for both initial and recurrent attacks (Wannamaker, 1972).

The possibility that viruses may be involved in the aetiology of acute rheumatic fever and rheumatic heart disease has been championed by Burch and his colleagues (1967). Burch (1967) hypothesized that latent viruses may be present which are activated by streptococcal infection at the time of an acute attack of rheumatic fever. While there are conditions caused by viruses acting synergistically with bacteria, the epidemiology of rheumatic fever does not appear to be related to outbreaks of viral infections. There is a good evidence that viruses especially of the picorna virus group (Coxsachie and Echo) can produce myocarditis and pericarditis in humans.

Coxsakial B viral antigen has been demonstrated in the valves by immunofluorescent techniques indication that the endocardium may be involved (Burch et al., 1967).