

(i)

THE PRINCIPLES AND PRACTICE OF HAEMODIALYSIS

THE EXPERIENCE AT THE KENYATTA NATIONAL HOSPITAL

BY

DR. SETH OUMAH MC'LIGEYO, M.B., CH.B. (NAIROBI), 1981

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DECLARATION

This thesis is my original work and has not been presented for a degree in any other University.

Signed 
DR. S. O. MC'LIGEYO, M.B., Ch.B. (NAIROBI)

This thesis has been submitted for the degree of Master of Medicine with my approval as a University supervisor.

Signed..... 
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DIALYSIS: A POEM

I

The Travenol salesman wears glasses and a dark suit:

"Do you
Take this machine
In sickness and in health
Till death do you part?"
I do.

II

Reclining
On the nausea-green hospital chair:
Below me children, playing in the street;
Above me old men, dying of coronaries
I am
The final essence of the technological age,
Flesh conjoined with plastic, vessels with
steel, coils, alarms, twisted tubing
turning scarlet
Deep within the machine dark blood
Mixing with fluid, cellophane separated,
Plugged in and turned on.
Dear God
Purify me.

By

MICHAEL SAPPERSTEIN (a patient on a programme of
maintenance haemodialysis) (150).

SUMMARY

Haemodialysis as a form of renal failure treatment was started at Kenyatta National Hospital in December 1972 with the opening of the Intensive Care Unit. Between that time and June 1982, the machines used were part of a commodity aid from Japan given to the Kenya Government. In that period haemodialysis was carried out irregularly, a total of 57 patients were dialysed. Of these 25 were females and 32 were males, 31 had acute renal failure, 18 had chronic renal failure and 8 had acute on chronic renal failure. The age range was 9 years to 50 years, with a mean age of 29.6 ± 3.6 ($\pm 2SD$), the commonest cause of acute renal failure was sepsis with disseminated intravascular coagulation and haemorrhage (32.3%) in various clinical settings. Chronic glomerulonephritis was the commonest cause of chronic renal failure (46.2%). At this time haemodialysis was geared mainly towards patients with acute renal failure who were expected to regain their renal function. 61.3% of the patients with acute renal failure started on haemodialysis died. The commonest cause of death was continuing uraemia (31.6%) due to irregular and inadequate dialysis.

The patients started on dialysis in this period were severely ill and had adverse biochemical parameters.

In the one year period, beginning August 1984 in the renal Unit at Kenyatta National Hospital, intermittent haemodialysis has been carried out regularly. This followed the installation of Gambro type haemodialysis machines in the Unit early in 1984. The machines were part of a commodity aid from West Germany given to the Kenya Government and the renal Unit where haemodialysis is being carried out was built by the Kenya Government. In the one year period, 42 patients have been haemodialysed. These include 20 with acute renal failure and 22 with chronic renal failure, deliberately started on maintenance haemodialysis. Haemorrhage in various clinical settings was the commonest cause of acute renal failure (50%). The commonest cause of chronic renal failure was chronic glomerulonephritis (59.1%). 30% of the patients with acute renal failure started on haemodialysis died. None of the deaths was related to the dialysis procedure. In the patients with chronic renal failure started on haemodialysis, 72.7% died. The commonest cause of death was interdialytic pulmonary oedema (31.3%). The concept of "prophylactic" dialysis was used in the patients with acute renal failure in the prospective period. Thus they were in a better clinical state and had less adverse biochemical parameters than the patients dialysed between December 1972 and June 1982. The outcome of the dialytic treatment was favourable.

The management of 22 patients with chronic renal failure on maintenance haemodialysis for periods of upto 36 weeks (170 patient-weeks) is described.

The clinical and biochemical states of the patients at the start of treatment were poor. The initial control of blood pressure and biochemical response to dialytic treatment were encouraging. The commonest recurring problem in the short term has been related to vascular access.

I N T R O D U C T I O N

I. (i) HISTORICAL BACKGROUND

Although modern active haemodialysis is almost 30 years old, dialysis was known to the Roman civilisation 18 centuries ago (1). At the time hot water bath was used to produce profuse sweating through the skin (semi-permeable membrane) making the patients lose their toxins into the water. This method apparently existed into the 1850s. Thomas Graham, a chemist, however, in 1854 defined dialysis as we know it today. He caused movements of solutes through semi-permeable membranes made from ox bladders (2).

The possibility of long-term haemodialysis may have been envisioned by Abel (3) in 1913 when he first described in vivo dialysis and speculated on its eventual use in the treatment of "numerous toxic states in which the eliminating organs of the body, more especially the kidneys, are incapable of removing at an adequate rate the natural or unnatural substances whose accumulation is detrimental to life" (4). The artificial kidney he used was composed of numerous parallel flow cellulose tubes. Hirudin obtained from the heads of leeches was used as an anti-coagulant. Thirty odd years of sporadic experimentation and limited technical advance passed before Abel's Prophecy was fulfilled in the primary sense of "tiding over a crisis" of acute renal failure. By successfully treating a number of temporarily oliguric patients with a rotating drum of his own design, Kolff (5-7) laid the foundation for the use of the artificial kidney in clinical medicine. In the middle 1940s Alwall (8) had developed the vertical drum kidneys, however Kolff's invention remained the most widely used and most effective of all artificial kidneys until 1960 when Kiil of Norway developed the flat plate parallel

flow artificial kidney.

General acceptance of the use of artificial kidney in the management of acute renal failure was slow. Dialytic procedures in their early days were inordinately complex (9-11). Results were variable, and convincing evidence that dialysis could reduce mortality rates was difficult to gather (12). For these reasons, together with an unfortunate polarisation of attitudes among nephrologists, more technical refinement and vastly expanded clinical experience was required to establish this procedures as an essential component of modern treatment for acute renal failure.

A realisation of the full potential of haemodialysis in the management of permanent irreversible renal failure was similarly slow to develop and for somewhat similar reasons. Early attempts to prolong life in chronic renal failure through repeated dialysis were uniformly disappointing. Alwall et al (13) succeeded in keeping several irreversibly oliguric patients for as long as 116 days. The intervals between dialytic treatment in these instances, however, were relatively long. In each case uraemia followed a sawtooth pattern of sweeping amplitude, and the patient's condition deteriorated with malnutrition and other complications, particularly infection, occurring. Although the therapeutic investment in these cases was truly heroic, motivation derived more from a hope, albeit dwindling with time, that adequate renal function would eventually return, than from a conviction that rehabilitation could be achieved through indefinite continuation of treatment.

A significant step towards more effective use of the artificial kidney in severe, prolonged renal failure was made by Teschan and associates (14). They espoused the concept of "Prophylactic" daily dialysis to prevent, rather than correct the symptoms of profound uremia. They also devised a technique for avoiding frequent cutdowns through the use of indwelling arterial and venous catheters flushed hourly with heparin. No deliberate attempt was made, however, to exploit this approach as indefinite substitution therapy for recognised, irreversible chronic renal failure. Accordingly, during the 1950s, haemodialysis was deemed of limited value in the treatment of chronic renal failure (15, 16). It was conceded to be helpful in treating the exacerbations of chronic mild uraemia and in preparing uraemic patients for surgery.

Despite the development of the hardware for effective dialysis, a major stumbling block to the long term application of dialysis remained. Each time a patient had to be dialysed a surgical cutdown was necessary and glass tubing had to be fired, shaped and inserted into the vessel. Attached rubber tubing completed the means for keeping circulatory access open between dialysis. It was not until 1960 that Scribner and his associates (17, 31) reported the first deliberate attempt to restore useful life through repeated dialysis of a patient with obviously terminal, irreversible renal disease. One critical factor which facilitated success in this pioneering effort was the development of an ingeniously simple method for gaining access to the patient's circulation repeatedly and conveniently without repeated cutdowns (18). The permanently indwelling Teflon-Silastic cannulas, con-

nected together when not in use to form an arterio-venous shunt, ushered in a new era for the artificial kidney.

As a further development in vascular access Cimino and associates (19, 37) developed an internal fistula - the artificial arteriovenous fistula as we know it today (20) by operative manouvre whereby a suitable artery and vein in the limbs were connected together in order to make the vessels appear prominent and hence easily accessible for frequent punctures for haemodialysis.

The development and improvement of haemodialysis also provoked thoughts on how helpful a patient could be to herself or himself. A London group in 1961 successfully trained a patient to look after himself at his home by knowing how to set up his machine and look after it during dialysis (21). This was followed by overnight dialysis at home by the same group in 1964 (22). In America home dialysis scheme had been proposed but was successfully put into operation also in 1964.

As a continuation in the development of artificial kidneys, Stewart, a chemist from Michigan, introduced the Hollow fibre for capillary flow dialysis in 1960 (23).

From the foregoing modern day dialysis is a challenge to today's nephrologist who may be amazed by the advance in haemodialysis which has accrued over the years.

(ii) HAEMODIALYSIS EQUIPMENT

Extracorporeal haemodialysis requires a satisfactory membrane package for solute and water transport and fluid cycling equipment (24). As haemodialysis therapy has become more common-

place, a large variety of dialysers and associated equipment has become available.

DIALYSERS

A dialyser is a device which separates two fluid media of differing composition and allows a partial exchange of their constituents from one to the other. The term haemodialyser is used to refer to a device which contains part of the circulating blood outside the body and which is so constructed as to allow the exchange of blood chemicals to take place across a membrane into another fluid commonly called dialysate, without the two mixing.

There are three designs of dialysers in clinical use today (24, 25):

1. The parallel flow dialyser (photograph 1)
2. The coil dialyser (photograph 2)
3. The hollow fibre dialyser (photograph 3)

Dialysers are basically membrane support systems. They must hold the membrane in such a fashion that blood and dialysate flow in an optimum manner on each of their respective membrane sides. Dialyser design and membrane properties maintain a degree of independence, for a variety of membranes can be used in most available dialyser designs; and the various designs can use different membranes, with the possible exception of the hollow fibre, ^{where} a limitation is imposed on the configuration of the dialyser, since the capillary fibre is its own support.

Cellulose base membranes of one sort or another constitute over 95 per cent of the membranes in current clinical use (24).



Photograph 1: A Gambro Lundia Plate (standard), a parallel flow dialyser.



Photograph 2: Two different types of coil dialysers.



Photograph 3: Two different types of hollow fibre dialysers.
A coil dialyser extreme left.

Cuprophane has been the standard against which other membranes are compared. Alternative membranes that are available include Polycarbonate, Polyacrylonitrile and Polysulfone membranes (26).

In considering a membrane for haemodialysis, the following properties are important (27):

1. Permeability properties; diffusive permeability of solutes and hydraulic permeability of the solvent.
2. Mechanical properties: tear strength, dimensional changes on wetting, resistance to elongation with time, and stress - strain resistance.
3. Toxic properties; release of toxic components from the membrane causing tissue damage.
4. Thrombogenic and haemolytic properties: alteration in blood clotting, platelet function and red blood cells damage by the membrane.

Aside from safety and nontoxicity of the materials used in the construction of the dialyser, other properties of major concern include:

1. The priming volume;
2. The ability to recover the blood after completion of dialysis;
3. The resistance of blood flow; and
4. The ability to clear waste products from the blood and to remove water.

DIALYSATE FLUID

Dialysate fluid is an aqueous mixture of the principal electrolytes in extracellular fluid. Dialysate is made by either mixing a premeasured quantity of salts in a reservoir of warm water, or proportionally mixing a concentrated salt solution with an appropriate amount of warm water continuously. The former method has been called the batch system, the latter the proportional delivery system.

One of the first considerations in preparing a dialysate solution is the quality of water used. When haemodialysis was in its infancy, untreated water from the city water was frequently employed. As chronic haemodialysis expanded, it became evident that careful attention to the quality of water could prevent certain serious medical problems (28, 29). Basically, a water treatment system consists, of a combination of filters, ion exchangers, or reverse osmosis devices. Sediment filters are routinely used to remove particulate matter above five microns in diameter. Absorptive carbon filters are advocated if free chlorine, chloramines or pyrogens are present in the water. Absorptive iron filters may be required if there is any iron present.

Ion exchangers are of two types: a water softener and a deioniser. A water softener is frequently used ahead of a reverse osmosis system because excessive hardness in the water feeding the reverse osmosis system may damage its membranes. Deionisers remove both cations and anions, exchanging them for hydrogen and hydroxyl groups in the water. They work most

efficiently to polish water after it is passed through the reverse osmosis process.

Reverse osmosis is a filtration process by which water is forced under high pressure against a membrane, allowing nearly pure water to pass. The process removes about 90 per cent of dissolved solids (28), and its effectiveness in delivering pure water is greatest when the feed water has been pretreated by filtration and softening.

The selection of a concentrate to mix with water to make the final dialysate has usually centred on its potassium, calcium and sodium content. The approximate concentration of ions in millimoles per liter in the final dialysate is most often 1.75 for calcium, 1.0 for magnesium, 2.0 for potassium, 33 for acetate, 132 for sodium, 105 for chloride, 200 mg/dl for dextrose. These figures are chosen to correct the electrolyte changes in various renal failure syndromes.

DIALYSATE SUPPLY SYSTEMS

The purpose of a dialysate supply system is to mix dialysis concentrate with water in an appropriate composition and to transport the final dialysate fluid along the membrane surface opposite the surface of the circulating blood.

There are individual batch and proportioning system, as well as central supply systems capable of supplying dialysate for as many as twenty patients simultaneously. The decision to use an individual supply system versus a central system is dependent mostly on economics. However, the flexibility of being able to vary the dialysate composition according to the patient needs is

retained only through the individual supply systems. One of the most widely used systems is the so-called recirculating single-pass dialysate delivery system used almost exclusively with the coil-type haemodialysers. This system consists of a cannister which holds the coil dialyser in place while dialysis fluid bathes the coil. The dialysis fluid is drained away from the cannister at a rate of 300 - 400 mls/mm and discarded as fresh dialysate is replenished in the cannister at the same rate. This type of system is adaptable to either a proportioning mixture or a batch mixer.

In parallel-flow dialysers and in hollow fibre dialysers, a system better suited to their use is the so-called single-pass dialysate delivery system. In principle, a fresh dialysate solution is passed countercurrent to the flow of blood and then discarded.

