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Peritoneal dialysis

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PERITONEAL DIALYSIS

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TABLE OF CONTENTS

BASIC PRINCIPLES OF DIALYSIS

From the early investigations the following basic principles concerning peritoneal dialysis were discovered (57). (1) Electrolytes will diffuse across a semipermeable membrane until the concentration is equal on both sides of the membrane. (2) Colloids do not usually diffuse across such a membrane. (3) The loss of electrolytes and fluids which are ordinarily resorbed in the tubules may be replaced by changing the dialysis fluid or can be replaced parenteraly (57). (4) Any biochemical abnormality will be partially corrected by using a physiologic solution as the dialysate (48).

PHYSIOLOGY OF THE PERITONEUM

Dialysis across a semipermeable membrane has been attempted in many portions of the body. Utilization of all levels of the gastrointestinal tract has been attempted by many with various results and complications (13, 55).

Of all the membranes used, the peritoneum seems best suited. The peritoneum is a semipermeable membrane with a filtering (dialyzing) surface of approximately 22,000 sq. cm which is comparable to the human adult renal glomeruli (1, 32, 33, 47, 55, 57). The glomerular function of dialysis through a semipermeable membrane with no selective tubular reabsorption is involved in peritoneal dialysis (76, 77). The peritoneal membrane allows

the passage of water and electrolytes from the extracellular fluid to the peritoneal cavity, but is relative impermeable to protein, erythrocytes, and leukocytes which are found in a limited amount in the dialysate (14). Merrill (52) states that there is no deterioration of the peritoneal membrane when used repeatedly - as exemplified in a case intermittently dialyzed for 9 months with no change in the clearance values. Grollman (32) also found this to be true. Frank, Fine, and Seligman (23) also substantiate this view, stating that the urea clearance does not diminish with time and exceeds the 10-15% renal function necessary for maintaining a physiologic internal environment.

REVIEW OF THE LITERATURE

A. Peritoneal Dialysis in Normal Animals

In 1877, Wegner (71) undertook the first known attempt at peritoneal lavage while studying the effects of the peritoneal fluid upon body temperature. Clark (12) in 1921, found that by increasing the temperature of the dialyzing fluid, the rate of absorption was increased by vasodilitation of the vessels. Through the experiments of Clark (12), Darrow and Yannet (18), Abbott (1), and Cunningham (17), it was discovered that the infusion of a salt poor hypotonic (hyposmolaric) solution into the peritoneal cavity resulted in a loss of electrolytes from the blood (primarily Na and Cl^-), rapid decrease in peritoneal fluid, shift of extracellular water into the cells and signs and symptoms of

dehydration. The injection of a hypertonic salt solution produced a rapid increase in water in the peritoneal cavity and extracellular space, hyperchloremia, hypernatremia, and tissue edema. It soon became shown by many experimenters, including Clark (17) and Darrow and Yannet (18), that many substances passed the peritoneal membrane, depending on their concentration gradient. Courtice (16) emphasized that the peritoneal aspect of the diaphragm is the most important absorbing area of the abdomen for particulate matter.

B. Peritoneal Dialysis in Uremic Animals

Ganter in 1923 was the first to introduce peritoneal dialysis for treatment of uremia in animals (1, 8). It was found that through repeated dialysis, the survival time could be lengthened, and Grollman et al. (32), have kept dogs alive up to 70 days. Odel et al. (55) and Abbott and Shea (1) stressed the importance of the composition of the irrigating solution so as to avoid major shifts in water and electrolytes. They attributed the failure of earlier investigations to this reason.

TYPES OF PERITONEAL DIALYSIS

Two methods of peritoneal dialysis (P.D.) exist. The continuous method was first used by Wegner (8) and most recently by Seligman, Frank, and Fine (71). See diagram No. 1. The intermittent method was introduced by Ganter (8), and later modified

by Grollman (32) and Maxwell (47). Ganter (8) performed the first peritoneal dialysis in a human in 1923. Since 1948, a number of publications have appeared on peritoneal dialysis in the human, using either the continuous method (2, 26, 31, 43) or the intermittent method (19, 32, 47).

CONTINUOUS:

The continuous type of peritoneal dialysis was first attempted in humans with renal insufficiency by Ganter in 1923 (14, 55). The setup, including the sump-drain as it came to be used, is shown in diagram No. 1 (after Frank, Fine, and Seligman (23).

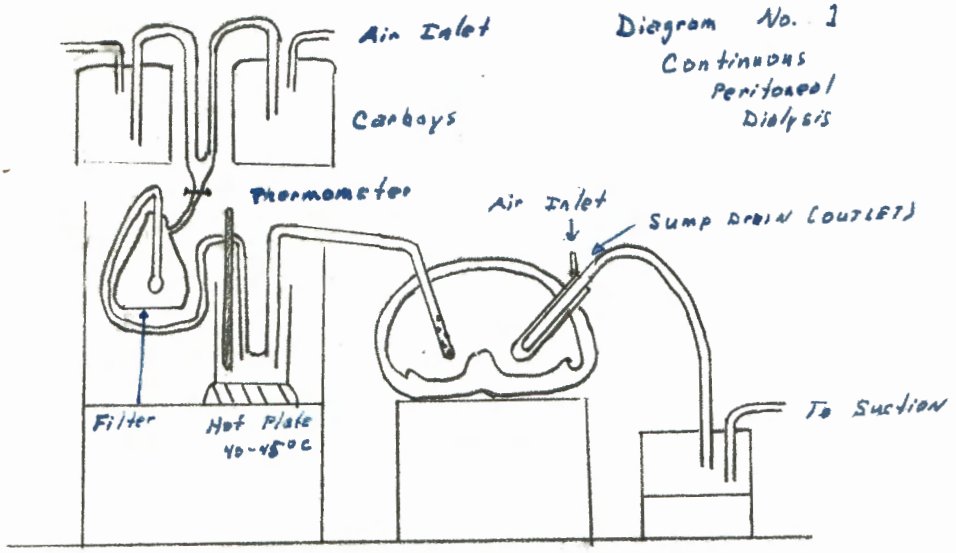


Diagram No. 1
Continuous Peritoneal Dialysis

The inlet and outlet tubes are placed in each flank just above the iliac crest (23).

The sump-drain is similar to the metal perforated suction tube used in modern surgery. Seligman, Frank, and Fine (71) discovered that the clearance of urea was proportional to the flow

rate and increased to a maximum at 1.5-3L (25-50 cc/min.); thereafter the value decreased. The same authors (26) stated that the principle of the sump-drain was to maintain atmospheric pressure in the sump cylinder (via air inlet) in spite of the slight negative pressure of the suction. This avoided drawing the surrounding omentum and tissue into the holes of the drain. Leakage of the dialysate around the inserted tubes was a major complication and was partially corrected by the development of a flexible sump-drain (26, 33, 57). Leakage promoted the development of peritonitis and attributed to the relaxation of the tissues of the anterior abdominal wall about the rigid tubes and to the respiratory and other movements of the patient (55, 57). Due to these factors, success was rarely obtained by this method (26, 57). (see p. 5. Intermittent P.D. vis. Continuous P.D.).

INTERMITTENT:

In 1951, Grollman, Turner, and McLean (32) introduced intermittent peritoneal dialysis in nephrectomized dogs and applied this to humans. (see p. 26 for its application). The intermittent procedure was found to be not only as efficient and technically simpler, but also lessened the chance of infection (33). The intermittent infusion of dialysate and withdrawal, allowing time for equilibrium to occur across the peritoneum, resulted in less need for complex drains, the use of smaller volumes of dialysate, and also decreased the incidence of pulmonary edema. (19, 33).

