URAEMIC PERICARDITIS IN RELATION TO BLOOD UREA LEVEL AND ECG CHANGES

THESIS

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BY

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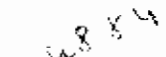

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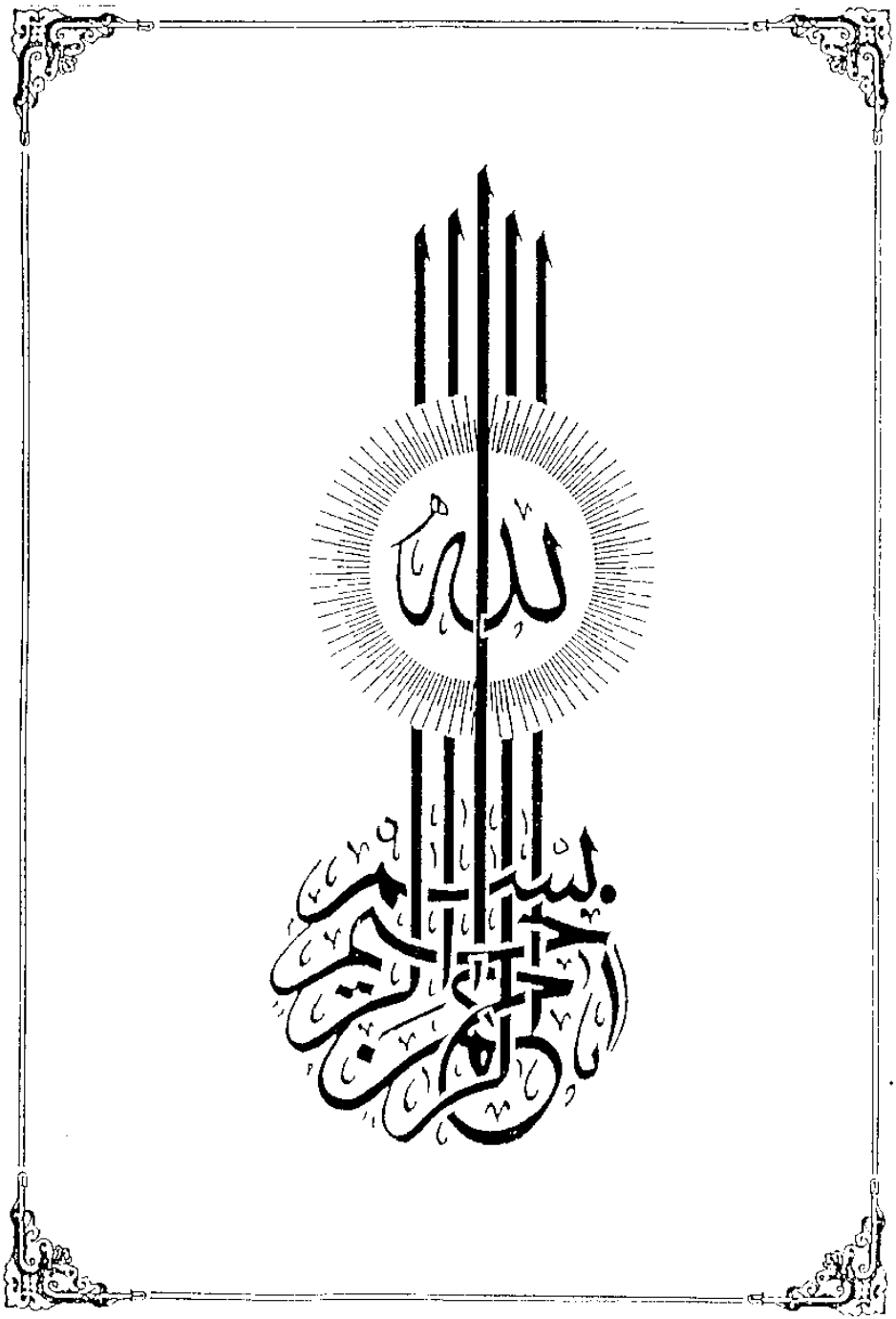

CONTENTS

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Introduction
&
AIM OF WORK

INTRODUCTION

The pericardium is a conical fibroserous sac which envelops the heart and roots of the great vessels. Although it is not essential to life, as it may be resected in treatment of some pericardial diseases, yet the normal pericardium has many physiological functions (Holt, 1970).