MONITORS

It is important that the haemodialysis procedures be monitored by various devices to ensure maximum safety. The parameters commonly monitored are:

1. The conductivity and temperature of dialysate
2. Blood leaks
3. Arterial and venous pressures
4. Tubing collapse
5. Air bubbles

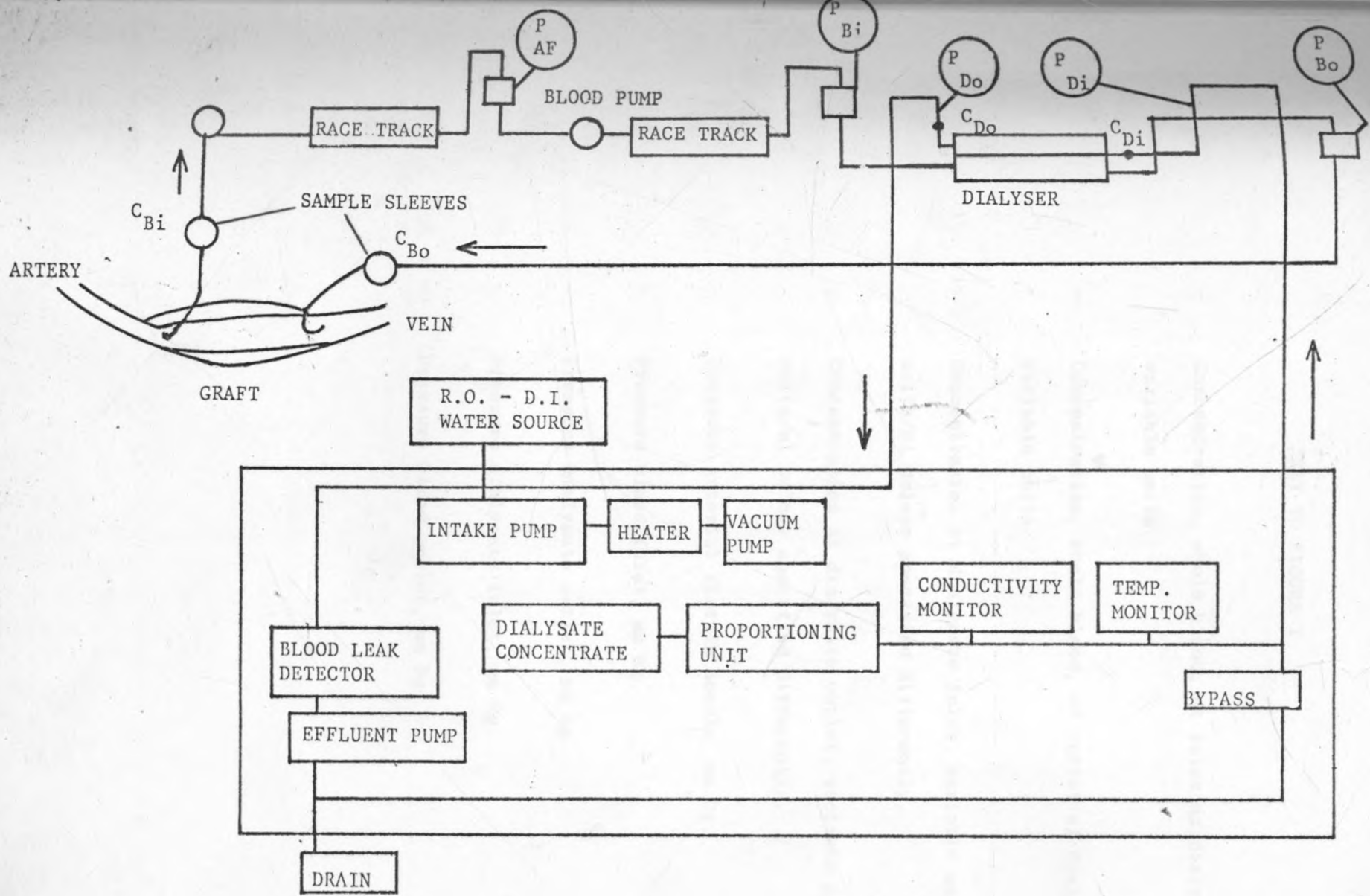


Figure 1: Schematic view of haemodialysis system.

KEY TO FIGURE 1

- C_{Bi} - Concentration, whole blood, at inlet of dialyser, variable units.
- C_{Bo} - Concentration, whole blood, at outlet of dialyser, variable units.
- C_D - Concentration at dialysate inlet, variable mass units/ml unless specified differently.
- C_{Do} - Concentration at dialysate outlet, variable mass units/ml unless specified differently.
- P_{AF} - Pressure arterial fistula needle, mm Hg.
- P_{Bi} - Pressure blood inlet, mm Hg.
- P_{Do} - Pressure dialysate outlet, mm Hg.
- P_{Di} - Pressure dialysate inlet, mm Hg.
- P_{Bo} - Pressure blood outlet, mm Hg.

BLOOD LINE TUBING

Connection of the dialyser to the patient requires additional equipment and supplies. Polyvinylchloride tubing is commonly used for blood tubing connections between the patient's vascular bed and the dialyser. These are fabricated with segments for blood sampling, heparin administration, bubble traps, segments which fit into blood pumps, and an arm of tubing for the administration of fluids during dialysis.

A filter in the venous bubble trap is necessary to minimise the risk of pulmonary embolization.

Blood pumps are almost always used to maintain flow through the dialyser. Blood pumping is performed with a roller pump. An inbuilt heparin pump is present in most dialysis machines.

SINGLE NEEDLE DIALYSIS

The invention of an oscillating system which allows blood to flow out of the body and back to the body through a single needle inserted into a peripheral vein has created a great deal of interest as a means of achieving extracorporeal circulation through a haemodialyser (30). The system consists of a cannula and a short Y connector. Fresh blood is drawn into the inflow line by a blood pump while the venous return is blocked. Alternatively, the inflow line is occluded by a clamp, while the outflow line is opened, allowing the return of blood from the dialyser to the patient. - This is in contrast to the double needle depicted in figure 1.

(iii) VASCULAR ACCESS FOR HAEMODIALYSIS

The historical landmark of successful semipermanent cannulation of blood vessels was described by Quinton et al in 1960 (31). Since that time several modes of access to the patients' vasculature have been described.

EXTERNAL SHUNTS

The Quinton-Scribner shunts provided the initial feasibility for chronic haemodialysis (17, 31). These shunts provide flow rates of approximately 150 to 350 ml/min and have generally failed to produce cardiac strain or complications. Because of the high incidence of attendant cannula complications, external shunt systems have largely been supplanted by internal A-V fistula procedures in most dialysis centres. External shunts are often preferable in specific clinical settings, e.g. short-term dialysis in patients awaiting renal transplantation; treatment of acute, reversible renal failures and whenever immediate haemodialysis is required (32). Arm locations permit technically easier operations but should be avoided in patients needing chronic dialysis. Placing an ankle shunt preserves arm blood vessels for future surgical construction of arteriovenous fistula.

Implantation of a teflon-silastic external shunt into the upper thigh (Groin shunt) is occasionally used (33). These are normally considered when confronting a patient who has no remaining access sites available in either arm or leg and who is in need of immediate dialysis.

Placing of teflon-silastic cannulas in the upper arm is

(iv) PRINCIPLES OF HAEMODIALYSIS

Currently, all extracorporeal haemodialysers that are practical for widespread clinical use employ a cellulose based membrane to separate blood from dialysis fluid. Mass transfer of solute and water across the membrane is dependent upon two fundamental processes, diffusion and ultrafiltration.

SOLUTE TRANSPORT BY DIFFUSION

Diffusion is the random thermal movement of molecules which, under the proper condition, can result in the transfer of mass from one position to another. When a solute such as urea is added to a fixed volume of water, the urea molecules when ~~added~~ dissolved are in constant random motion and become distributed uniformly throughout the volume of water. The rate of this random movement of solute in water relates to molecular size, molecular shape, and ionic charge and is described by the diffusion coefficient of the species in water. The water molecules, too, are in constant random motion at a rate described by the diffusion coefficient for water in water. If the solution of urea is connected by aqueous channels that permit diffusion of the solute to another solution of different urea concentration, then the process of diffusion will result in a net transfer of urea (mass transfer) from the compartment of higher concentration to the compartment of lower concentration.

DIALYSANCE BY THE ARTIFICIAL KIDNEY

The function of haemodialysers may be described in the same way as the function of the kidney with respect to the removal of

substances from blood. In Fig. 2 the schematic representation of the kidney emphasizes the roles of hydraulically determined filtration by glomeruli and a variety of tubular processes that finally determined the output or clearance. In contrast, in the absence of ultrafiltration the haemodialyser operates as a simple diffusion device but nevertheless produces a calculable output or clearance (43).

For the kidney,

$$\text{Clearance} = \frac{UV}{P} = \frac{Q_u C_u}{C_{Bi}} = \frac{\text{mass out}}{\text{Blood concentration}}$$

and for the haemodialyser

$$\text{Clearance} = \frac{Q_{Bi} (C_{Bi} - C_{Bo})}{C_{Bi}} = \frac{\text{mass out}}{\text{Blood concentration}}$$

Dialysance (D_B) has been defined as "the minute rate of net exchange of a substance between blood and bath fluid per unit blood-bath concentration gradient" (43). From Fig. 2 this description may be expressed mathematically as

$$D_B = \frac{Q_{Bi} C_{Bi} - Q_{Bo} C_{Bo}}{C_{Bi} - C_{Di}}$$

In the absence of ultrafiltration the flow rates for blood entering and leaving the system are equal ($Q_{Bi} = Q_{Bo}$), and if single-pass dialysis is employed the concentration of solute in the entering dialysis fluid (C_{Di}) is always zero. Therefore the expression can be simplified and re-written in a form similar to the usual clearance equation.

$$D_B = \frac{Q_B (C_{Bi} - C_{Bo})}{C_{Bi}}$$

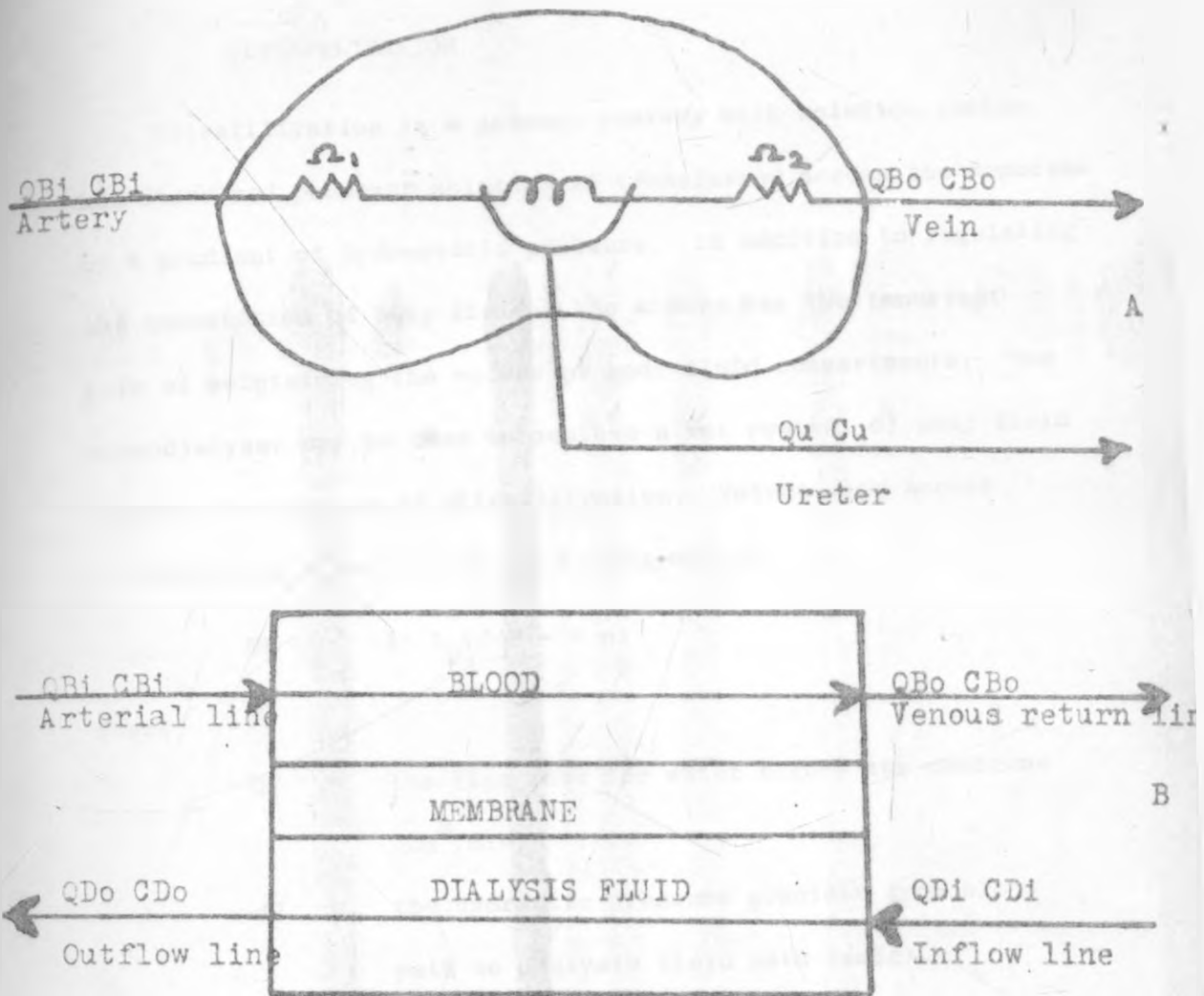


Figure 2. Diagrammatic representations of the natural and artificial kidney. The natural kidney (A) moves solute across the glomerular basement membrane convectively in response to a hydraulic force generated by blood pressure and the afferent (r_1) and efferent (r_2) resistance on either side of the glomerular capillary. In contrast, the artificial kidney (B) operates by diffusion across a membrane.

KEY TO FIGURE 2

- QB - Blood flow rate.
- CB - Concentration of substance in blood.
- Qu - Urine flow rate.
- Cu - Concentration of substance in urine.
- QD - Dialysate flow rate.
- CD - Concentration of substance in dialysate.
- i - In
- o - Out

ULTRAFILTRATION

Ultrafiltration is a process whereby bulk solution (water and dissolved permeant solutes) is transferred across the membrane by a gradient of hydrostatic pressure. In addition to regulating the composition of body fluids, the kidney has the important role of maintaining the volume of body fluid compartments. The haemodialyser may be used to achieve a net removal of body fluid through the process of ultrafiltration. Volume flow across a semipermeable membrane may be expressed as,

$$QF = A \cdot L_p (dP - d\pi)$$

where

QF = the flux rate for water across the membrane
(CM^3/min)

dP = the hydraulic pressure gradient from blood path to dialysis fluid path (mmHg).

$(d\pi)$ = the osmotic pressure gradient from blood path to dialysis fluid path (mmHg).

L_p = the hydraulic permeability, i.e. the volumetric flow rate of water per unit area of membrane per unit pressure gradient
($CM^3/(min. CM^2 mm Hg,)$)

A = the area of the membrane.

Ultrafiltration in most haemodialysers is determined for all practical purposes by the hydraulic permeability of the membrane and the hydrostatic pressure applied to the blood side of the dialyser.

SOLUTE TRANSPORT BY CONVECTION

Convective transfer of solute occurs when permeant solute moves as a solution with water in response to either a hydrostatic or an Osmotic gradient.

(v) INDICATIONS FOR HAEMODIALYSIS

The well established indications for haemodialysis include:-

- (1) Acute renal failure
- (2) Chronic renal failure
- (3) Acute intoxications (inadvertent drug over dosage and poisonings, acute metabolic derangements resulting in a toxic level of metabolites - lactic acidosis, hyperuricaemia, hypercalcaemia).
- (4) Overhydration

Proposed indications for dialysis have ranged from schizophrenia and myasthenia gravis (44) to Psoriasis (45). Early in the development of haemodialysis methodology the preferred therapeutic approach was to withhold haemodialysis until pressed to carry forward by some dire clinical or laboratory event. With the high level of technical competence now widely available, early application of haemodialysis is the rule. "Prophylactic" haemodialysis as advocated by Teschan et al (44) in 1960 is now routinely practised in acute renal failure.

In chronic renal failure, at a minimum, the following factors must be weighed in each case when deciding the time to initiate maintenance dialysis:

- (1) Degree of incapacity from uraemic symptoms.
- (2) Long-term expectations for maintenance dialysis: Is transplantation planned? if so, when?