Doolan et al. (19) conducted experiments in which 2 liters of dialysate was infused intra-peritoneally and triated water (HTO) was given intravenously. It was found that the urea concentration was practically equal in the dialysate and plasma in one hour , the dialysate (peritoneal fluid) being an expansion of the ECF compartment. The diffusion of the HTO came to equilibrium in 1 hour and at the end of 40 minutes was 80% complete. These studies thus indicated the need for the dialysate to be left within the peritoneal cavity from 40-60 minutes. Maxwell et al. (46) discovered that the equilibration of the solutes across the peritoneal membrane occurred in 1-1½ hours. Boen (8) found that after 80 minutes, the electrolyte composition of the peritoneal fluid approximated that of plasma and the urea concentration was 89% that of plasma.

Therefore, from the above experiments, the optimum time to leave the dialysate within the peritoneal cavity occurs at approximately 30-45 minutes. By changing the dialysate at this time interval an optimal maximal exchange of electrolytes and waste products can be achieved over a shorter period of time.

INTERMITTENT PERITONEAL DIALYSIS VS. CONTINUOUS

Abbott and Shea (1) viewed intermittent dialysis as preferable to continuous dialysis as equilibration across the membrane requires 2-4 hours. Odel et al. (57) state that continuous and intermittent peritoneal lavage are equally

effective but prefers the continuous method. The reports of many investigators (1, 8, 25, 26, 32, 37, 43, 47, 55) emphasize that intermittent peritoneal dialysis (IPD) has the advantages of: (1) less channeling (short-circuiting between inflow and outflow tracts, thereby decreasing dialyzing area), (2) less peritoneal contamination and infection due to less leakage (3) using smaller volumes of dialysate (4) less chance of perforating the intestines during the insertion of the catheter and (5) requiring simpler apparatus. The increasing use of peritoneal dialysis since the introduction of the technique of Maxwell et al. (11) substantiates the success of intermittent over continuous peritoneal dialysis.

RENAL FAILURE AND UREMIA:

Acute renal failure (ARF) is the syndrome characterized by the sudden deterioration of renal function (anuria or oliguria) and anatomically by the areas of tubular degeneration (78). Strauss (75) emphasizes most nephrotoxins result in the necrosis of only the tubular cells, leaving an intact basement membrane as a scaffolding for regenerating tubular cells. Franklin and Merrill (27) found that with the development of ARF of parenchmyal origin, the urinary osmolarity falls to plasma values, urinary urea decreases markedly (the urinary urea/plasma urea being 5 or less) and the sodium concentration of urine increases 40-90 mEq/1. The urinary sodium excretion usually

always exceeds 30 mEq/l and the specific gravity drops in both acute tubular necrosis and chronic renal failure (60). According to Grollman (33), the majority of the cases of ARF are characterized by a period of severe oliguria usually lasting 3-10 days with a few cases lasting as long as 25 days. Swann and Merrill (78) also found this to be true. Clinical symptoms other than that of the associated disease and some lethargy and nausea may be prevented during the 1st week by the limitation of salt and water (78). Thus, a conservative management is important and should be utilized before the need of dialysis arises.

CLINICAL FEATURES:

During the first week lethargy, anorexia, weakness, pruritus, and a bad taste to cigarettes develops. Emesis usually begins towards the end of the first week (78) and clinical deterioration usually occurs during the second week of oliguria. Cardiovascular symptoms include tachycardia, increase in pulse pressure, tic-tac rhythm, a heaving precordium, and accentuation of the cardiac sounds, particularly of the 2nd pulmonic HS (78, 40). Heart failure is usually preceded by an acute rise in blood pressure (hypertension) consisting primarily of rise in systolic pressure; this is accompanied by an apical systolic murmur due to functional mitral insufficiency (33, 78). A gallop rhythm and cardiac enlargement are also commonly seen. Dyspnea, rónchi, rales, and pulmonary edema may supervene according to the above

authors. Pericardial friction rub may occur if the patient survives long enough (24, 78).

Anemia (normocytic, nomochromic) is due to hemolytic and supression phenomenon (14, 32, 33, 78), and the presence of pallor may be moderately severe. Early, the patient may be extremely irritable (65). Later the mental state is characteristically dulled with the patient showing signs of apathy, listlessness, and a tendency to move slowly and speak softly (46). Headaches are common (36). Muscular twitching is common and is not the result of hypocalcemia unless alkalosis is present (46). Convulsions occur in approximately 5% of these patients and are most often the manifestations of hypertension (24, A).

GENERAL MANAGEMENT:

The early treatment of these cases is of major importance. The cause of oliguria due to prerenal azotemia (circulatory insufficiency, dehydration, or salt depletion) are self explanatory insofar as treatment (33, C). In the above cases of ARF, water and electrolyte balance is essential (40). The best guides to the hydration of the patient are (1) the restriction of fluid to equal output plus the difference between the insensible loss and gain (75, 78), (2) daily weight of the patient (74) and (3) the serum sodium concentration. See water intoxication p. 19. Approximately 500 cc of insensible water loss occurs for every degree of fever over 100 degrees F.

The elimination of exogenous dietary proteins and reduction of endogenous catabolism by supplying needed calories of dextrose (8, 74, 87) and complete bed rest will help prevent the rise in the toxic products of protein metabolism (75). If a minimum of 100 grams of glucose (75) is administered daily in divided amounts, ketosis will be prevented and endogenous catabolism will be reduced by almost one-half (84, 87). Gamble (C) has demonstrated that 300 grams of carbohydrate per day is necessary to produce a maximal protein sparing effect in patients with ARF. However, most patients with uremia are prevented from taking a high caloric diet due to anorexia and vomiting (9). Anabolic steroids (norethandrolone - Nilevar®) may be of some value in reducing the rapid breakdown of protein (8, 87). Since they effect a rise in BSP and LDH, one must carefully evaluate their use in a patient with liver damage (75).

Potassium intoxication may be temporarily treated by the infusion of glucose which will cause the K^+ to move intracellularly with it. The infusion of glucose (crystalline insulin may (24, 78) or may not (19) be added - 1 unit for each 1.5-2.0 gms) will reduce the serum K^+ concentration by increasing the total body stores as much as 50-150 mEq/l of K^+ (68, 75). This effect must also be kept in mind during dialysis as well as during the diuretic phase of ARF as K^+ may then be needed (68). Another means of reducing the K^+ concentration is through cation - sodium

exchange resins per mouth or rectum (19, 74, 75). The ammonium or hydrogen series may accentuate the metabolic acidosis (24). The end products in the metabolism of fat and carbohydrate consist primarily of carbon dioxide and water which may be eliminated by the respiratory tract (75); thus it is from this that the diet is chiefly composed. Boen (8) gives his patients a diet of butter, sugar, custard, rice, and cream - 1300 calories. The food must be poor in protein and potassium (20) and it is important to make the diet as appetizing as possible. If the patient is unable to ingest the foods, an intravenous solution of dextrose or an Upjohn product Lipomul® (a 15% emulsion of cottonseed oil - 160 cal/100 ml) have been used (40).

Sodium is given only to replace losses in acute renal failure and medications that are eliminated by the urinary tract are withheld where possible (75). The anemia in uremia is iron and vitamin resistant (24); transfusions, if required, are to be with fresh blood (14, 80). Administration of vitamins, especially of water soluble vitamin B complex and vitamin C is recommended due to the removal of these substances during dialysis and for general body needs (8, 74). Vomiting may be controlled effectively in some cases of uremia by the administration of 25-50 mgm. chlorpromazine IM - four times per day (24).