Diseases of the pericardium are numerous although their incidence among heart diseases is lower than other non-pericardial diseases. Pericardial inflammation or pericarditis is one of the major diseases which affect the pericardium. It can occur as a separate disease or as a part of a systemic disorder. Renal failure is one of those systemic diseases which could be commonly complicated by pericarditis (Hager, 1965).

Uraemic pericarditis was described more than 100 years ago. Bright (1836), described eight cases of pericarditis occurring in his classic study of 100 uraemic patients. Since then, pericarditis has been recognized as a frequent complication of acute and chronic uraemia (Comty et al., 1971). It has been reported in up to 50% of autopsied patients with chronic renal failure, and has been diagnosed clinically in nearly 20% of patients with acute renal failure. Anyhow,

pericarditis commonly accompanied terminal uraemia and the presence of this complication was previously considered to mean that death was impending (Wacker and Merrill, 1954). But, intermittent dialysis and renal transplantation have however, changed the prognosis of terminal uraemia and of the complications that accompany it, so that uraemic pericarditis can now be considered accessible to treatment (Symons and Wrong, 1964; Beaudry et al., 1966; Hager, 1965; Alfrey et al., 1968; Bailey et al., 1968).

AIM OF THE WORK

A clear relation between blood urea level and existence of a pericardial rub is not well known. The blood urea level might have a close relation to pericarditis in a general way or in the particular patient. The same argument may hold true for the ECG changes in uraemic pericarditis. The aim of this work is to find the relation between the blood urea level and ECG changes and uraemic pericarditis.

A study on 15 patients undergoing regular dialysis at Ain Shams Kidney Dialysis Centre was made as a trial to clear this point.

Review of Literature

THE NORMAL PERICARDIUM

ANATOMY:

The pericardium is normally found in vertebrates from the lower forms to man and has been studied in a wide variety of mammals (Parker and Haswell, 1940).

It is placed in the middle mediastinum behind the body of the sternum and the cartilages of the ribs from the second to the sixth; and in front of the thoracic vertebrae from the fifth to the eighth. It is conical in shape and consists of a serous membrane and a fibrous sac; the serous membrane covers the outside of the heart (visceral pericardium), extending a short distance beyond the atria and ventricles on the great vessels and lines the inside of the fibrous sac (parietal pericardium) (Gardner et al., 1969). The heart is thus, considered to invaginate the wall of the serous sac from above and behind and practically obliterates its cavity, the space being a potential one (Warwick, 1973). The pericardial cavity is that space between the visceral and parietal pericardium, and contains a varying amount of fluid. The tough, thick fibrous pericardium forms a flask-like sac, the neck of which is closed by its attachment to the great vessels; it is attached ventrally to the manubrium of the sternum and the xiphoid process,

dorsally to the ventral column, and caudally to the central tendon of the diaphragm (Gray, 1959). In different species and in different animals within a given species, the wall thickness and the attachments to the diaphragm and sternum vary in magnitude. In man, the fibrous layer is well developed and the attachments to the diaphragm are extensive (Elias and Boyd, 1960). The fibrous sac is connected to the upper and lower ends of the sternum by weak sternopericardial ligaments. The fibrous pericardium provides slippery surfaces for the heart to beat inside and the lungs to move outside within a densely adherent parietal pleura on which the breathing lungs glide (Last, 1978).

HISTOLOGY:

The serous layer is smooth and glistening, covered with flattened irregular polygonal mesothelial cells (Kluge and Hoving, 1967); and attached to the fibrous layer by delicate connective tissue rich in elastic fibres (Bremer and Weatherford, 1944). A superficial, middle and deep layer of collagenous fibres interlaced with elastic fibres can be distinguished in the fibrous layer (Elias and Boyd, 1960). In man, the geometric configuration of the collagenous fibres in the parietal pericardium varies with age; the fibres which are particularly straight in the foetus,

become wavy after birth, reaching the largest wave amplitude in young adults and becoming nearly straight again in old age (Hort and Braün, 1962). The waves in the collagenous fibres are closely related to the development of the elastic fibres. Elastic fibres in the foetus are few, but they increase in number and thickness from birth to adult life. The total quantity of elastic fibres in older persons is approximately the same or somewhat greater than that in younger adults.These findings suggest that the pericardium of young adults is more elastic than that of the elderly (Holt, 1970).