- (3) Presence of uraemic signs or symptoms or both: Uraemic neuropathy, Pleuritis, Pericarditis, recurring infection and so on.
- (4) Change in life-style imposed by beginning maintenance haemodialysis.
- (5) The wishes (informed consent) of the patient and supporting family members.

Selection criteria for admission into a maintenance dialysis programme differ from centre to centre depending on such variables as availability of manpower and facilities for dialytic therapy.

(vi) COMPLICATIONS IN HAEMODIALYSIS

The complications of haemodialysis can be divided into those that are:

- (1) Consequences of the usual dialysis procedure;
- (2) Mechanical, iatrogenic or both, and
- (3) Manifestations of persistent or incompletely treated uraemia.
- (4) Problems of undetermined causes.

CONSEQUENCES OF THE USUAL HAEMODIALYSIS PROCEDURES

Haemodialysis can be viewed as two distinct phenomena: Clearance and ultrafiltration. Conceptual separation of clearance or diffusion and ultrafiltration is necessary for understanding the dialysis procedure and developing the proper approach to individual patients. One can utilize a rapid blood flow rate and large surface area dialyser to increase or maintain clearance, yet minimise fluid removal either by using low hydro-

static pressure or replacing a quantity of fluid equal to that removed. On the other hand, one can minimise clearance utilising a small dialyser with a slow blood flow rate while using maximum hydrostatic ultrafiltration to achieve greater fluid removal.

Changes In Vascular Volume: The most critical change that occurs with haemodialysis is reduction of extracellular volume by ultrafiltration. Insidious visceral engorgement, third space collection, and pulmonary aedema result from fluid overload. Hypertension in dialysis patients is also usually volume dependent (46), and the high incidence of cardiovascular disease in uraemic patients largely results from the persistent fluid overload and resulting hypertension (47).

Several factors influence the likelihood of complications during fluid removal by dialysis:

- (1) Volume of the dialyser and blood lines;
- (2) Compliance of dialyser, that is the degree of expansion of the blood compartment with an increase in resistance to flow, and
- (3) Magnitude of ultrafiltration.

A large extracorporeal circulation, a highly compliant dialyser, and an increased ultrafiltration coefficient are more likely to lead to hypotension than is a small surface area dialyser with a low ultrafiltration coefficient. Newer dialysing membranes allow a degree of ultrafiltration such that fluid can be removed from the vascular compartment faster than it can be replenished from interstitial and intercellular spaces, other

factors such as uraemic autonomic neuropathy (48), hormonal changes, and myocardial disease complicate efforts at reducing vascular volume. The potential adverse effects of antihypertensive medications as well as changes in osmolality and blood acetate concentration also should be assessed when one attempts ultrafiltration.

Controlled sequential ultrafiltration and haemodialysis, in which diffusion and ultrafiltration are separated, might allow a more stable dialysis and facilitate the removal of larger quantities of fluid (49, 50).

Electrolyte and Osmolar shifts: Abrupt changes in the concentration of sodium, potassium, and calcium during haemodialysis can induce serious consequences. Diffusion or clearance of electrolytes and other substances can create changes in intracellular and extracellular concentrations and can alter osmolality.

Most dialysate solutions have sodium concentrations between 130 and 140 mm ol/litre. The lower sodium concentrations are used to remove sodium and thereby reduce blood pressure. However, patients frequently develop mild hyponatraemia, a chemical disorder that might be related to the development of side effects such as muscle cramps and dialysis disequilibrium. Severe dialysis disequilibrium is manifested by marked mental and psychiatric changes and grandmal seizures; a mild disequilibrium occurring after dialysis produces headache, fatigue and lethargy (51, 52). Rapid changes in the concentration of urea or other unidentified osmotically active agents (idiogenic osmoles) and changes in the PH of the central nervous system contribute to

the pathogenesis of this syndrome (52, 53). The rate of change of serum sodium, urea or other substance is probably more important than is the quantity of any substance removed. The syndrome usually can be prevented by avoiding prolonged and efficient dialysis, particularly when the blood urea nitrogen is markedly elevated. Intravenous osmotic agents such as mannitol or dextrose reduce the intensity of osmolar change and are believed by some to be effective in preventing muscle cramps and disequilibrium (54).

The most critical electrolyte shift in patients undergoing haemodialysis involves potassium. Hyperkalaemia often occurs in patients with advanced renal failure. This can be lowered by utilising a dialysate potassium concentration between 1.5 and 3.0 mmol/litre. In the patient with cardiac failure who takes digitalis, the patient with coronary disease and an irritable conduction system, or the small, elderly patient whose dietary intake is inadequate, a low potassium concentration in the dialysate can lead to transient hypokalaemia and severe rhythm disturbances.

Acetate Accumulation: In the early 1960s, acetate was developed as a dialysate buffer to avoid the use of complicated systems containing bicarbonate and bubbled carbon dioxide. It has been demonstrated that patients treated for long periods with acetate-buffered dialysate become bicarbonate depleted (55). In addition, transient acetate accumulation occurs during and immediately after dialysis in a significant percentage of patients (56). The acetate ion has been cited as a cause of myocardial depression (57) and cardiovascular instability in seriously ill patients (58).

Anticoagulation: Abnormalities of platelet factor III

activity and platelet adhesiveness due to uraemia can be reversed by haemodialysis (59). Bleeding remains a risk, however, because systemic administration of heparin is necessary in carrying out extracorporeal circulation. Also, many dialysis patients are treated with warfarin, aspirin, and antiplatelet drugs to help preserve the patency of arteriovenous shunts and fistulas. These agents can induce a variety of bleeding complications including gastrointestinal haemorrhage; bloody pericardial, pleural, and joint effusion; subdural haematoma; retroperitoneal haematoma; ocular and retinal haemorrhage; and increased menstrual flow (60).

Shunt and fistula problems: The reported problems include:

- (1) Repeated thromboses requiring multiple operations (61).
- (2) Vascular compromise and vascular "steal"^{syndrome} (62).
- (3) Infections (61)
 - (a) Local
 - (b) Endarteritis with septicaemia and septic emboli.
 - (c) Aneurysm with rupture
- (4) Increased cardiac output
- (5) Carpal tunnel syndrome (63)
- (6) Recirculation yielding inadequate dialysis

MECHANICAL AND IATROGENIC COMPLICATIONS OF HAEMODIALYSIS

Air embolus and the consequences of improper composition of the dialysate were devastating complications in the past, but improved automated equipment and safety features largely eliminated these problems. Haemodialysis can occur from inadequate water treatment or contamination of the dialysate (64).

Other clinical problems such as dementia (65) hypertension and sudden death can result from dialysate contamination with substances such as aluminium, calcium, and fluoride. Toxic chemicals such as diethylthalamate and polyvinyl chloride can be leached from membranes, dialyser shells, or blood lines and have been suggested as causes of cardiotoxicity (66), necrotizing dermatitis (67) and hepatitis (68).

The precipitous drop in the leukocyte count during haemodialysis is thought to result from activation of the complement system during exposure of blood to dialysis membranes. Leukocyte accumulation in the pulmonary vasculature has been held responsible for the pulmonary hypertension and hypoxaemia that occur in some patients (69). A fall in $P_a O_2$ also might be related to hypoventilation induced by the hypocapnia that results from a loss of carbon dioxide during dialysis (70).

Some deficiency states may occur during haemodialysis. This may be related to loss of folate, soluble B vitamins and amino acids across the dialysing membrane.

MANIFESTATION OF PERSISTENT OR INCOMPLETELY TREATED URAEMIA

Variable degrees of anaemia persist in most patients undergoing maintenance dialysis (71). Although this normochromic, normocytic anaemia primarily results from decreased erythropoiesis, other factors including endogenous haemolysis, low-grade blood loss, iron deficiency, and folate deficiency also contribute. Uraemic patients have compromised immune systems, and increased frequency of infection has been reported (72). Although haemodialysis tends to correct this immune deficiency

dialysis patients still seem to have an increased incidence of infection.

Pericarditis occurs at two different periods in patients with kidney failure. First, it often occurs just prior to, or soon after the induction of dialysis. Second, pericarditis can occur in patients who are dialysed chronically. In this setting, it can result from viral infection, or an underlying systemic disease. Gelfand et al (73) have reported an increased incidence of pericardial effusion in uraemic patients being treated with minoxidil.

Some researchers have reported an acceleration of atherosclerotic cardiovascular disease in uraemic patients on dialysis (74), but some studies argue that atherosclerosis is not accelerated in this population (75).

Hyperparathyroidism does not respond to haemodialysis alone. In the patient with far advanced hyperparathyroidism, subtotal parathyroidectomy is sometimes necessary. In patients not treated appropriately, the end results can include severe proximal myopathy; severe bone disease with pathologic fractures and pain; and subcutaneous, joint, visceral, and vascular calcification (76).

Several types of encephalopathy can occur in patients undergoing chronic dialysis (77). Uraemic encephalopathy is common in patients with advanced renal insufficiency, and mild forms can appear in inadequately dialysed patients. A severe encephalopathy consisting of myoclonic jerking, speech impairment, dementia, and status epilepticus leading to death has

been recognised in long-term dialysis patients (78). Some researchers have suggested that orally administered Aluminium gels cause this syndrome of "Dialysis Dementia" (79). Others have indicated that the syndrome is related to aluminium in water used for dialysis (65, 80). Atherosclerotic cerebrovascular disease and subdural haematomas (81) must be distinguished from this syndrome.

Many chemical abnormalities can persist in patients on chronic dialysis, including hyperlipidaemia, mild acidosis, hyperuricaemia, hypermagnesaemia, hyperamylasaemia and elevated blood urea nitrogen and serum creatinine levels. Hypertriglyceridaemia and decreased high-density lipoprotein levels might contribute to premature atherosclerotic disease; hyperuricaemia might provoke gouty arthritis; and chronic acidosis probably accentuates metabolic bone disease.

PROBLEMS OF UNDETERMINED CAUSES

Intractable ascites is a puzzling and very rare complication in patients treated by maintenance haemodialysis, the aetiology and pathogenesis of which remain a mystery in most instances (82).

Pruritis is common and often is severe and unrelenting (83).

Psychiatric, emotional, and social problems are major complications in this population. Finally, many serious complications result from the use of inappropriate drugs and dosage levels in dialysis patients.

(vii) MORTALITY IN PATIENTS ON HAEMODIALYSIS

The average death rate in patients on long-term haemodialysis in many well established centres is estimated to be about 10 per cent per year (84). Prominent causes of death are cardiac disease, cerebrovascular disease and infections (85). Other causes of death have included respiratory disease, "kidney disease and uraemia", endocrine and metabolic diseases; complications of treatment and accidental death; diseases of digestive system and liver; Neuropsychiatric diseases, and neoplasms.

(viii) HAEMODIALYSIS - THE AFRICAN SITUATION

Regular haemodialysis programmes are expensive and might drain a disproportionately high portion of the health budget in countries with limited resources. This is probably the major reason why such centres have not found place in most developing countries (86).

There is, however, evidence that haemodialysis, in one form or another, has been practised as a form of therapy in Egypt since 1966 (87), in the Sudan since 1974 (88) and in Kenya since 1973 (89, 90).

11. MATERIALS AND METHODS

RETROSPECTIVE STUDY

The records of all patients started on haemodialysis at Kenyatta National Hospital, between July 1973 and June 1982, were analysed retrospectively. The total of 57 patients included 31 with acute renal failure and 26 with chronic renal failure.

During that period, haemodialysis was used in three categories of patients:

- (a) those with acute renal failure who were expected to recover soon with supportive treatment.
- (b) those with stable chronic renal failure, who destabilised after developing an acute episode precipitating rapidly increasing uraemia.
- (c) those patients whose renal disease was uncertain and awaited confirmation of diagnosis.

Thus haemodialysis, as a form of replacement therapy for chronic uraemia, was never envisaged at that time.

Patients were dialysed three times weekly for five to seven hours, following a variable period (four to seven) of daily dialysis. The MC-20 coil dialyser was used throughout the period. The water supply used was tap water from the city supply system. There was no additional treatment. Batch production of dialysate requiring the mixing of dry chemicals with water in a container large enough to provide for the entire treatment was in use then. Photograph 4 shows the haemodialysis equipment used between July 1973 and June 1982.

The data on the patients thus treated are analysed with respect to:

- (a) Age and sex
- (b) Renal failure syndrome necessitating institution of haemodialysis therapy.
- (c) Primary cause of renal failure syndrome.
- (d) Clinical features at the onset of haemodialysis therapy.
- (e) Some biochemical parameters at the onset of haemodialysis therapy.
- (f) Outcome of haemodialysis therapy.



Photograph 4: Haemodialysis equipment used at the Kenyatta National Hospital between July 1973 and June 1982.

(ii) PROSPECTIVE STUDY

The author was actively involved in the management of patients with renal failure syndromes in the period between August 1984 and August 1985.

(1) PATIENTS: Between August 1984 and August 1985, forty two patients were started on haemodialysis therapy. These included twenty with acute renal failure and twenty two with chronic renal failure. All patients with acute renal failure were eligible for haemodialysis. The selection of patients with chronic renal failure for maintenance haemodialysis was the sole responsibility of the hospital nephrologists. The guidelines outlined in Appendix I were used in the selection.

Before the start of dialysis, the following data were obtained from the patients.

- (a) Age and sex
- (b) Renal failure syndrome
- (c) The duration of the illness (renal failure) that is, the duration between the time at which the patient first developed symptoms of renal failure and the time of initiation of dialysis.
- (e) Laboratory data viz:
 - (i) serum creatinine
 - (ii) Blood urea nitrogen
 - (iii) serum potassium and sodium levels

- (iv) serum calcium and inorganic phosphate levels where indicated.
- (v) creatinine clearance
- (vi) full haemogram
- (vii) urinalysis
- (viii) others:
 - Chest X-rays
 - Electrocardiograms and Echocardiograms
 - Renal ultrasounds
 - Intravenous Urograms (where possible)

Once dialysis was started, the predialysis serum potassium and sodium and the blood urea nitrogen were done at every dialysis. The post-dialysis urea and electrolytes and other investigations were done as required.

The short-term complications and the outcome of dialytic therapy were looked into.

(2) ARTERIOVENOUS SHUNTS: Once the decision to start a patient on haemodialysis was made, a limb arteriovenous shunt (Scribner-Quinton type) was fashioned. This was done by urological surgeons, nephrologist or senior house officers in medicine and paediatrics going through their rotation in nephrology.

During the study period, complications arising by virtue of the presence of the shunts were looked into. Thus,

- (a) The clotting of a shunt was noted any time it occurred.
- (b) Any significant bleeding from the shunt site or following a disconnection of the shunt was noted.
- (c) Swabs for cultures were taken from the shunt site in the second week after placement and at any time there-

after when there were signs of acute inflammation at the shunt site, such as pain, tenderness, and redness ("shininess") with or without pus formation.

The cannula replacement rate following complications was worked out. Photograph 5 shows an arteriovenous shunt in situ in one of the patients dialysed during the prospective study period.

(3) DIALYSIS EQUIPMENT: In the prospective study period the Gambro (R) machines were used exclusively. These were equipped with monitors for conductivity and temperature of dialysate, blood leaks, arterial and venous pressures, tubing collapse and air bubbles. The tubing and dialysers were for single use. For all the forty two patients except two, the Gambro Lundia plate dialysers (1.25 m² surface area, 11.5 microns membrane thickness) was used. One patient, 9 years old, required the use of a Gambro Lundia minor dialyser (0.51 m² surface area, 13.5 microns membrane thickness) and the other, a grossly muscular man, required the use of a Gambro Lundia major dialyser (1.7 m² surface area, 11.5 microns membrane thickness). Photograph 6 shows a patient on haemodialysis.