In these patients a very high blood pressure and changing

neurologic signs called acute hypertensive encephalopathy may occur (22, 32, 75, 82) and the blood pressure must be controlled - but gradually so as to avoid a hypertensive collapse with subsequent renal damage (28, 29). Freis (28, 29) recommends reserpine, an initial close of 2 mgm. IM whenever the BP 230 systolic or 120 diastolic. Hydrolazine may be used in conjunction with reserpine.

LONG TERM MANAGEMENT:

Patients who have not recovered from ARF after two weeks (time required for endogenous protein breakdown to fall to a minimum) may be started on a minimum protein diet. In the long term management of individuals with chronic renal failure, the limitation of protein should not be too severe as many of these individuals are already suffering from anemia, hypoalbuminemia, and edema (9).

BIOCHEMICAL CHANGES ENCOUNTERED IN RENAL FAILURE

I. Unadvoidable Changes Oliguria

- A. Uremia
- B. Hyperkalemia
- C. Hypermagnesemia
- D. Hyperphosphatemia
- E. Hypocalcemia
- F. Acid - Base - Balance - Acidosis

G. Hypochloremia

II. Advoidable

A. Water Intoxication

B. Sodium Excess and Edema

Uremia: The compounds in serum that contain nitrogen in their structure may be classified as either (1) protein or (2) non-protein (54). The nonprotein nitrogen (NPN) of blood is a collective concept of a mixture of substances of relatively small molecular weight, which are found in a protein free filtrate. These substances include urea, uric acid, creatinine, creatine, amino acids, ammonia, and many others, some of which are unidentifiable. Of this group, urea constitutes approximately 45% (54).

Urea is the end product of protein catabolism within the body and it is formed in the liver. Wynn (87) stated that one gram of urea resulted from the catabolism of three grams of protein and one gram of urea equals approximately .5 gm urea nitrogen. Boen (8) has calculated that 1.35 mEq/l of K^+ is released during the production of one gram of urea.

The actual role of urea in the uremic syndrome or its manifestations is not definitely known. Urea has generally been regarded as harmless (20, 64, 78), but complications such as hemorrhage and sepsis increase if the blood level rises above 400 mgm % (20). The concentration of urea in plasma is important, as it is an indicator of the protein catabolism (59). However,

as the urea level rises, more urea is passed into the gastro-intestinal tract and is broken down into ammonia by micro-organisms. The ammonia may produce a local irritant effect and be responsible for gastritis, colitis, anorexia, nausea, and emesis. Infection, trauma, starvation, and low carbohydrate metabolism - all increase protein catabolism.

HYPERKALEMIA:

The normal plasma potassium (K^+) concentration ranges from 3.5-5.0 mEq/1, the intracellular concentration being 25-35 times greater than the extracellular component (87). Wynn (87) states that this gradient is maintained by metabolic processes which, if interfered with, will allow the escape of K^+ into the extra-cellular fluid (E.C.F.). In renal failure, acidosis, anoxia, and most important, the release of K^+ from the cell during the catabolism of protein contribute to its rise. One study revealed a .4 to 1.5 mEq/1 change in serum K^+ concentration for every .1 unit change in extracellular pH (68). The ratio extracellular/intra-cellular K^+ concentration varies inversely with the pH of the plasma, i.e. acidosis increases and also alkalosis decreases the ratio (67). The intracellular cations (K^+ and Mg^{++}) bears a constant ratio to the IC protein anions and as the protein is broken down, these cations are released. Wynn (87) also states that the normal ratio of K^+ to IC protein is approximately

3.0 mEq K^+: 6 grams protein.

The level at which K^+ produces toxic manifestations is said to be above 7 mEq/l in the adult and somewhat higher in infants (87). Patients with serum K^+ concentration >9 mEq/l rarely survive (40). Acidosis, hyponatremia, and hypocalcemia are said to potentiate the effects of hyperkalemia (64, 68, 87). It has been found that in prolonged oliguria or chronic renal insufficiency, the cardiotoxic effects of hyperkalemia become manifested at progressively lower concentrations (87).

Clinically, the effects of hyperkalemia are the development of cardiac irregularity, tachycardia, ventricular fibrillation, and sudden death due to cardiac arrest (64, 87). Paraesthesia, muscular irritation, hiccoughs, weakness, and flaccid paralysis may precede the above (33, 87). The person may become apathetic, listless, mentally confused, and hypotension may occur (33). The electrocardiagram is a more reliable method of determining the effect of K^+ on the body than the serum K^+ level (74, 75). The earliest changes include the appearance of tall, peaked, symmetric T waves (6). Disappearance of the P waves due to auricular standstill, followed by prolongation of the QRS and PR interval occur (6, 40, 86). Depression of the ST segment then occurs with the final disappearance of the usual components and the EKG assumes the appearance of a biphasic sine wave, followed by cardiac arrest (6, 40, 33, 87). In contrast, in hypopotassemia, the EKG shows a progressive broadening and

decrease amplitude and finally inversion of the T wave (76).

Boen (8) et al. (86) have found a marked reduction in plasma potassium concentration during the first few hours of dialysis. The K^+ reaches a lower level of which improvement of the EKG abnormalities occur and little further change occurs in the K^+ extracellular concentration. Boen (8) states that due to the shift of the potassium into ECF, hypokalemia is prevented and peritoneal dialysis may be performed with a potassium free dialysate for approximately 24 hours.

ELECTROLYTES:

Serum hypermagnesmia results from the catabolism of protein (see above p. 15). In most cases uremia is associated with a chloride deficiency (8, 36) and this is due primarily to the vomiting, other factors being polyuria, isothenuria, and diarrhea - all of which decrease total body base (36). Due to the constant $Ca^{++}/HPO_4^{=}$ level within the plasma, the accumulation of $HPO_4^{=}$, resulting from the catabolism of phospholipids and nucleo proteins, will produce a fall in the serum calcium level (hypocalcemia) and hyperphosphatemia. However, due to the associated metabolic acidosis, the ionized Ca^{++} level does not fall to a level at which tetany will occur (36). The phosphate concentration may be reduced by the oral administration of aluminum hydroxide (100 ml/meter squared QID (40), and the

Ca^{++} concentration will, thus, increase. During dialysis, hypocalcemia and tetany may occur if calcium is not added to the irrigation medium.

ACID BASE BALANCE

The total serum base is equal to the sum of sodium and potassium (cations); whereas the buffer base is equal to the difference between the total base (cations) and the nonbuffer anions (85). The anions are acid or potential acid radicals and they are grouped into two main groups (72, 82). The anions (potential acid) and cations (potential base), as found in normal plasma are listed in table No. 1.

The normal venous plasma CO_2 content is 28.3 mM/l (24-33 mM/l (72). Wallace and Hastings (84) and others (8, 63) discovered that the intracellular HCO_3^- concentration is impermeable to CO_2 bound as HCO_3^- and is thus not changed by elevations of serum HCO_3^- if the pCO_2 is unchanged. Rosenbaum (63) stated that intravenous bicarbonate is limited in its distribution to the ECF. Weller, Swann, and Merrill (85) stressed that though a rapid change in pH and rise in plasma $HCO3^-$ occurred, no change occurred at the respiratory center and hyperventilation continued. This may result in the continuation of the secondary respiratory alkalosis (at the beginning of dialysis) to be the sole (primary) metabolic error at the end of dialysis after the metabolic acidosis has been corrected. Respiratory adjustment will occur after a certain delay (8).

