

# CARDIAC EMERGENCIES DURING HAEMODIALYSIS

THESIS

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by

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... TO MY WIFE

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**I N T R O D U C T I O N**  
**A N D**  
**A I M O F T H E W O R K**

## INTRODUCTION

During the past two decades, cardiovascular mortality has decreased by 20-30% (Stern, 1979). In contrast, during the same period of time, no such reduction in mortality has been reported in the dialysis population, where the death rate from heart disease remained constant at about 30% (Rostand et al., 1979). Several demographic factors may account for this sustained high cardiac mortality and for the even larger number of patients who develop non-fatal heart disease. In addition, patients with end-stage renal failure have a constellation of disease- and treatment-related alterations in haemodynamics, metabolism and cardiac oxygenation, myocardial contractility and left ventricular performance. The relationships of these factors to the development of myocardial ischaemia are well established (Rostand and Rutsky, 1984).

Age is one of the most important demographic factors influencing cardiac morbidity and mortality (about 46% of the patients on regular haemodialysis are above 55 years) (Evans et al., 1981). Thus, progressively increasing age of the dialysis population may contribute significantly to the sustained cardiac risk seen in these patients (Rostand and Rutsky, 1984).

In addition, the race and sex of dialysis patients may also contribute to their cardiac risk. It was found that white dialysis patients had a greater risk for manifesting ischaemic heart disease symptoms than black patients, and that women undergoing



dialysis may have had an accelerated appearance of symptomatic ischaemic heart disease (Rostand et al., 1982). Hutchinson et al. (1982) found that the presence of pre-existing heart disease in patients entering dialysis increased their death risk, and therefore considered it to be an important prognostic variable.

The nature of the disease which causes renal failure may also be important. Accelerated hypertension, which accounts for the high percentage of all renal failure conditions in the United States, is known to accelerate the atherosclerotic process and produce increased left ventricular work, hypertrophy and ultimately failure (Burton et al., 1979). Diabetic nephropathy, which is an important cause of chronic renal failure, may be associated with congestive cardiomyopathy, with no evidence of coronary artery disease or hypertension. Thus, the increased number of diabetic patients may represent another contribution to the sustained mortality from heart disease seen in end-stage renal disease programs. Amyloidosis, Fabry's disease, scleroderma and other vasculitis, though infrequent as a cause of renal failure, can affect myocardial function, cardiac conduction and coronary perfusion. Nephrotic syndrome is usually accompanied by increased total lipid, thus representing a risk for accelerated atherosclerosis. However, interstitial nephritis was found to be associated with a greater incidence of ischaemic heart disease (Rostand and Rutsky, 1984).

While the above mentioned diseases may affect the heart directly, the ultimate development of the uraemic state leads to multiple metabolic and haemodynamic alterations, which may have serious cardiovascular consequences. Hypertension occurring either primarily or secondarily to progressive deteriorating kidney function is clearly the best identified risk factor for both dialysis associated ischaemic heart disease and for the development of altered left ventricular performance. Secondary hyperparathyroidism may accelerate the rate of peripheral vascular and coronary calcification, calcification of the conducting system and myocardial calcification. Anaemia associated with chronic renal failure may contribute to a high output state which may increase cardiac work. Electrolyte abnormalities, such as hyperkalaemia, may contribute to abnormal myocardial conduction and contractility. The haemodialysis procedure itself may produce significant haemodynamic stress on the heart, as it is frequently associated with hypoxaemia, hypotension and arrhythmias, thus producing symptoms of myocardial ischaemia (Rostand and Rutsky, 1984).

## **AIM OF THE WORK**

A study of cardiac emergencies encountered during haemodialysis searching for their incidence, nature and type, effective treatment and incidence of mortality.

# **REVIEW OF LITERATURE**

## **HAEMODIALYSIS**

The primary purpose of haemodialysis treatment is to remove toxins which are normally eliminated from the body by the kidney, and to maintain mineral and water homeostasis. Haemodialysis interposes a semi-permeable membrane between a flowing stream of blood and an appropriate rinsing solution. Conventional haemodialysis includes solute transport and ultrafiltration.

### **ULTRAFILTRATION:**

Solvent, which in the case of clinical dialysis is water, can be transported from one side of the semipermeable membrane to the other side. Clinically, it is necessary to remove fluids from the blood to the dialysate. This is called ultrafiltration, and is accomplished by creating a pressure gradient across the membrane. In haemodialysis ultrafiltration is done hydrostatically, either by creating a negative pressure on the dialysate, or by increasing the positive pressure in the blood compartment (Van Stone, 1983).

**SOLUTE TRANSPORT:**

Solutes cross the semipermeable membrane by two basic mechanisms: The first mechanism - diffuse transport - where the molecular motion of the solute results in its movement across the membrane. This type of transport depends on membrane permeability, surface area, molecular weight and concentration gradient. The second mechanism is conductive transport, which is the movement of solute across a membrane caused by the passage of solvent containing the solute, which in turn depends on the rate of solvent transport, sieving properties of the membrane and concentration of the solute in the solvent (Van Stone, 1983).

**SEQUENTIAL ULTRAFILTRATION AND DIALYSIS:**

This technique is a modification of conventional haemodialysis that requires the divorcing and sequencing of fluid and solute removal, these two processes being simultaneous during haemodialysis. Isolated ultrafiltration, i.e. fluid removal without solute removal, may precede or follow the phase of pure solute removal. During isolated ultrafiltration, dialysis fluid does not flow through the dialyzer, but the required hydrostatic pressure gradient is established across the dialyzer membrane for the desired rate of fluid removal (Van Stone, 1983).

**HAEMOFILTRATION:**

Haemofiltration removes large quantities of fluid from the blood stream through a semipermeable membrane with the majority of the fluid volume being replaced back into the blood in the form of infusion solution. The main advantage of this technique over conventional haemodialysis is that it provides more cardiovascular stability and control of hypertension (Van Stone, 1983).

**HAEMODIAFILTRATION:**

This technique includes haemofiltration and haemodialysis performed simultaneously. This offers a combination of the advantages of haemodialysis (high clearance of small molecular weight substances) and those of haemofiltration (fluid removal without circulatory impairment) (Wizeman et al., 1978).