(4) WATER TREATMENT AND DIALYSATE SUPPLY: In the prospective study period, the water used for dialysis was softened, sedimented and filtered then reverse osmosed before being pumped to the continuous proportioning system which used a fixed proportioning pump to mix liquid concentrate with the water to produce the final dialysate as required, generally in 1/34 proportion. The dialysate solute concentration for all patients was sodium, 133.0 mmol/l; potassium, 3.0 mmol/l; calcium

1.75 mmol/l; magnesium, 1.0 mmol/l; chloride, 108.5 mmol/l; Acetate, 38.0 mmol/l; and glucose, 3.0 g/l. Photograph 7 shows part of the reverse osmosis equipment at the Kenyatta National Hospital.

(5) THE DIALYSIS PROCEDURE: All patients with acute renal failure underwent an initial four to five days daily dialysis and thereafter thrice-weekly, four hour dialyses until recovery or death. The patients with chronic renal failure underwent thrice weekly, four hour dialyses. The initial one or two dialyses, in all patients, were of short duration (two or three hours) to avoid dialysis disequilibrium. Emergency dialysis was carried out mostly on patients who developed severe pulmonary oedema in the interdialytic period.

The blood flow rate during most of the dialyses has been 150 to 200 mls/min. The transmembrane pressure (ultrafiltration or hydrostatic pressure) has been personalised according to individual patient need, that is, the degree of fluid overload. In all the dialyses heparin infusion at a rate of 500 units/hr has been used, except on one patient who required 1000 units/hr, to prevent clotting of blood in the tubing. In most of the cases the bedside whole blood clotting time has been relied upon for adequate heparinisation.

All dialyses, except emergency dialyses, were carried out during the day, mostly in the mornings. The dialyses were all nurse-attended, with the doctors coming in as necessary, following the review of patients before the start of each dialysis.



Photograph 5: An arteriovenous shunt in situ in one of the patients dialysed during the prospective study. The Shunt has been in situ for 20 weeks, required declotting only once.



Photograph 6: A patient on haemodialysis during the prospective study period. A Gambro Machine in use.



Photograph 7: Part of the reverse osmosis equipment for water treatment at the Kenyatta National Hospital dialysis Unit.

IV.

R E S U L T SRETROSPECTIVE STUDY

A total of fifty seven patients were haemodialysed between July 1973 and June 1982. Of the 57 who were haemodialysed 26 (45.6%) were females and 31 (54.4%) were males. Their ages were between 9 and 50 years with a mean of 29.6 years (Fig. 3). Thirty one patients (54.4%) had acute renal failure, eighteen (31.6%) chronic renal failure and eight (14%) had acute on chronic renal failure (Fig. 4). Fig. 5 shows the number of patients dialysed during each of the years beginning from July 1973.

Causes of renal failure in patients who underwent haemodialysis
(July 1973 - June 1982) - Tables 1 and 2

Table I:

ACUTE RENAL FAILURE

Primary cause	Number	Percentage
Disseminated Intravascular coagulation, sepsis and haemorrhage	10	32.3%
Antepartum/postpartum haemorrhage	6	19.4%
Surgery with haemorrhage	4	12.9%
Mismatched transfusion	3	9.7%
Multiple injuries and haemorrhage	3	9.7%
Acute glomerulonephritis	2	6.4%
Not known	2	6.4%
Haemolytic Uraemic syndrome	1	3.2%
Total	31	100%

NUMBER OF PATIENTS

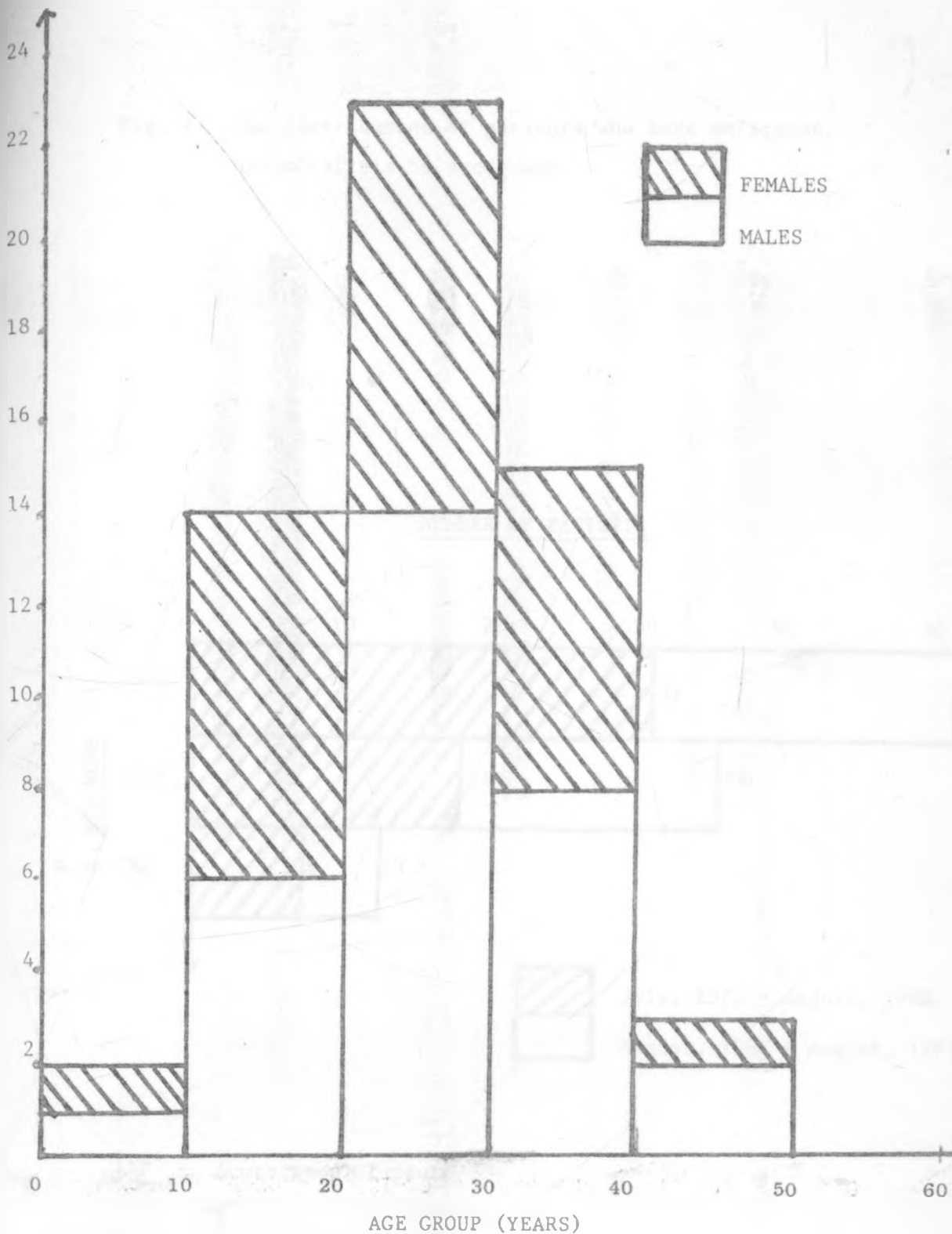
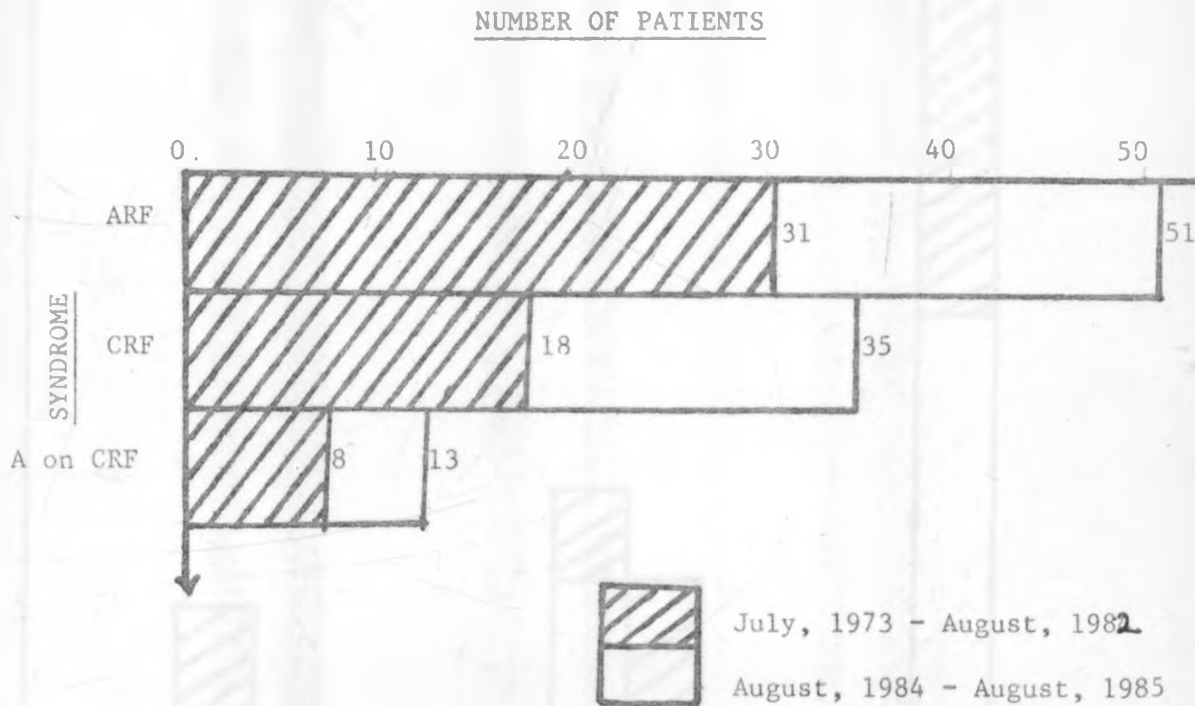


Fig. 3: Age and Sex distribution of the patients who underwent haemodialysis between July 1973 and June 1982 (Retrospective period).

Fig. 4: The distribution of patients who have undergone haemodialysis by syndromes.



ARF - Acute renal failure

CRF - Chronic renal failure

A on CRF - Acute on chronic renal failure

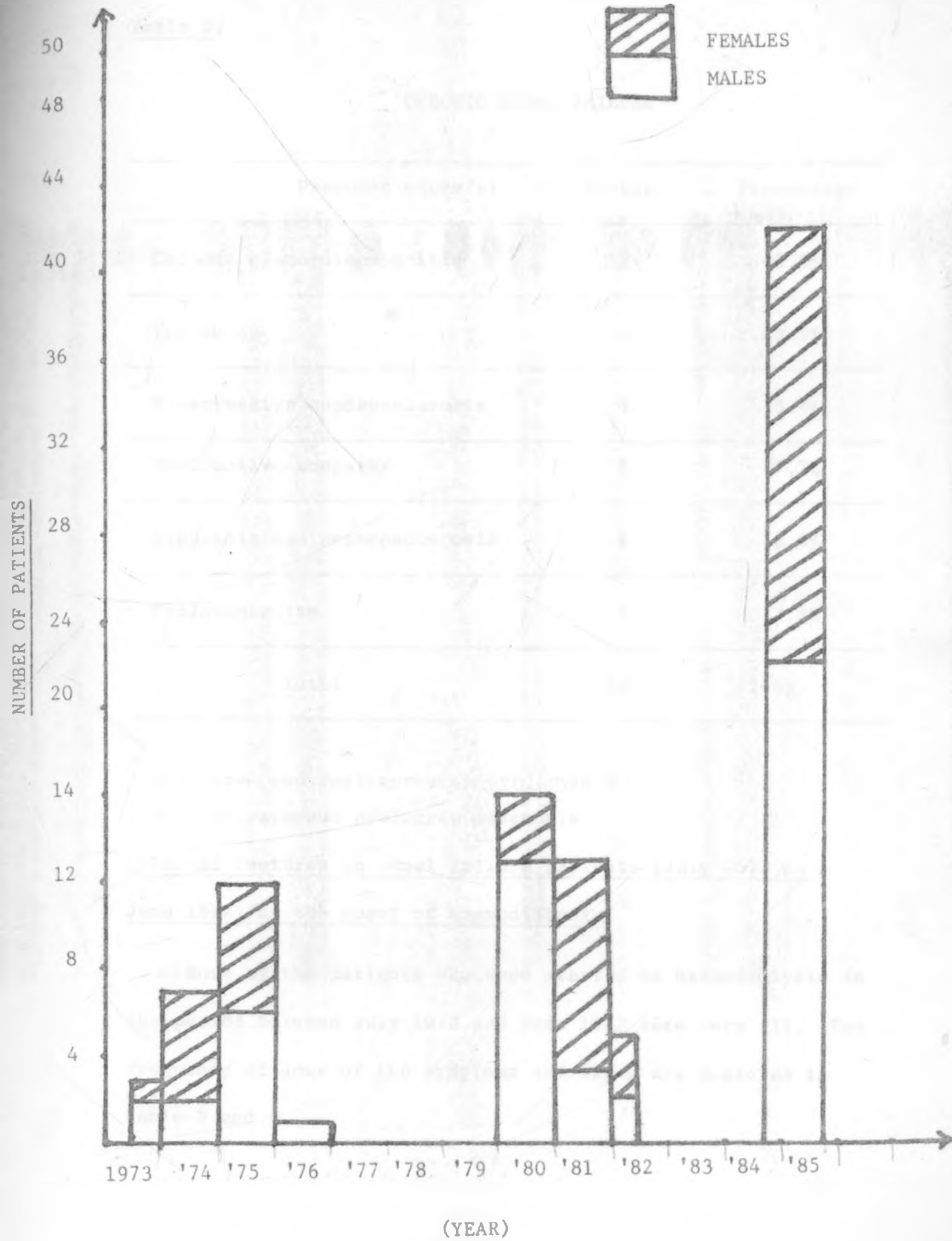


Fig. 5: Number of patients dialysed during each year beginning from July, 1973.

Table 2:

CHRONIC RENAL FAILURE

Presumed cause(a)	Number	Percentage
Chronic glomerulonephritis	12+	46.2%
Not known	6	23.1%
Hypertensive nephrosclerosis	4	15.4%
Obstructive uropathy	2	7.7%
Hypoplasia and nephrosclerosis	1	3.8%
Pyelonephritis	1*	3.8%
Total	26	100%

+ Pre- and Post-mortem histology in 8

* Intravenous pyelogram diagnosis

Clinical features in renal failure patients (July 1973 to June 1982) at the onset of haemodialysis

Most of the patients who were started on haemodialysis in the period between July 1973 and June 1982 were very ill. The frequency of some of the symptoms and signs are depicted in table 3 and 4.