TABLE NO. 1 (NORMAL VALUES FOR PLASMA)

Blood Plasma mEq/l

BASE		ACID	
		HCO_3^-	27
		Cl^-	103
Na^+	142	HPO_4^-	2
K^+	5	$SO_4^=$	1
Ca^{++}	5	Org. Acid	6
Mg^{++}	3	Protein	16
	155		155

ACIDOSIS:

A normal individual has a pH (H^+) ion concentration 7.35-7.45 (extreme range 7.0-7.8) (30). In oliguric states metabolic acidosis occurs and is primarily due to the catabolism of protein (oxidation of protein sulphur and phosphate) with the resulting production of sulfate and phosphate radicals and other organic anions (acids) (36, 87). In response to the primary metabolic acidosis, a secondary respiratory alkalosis occurs (82).

WATER INTOXICATION

This entity may be eliminated with proper management of the patient. Symptoms include apathy, weakness, nausea, vomiting, delirium, frank convulsions, coma - death. The difference between the insensible loss (skin and lungs - 10 ml/Kg/day in an afebrile

adult in a temperate climate (87) and the insensible gain from the combustion of foods (1 ml H_2O/gm of oxidized fat, 0.4 ml H_2O/gm of protein and CHO) must be considered with the sensible (visible) loss or gain (75, 87). These calculations become involved and at best are only an approximation. It was discovered that changes in body weight correlated well with the state of hydration over a short period of time and, thus, the most accurate means of water balance is the careful weighing of the patient (19, 75, 87). Satisfactory fluid balance may be assumed if a daily loss of body weight of 200-300 grams occurs (11, 74).

SODIUM EXCESS AND EDEMA:

In renal insufficiency, the kidneys cannot excrete a large load of sodium, resulting in its accumulation along with water in the ECF. Pulmonary edema, cerebral edema, convulsions and coma may occur (87).

INDICATIONS FOR DIALYSIS:

INDICATIONS:

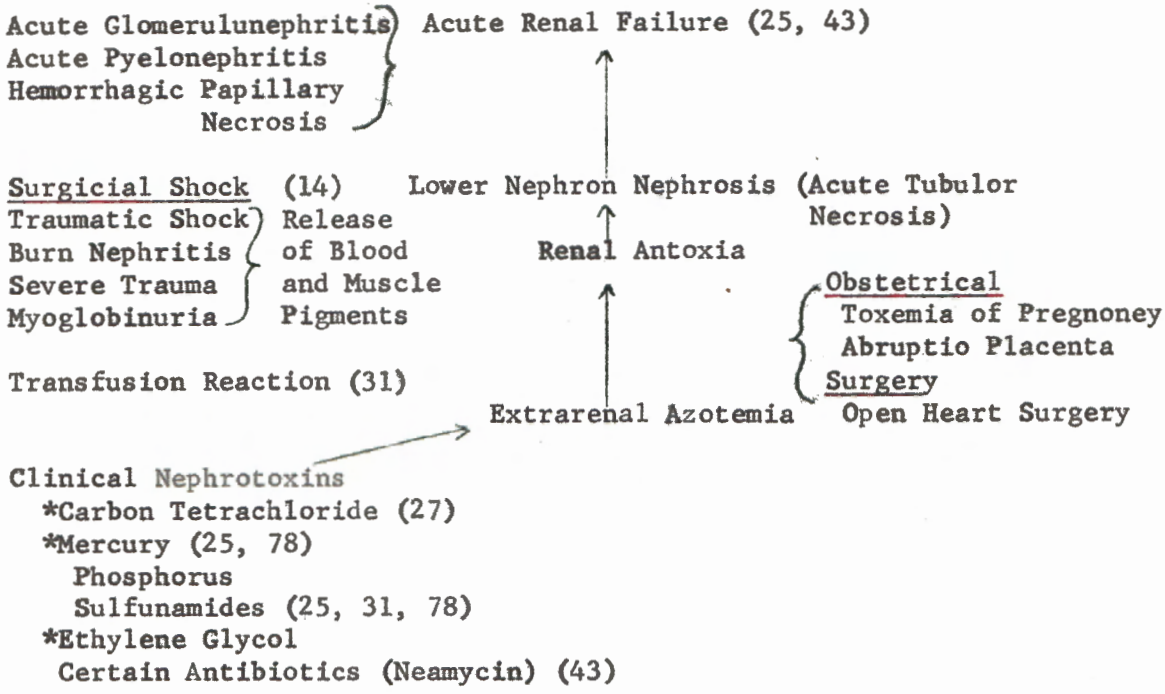
1. Acute renal failure
2. Chronic renal insufficiency following
   A. Trauma
   B. Infection

C. Operation

3. Chronic edema (intractable edema)

4. Intoxications

TABLE NO. 2 (ACUTE RENAL FAILURE)



The primary indication for intermittent peritoneal dialysis is acute renal insufficiency (43). See table No. 2 modified from Swann and Merrill (78). As to the time of dialysis in ARF, there is no uniform agreement except for two (1) hyperkalemia (serum K^+ concentration >7, especially if EKG abnormalities are present (8, 37). (2) Severe acidosis, the plasma HCO_3^- concentration <10 mEq/l or the CO_2 CP of <12 (8, 37). (3) Others include a very high BUN which is rising (37). A criterion used by many is

a BUN >3.5 to 4 gm/1 (59). Parson (8) is in this group who states that a BUN 180-200 mgm % (blood urea 386-428 mgm %) is an indication for dialysis. However, in the minds of others, the BUN by itself forms no basis for dialysis (8). Boen (8) indicates that only when viewed with the other laboratory data and clinical signs is the BUN considered. (4) The rate of progression of the chemical abnormalities is a key factor and dialysis must be performed earlier when this occurs to avoid reaching lethal levels. (5) A fifth criterian is the rapid clinical deterioration of an individual (8, 37). The last major criteria is an individual who has been grossly overhydrated. Indications used by some, according to Boen (8), include a chloride concentration < 85 mEq/1, a plasma $SO_4^=$ concentration >10 mEq/1, or when anuria has persisted for more than five to six days.

The indications for peritoneal dialysis in the patient with chronic renal insufficiency is definitely not settled at the present time, although for practical reasons it is - it should not be performed (8, 55). Peritoneal dialysis is performed in these patients when the renal failure becomes accentuated by trauma, vomiting, diarrhea, or excessive protein intake (5, 8, 37). Dialysis may be required in these patients as well as in cases of ARF when conservative measures fail - as in sepsis, crush syndrome, gastrointestinal bleeding (8). A re-exacerbation

of their underlying kidney disease would also be an indication. Dialysis has been used in patients to improve them for surgery on their kidneys, as in a case of a stag horn calculus (nephrolithotomy) (1). Boen (8) implies that even with these complicating factors, dialysis will only be considered if renal function was 15% of normal prior to recent stress. He stresses that a patient with a urea clearance of 10 ml/min need not be dialyzed unless complications are severe (52). Frank, Fine, and Seligman (23) also quote 10-15 as the minimal renal excretory function necessary to prevent reaccumulation of the products of protein breakdown. Wynn (87) also corroborates the statement that with proper management, patients with 10% of their renal function can be maintained with conservative management.

Intoxication due to methyl alcohol poisoning, boric acid poisoning (69, 70), barbituates intoxication (48), salicylism (17, 19, 22, 70) or diabetic acidosis with oliguria may be indications for peritoneal dialysis. There conditions have all been successfully treated with peritoneal dialysis as well as hypercalcemia (48). Severe jaundice and hepatic coma have both been treated with dialysis with temporary relief (47, 48).