BLOOD, LYMPH AND NERVE SUPPLY:

In man, the pericardium receives its arterial blood supply from the pericardial branches of the internal thoracic and musculophrenic arteries and the aorta. The venous drainage of the pericardium is mainly through the tributaries of the azygos vein which ends in the superior vena cava just before it pierces the pericardium. Some of the pericardial veins end directly into the superior vena cava or into the brachio-cephalic veins through the internal thoracic vein and/or the left superior intercostal vein. The presence of pacinian corpuscles in the pericardium has been reported. Lymphatics are found in both the endocardium and epicardium, and they drain into nodes at the base of the heart (Warwick, 1973).

The parasympathetic nerve supply is through nerve fibres from the vagus, the left recurrent laryngeal and the oesophageal plexus which penetrate the pericardium to supply all its layers. The sympathetic nerve supply is from the first dorsal ganglion, the stellate ganglion and the aortic (cardiac and diaphragmatic plexuses) (Elias and Boyd, 1960). The phrenic nerve supplies the fibrous and parietal layers only, but the visceral layer on the heart surface, is insensitive. The pain of pericarditis originates in the parietal layer only, and is transmitted by the phrenic nerve, while pain of ischaemic heart (angina pectoris) originates in the muscle or vessels and is transmitted by the sympathetic nerve fibres (Last, 1978). Afferent impulses from several types of receptors in the pericardium, connective tissue, adventitia, and heart wall pass by peripheral sensory axons through sympathetic plexus and through the lower two cervical and upper four thoracic sympathetic ganglia to the thoracic dorsal ganglia, where the cell bodies of the neurones are located. Lastly, the impulses ascend through the spinothalamic tract to terminate in the postero-ventral nucleus of the thalamus (White, 1957).

PERICARDIAL FLUID:

The visceral and parietal layers are separated by various amounts of pericardial fluid, the volume of which in an average adult man is in a range between 20-60 ml. The fluid is said to be an ultrafiltrate of blood serum and, containing between 1.7 and 3.5 percent protein, with a variable albumin to globulin ratio and a colloidal osmotic pressure approximately 25 percent that of blood serum. Protein appears to diffuse into the pericardial space from the blood stream (Maurer et al., 1940; Soliman et al., 1966).

FUNCTION:

During the past 300 years, widely different views have been expressed on the functional significance of the pericardium. These range from the statement that it "serves no vital function" (Moore and Shumacker, 1953), to the view that "from clinical and experimental studies it is, then, of interest that the clinical picture of cardiac failure is to a great extent the result of pericardial constriction or limitation.." (Duomarco et al., 1962). Since human beings with congenital absence of the pericardium and pericardiectomized animals survive indefinitely and appear to carry out their gross physiologic functions

in a more or less normal manner, it is not surprising that the pericardium has been thought to have little functional significance (Moore and Shumacker, 1953). However, this conclusion is unwarranted until the appropriate studies have been made for the relation of atrial and ventricular dynamics to the presence or absence of the pericardium in health and disease. Any restrictive function that the pericardium might serve, would be expected to affect ventricular and atrial volumes; since techniques for estimating these volumes have been in use for many years, it is not surprising that views differ as to the functional significance of the pericardium (Holt, 1970).

Functions that various investigators have attributed to the pericardium include prevention of overdilatation of the heart (Holt, 1967; Duomarco, 1962), protection of the heart from infection and from adhesions to surrounding tissues, maintenance of the heart in a fixed geometric position within the chest (Holt, 1967), regulation of the interrelations between the stroke volumes of the 2 ventricles, and prevention of right ventricular regurgitation when ventricular diastolic pressures are increased (Berglund et al., 1955).