RETROSPECTIVE

(JULY 1973 - JUNE 1982)

3A:	ARF	3B:	CRF	3C:	A on CRF
SYMPTOMS	% (NUMBER)	SYMPTOMS	% (NUMBER)	SYMPTOMS	% (NUMBER)
Oligoanuria	100% (31)	Dizziness	100% (18)	Bleeding	100% (8)
Dyspnoea	79% (24)	Anorexia	94% (17)	Dyspnoea	100% (8)
Anorexia	72% (22)	Nausea	89% (16)	Oligoanuria	100% (8)
Dizziness	64% (20)	Headache	72% (13)	Anorexia	88% (7)
Nausea	63% (19)	Bleeding	67% (12)	Nausea	88% (7)
Swelling of body	32% (10)	Dyspnoea	56% (10)	Dizziness	88% (7)
Cough	21% (6)	Pruritis	56% (10)	Vomiting	67% (6)
Headache	13% (4)	Vomiting	50% (9)	Cough	63% (5)
Vomiting	10% (3)	Swelling of body	50% (9)	Headache	63% (5)
Bleeding	10% (3)	Convulsions	28% (5)	Pruritis	63% (5)
Convulsions	6% (2)	Coma	28% (5)	Swelling of body	50% (4)
Coma	3% (1)	Chest pain	11% (2)	Coma	38% (3)
Chest pain	0% (0)	Oligoanuria	0% (0)	Convulsions	25% (2)
Pruritis	0% (0)			Chest pain	0% (0)

Table 3: The frequency of some symptoms in renal failure patients at the onset of haemodialysis.

RETROSPECTIVE

(JULY 1973 - JUNE 1982)

4A: ARF

4B: CRF

4C: A on CRF

SIGNS % (NUMBER)	SIGNS % (NUMBER)	SIGNS % (NUMBER)
Moribund 48% (15)	Pallor 100% (18)	Pallor 100% (8)
Stupor or coma 42% (13)	Hypertension 89% (16)	Hypertension 100% (8)
Pallor 39% (12)	Moribund 78% (15)	Poor Nutrition 100%
Infection 32% (10)	Stupor or coma 78% (15)	Moribund 88% (7)
Bleeding 23% (7)	Bleeding 56% (10)	Stupor or coma 88% (7)
Oedema 6% (2)	Poor Nutrition 56% (10)	Bleeding 75% (6)
Hypertension 3% (1)	Pericarditis 33% (6)	Bone tenderness 63% (5)
Poor Nutrition 3% (1)	Bone tenderness 28% (5)	Pericarditis 50% (4)
Pericarditis 0% (0)	Pulmonary Oedema 22% (4)	Pulmonary Oedema 50% (4)
Heart failure 0% (0)	Heart failure 22% (4)	Heart failure 50% (4)
Pulmonary Oedema 0% (0)	Infection 22% (4)	Oedema 50% (4)
Pleural effusion 0% (0)	Oedema 22% (4)	Infection 38% (3)
Ascites 0% (0)	Pleural effusion 17% (3)	Pleural effusion 25% (2)
Bone tenderness 0% (0)	Ascites 11% (2)	Ascites 13% (1)

Table 4: The frequency of some clinical features in renal failure patients at the onset of haemodialysis.

BIOCHEMICAL PARAMETERS IN RENAL FAILURE PATIENTS
(JULY 1973 TO JUNE 1982) AT THE ONSET OF DIALYSIS

The biochemical parameters for patients with acute renal failure are shown in Table 5. The mean potassium levels were 5.6 mmol/L (Range 3.4 - 8.4 mmol/L), blood urea nitrogen 55.5 mmol/L (Range 22 - 105 mmol/L) and serum creatinine 417~~m~~mmol/L (Range 130 - 1000 mcmol/L). For chronic renal failure patients (Table 6) the corresponding figures for serum potassium, blood urea nitrogen and serum creatinine were 4.1 mmol/L (2.8 - 6.2 mmol/L), 59.3 mmol/L (26 - 100 mmol/L) and 650 (215 - 1300 mcmol/L).

KEY TO TABLE 5 AND 6

Na ⁺	-	Sodium
K ⁺	-	Potassium
Cl ⁻	-	Chloride
Po ₄ ³⁻	-	inorganic phosphate
Ca ²⁺	-	Calcium
HCO ₃ ⁻	-	bicarbonate
BUN	-	Blood urea nitrogen
Cr	-	creatinine

PRE-DIALYSIS BIOCHEMICAL PARAMETERS IN ACUTE RENAL FAILURE PATIENTS
(JULY 1973 TO JUNE 1982)

Serum Concentration in mmol/L

Patient	Na ⁺	K ⁺	Cl ⁻	PO ₄ ³⁻	Ca ²⁺	HCO ₃ ⁻	pH	BUN	Cr (mcmol)
I . A	146	5.8	105	1.6	2.5	15	7.32	48	266
V . S	132	5.6	100	1.6	2.3	11	7.20	105	1000
E . N	140	4.2	96	1.4	2.3	19	7.30	87	650
J . N	128	3.5	102	1.2	2.0	17	7.27	62	500
J . G	155	3.4	98	1.7	2.1	15	7.28	44	208
K . G	130	6.0	100	1.0	1.9	16	7.27	39	200
A . E	130	5.0	94	0.8	2.4	20	7.30	36	167
S . N	142	4.6	103	1.2	1.7	20	7.33	50	289
R . N	136	4.5	106	1.5	1.8	14	7.26	67	500
M . K	150	6.7	105	1.1	1.5	21	7.38	54	350
G . O	128	3.6	95	1.8	2.0	16	7.28	60	482
M . M	144	7.2	101	0.9	1.6	11	7.25	74	591
S . M	150	6.4	98	1.4	2.4	15	7.27	49	380
C . I	134	5.4	99	1.4	2.0	20	7.32	55	400
C . R	136	8.0	100	0.9	1.8	16	7.30	63	567
S . A	130	4.0	100	1.3	2.1	18	7.32	50	300
R . M	142	4.0	104	1.6	2.7	22	7.36	41	250
J . O	150	7.3	97	1.7	1.5	10	7.22	89	700
E . K	146	6.6	92	1.0	1.7	20	7.36	42	290
J . A	130	5.7	106	1.4	2.0	14	7.28	70	574
M . F	126	3.7	100	1.2	1.9	16	7.28	66	600
S . A	142	3.6	94	2.0	2.1	17	7.30	49	293
A . K	150	8.4	99	0.6	2.2	15	7.26	52	340
B . M	150	5.8	98	1.1	2.4	18	7.32	39	320
M . S	134	6.5	110	1.4	1.8	24	7.40	26	130
W . J	128	7.2	101	0.9	2.0	19	7.36	51	400
M . L	142	6.2	93	1.3	2.4	16	7.30	58	326
W . H	140	8.0	103	1.3	2.1	20	7.34	32	283
O . O	136	5.6	96	1.8	1.6	21	7.36	22	160
J . M	150	4.1	105	1.8	1.8	15	7.28	61	540
A . C	148	6.0	94	1.0	2.0	12	7.28	100	874

Table 6:

PRE-DIALYSIS BIOCHEMICAL PARAMETERS IN CHRONIC RENAL FAILURE PATIENTS
(JULY 1973 TO JUNE 1982)

Patient	Serum concentration in mmol/L								
	Na ⁺	K ⁺	Cl ⁻	PO ³⁻	Ca ²⁺	HCO ₃	PH	BUN	Cr (micromol)
W . W	130	3.0	96	2.0	1.1	12	7.23	41	650
J . A	128	3.0	105	1.8	1.4	18	7.30	45	742
D . A	132	3.4	98	2.2	2.0	22	7.38	55	536
R . B	126	3.6	99	1.6	1.6	16	7.34	87	430
T . S	132	4.1	98	1.5	1.5	10	7.21	90	870
M . G	130	3.4	96	1.6	2.5	16	7.26	26	100
O . P	130	4.5	100	1.7	2.0	15	7.28	36	769
K . W	128	4.4	95	3.0	1.2	18	7.34	60	-
A . H	134	4.3	94	1.9	2.7	20	7.36	81	630
J . A	122	3.6	98	2.4	2.1	17	7.32	72	1300
R . W	136	3.8	97	3.1	1.8	16	7.34	80	450
K . S	130	4.5	99	2.2	2.0	19	7.30	45	750
M . O	140	5.0	90	2.6	1.9	20	7.38	86	624
W . M	138	5.5	106	2.1	2.5	15	7.28	45	315
N . R	130	4.2	102	1.7	2.0	23	7.38	47	700
K . I	128	3.6	94	1.5	1.2	19	7.26	39	240
M . W	134	6.0	102	1.8	1.0	10	7.22	57	520
C . S	136	4.4	100	2.0	1.0	15	7.24	61	670
M . W	134	3.8	99	2.0	2.3	14	7.24	100	834
K . C	130	6.2	95	2.4	0.8	11	7.21	96	912
M . M	128	2.8	102	1.8	2.0	22	7.38	54	365
R . K	142	4.6	99	2.0	1.1	20	7.36	65	215
O . H	130	5.0	98	2.8	1.6	17	7.30	74	946
A . G	144	5.0	99	1.3	2.0	19	7.36	56	870
P . A	130	4.8	92	2.6	1.4	19	7.34	43	912

Outcome of haemodialysis

In the period from July 1973 to June 1982 the patients were haemodialysed for periods varying from 1 to 56 days with a mean duration of 10 days. The number of dialyses per patient varied between 1 and 30, mean 8.

Of the 31 patients with acute renal failure dialysed, 61.3% died, the rest recovered and were discharged for follow up. 31.6% of the deaths were considered due to uraemia, the deaths could not be related to any biochemical aberration, 47.3% due to the primary cause of acute renal failure, 15.8% due to dialysis disequilibrium and 5.3% due to haemorrhage related to heparin infusion during haemodialysis.

55.6% of the patients with chronic renal failure on haemodialysis died, the rest were discharged to the home district hospitals for terminal care after confirmation of diagnosis. Of those who died, 50% were due to uraemia, 40% due to dialysis disequilibrium and 10% due to septicaemia.

Of the patients with acute or chronic failure 50% died, the rest gained their residual renal function and were discharged. 50% of the deaths were due to uraemia, 25% due to dialysis disequilibrium and in the rest, the cause of death was not established.

The clinical and biochemical response to haemodialysis in these patients was difficult to analyse due to the limited numbers of dialyses and lack of documentation.

PROSPECTIVE STUDY

Forty two patients have been haemodialysed in the one year period between August 1984 and August 1985. Of these 54.8% were females and 45.2% were males. Their ages ranged between 9 and 56 years with a mean of 30.2 years (Fig. 6). 47.6% of the patients had acute renal failure, 40.5% had chronic renal failure and 11.9% had acute on chronic renal failure.

Tables 7 and 8 show the frequency of some symptoms and signs at the onset of haemodialysis in the prospective period.

Table 9 shows a summary of experience with acute renal failure patients on haemodialysis. Of the 20 patients with acute renal failure dialysed, 30% died. In all cases the cause of death was unrelated to the haemodialysis procedure (Table 10).

In the acute renal failure patients, the average pre-dialysis blood urea nitrogen, serum potassium and serum creatinine were 32.5 mmol/L (range 19 to 64.5) 6.2 (range 4.0 - 8.0) and 345 ^{mcmmol/L} (range 133 to 640) respectively.

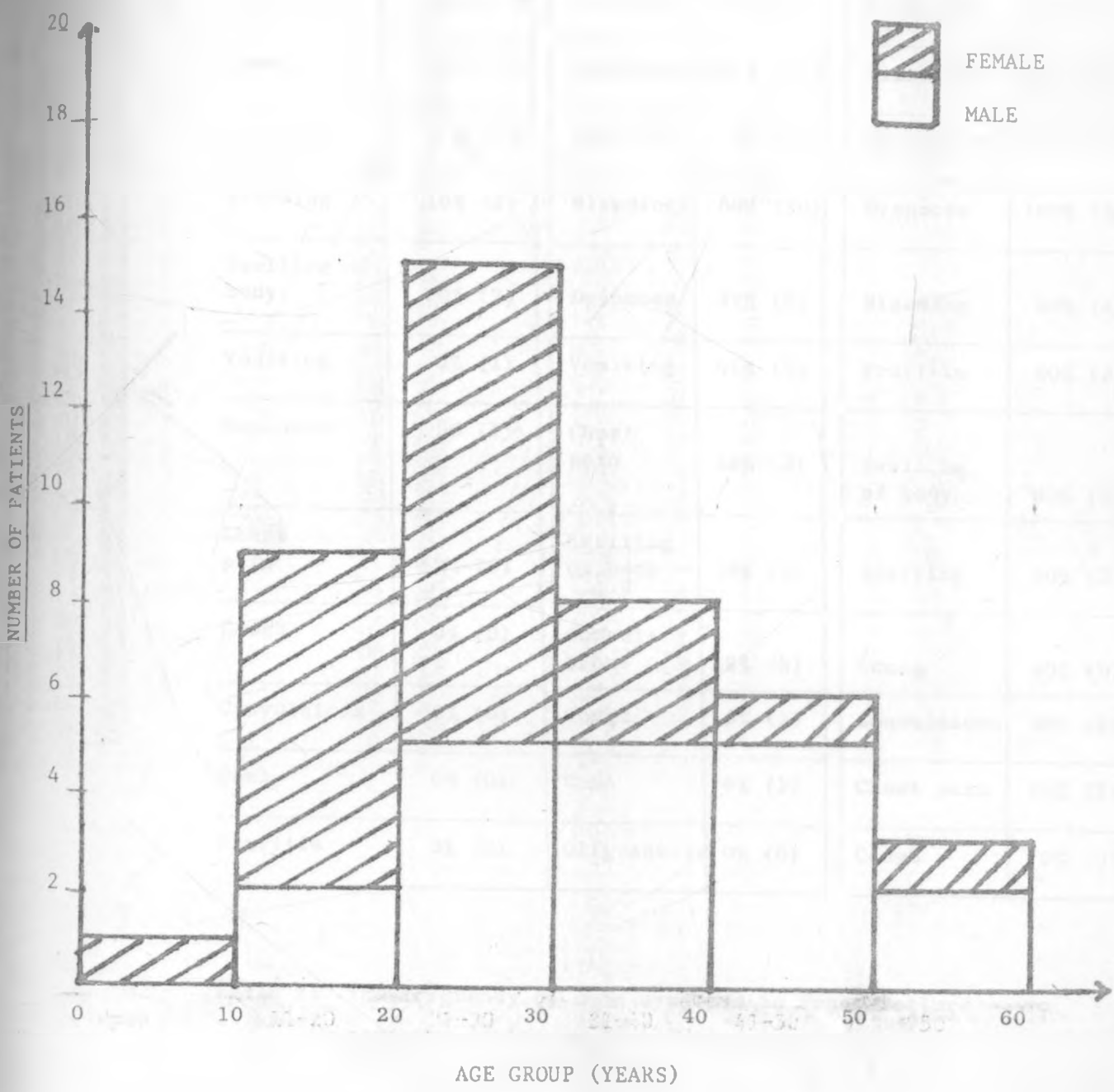


Fig. 6: Age and Sex distribution of the patients who underwent haemodialysis between August, 1984 and August, 1985 (prospective period).

PROSPECTIVE
(AUGUST 1984 - AUGUST 1985)

7A: ARF		7B: CRF		7C: A on CRF	
SYMPTOMS	% (NUMBER)	SYMPTOMS	% (NUMBER)	SYMPTOMS	% (NUMBER)
Oligoanuria	85% (17)	Anorexia	100% (17)	Anorexia	100% (5)
Anorexia	60% (12)	Nausea	100% (17)	Nausea	100% (5)
Dyspnoea	60% (12)	Headache	88% (18)	Headache	100% (5)
Nausea	50% (10)	Dizziness	71% (12)	Dizziness	100% (5)
Dizziness	50% (10)	Pruritis	59% (10)	Oligoanuria	100% (5)
Bleeding	10% (2)	Bleeding	59% (10)	Dyspnoea	100% (5)
Swelling of body	10% (2)	Dyspnoea	47% (8)	Bleeding	80% (4)
Vomiting	5% (1)	Vomiting	41% (7)	Pruritis	60% (3)
Headache	5% (1)	Chest pain	18% (3)	Swelling of body	60% (3)
Chest pain	0% (0)	Swelling of body	18% (3)	Vomiting	60% (3)
Cough	0% (0)	Convulsions	12% (2)	Coma	40% (2)
Convulsions	0% (0)	Cough	6% (1)	Convulsions	40% (2)
Coma	0% (0)	Coma	6% (1)	Chest pain	40% (2)
Pruritis	0% (0)	Oligoanuria	0% (0)	Cough	0% (0)

Table 7: The frequency of some symptoms in renal failure patients at the onset of haemodialysis.