CONTRAINDICATIONS TO PERITONEAL DIALYSIS:

A few years ago this question was well settled, but at present only one or two actually remain: (1) diffuse infection

of the skin of the anterior abdominal wall (57) and (2) diffuse peritoneal adhesions (43, 47). Peritonitis, which was once believed to be an absolute contraindication to peritoneal dialysis (43, 47), has now been treated by peritoneal dialysis (10, 11) with good results. Burnett (10) has been able to decrease fatalities where severe peritonities is present. The lavage removes huge quantities of toxin and bacteria from the body, thereby enhancing the body defenses on the remaining toxin and bacteria. Recent or extensive abdominal surgery also was once believed to be an absolute contraindication to peritoneal dialysis, and it has been successfully utilized in four cases, five to eight days after surgery (19, 47). Doolan et al. (19) state that the cases requiring dialysis and who have had recent abdominal trauma or surgery need to be considered individually. Even in these patients if no other means are available, a therapeutic trail may be performed as the risk is small.

TECHNIQUE

Equipment:

The necessary equipment needed for peritoneal dialysis includes: (1) a routine paracentesis set (43, 47) with a trocar set. Maxwell et al. (47) recommend a 17 F Duke trocar set. Sterile tubing and one or two sterile catheters will be needed. The catheter must be stiff enough to allow

some manipulation without kinking and Maxwell et al. (47) prefer a catheter with a permanent curve at the distal end. Various catheters are placed on the market, the new polyvinyl catheters (smooth or transversely ridged at the distal end with multiple tiny perforations) are very adequate (14, 19, 47). These tubes may be autoclaved and used again.

Solutions:

The proper selection of a dialyzing solution centers around (a) one which would permit maximum diffusion of nitrogenous and other waste products (b) one which simulate the normal extracellular and plasma electrolyte concentration and act as a buffer (c) one which would be nonirritating, thus allowing maximal efficiency of the filtering membrane (55, 56).

In the past, it was recommended that the various solutions be prepared in the pharmacy; however, precipitation of salts occurred if the pH was not carefully adjusted (23, 55) and many of the earlier failures were due to water and electrolyte imbalances of the dialysis solutions (55). Solutions are now available commercially (See table No. 3) and may be stocked in the hospital pharmacy in ordinary liter infusion flasks. The use of these commercially prepared solutions dispense with their time consuming preparation and reduces the chance of introducing infection (47). Due to the readibly available solutions and the simplicity of the technique, a dialysis may be started within

30 minutes after the decision is made (48).

TABLE NO. 3 DIALYSATES

	Milli. equivalents per liter						mos M per liter				
SOLUTION	Na^+	K^+	Ca^{++}	Magnes	Cl^-	HCO_3^-	no Gluc.	1.5%	3%	5%	7%
Grollman's Solution	134.5	2.7	3.6	1.1	106.1	35.8	281.5	364.5	448.2	559	670.4
Lactated Ringers	130	4.0	3		109	28	272.5	355.5	438.7	550.3	661.4
Impersol (Abbott)	140.5		3.5	1.5*	101	44.5		371.5			677.4
Peridial (Cutler)	140		4.0	1.5	102.5	43		371			677
Dianeal (Baxter)	141		3.5	1.5	101	45		372.5			678.4

*The 1.5 mEq/l is within the lower normal range for humans

Lograin, Marcell, and Merrill (43) emphasize the importance of using a solution which is hypertonic to plasma. To prevent the uptake of water by the body, thus, preventing hypervolemia cardiac failure, and pulmonary edema (8, 22, 23), the perfusing solution should have a tonicity of 360-390 mos M/1 (43, 48, 70). Normal plasma is 280 mos M/1 but enough dextrose must be added to increase the osmolarity above the elevated level found in uremic patients (48, B). A one percent glucose solution (10 gm/1) raises the osmolarity approximately 55 mos M/1. Since the osmolarity is greater in the dialysis fluid, the absorption of the administered fluid will be prevented (47, 70). A 4.25% (525 milliosmol/1

solution may be prepared by mixing a 1.5% (372 mos M/1) and a 7% (677 mos M/1) solution (11).

With the exceptions of the first two solutions tested, the dialysis solution is essentially a potassium free extracellular solution. The omission of potassium can be corrected in those cases in which a normal or hypokalemic level exist by adding potassium chloride concentrate by hypodermic syringe to bring the concentration to 4 mEq/1. This is also done during dialysis when the K^{+} concentration falls below 3.5-4 mEq/1 (8).

The dialysis technique employed was patterned after that of Grollman and associates as modified by Maxwell et al. (19, 22, 48). Two liters of fluid are given simultaneously, necessitating the addition of drugs of electrolytes to only one bottle, thereby lessening the chance of infection. To each 2 liters of solution are added 25 mgm. of tetracyclines, 8 mEq/1 of potassium chloride (if required) and 10 mgm. of aqueous heparin - which is discontinued after three exchanges if the outflow tube is not grossly sanguineous.

The following paragraph is taken directly from Maxwell (47):

PROCEDURE:

"The patient should be supine or semi-supine with his bladder emptied (in dwelling catheter) just prior to the procedure. No special sedation is necessary unless the patient is unduly apprehensive or agitated. The abdomen is shaved, prepared, and draped as for a laparotomy. After local infiltration with procaine

hydrochloride, an incision is made in the midline about one-third of the way from the umbilicus to the pubic bone. The incision should be small enough so that the skin fits very snugly around the trocar. The scapel blade (no. 11 Bard Parker) is inserted into the anterior abdominal wall until it is felt to "grate" on the linea alba, and a small incision is made into the latter. The midline is relatively avascular, and any slight superficial bleeding which occurs is usually controlled by lateral pressure exerted by the trocar and catheter."

The trocar with stylet in place is inserted through the incision into the peritoneal cavity. A sharp thrust is occasionally necessary and the patient may experience pain as the perietal peritoneum is stretched. The stylet is then removed and the trocar is advanced to its full length. The plastic catheter is then fitted through the trocar and is manipulated dorsally towards either the right or left paravertebral sulcus (47). Boen (8) stresses that the catheter must be positioned in the small pelvis to insure a satisfactory return of dialysate. Ideally the catheter should extend well into the peritoneal cavity, necessitating rotating the catheter in various directions before the omentum is pushed aside as a natural pathway is found to admit the full length of the catheter. It may be helpful to run in 1 or 2 liters of solution if the insertion of the catheter becomes completely impeded. Twiss and Maxwell (83) state that the patient is usually supine, but may be slightly flexed (head raised) to

relieve pressure of fluid on the diaphragm and thus relieve breathing.

The trocar is then withdrawn, purse string sutures may be placed around the catheter to prevent subcutaneous bleeding if this is a problem. The catheter is then connected to the Y-tubing and the solution (previously warmed to body temperature (8, 32, 52) is permitted to flow into the peritoneal cavity as rapidly as possible, usually requiring 5-10 minutes. If the process requires longer then this, the catheter should be repositioned as the perforations may be buried in omentum. The tubing should be clamped at the point where the bottles are empty and the tubing is filled with solution.

The fluid is allowed to remain in the peritoneal cavity for approximately one hour, after which the bottles are placed on the floor beside the patient's bend and the clamp is removed. Siphoning or gravity drainage should require no more than 10-20 minutes (47) and the stream should be forceful and seen entering both bottles. Drainage is facilitated by inserting an airway needle through the inlet stopper of the liter container (19). Two more liters of solution are prepared during periods of equilibration within the abdominal cavity so that as soon as drainage ceases, a new infusion may be started. The composition of these irrigating solutions will depend on the subsequent chemistries and fluid balance (8).