PROSPECTIVE

(AUGUST 1984 - AUGUST 1985)

8A: ARF		8B: CRF		8C: A on CRF	
SIGNS	% (NUMBER)	SIGNS	% (NUMBER)	SIGNS	% (NUMBER)
Pallor	100% (17)	Pallor	100% (17)	Pallor	100% (5)
Bleeding	15% (3)	Blood pressure >160/90 mmHg	100% (17)	Blood pressure >160/90 mmHg	100% (5)
Infection	15% (3)	Poor Nutrition	59% (10)	Poor Nutrition	100% (5)
Oedema	10% (2)	Bleeding	35% (6)	Bleeding	60% (3)
Pericarditis	5% (1)	Moribund	24% (4)	Bone tenderness	60% (3)
Pulmonary Oedema	5% (1)	Stupor or coma	18% (3)	Pericarditis	40% (2)
Blood pressure >160/90 mmHg	0% (0)	Bone tenderness	18% (3)	Heart failure	40% (2)
Heart failure	0% (0)	Pericarditis	18% (3)	Pulmonary Oedema	40% (2)
Pleural Effusion	0% (0)	Oedema	12% (2)	Moribund	40% (2)
Stupor or coma	0% (0)	Heart failure	12% (2)	Stupor or coma	40% (2)
Ascites	0% (0)	Pulmonary Oedema	12% (2)	Infection	40% (2)
Poor Nutrition	0% (0)	Ascites	12% (2)	Oedema	20% (1)
Bone tenderness	0% (0)	Pleural Effusion	6% (1)	Ascites	20% (1)
Moribund	0% (0)	Infection	0% (0)	Pleural Effusion	0% (0)

Table 8: The frequency of some clinical features in renal failure patients at the onset of haemodialysis.

TABLE 9:

SUMMARY OF EXPERIENCE - ACUTE RENAL FAILURE PATIENTS ON HAEMODIALYSIS

PATIENT	AGE (YEARS)	SEX	CAUSE OF RENAL FAILURE	NUMBER OF DIALYSES	OUTCOME
L . Z	18	F	DIC, Haemorrhage	5	Dead
C . K	17	F	Haemorrhage (surgical)	2	Dead
M . G	26	F	DIC, Haemorrhage	1	Living
H . W	30	F	Haemorrhage (hydatidiform mole)	4	Living
F . K	30	F	Haemorrhage (surgical)	15	Living
J . N	37	F	Haemorrhage (DIC)	6	Living
M . M	28	F	DIC Haemorrhage	6	Living
F . M	28	F	Haemorrhage (DIC)	4	Living
M . W	56	F	Mismatched Transfusion	2	Living
F . A	30	F	Obstruction (uterine fibroids)	4	Living
M . K	30	F	Obstruction (ovarian cancer)	4	Dead
M . N	35	F	Haemorrhage (Gastrointestinal)	5	Dead
N . W	35	M	Malaria (DIC, Haemorrhage)	5	Living
E . W	34	F	Haemorrhage (surgical)	5	Dead
S . W	27	F	Acute glomerulonephritis	5	Living
J . O	41	M	Cholera	16	Living
H . M	25	M	Cholera	2	Dead
J . W	27	F	Mismatched Transfusion	7	Living
S . K	33	F	Acute glomerulonephritis	4	Living
P . A	24	F	Haemorrhage (fibroids)	12	Living

TABLE 10:

CAUSES OF DEATH - ACUTE RENAL FAILURE PATIENTS ON HAEMODIALYSIS

PATIENT	SEX	AGE (YEARS)	NUMBER OF DIALYSES	CAUSE OF DEATH
L . Z	F	18	5	Haemorrhage, DIC (Diffuse)
C . K	F	17	2	Peritonitis Ovarian cancer
M . K	F	30	4	Uterine cancer
M . N	F	35	5	Haemorrhage (Gastro-intestinal)
E . W	F	34	5	Uraemia
H . M	M	25	2	Pulmonary Oedema

MAINTENANCE HAEMODIALYSIS FOR CHRONIC RENAL FAILURE

(a) The patients before the start of haemodialysis

Between August 1984 and August 1985, 22 patients with chronic renal failure were placed on maintenance haemodialysis. By August 1985 the total duration of dialysis was 170 patient-weeks. On the 24 August 1985 four patients were being dialysed in hospital, two had been transplanted, and sixteen had died (Table 11). There were 16 men and 6 women. At the beginning of treatment their ages ranged from 9 to 55 years, mean 25.4 years.

The frequency of symptoms and signs in these patients is summarised in tables 7 and 8. At the time maintenance dialysis was started six patients were in moribund condition, 8 patients were considered to have started treatment at the optimum time, and the remaining 8 patients were somewhere between these two groups.

Of the six moribund patients, three had had generalised fits, three had pericarditis and one severe gastrointestinal bleeding. One of this group was treated with peritoneal dialysis before maintenance haemodialysis was started. The 8 patients considered to have started maintenance haemodialysis at the optimum time had none of these complications; only one required peritoneal dialysis before haemodialysis was started. Of the remaining 8 patients whose clinical state was somewhere between the moribund and optimum group, two had pericarditis and two patients required peritoneal dialysis, and in both this was complicated by peritonitis. Pericarditis was diagnosed on clinical, electrocardiographic and echocardiographic evidence.

The endogenous creatinine-clearance on starting maintenance haemodialysis was less than 5 ml/min in 8 patients, and between 5 and 10 mls/min in four patients; no clearances were available in ten patients. The plasma creatinine levels prior to starting haemodialysis varied between 500 and 1800 $\mu\text{mol/L}$, with a mean of 774 $\mu\text{mol/L}$. In fourteen patients the plasma creatinine was between 500 and 1000 $\mu\text{mol/L}$, in six patients between 1000 - 1500 $\mu\text{mol/L}$ and in two patients above 1500 $\mu\text{mol/L}$. The blood urea nitrogen levels prior to starting haemodialysis varied between 20 and 116 mmol/L , with a mean of 58.5 mmol/L . In two patients the blood urea nitrogen was more than 100 mmol/L , in four patients between 60 and 100 mmol/L , and in sixteen patients between 20 and 60 mmol/L .

Chronic glomerulonephritis was the presumed (clinical and laboratory evidence) underlying renal disease in 13 patients, chronic obstructive uropathy in two, hypertensive nephrosclerosis in three, two patients had unilateral renal hypoplasia with nephrosclerosis in the other and in two patients no presumed renal disease was assigned. Malignant hypertension with papilloedema was present in three patients, severe hypertension without papilloedema in ten patients and moderate hypertension in nine patients.

Table II

SUMMARY OF EXPERIENCE - CHRONIC RENAL FAILURE PATIENTS ON HAEMODIALYSIS

Patient	Age (Years)	Sex	Presumptive Diagnosis	Weeks of Dialysis	Numbers of Dialysis	Outcome
R . C	20	F	Chronic Glomerulonephritis	4	11	Dead
B . O	39	M	Hypertensive nephrosclerosis	0.5	2	Dead
M . W	12	F	Chronic Glomerulonephritis	19	55	Dead
L . K	18	F	Hypertensive nephrosclerosis	4	12	Dead
A . M	28	M	Chronic Glomerulonephritis	36	110	Living
S . M	15	M	Chronic Glomerulonephritis	5	13	Dead
R . A	15	F	Chronic Glomerulonephritis	13	37	Dead
M . M	9	F	Chronic Glomerulonephritis	0.5	1	Dead
A . H	30	M	Chronic Glomerulonephritis	0.5	1	Dead
K . M	42	M	Chronic Glomerulonephritis	0.5	1	Dead
G . E	42	M	Chronic Glomerulonephritis	10	31	Dead
M . M	55	M	Obstructive Uropathy	5	16	Dead
J . G	25	M	Chronic Glomerulonephritis	1	3	Dead
G . G	51	M	Obstructive Uropathy	15	44	Living
J . K	45	M	Diabetic Nephropathy	1	2	Dead
M . A	15	M	Tumour infiltration	3	8	Dead
J . K	35	M	Hypertensive nephrosclerosis	20	60	Living
J . A	35	M	Hypoplasia, Nephrosclerosis	10	31	Living
J . G	49	M	Chronic Glomerulonephritis	4	12	Dead
M . M	18	F	Hypoplasia, Nephrosclerosis	4	12	Dead
S . T	24	M	Chronic Glomerulonephritis	7	21	Living
H . M		M	Chronic Glomerulonephritis	7	20	Living

(b) The patients after the start of maintenance haemodialysis

Only patients dialysed for more than four weeks are considered in the analysis. The number totalled 15.

(i) Changes in weight and the concentration of certain plasma constituents

During the initial weeks of dialysis in order to control the blood-pressure, it was often necessary to reduce the patients weight by ultrafiltration during dialysis, and by restricted salt and fluid intake between dialyses.

A mean of 4.5 kg (range 0.5 to 12.5 kg.) was removed from 15 patients in their first 4 - 8 weeks of dialysis. Subsequently, the weights of the patients have been variable with some gradually increasing (4 patients) some static (8 patients) and some continuing to loose weight (3 patients).

In the patients dialysed for four weeks and above, the biochemical data for the last three dialysis were averaged. The mean plasma creatinine for all the patients was 250 $\mu\text{mol/L}$ range (129 - 450 $\mu\text{mol/L}$). The mean blood urea nitrogen was 14.7 mmol/L (range 7.0 - 32.5 mmol/L).

(ii) Survival

Of the four patients on haemodialysis on 24th August 1985, one had been on dialysis for 20 weeks, two for seven weeks and one for six weeks.

Between August 1984 and August 1985 sixteen (72.7%) of the 22 patients placed on haemodialysis had died. Two

patients had renal homotransplants following 14 and 36 weeks of haemodialysis, One of the two is doing well and is being followed up. The other developed acute renal failure due to vessel thrombosis and is back on the dialysis programme awaiting a second transplant.

(iii) Deaths

There were 16 deaths in the one year period. The causes of deaths are shown in Table 12. Pulmonary oedema was the commonest cause of death accounting for 31.3% of all deaths. Pulmonary thromboembolism, uraemia, cardiac tamponade and intracranial haemorrhage each caused 12.5% of the deaths. Other causes of deaths included dialysis disequilibrium and septicaemia.

(c) Problems in haemodialysis

(i) Arteriovenous shunt problems

Upto 24th August 1985, 39 arterial cannulae have been put in place in 22 patients in 170 patient-weeks. Seventeen of these cannulae, therefore, were replacements. The mean arterial cannula survival time was therefore 4.1 weeks, otherwise expressed as an arterial cannula replacement rate of one arterial renewal per 10 patient-weeks. Similarly, 46 venous cannulae were put in place in these 22 patients. Twenty four of these venous cannulae, therefore, were replacements, and the venous cannula replacement rate was one per 7.1 patient-weeks (mean venous cannula survival time of 3.8 weeks) (Fig. 7).

CAUSES OF DEATH - CHRONIC RENAL FAILURE PATIENTS ON HAEMODIALYSIS

Patient	Age (Years)	Sex	Weeks of Dialysis	Cause of Death
R . C	20	F	4	Septicaemia
B . O	39	M	0.5	Pulmonary Embolism*
M . W	12	F	19	Uraemia
L . K	18	F	4	Pulmonary oedema
S . M	15	M	5	Pulmonary oedema
R . A	15	F	13	Cardiac Tamponade*
M . M	9	F	0.5	Uraemia, septicaemia
A . H	30	M	0.5	Disequilibrium Syndrome
K . M	42	M	0.5	Pulmonary oedema
G . E	42	M	10	Cardiac Tamponade*
M . M	55	M	5	Cerebral Haemorrhage*
J . G	25	M	1	Pulmonary oedema
J . K	45	M	1	Pulmonary oedema
M . A	15	M	3	Luekaemia, Haemorrhage*
J . G	49	M	4	Cerebral Haemorrhage*
M . M	18	F	4	Pulmonary Embolism

* Postmortem examination done

There have been 80 clotting episodes in 22 patients in 170 patient-weeks, that is, one clotting episode per 2.1 patient-weeks.

Significant bleeding due to the presence of an arteriovenous cannula has occurred 34 times in 22 patients in 170 patient-weeks, that is one bleeding episode per 5.0 patient-weeks. One bleeding episode was due to a disconnection of the arteriovenous cannula, ten have been due to poor ligation of bleeders during institution of cannula and the rest were due to capillary oozing either due to uraemia or associated with the use of heparin infusion during haemodialysis.

A shunt infection was diagnosed when there were signs of acute inflammation at the shunt site, such as pain, tenderness, and redness (shininess) with or without pus formation. Between August 1984 and August 1985 40 shunt infections were seen; a rate of one infection per 4.3 patients weeks.

Staphylococcus aureus was responsible for 62.5% (25) of the infections; Staphylococcus albus for another 20%, 7.5% of the infections was attributed to Pseudomonas aeruginosa; 5% to Escherechia coli; and in 5% no pathogens were isolated, despite obvious signs of acute inflammation and pus formation. Photograph 8 shows one of the infected cannula sites.

Oedema of varying amount occurred in the cannulated limbs in virtually all the cases. In 5 cases the oedema was very marked and required the use of antiinflammatory drugs including chymoral (Photograph 9).

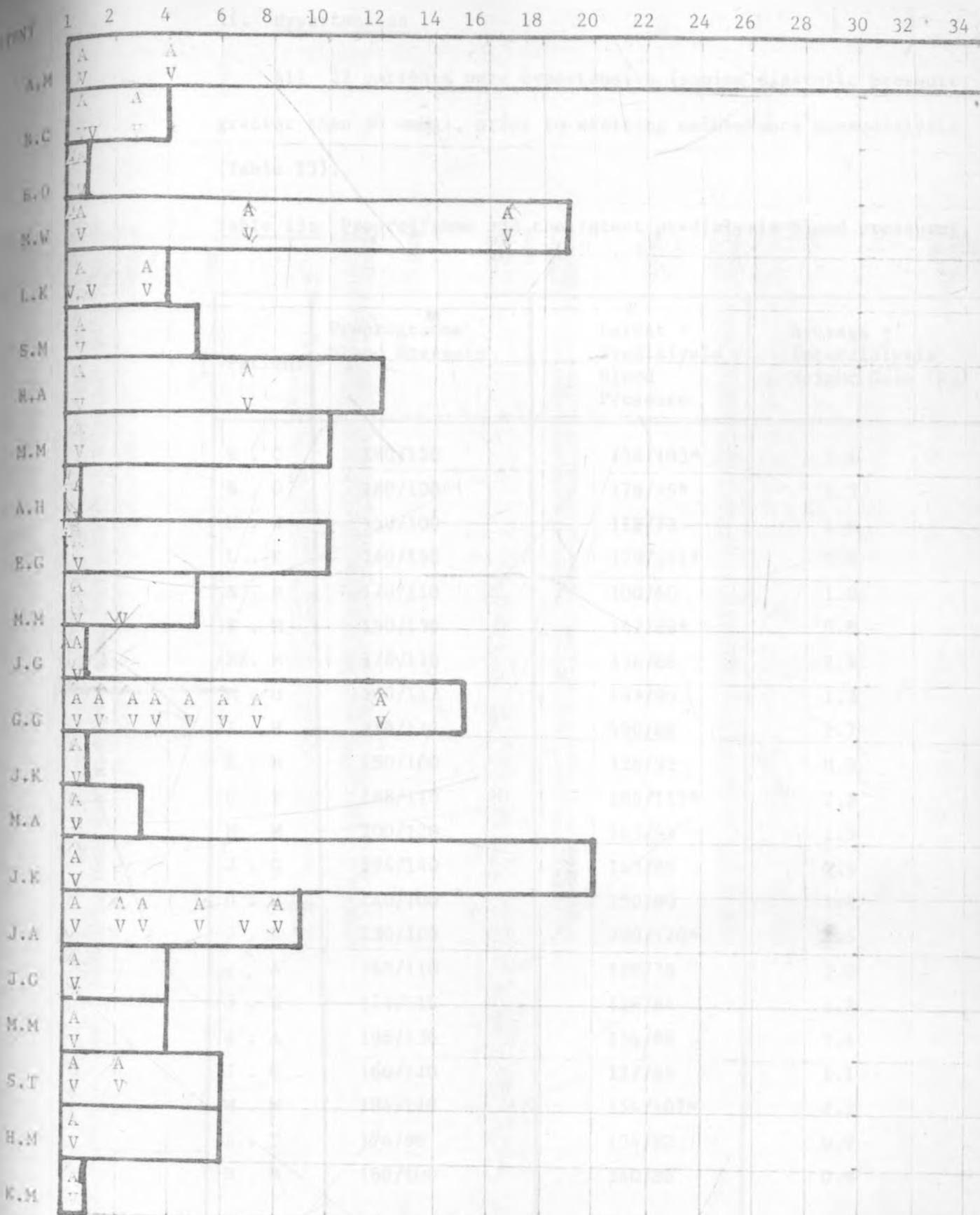


Photograph 8; One of the infected cannula sites. Infection led to cannula replacement. Pus swab from this site grew staphylococcus aureus.



Photograph 9: Gross oedema of the shunted limb occurring after three days.

CANNULA SURVIVAL (WEEKS)



MEAN CANNULA SURVIVAL TIME (WEEKS)

ARTERIAL - 4.1

VENOUS - 3.8

Fig. 7: Representation of cannulations related to individual dialysis programs. Program weeks are indicated at the top of the chart (A - arterial cannulation, V - venous cannulation).

ii. Hypertension

All 22 patients were hypertensive (supine diastolic pressure greater than 90 mmHg), prior to starting maintenance haemodialysis (Table 13).

Table 13: Preprogramme and the latest predialysis blood pressures.

Patient	Preprogramme Blood Pressure	Latest + Predialysis Blood Pressure	Average + Interdialysis Weight Gain (Kg)
R . C	180/120	158/103*	1.1
B . O	180/100	178/95*	2.3
M . W	150/100	118/73	1.6
L . K	240/150	179/111*	0.9
A . M	170/110	100/60	1.0
S . M	190/136	147/89*	0.8
RR. M	178/110	136/86	2.9
M . M	160/112	143/90	1.3
A . H	210/112	150/88	1.7
K . M	150/100	128/92	0.9
G . E	188/110	185/117*	2.5
M . M	200/120	163/95	1.5
J . G	196/140	143/89	1.9
G . G	140/100	150/90	1.4
J . K	230/160	200/120*	2.6
M . A	160/110	128/78	1.0
J . K	174/110	126/84	1.2
J . A	198/130	134/88	1.4
J . G	160/110	127/85	1.1
M . M	184/110	154/107*	2.2
S . T	176/96	134/82	0.8
H . M	160/100	140/80	0.9

+ Average of three latest dialyses

* On hypotensive drugs

A degree of blood pressure control was achieved in 13 of 22 patients without the routine use of antihypertensives. The rest of the patients were either on antihypertensives or had been dialysed for a very short period. The patients whose blood pressures remained high tended to have the greater interdialytic weight gain with the tendency to developing pulmonary oedema.

(iii) Transfusion requirements

Patients were transfused when the haematocrit fell to below 21% or if it was higher but the patient was symptomatic. All patients were suffering from the anaemia of chronic uraemia. Subjectively they felt better at haematocrit readings above 24%.

The requirement for blood transfusion varied from patient to patient but averaged approximately 2 units of whole blood per month.

(iv) Pericardial effusion

Pericardial effusion was present in 5 of the 22 patients at the onset of maintenance dialysis. In two other patients the pericardial effusion was first diagnosed after 10 and 14 weeks of dialysis.

(v) Haemorrhage (haemoptysis and haematemesis)

One patient of the twenty two had recurrent haematemesis. Endoscopic examination of the upper gastrointestinal tract revealed no significant lesion. Another patient had haemoptysis on and off, but the radiological and spirometric examinations were within normal limits. Bronchoscopy was not done. Hae-

moptysis was thought to be due to the bleeding tendency of uraemia.

(vi) Febrile reactions

No significant temperature elevations were observed during dialysis, except in four occasions during transfusions, but these tended to settle in 1 - 2 hours after stopping the transfusion,

(vii) Ascites

8 of the 22 patients had ascites during haemodialysis. Three of these had had prior peritoneal dialysis with peritonities, two had an associated pleural effusion and five had congestive cardiac failure at the time of development of ascites. In seven of the patients the ascites responded to therapy with diuretics, antibiotics where indicated and to ultrafiltration during dialysis. In one patient the ascites developed in the absence of any predisposing cause and was not responsive to any of the above forms of therapy and repeated tapping. The latter patient had progressively increasing ascites and cachexia and was considered to have refractory ascites of haemodialysis.

Tables 14 and 15 show the clinical features and pertinent laboratory data on the eight patients. Figure 8 shows the temporal relationship between haemodialysis and ascites.

Photograph 10 shows the patient considered to have refractory ascites of haemodialysis.

CLINICAL FEATURES OF EIGHT PATIENTS WITH ASCITES DURING HAEMODIALYSIS

patient	Age in Years (SEX)	Presumed Diagnosis	Peri- toneal Dial- ysis	Duration, weeks		Severity of Ascites	Peri- pheral Oedema	Pleural effusion	Peri- card- itis	Con- ges- tive Heart fail- ure	Hyper- ten- sion	Outcome
				of Ascites	on HD							
R . C	20 (F)	Chronic glomeru- lonephritis	YES	10	-6	Moderate	YES	YES	YES	YES	NO	Dead
L . K	18 (F)	Hypertensive Ne- phrosclerosis	NO	2	+2	Mild	NO	NO	NO	YES	YES	Dead
A . M	28 (M)	Chronic glomeru- lonephritis	NO	3	+14	Mild	NO	NO	NO	NO	NO	Alive
R . A	15 (F)	Chronic glomeru- lonephritis	YES	36	-22	Moderate	YES	YES	YES	YES	YES	Dead
E . G	42 (M)	Chronic glomeru- lonephritis	YES	16	-6	Moderate	NO	NO	YES	NO	YES	Dead
M . M	18 (F)	Renal Hypoplasia with Nephros- clerosis	NO	6	-2	Mild	YES	NO	NO	YES	YES	Dead
J . K	35 (M)	Hypertensive Ne- phrosclerosis	NO	8	+12	Mild	YES	NO	YES	YES	YES	Alive

1. Duration of Haemodialysis before appearance of Ascites.

Table 15:

PERTINENT LABORATORY DATA ON PATIENTS WITH ASCITES DURING HAEMODIALYSIS

Patient	Age in Years (SEX)	Serum		ASCITIC FLUID								
		Total	Protein Albumin	Culture	Specific gravity	Total Protein	Albumin globulin ratio	White blood cells	%Lymphocytes	Proteinuria	Prothrombin index	ALT.
R . C	20 (F)	67 g/L	32 g/L	Pseudo-aeruginosa	1.020	32 g/L	1.38	750/mm ³	34%	Trace	78%	26K.U
L . K	18 (F)	62 g/L	34 g/L	Negative	1.014	20 g/L	-	10/mm ³	100%	Nil	100%	15K.U
A . M	28 (M)	66 g/L	33 g/L	Negative	-	26 g/L	-	5/mm ³	100%	Nil	70%	38K.U
R . A	15 (F)	60 g/L	31 g/L	Staph-Aureus	1.023	33 g/L	1.13	1050/mm ³	40%	Trace	84%	52K.U
E . G	42 (M)	71 g/L	35 g/L	Negative	1.019	28 g/L	1.00	1180/mm ³	30%	Nil	100%	45K.U
G . G	51 (M)	75 g/L	33 g/L	Negative	1.021	35 g/L	2.60	500/mm ³	80%	Trace	88%	43K.U
J . K	35 (M)	68 g/L	34 g/L	Negative	1.012	20 g/L	-	20/mm ³	100%	Nil	80%	48K.U
M . M	18 (F)	63 g/L	36 g/L	Negative	1.014	26 g/L	-	10/mm ³	100%	Nil	67%	50K.U

ALT - Alanine aminotransferase



Photograph 10: G.G., A patient thought to have developed Idiopathic ascites of haemodialysis. Note the gross ascites and marked wasting.

— Ascites

* - Prior peritoneal dialysis



Fig. 8: The duration of haemodialysis (shaded bars) and Ascites (solid bars) and the temporal relation of each is shown for

(viii) Hepatitis

Of the 22 patients with chronic renal failure so far dialysed, one has had serological (Hepatest) evidence of hepatitis B virus infection, None has had any clinical or biochemical evidence of hepatitis.

After working in the dialysis unit for about nine months one staff member has developed clinically, biochemically and serologically proven hepatitis B virus infection.

(ix) Hearing loss

Audiograms were obtained in 4 of the patients. All the four were abnormal, showing varying degrees of bone conduction abnormalities. In one patient who clinically had a hearing defect, the gross abnormality was temporarily related to the administration of large doses of lasix at the initiation of dialysis.

(x) Pulmonary Oedema between dialyses

Ten of the 22 patients developed sixteen episodes of pulmonary oedema in ^{the} interdialytic period. In 5 of them this was fatal. The pulmonary oedema was diagnosed clinically and with the help of radiology in some cases. The cause of the pulmonary oedema was considered to be due to fluid overload in patients who were anaemic, hypertensive and had impaired cardiac function.

(xi) Disequilibrium Syndrome

This was seen in 3 of the 22 patients. In all it occurred during the first dialysis. It was characterised by headache, nausea, vomiting, hypertension, restlessness, twitches, convulsions, confusion and coma. One of the patients died. The other two were salvaged by infusion of mannital. The three patients who developed disequilibrium syndrome had high pre-dialysis blood urea nitrogens (87.5, 77 and 166 mmol/L) and correspondingly high serum creatinine level (1000, 874, 1800 mcmol/L). These dropped to (45.6, 44.0 and 56.0) and (820, 546 and 1050) respectively, after dialysis.

(xii) Hypotension, Muscle cramps, Nausea, vomiting, headache

In a total of 503 dialyses carried out in patients with acute and chronic renal failure, hypotension was observed in 26%, Muscle cramps in 20%, Nausea and vomiting in 15%, Headache in 18%.

(xiii) The first - use Syndrome

This occurs as a feeling of ill-being at the start of dialysis with a new dialyser. The syndrome is characterised by dyspnoea, chest constriction, nausea, vomiting and hypotension, sometimes culminating in cardiopulmonary collapse. This has occurred in one patient of the 42 who have been started on haemodialysis.

V.

DISCUSSION

That haemodialysis is of value in acute renal failure, chronic renal failure, acute intoxications and in states of overhydration is uncontestable (24). Less well defined indications of haemodialysis have included myasthenia gravis (44), Schizophrenia (44) and Psoriasis (45),

At the Kenyatta National Hospital haemodialysis has exclusively been carried out in patients with various renal failure syndromes and in states of overhydration related to the presence of renal diseases. No patient with acute intoxication has so far been started on haemodialysis. This has been in part due to the limited availability of dialysis and staff. On the other hand most intoxications have occurred in the paediatric age groups and peritoneal dialysis and/or conservative management have been given preference because of the problems encountered with angioaccess for haemodialysis in this age group.

In the period between July 1973 and June 1982, haemodialysis at Kenyatta National Hospital was specifically meant for patients with acute renal failure in whom recovery was anticipated (90). The other patients dialysed either had acute or stable chronic renal failure and had haemodialysis to tide them over the acute phase or had unidentified renal disease and were dialysed to allow time for confirmation of diagnosis. Recently, with the establishment of a dialysis centre at the hospital, there has been a deliberate effort to start patients with confirmed chronic renal failure on maintenance haemodialysis, all with a view to giving them renal transplantation at some time or another. The choice of the patients has been made using the

guidelines outlined in appendix I.

For the past two and a half decades, the emphasis in most centres has been on prophylactic dialysis for acute renal failure rather than waiting until pushed by a catastrophic biochemical or clinical occurrence (14, 91 - 93). This principle has been followed at Kenyatta National Hospital recently, where of 20 patients with acute renal failure in a one year period (August 1984 to August 1985), none was moribund at the onset of haemodialysis and ^{They} had biochemical parameters as follows: potassium 6.2 ± 1.3 mmol/L (mean \pm 2sD), blood urea nitrogen 32.5 ± 5.4 mmol/L (mean \pm 2sD) and serum creatinine 345 ± 100 μ mol/L (mean \pm 2sD). In the earlier period (July 1973 to June 1982), 48% of the 31 patients with acute renal failure started on haemodialysis were moribund and the biochemical parameters were 5.6 ± 2.8 mmol/L (mean \pm 2sD) for potassium, 55.5 ± 10.4 mmol/L (mean \pm 2sD) for blood urea nitrogen and 417 ± 216 μ mol/L for serum creatinine.

The mortality in patients with acute renal failure on haemodialysis was 30% for the patients started on haemodialysis between August 1984 and August 1985 as compared to 61.3% in patients with acute renal failure dialysed between July 1973 and June 1982. The former figure is closer to the ones reported in other series (14, 92, 94 and 95).

The mortality in acute renal failure is influenced by various factors apart from timing and frequency of dialysis. Thus, Teschan and associates (14) in 1960 found that when renal

failure is due to obstetrical accidents, the mortality was 7% with prophylactic dialysis and 15% without it. The corresponding figures for acute renal failure of medical aetiologies was 29% and 40%. The mortality in postsurgical and post traumatic renal failure ranges from 47% (14) to 67% (95). These figures are higher in the presence of associated sepsis (44). Age has been a major factor associated with increased mortality in some series (96). In the retrospective study, 46.3% of the patients who died had gynaecological problems associated with sepsis and disseminated intravascular coagulation, 6.4% obstetrical haemorrhage and 10.3% surgery with haemorrhage. All the patients were below 50 years of age. The mean blood urea nitrogen for the patients who died was 64.3 ± 12.2 mmol/L. Thus the major causes of mortality seemed to be the degree of uraemia and the nature of the primary illness causing the uraemia. In the 6 patients with acute renal failure who died in the prospective study period, all developed acute renal failure post-surgery, except for one who had cholera, but died of pulmonary oedema due to overzealous fluid replacement without close monitoring.