This procedure is repeated continuously from 12-36 hours, depending upon the case usually 30-50 L are exchanged (47).

At the termination of the procedure 400-1000 cc of dialysate may still remain and cannot be removed even with abdominal pressure (19). If over hydration is a potential danger, one should use a hyperosmolaric solution for the last 2-3 dialysis (85).

Dialysis of the intermittent type, limiting the lavages to only 3-4 a day, have been performed over a period of up to three weeks, using the original catheter which is left in place (14, 19). Following the removal of the catheter, the surrounding skin is disinfected and a small dressing is placed over the site (8, 83).

In infants and small children the amount of dialysate necessary will vary from 70-100 ml/Kg or (.5-1.5l), depending upon (1) size of the patient (2) influence upon respiration (3) degree of discomfort (33, 70, 83).

RECORDS:

To begin the procedure, a signed operative permit is necessary (83). During the dialysis the following information must be carefully recorded.

1. Time at which infusion was started, finished, and amount of infusion. Total exchange recorded.

2. Fluid balance (see p. 19).
3. BP and P (regular or irregular), RR every 15 min. during first exchange, if stable every 30 minutes (8, 83). When using a hypertonic solution, hypovolemic shock may be precipitated, necessitating careful, frequent checks on BP and P (11).
4. Base line laboratory studies and periodic values throughout dialysis procedure (47). (Ht, Hb, WBC, BUN, Cl^-, CO_2, CP, Ca, K^+, Na, PO_4, plasma proteins, coagulations.
5. Drugs added.

RESULT OF DIALYSIS - CLEARANCES:

Boen (8) found that during dialysis, urea diffuses most rapidly into the dialysate and the diffusion rate of potassium is almost equal to urea. Thereafter, follow creatinine, phosphate and uric acid at approximately the same rate. The diffusion rate of calcium and phosphate are lower due to the binding of these electrolytes with plasma proteins. The sharpest rise in concentration of the electrolytes occurred during the first thirty minutes and then proceeded at a slower rate as equilibrium was approached (18, 12).

Using intermittent peritoneal dialysis and allowing the fluid to remain in the abdomen 30 minutes, Boen (8) obtained an urea clearance of 26 ml/min by using a dialyzed volume of 2.5 L/hr. Boen (8) et al. (25, 26), found a urea clearance of 10-11.1 ml/min.

at a diffusion rate of 1 L/hr. According to Boen (8) the clearance increased until 3.5 L/hr (28.5 ml/min) and decreased after this point. At a dialyzed volume of 2.5 L/hr which Boen (8) considers most economical, the clearance of urea is 26/ml/min, K^+ 19, inorganic $PO_4^=$ 16, uric acid 14, creatinine 15, and Ca^{++} 9.5.

DURATION OF PERITONEAL DIALYSIS:

The duration of the dialysis will depend on several factors:

1. If hyperkalemia is the indication for dialysis, dialysis should be continued until the plasma level is approximately 4 mEq/1. In this way a second dialysis is postponed as long as possible (8).

2. Until the BUN <100 mgm % (57).

3. In a state of accelerated protein breakdown, dialysis must be continued to prevent accumulation of anions, organic acids, and potassium.

4. In chronic renal insufficiency, dialysis must be continued until physiologic concentrations are achieved.

APPEARANCE OF THE RETURN FLUID:

The return fluid is usually clear and colorless, but it may be opalescent or opalescent and some color varying from yellow to grossly bloody (32). Opalescence is best correlated with the number of cells present, predominately polymorphonuclear leukocytes in cells count varying from 10-400 per cu. mm. (32,). These cells are always found in the peritoneal dialysate

and are probably due to the enhanced diapedesis of the leukocytes due to the chemical irritation of the irrigating fluid. The presence of there cells therefore do not indicate infection and probably exert a protective action (8, 32).

COMPLICATIONS:

The most frequent complication of peritoneal dialysis reported in the literature was peritonitis. This complication, although now not the most frequent, remains the most serious complication (19, 43). Infection may be introduced into the peritoneal cavity around the catheter as some leakage occurred at one time or another so that the bacteria had free access to the peritoneium (8, 19, 57). It has been established that transmural migration of bacteria from the gastrointestinal tract does occur (66, 79), and Frank and associates (26) have suggested that the intestinal wall of uremic patients is more permeable to bacteria than that of a normal person, especially with the added serosal irritation due to the dialysate.

A positive culture in the outflow fluid (old-open sump method) did not always indicate peritonitis of its inevitable development (8, 26, 57). This can be explained by the outflow fluid being in contact with non-sterile environment such as a graduated cylinder (57). However, with the method described above by Maxwell et al. (47) the threat of peritonitis has been eliminated (clinical peritonitis occurring in 76 cases) by (a) the addition

of a broad spectrum antibiotic to the dialyzing solution (b) the use of new sterile tubing with each infusion (c) the procedure being a closed system with drainage of fluid into the original bottles and (d) limiting the dialysis procedure to 36 hours. Frank, Seligman, and Fine (26) state that the likelihood of infection increases with the duration of the irrigation period and thus should be limited to 2-3 days.

It has also been pointed out by the above group (57) that peritonitis may develop in the absence of the classic signs. Emphasis should, thus, be placed on the early detection and treatment of peritonitis if it develops. Doolan et al. (19) recommends culturing every third dialysate, frequent Gram smears, carefully washing or wearing sterile gloves and cleansing the area of connection when changing the tubing on the catheter. If infection does occur terramycin, penicillin, and streptomycin or as advocated by Fine (19) neomycin should be added to the next lavage. Eschericia coli has been found to be the usual pathogen for peritonitis and the above antibiotics should be sufficient (26).

The most troublesome complication reported by Doolan et al. (19) was obstruction of the peritoneal catheter. This complication has been reported by numerous authors. The use of heparin to decrease clot and fibrin formation in the catheter and perforation

has been recommended (8, 22, 47, 55). If the outflows stream stops or slows down, twisting or repositioning of the catheter may restore the flow. If this fails approximately 20 cc. sterile saline may be injected under pressure to free the fibrin clot (57). Placement of a new catheter along original path is only a measure of last resort.

The presence of adhesions may prevent catheter insertion or adequate drainage from loculated cavities (47). The only adhesions which have been seen by Doolan et al. (19) have been at sites of former insertions, thus making each insertion more of a problem in chronic cases.

A bleeding tendency is present in many patients with uremia and thus calls for a small surgical incision in placment of the trocar (19). If bleeding persists, a purse string suture may be placed in the subcutaneous tissue to help approximate it to the catheter. It is best to avoid the suturing of the catheter to the skin in order that one may be able to manipulate it later if required (47).

Hemorrhage has been recorded (37, 57) where a blood vessel within the abdomen was eroded by a rigid catheter. Perforation of the bowel (viscus) by the trocar has also been recorded (37, 43, 47). According to Boen (8), this is especially likely to occur in the presence of severe tympanitis, a large uterus, or

or adhesions due to past operations.

Subcutaneous edema has been a complication in cases where the catheter has not been properly inserted into the peritoneal cavity (43, 47). Over hydration (see pulmonary edema) is associated with the infusion of a hypotonic solution. The incidence of abdominal distention or ileus has been small, usually disappearing within 24 hours after the procedure was terminated (19, 43, 47).

A major complication has been the leakage of fluid around the catheter site (19, 47). This is commonly associated with excessive abdominal distention and is seen in most patients at one time during the procedure, thereby producing errors in the fluid balance towards falsely high values. Accuracy of the fluid balance is also influenced by vomiting, diarrhea, perspiration, hemorrhage, and weeping lesions.

Hypoproteinemia has been a frequent complication in peritoneal dialysis. When larger volumes of fluid are used than are necessary even greater losses of protein will occur (22). Even without complications, the protein content of the dialysate will range .03-0.8 gm % with a mean of 0.2 gr % per 2000 ml of dialyzing fluid (22). Doolan et al. (19) reports the protein concentration averaged approximately 50 mg/100 ml or .5 gram/l.

Boen (8) also reported the protein concentration of .5 gm/l was the most common finding during peritoneal dialysis. This

may account for a loss of up to 40 gms of protein during peritoneal dialysis. Burns et al. (14) using Maxwell et al. (47) procedure have found the protein loss to amount to 20-30 gms/24 hours.

These findings are evidence for the high degree of permeability of the peritoneal membrane (32) and also points to the complications (peripheral and pulmonary edema) resulting from the depletion of proteins (57). Replacement therapy (intravenous albumin) is usually indicated in patients with chronic renal disease with prolonged or repeated dialysis.

Abdominal pain is occasionally encountered, beginning at about the 8th to 10th hour of the procedure (43). The pain is not intolerable and is most likely to occur towards the end of the outflow phase (47). This pain is nearly always alleviated by the next inflow of fluid, but, if severe, may require the infiltration 5 cc of 2% procaine hydrochloride. Legrain etal. (43) state that severe abdominal pain may be encountered if the peritoneum is suddenly distended or if the fluid is excessively hyper or hypotonic. In a few cases, mild abdominal pain is present for up to 24 hours post-dialysis.

DEATH DURING DIALYSIS

Cardiac failure is the major cause of death and is due to K^+ intoxication, congestive failure (accompanied by pulmonary edema)

or circulatory collapse (40, 78). Peritonitis, septicemia, shock, and pulmonary embolism are also leading causes (57).

PROGNOSTIC AIDS:

The concentration of urinary urea nitrogen divided by the serum urea nitrogen is called the urine serum urea nitrogen ration (USUR) (60). Normally the ratio is above 20. A value exceeding 20 was found in cases with acute tubular necrosis (ATN) and if below 10, this was a very poor sign. Values below 10 were found in severe cases of chronic renal insufficiency and in patients near death with ATN. See table No. 4.

TABLE NO. 4

		Urea NITROGEN mgm %	
	CASES	URINE	SERUM
Post-OP UNCOMPLICATED COURSE	18	250-1750	8-24 14-113
Chronic Nephritis	12	87-750	23-290 1-13
Transient Nephritis	22	200-2465	10-62 12-25
Acute Tubular Necrosis	21	139-1436	10-156 2-32

The endogenous creatine clearance is a clinical test of GFR and may be used as a prognostic guide in chronic renal failure (81). It was found that electrolyte abnormalities developed with C_{cr} below 30 ml/min and if below 10 ml/min, the patients longevity was usually less than nine months.

COMPARISON TO THE ARTIFICIAL KIDNEY:

The major differences between hemodialysis and peritoneal dialysis according to Boen (8): (1) the artificial kidney has

a larger dialyzing surface than the peritoneum and (2) the artificial kidney has a larger clearance than the peritoneum. The blood flow may be regulated and increased by means of pumps in the artificial kidney (8, 53) whereas in peritoneal dialysis, the flow is dependent on several factors. The urea and potassium clearance of hemodialysis is approximately 4 times greater than the peritoneal clearance (8, 47). Both means of dialysis remove substances at corresponding rates, i.e., if they are easily removed by one method, they are by the other. Calcium and magnesium are protein bound and pass through the membrane of the artificial kidney or peritoneum only in small quantities.. The difference therein between the two procedures lies in the duration of the dialysis required to attain the same reduction in the plasma, peritoneal dialysis being approximately four times slower (8, 47).

Disadvantages of the artificial kidney are:

1. Need for 5 or more units of fresh blood; thereby also increasing the risk of serum hepatitis or a transfusion reaction (8, 47).
2. Permanent trained team (47).
3. Heparinization is necessary and may prevent the use of this procedure in patients who are bleeding (8, 64). However, regional heparinization may be used.
4. Danger of air embolism (8).
5. The necessity of using an artery with each procedure and the

eventual inability to find one in patients who may need several dialysis (19, 47, 70).

6. The procedure may prove technically impossible in younger children, especially if under 24 months, in attempting to cannualize these patients (21, 38). Also serious deterrents include the lack of a team with pedatric experience and the greater degree of major fluctuations in blood volume (38, 70).
7. The artificial kidney is ineffective in the presence of shock (11).

Disadvantages of peritoneal dialysis are:

1. Somewhat greater chance of peritonitis (8). See p. 31.
2. See Complications p. 33.
3. Intestinal perforation, recent surgery, protein loss. (8).
4. Sterile dialysate is required for peritoneal dialysis, whereas it is not with the artificial kidney. (8).
5. Fatigue resulting from longer duration of the procedure (8).
6. The deposition of intracellular potassium will be cleared the day following dialysis (8).
7. Slower rate of attaining physiologic equilibrium of electrolyte and water.

Advantages of Peritoneal Dialysis:

1. Equipment and materials necessary may be found in a community hospital (8, 43). Many patients are too ill to be transported

to a center where an artificial kidney is available.

2. The procedure does not call for a specialized team, as does the artificial kidney (43) nor does it require the number of personnel (48).
3. Rapid changes in blood volume can be prevented easier, thus preventing shock as occasionally occurs with the artificial kidney (8).
4. Due to the longer duration of the procedure, time permits for the adjustment of the irrigating solution (8).
5. In patients with increase protein catabolism, it is more advantageous to dialyze them over a longer period of time (8).
6. Procedure may be used successfully in any age group, including small infants (35).

Thus, as stated above, there are no essential differences between what can be accomplished by hemodialysis or the artificial kidney, the differences being only in the rate at which this is achieved. Maxwell (47) reported that it requires approximately four times the amount of time to obtain the same results with peritoneal dialysis as with hemodialysis.

Most of the earlier methods of peritoneal dialysis have been eliminated by the recent modifications - as outlined in the paper. Both procedures (P. D. and hemodialysis) have their advantages and disadvantages, and both measures must be considered useful and potential life saving procedures in ARF and certain other states (74). See p. 21. The present role of peritoneal

dialysis at one of the leading centers is seen in table No. 6, which demonstrates that the number of peritoneal dialysis performed today is greater than the number of hemodialysis.

TABLE NO. 5 (Ref. - 11)

YEAR	HEMODIALYSIS	PERITONEAL DIALYSIS
1959	85	3
1960	102	12
1961	37	121

The two procedures must not be thought of as separate entities but as valuable adjuncts to be used as needed. (Refer to cases No. 3 and 4).

DISCUSSION:

Result of Dialysis:

- Following dialysis maximal clincial improvement may not be evident for one or two days (46) and this may occur even with the return of the electrolyte picture to an abnormal state (82). The purpose of dialysis is of course to return the electrolyte picture to normal. The BUN is not decreased to normal levels but is lowered just as effectively as does hemodialysis (80-90 mgm %) (refer to case No. 4). The BUN in chronic glomerulonephritis is also lowered to 60-90 mgm % with peritoneal dialysis. See cases No. 1 and 2.