The first deliberate attempt to restore useful life through repeated dialysis of a patient with obviously terminal, irreversible renal disease was reported by Scribner and his associates in 1960 (17). Before then haemodialysis was deemed of limited value in the treatment of chronic renal failure (15, 16). Two and a half decades after the recognition of the usefulness of haemodialysis in the management of chronic renal failure, little information exists as to its application in the

African continent. There is evidence, however, that it has been used in South Africa extensively (97, 98) and to some extent in Egypt (87). At the Kenyatta National Hospital, deliberate use of haemodialysis in patients with chronic renal failure has been going on for only one year (August 1984 to August 1985). Before then haemodialysis was restricted to patients with reversible renal disease (89, 90),

The criteria for selection of patients for the chronic haemodialysis and transplant programme are outlined in Appendix I. There is no accurate data as to the mortality rate of chronic renal failure in Kenya at the moment, but so long as treatment capability remains low, the onerous task of patients selection must go on. There is as yet no universally acceptable set of criteria by which such decisions can be made, nor is one likely to be developed which is consonant with all values of civilised man (99, 100). Human worth is not measurable except by arbitrary standards, with or without the aid of "Selection committees" and prediction as to total response to any patient prior to clinical trial remain precarious.

Age alone is no longer a leading consideration in selecting patients for most haemodialysis programmes (101, 102). The presence of other diseases which might limit the clinical benefits of dialysis constitute a contraindication. In some instances, diabetes (103), Goodpastures syndrome (104), and lupus erythematosus (115) have not precluded the success of chronic dialysis. Estimates of the patients' intelligence, discipline, motivation and family setting, tend to enter into the decision-making process (99, 100, 107). In truth, however, it must be

recognised that a major determinant of patient selection is simply whether or not a chronic dialysis facility exists in the area, and whether or not it can accept another patient. It can be argued that a "first come first served" basis may be the only ethically acceptable one. Examination of our total experience in the area of shunt survival reveals a mean survival of 4.1 weeks for arterial cannulae and 3.8 weeks for venous cannulae. This is considerably less than the 12.4 and 13.9 months survival reported by Pendras and associates (108) and 4.8 and 4.5 months survival reported by Schupak et al (109).

The commonest shunt complication in this study has been clotting, occurring at a rate of one episode per 2.1 patient-weeks. Infections occurred at a rate of one per 4.3 patient-weeks and significant bleeding at a rate of one episode per 5.0 patient-weeks. The commonest infecting organism in this study was Staphylococcus aureus, accounting for 62.5% of the infections. This is similar to what is reported in other series (110, 111, 112). The short survival time for our cannulae can be explained on the basis of the complications above, which in turn can be explained on the basis of (a) hesitancy to use specific antibiotics for specific infections, (b) insertion of cannulae by varying groups of doctors, most with little experience in cannula surgery, and (c) lack of appropriate cannulae care once inserted because of shortage of staff experienced in cannula care.

In this study all the patients with chronic renal failure started on haemodialysis were hypertensive (supine diastolic pressure greater than 90 mm Hg). The mean blood pressures of all

the patients before the start of haemodialysis was 181 ± 12 mm Hg systolic by 116 ± 6 mm Hg diastolic. Following a minimum of four weeks dialysis the corresponding figures were 146 ± 5.5 mm Hg and 91 ± 6.2 mm Hg. In the patients in this study blood pressures control was achieved by ultrafiltration alone in 59% of the patients. The other patients required to be on hypotensive drugs for control of blood pressure, but none seemed resistant to most antihypertensives, except one.

There are two main factors in the aetiology of hypertension in patients with chronic renal failure: Volume and salt overload and the renin-angiotensin system (113). In 80-90% ^{of} patients ^{the} control of hypertension is normally achieved by dietary restriction of salt and water and ultrafiltration during dialysis. In the remaining 10-20%, antihypertensives and occasionally bilateral nephrectomy is required for control of blood pressure (113). This latter group is usually considered to have hypertension due to elevated plasma renin levels. In this study it has not been possible to estimate the plasma renin levels in the patients. Additionally, dietary restriction in the patients has been very difficult. This could account for the low percentage of patients whose hypertension have been controlled by ultrafiltration. Bilateral nephrectomy is currently being considered for one patient (J. K.) who has been on dialysis for twenty weeks and has been treated with maximum dosages of antihypertensives without achieving control of blood pressure.

In patients in this study, hypotension has occurred in 26% of the 503 dialyses, while muscle cramps have occurred in

20%. Reports from other centres give prevalence of hypotension as 22 - 44% and that of muscle cramps as 13 - 54% (58, 114 - 117).

Hypotension is a frequent occurrence in chronic haemodialysis patients during the dialysis procedure. The subject has been reviewed in detail by Rubin and Gutman (118), who summarised the well known cardiac factors (coronary-artery disease, cardiomyopathy, pericardial involvement, arrhythmias), haemodynamic factors (anaemia, arteriovenous fistulae, extracellular fluid volume changes, altered peripheral resistance), pulmonary factors (pulmonary oedema, calcification, infection), and dialysis-related factors (ultrafiltration, blood loss, endotoxaemia, bacteraemia, haemolysis, hypoxaemia, acetate infusion, osmolality changes). In this study, episodes of hypotension occurred during attempts at ultrafiltration in 70%. In the other cases no obvious predisposing factor was detectable, except for anaemia which was present in all the patients and acetate which was present in the dialysate used in all the patients.

It is generally accepted that muscle cramps are unavoidable accompaniments of dialysis, especially when a lot of fluid is removed, and that palliative treatment is all that is required for these complication (119). In this study the sodium concentration in the dialysate was 138.0 mmol/l and the glucose was 3.0 g/l. The high strength of the dialysate in this study could explain the relative infrequency of muscle cramps.

In this study transfusion requirement averaged 2 units of whole blood per month. Patients were not transfused unless they were symptomatic or the haematocrit fell to below 21%. All the

patients had anaemia attributable to their chronic renal failure. Other possible causes of anaemia included blood loss from the shunt site, withdrawal of blood for analysis at each dialysis, blood loss into the dialyser at the end of dialysis, inadequate nutritional intake due to inadequate dialysis and possible infections. Thomson and associates (120) report a requirement of 2.5 units of packed red blood cells per month in adequately dialysed patients. In the recent past, deliberate transfusion, based on immunetolerance has been advocated to promote graft survival in patients due to have homotransplants (121).

23% of the patients in this study had pericarditis with effusion before the onset of haemodialysis. All of these resolved on dialysis. Two patients however developed pericarditis with effusion 10 and 14 weeks after the onset of haemodialysis. These were attributed to inadequate dialysis with ongoing uraemia. Pericarditis has become recognised as an important cause of morbidity in patients on a chronic, intermittent haemodialysis programme. Barber and colleagues (122) found evidence of pericarditis in four out of thirteen post-mortem examinations performed after prolonged haemodialysis. Comty and associates (123) reviewing pericarditis in chronic uraemia found an incidence of 16% in all patients accepted for haemodialysis. Mitchell (124) reported pericarditis in 18% of patients accepted for haemodialysis.

It is possible that chronic dialysis modifies the natural history of uraemic pericarditis to produce a distinct form of illness. It is felt, though, that ^{This} is a separate disease. The aetiology remains obscure and while infective illnesses pre-

dispose to pericarditis, there is little evidence that the pericarditis is infective (24). Pabico and associates have suggested that cytomegalovirus may act as a causative agent (125).

Ascites resistant to treatment has become an increasingly recognised problem in many dialysis centres. In the majority of patients described, no specific cause of ascites has been described (81, 82). Previous peritoneal dialysis has been considered to play an aetiological role in such patients (82). Investigators have treated these patients in several ways: with continuous drainage, frequent paracentesis, exploratory laparotomy and total ascites drainage, bilateral nephrectomy, or kidney transplant all with different results (126 - 131). Recently, the use of non-absorbable triamcinolone, oral indomethacin and intraperitoneal radioactive chromium have been tried with some positive results (132).

In this study eight of the 22 patients had ascites during haemodialysis. In five of these the ascites was considered to be due to fluid overload and congestive cardiac failure. In another two, previous peritoneal dialysis and peritonitis were considered responsible. In one patient (G. G) exudative ascites developed without any obvious precipitating cause. The ascites progressively increased in size and was associated with marked cachexia. It showed no response to frequent attempts of ultrafiltration. This patient was considered to have idiopathic ascites of haemodialysis. Becerra and Ontiverous (132) postulate that ascites of uraemic patients on haemodialysis is a part of what they call "Uraemic polyserositis" and that it is caused by inflammation. In some published reports of patients with ascites

undergoing long-term haemodialysis, hypoproteinaemia was the rule (128 - 131). The possibility of altered peritoneal permeability because of repeated peritoneal dialysis has been advanced as a cause (130, 131). Overhydration has been the constant feature in some reported cases (127 - 129).

Dialysis disequilibrium syndrome was first described by Kennedy in 1962 (133). It was him who proposed the pathophysiological explanation of a "reverse urea effect" the mechanism of which is the rapid build-up of an osmotic gradient across each side of the blood-brain barrier. Confirmation of this hypothesis was obtained by the work of Dossetor and Zborowski (134) who prevented or reduced the clinical disorder by addition of urea to the dialysis bath. However, more recent studies have shown in fact that this simple mechanism is not sufficient to explain the disequilibrium syndrome. Arieff and associates (52) have demonstrated in the dog that a rapid dialysis creates an osmotic gradient between;

- (1) Plasma and cerebrospinal fluid, and
- (2) Cerebrospinal fluid and brain cells, the water content of which increases. However, they failed to identify the osmotically active substance(s) which they simply defined as "idiogenic osmoles".

Evidence has also been presented for mechanisms other than merely "osmotic", for example, rapid swings of acid base balance (51), of blood glucose (135), of serum calcium (51), of serum sodium and of the hydration state (51). Another interesting hypothesis deals with the correction of acidosis and hyperphosphataemia which is able to shift the HbO_2 dissociation curve

to the left whereby tissue hypoxia due to renal anaemia might be aggravated (136),

The usual clinical descriptions of this syndrome are relatively simple (133) and consist of headache, nausea and vomiting, drowsiness or agitation, muscular cramps and twitching. More severe disorders such as disorientation and mental confusion with occasional asterixis can occur and in some cases acute psychosis, seizures, stupor and coma are also seen.

The clinical signs and symptoms occur mainly during the first haemodialysis (137). They disappear in less than 36 hours. The rate of change of serum sodium, urea or other substance is probably more important than is the quantity of any substance removed (138).

In this study three of the twenty two patients developed disequilibrium syndrome during or immediately after the first dialysis. One of the patients died while the other two were salvaged by mannitol infusion. The patients who developed disequilibrium syndrome had very high predialysis blood urea nitrogen and were extremely acidotic. Attempts at preventing dialysis disequilibrium have included prior peritoneal dialysis or haemodialysis of short duration and low blood flow rates to facilitate a gradual fall in the serum concentration of the osmotically active substance.

Acute pulmonary oedema is a frequent emergency in dialysed patients (139). It may be a reflection of pericarditis, masked valvular heart lesion and cardiomyopathy associated with uraemia (123, 139, 140). However, the commonest causes of pulmonary

oedema between dialysis are inadequate dialysis and poor fluid and salt control (141) in patients who are already anaemic, have heart muscle disease due to uraemia and/or hypertension and are bound to be hypoproteinaemic due to their renal disease. Robson and associates consider the use of high dialysate sodium concentration as a possible cause of pulmonary oedema in patients on haemodialysis (142). An interesting pulmonary oedema associated with blood membrane complement mediated pulmonary leucostasis evoking mild pulmonary oedema has been described (143).

In this study ten of the twenty two patients on maintenance dialysis developed sixteen episodes of pulmonary oedema. 31.3% of these episodes were fatal. The common reasons for developing pulmonary oedema were severe hypertension (diastolic blood pressure greater than 120 mm Hg), anaemia (Haemoglobin less than 8 g/dl), inadequate dialysis with poor removal of fluid, clotted shunts with inability to get access sites and in some situations, inability to get dialysis fluid in time.

The syndrome of "first-use" is characterised by dyspnoea, chest constriction or angina, nausea, vomiting and hypotension, sometimes culminating in cardiopulmonary collapse (144). Hakim and associates (145) described this syndrome in 3% to 5% of their dialysis population and attributed its occurrence to the in vivo complement activation by a new cuprophane membrane. This has been confirmed by several investigators (146 - 148), who presented convincing evidence by quantitative determination of the generation of complement fraction C_{3a}.

In the current study, one patient of the 22, developed the syndrome at each dialysis, stabilising only after some period of dialysis, usually 30 - 60 minutes. At our centre all dialysers are currently used only once and thereafter discarded.

In the current study 72.7% of the twenty two patients with chronic renal failure started on maintenance haemodialysis have died. This is in sharp contrast to the annual mortality of 7 - 10% reported in other series (84, 85). The commonest causes of death in this study were congestive cardiac failure and pulmonary oedema due to inadequate dialysis and fluid overload (31.3%), pulmonary embolism (12.5%), uraemia (12.5%), cardiac tamponade (12.5%) and intracranial haemorrhage (12.5%). Other causes of death were dialysis disequilibrium, ^{and septicæmia.} This compares with causes of death in European dialysis centres in the late sixties where prominent causes of death were congestive cardiac failure, electrolyte imbalances and infections (149).

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VII.

APPENDIX I

KENYATTA NATIONAL HOSPITAL RENAL UNIT

POLICY FOR ACCEPTABILITY OF DIALYSIS TREATMENT AND TRANSPLANTATION

Acute Renal Failure (ARF)

1. All patients with reversible acute renal failure without advanced malignancies.
2. Patients will be individually assessed by the Renal Team eventually, but under emergency situation the nephrologist on call will decide on the immediate treatment.
3. The guidelines for immediate treatment of acute renal failure should include too much fluid accumulation, elevation of potassium, urea, creatinine and presence of acidosis.

Chronic Renal Failure (CRF)

General criteria for acceptance of dialysis:

1. The patient must be transplantable.
2. Patients must have no serious medical problems (advanced malignancies, elderly or complicated diabetics, nor beyond 55 years and complicated collagen diseases).
3. Must have no serious mental or physical handicap.
4. Patients who are socially, educationally and economically useful to the society.
5. The patient must be well motivated.
6. The family must not only be well motivated but also concerned and actively involved.

7. The home and social circumstances must be favourable.²
8. The final decision on the acceptability or non-acceptability of the individual patient must remain with the renal team.

Peritoneal Dialysis for CRF

All patients who meet the general criterial set above.

Haemodialysis

1. All the criteria for ARF and CRF must be met.
2. Patients must be HBs Ag. negative but those who are positive will be put on an identified machine in isolation room.

Acceptability for Transplantation

1. Must meet all the general criteria.
2. Should not be beyond 55 years.
3. Should have live related donor.
4. Should ideally be HBs Ag negative. (Hv)
5. Should accept alternative treatment should transplantation fail.

CAPD (Continuous Ambulatory Peritoneal Dialysis)

1. General criteria should be met (for CRF).
2. This treatment can be offered to CRF patients awaiting transplantation or those in whom transplantation has failed, or those who have no vascular access or those who cannot tolerate haemodialysis.

IPD (Intermittent Peritoneal Dialysis)

1. All general criteria should be met (for CRF).
2. Suitable for patients who are over 55 years old or those who for other reasons cannot be offered other forms of dialysis or transplantation.