Antibiotics:

A controversial issue arises concerning the use of antibiotics in the dialysate. Most authors advocate the withholding of antibiotics in the irrigating medium, unless definite signs of infection occur (8, 19, 24, 52, 59, 70). Maxwell (47), however, uses small amounts of antibiotics prophylactically and this is advocated by others (55). However, in the series of four cases after the technique of Maxwell et al. (47), reported on at the end of this paper, infection occurred once (case No. 4). The use of antibiotics in these small amounts may be of greater danger as once infection does occur, it is apt to be resistant to higher doses of the antibiotic. To evaluate the effectiveness of the prophylactic addition of antibiotics, it will be necessary to perform control parallel studies under similar conditions.

If infection does occur, the addition of penicillin and streptomycin to the dialysate is recommended (10, 32, 57). MacLeon (45) stresses the danger of intraperitoneal neomycin - both respiratory arrest and renal failure having been reported.

FUTURE

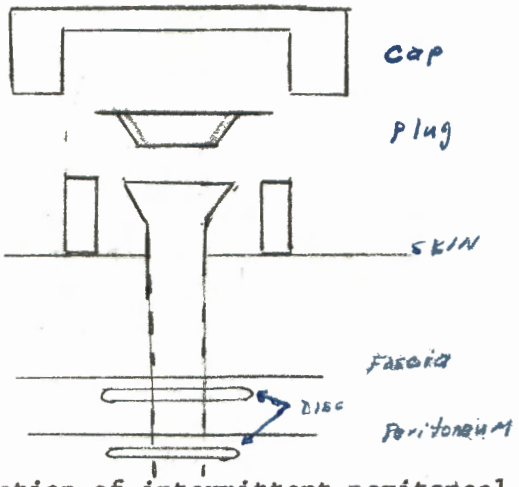
Peritoneal dialysis is now being used in ARF and chronic renal failure to prevent the clinical manifestations of uremia (11). This means earlier and repeated dialyses, especially in cases of ARF. After the initial control in a patient with ARF, the dialysis

may be carried out from 8 to 24 hours per day, depending on the severity of the process. The catheter may be left in place and clamped when not in use, retaining a sterile tip (11). Burns et al. (11) report of a case where a catheter was left in 34 days without producing peritonitis, intestinal perforations or other complications. (See complications p. 31.)

The increasing scope of medicine in its attempt to prolong the life of mankind is now even looking at patients with chronic renal failure. Merrill (D) reports the current concept at one of the hemodialysis centers is to effect a more gradual equilibration in patients with chronic renal failure. Peritoneal dialysis is thus particularly suitable in assisting patients with chronic renal failure over acute episodes (19). The further use of peritoneal dialysis may be of some value in the patient with chronic renal failure whose kidneys maintain some renal function but not of sufficient degree to maintain his life over a long period of time. Such an individual may be aided by periodic periods of dialysis to return the plasma metabolites and electrolytes to physiologic levels and thus offer a means of prolonging ones life. In fact, Merrill (52) has developed a peritoneal access fitting which is left in place permanently and allows for an unlimited number of placement of the catheter and dialysis. (See diagram No. 2). Between dialysis, the mouth of the fitting is covered with a sterile plug and cap to prevent

access of micro-organisms.

Diagram No. 2



The scope of application of intermittent peritoneal dialysis is thus expanding. Modifications in recent years has enabled its widespread general use and future advances such as above may further its use in cases of chronic renal failure.

SUMMARY

The basic principles of dialysis and the history leading to the use of primarily intermittent vs. continuous peritoneal dialysis was discussed. The function of the peritoneum is similar to the glomerulus, being a semipermeable membrane (22,000 sq. cm) and lacking the selective tubules reabsorption of the kidney. Repeated dialysis has not been found to result in deterioration of the peritoneal membrane.

A discussion concerning the various etiologies, clinical features, and management of renal failure is presented. Restriction of fluid is stressed as is the importance of the daily weighing of these individuals as a guide to their hydration. The administration of calories (glucose) to diminish the endogenous catabolism of protein and the elimination of exogenous protein is also stressed.

The biochemical picture of patients with anuria chloride, anuria - a lowered concentration of sodium, calcium, chloride, and bicarbonate and elevated concentration of potassium, magnesium, phosphate, and residual anions (sulphate and organic acid) is discussed. The indications for dialysis of which the primary indication is acute renal failure (ARF) are presented in the paper. Absolute contraindication to P.D. are only two: (1) diffuse infection of the anterior abdominal wall and (2) diffuse peritoneal adhesions.

The technique used by Maxwell et al. (47) was discussed, emphasizing the importance of sterile technique and procedure and the use of hyperosmolaric solutions as dialysate.

The most serious complication of dialysis reported was peritonitis. Significant other complications which were discussed included obstruction of the catheter, bleeding, leakage of the fluid about the catheter site, hypoproteinemia, abdominal pain, and adhesions.

The advantages and disadvantages of P.D. and hemodialysis are discussed. The only essential difference between the two methods being only the rate at which the dialysis is achieved, hemodialysis being approximately four times faster. The importance of considering the methods as valuable adjuncts and not totally unrelated means of therapy is stressed.

Most of the authors recommend the withholding of antibiotics to the dialysate unless infection has developed. The prophylactic use of antibiotics appears to be unwarranted, only leading to later difficulties if an infection occurs with a resistant organism.

The role of peritoneal dialysis in acute renal failure is well recognized and its role in assisting patients with chronic renal failure over acute episodes is also well known. The role yet to be evaluated is the maintenance or the prolonging of the life of individuals with chronic renal failure. Recent advances as mentioned in the paper and future advances may help us attain this end.

ACKNOWLEDGEMENT:

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CASE STUDIES

The technique of Maxwell et al. (47) was used in the following dialyses. Tetracyline was added in the amount of 25 mgm to each liter of infusion. Hypertonic dialysate solution was used in the first few infusions in each of the dialysis.

Case 1 #46667 UNH B.S.

History: A 25 year old W F was admitted to UNH for the first time on 6-18-62 with a C.C. of extreme shortness of breath accompanied by weakness, chills, nausea and emesis. Patient stated that two months PTA she had noticed easy bruisability and swelling of hands, feet, and face. Menstruation at this time was prolonged and epistaxis occurred one week prior to admission. Past history revealed repeated sore throats followed by "Brights Disease" of kidneys at age 15. This was associated with nephrotic syndrom (edema of 8 months duration) and patient was hospitalized on several occasions. Patient became edematous at time of pregnancy five years later and has been treated with vitamins and anti-hypertensive agents since 1952.

Physical: BP 185/115 P 84 RR 24 T 36.4°C.

Ht. 5' 6" Wt. 183.5

On admission, the patient was dyspneic, very weak, drowsy, and apprehensive. Skin presented a yellowish cast with multiple

ecchymotic areas on trunk and extremities. Pitting edema of extremities was present as well as periobital edema. Sclera was pale and hemorrhages and exudates of eye grounds were present. A uremic odor was present upon her breath - minimal moist roles were present bilaterally.

Clinical Course: The evening of her admission, the patient went into pulmonary edema and was treated by the usual measures. The following day peritoneal dialysis was decided upon.

TABLE NO. 1A

DATE	BUN	CO_2CP	K^+	Na^+	CL^-	Ca^{++}	$PO_4^{=}$	Hb	DATE
6-19 1st P.D.	185	8	3.8	115	85	8.2	24	5.7	(1) 48 L/43 hrs.
6-21 END	82.5	24	3.6	140	95	8.7	6		(2) 5415 cc fluid removed
2nd P.D. 7-1	150	15.1	3.5	108	87	8.0	11.4	7.2	(1) 48 L/48 hrs.
END 7-2	67.5	30	4.5	133	91	7.7	6.3		(2) 972 cc removed
3rd P.D. 8-4	118	17	4.5	105	69	8.6	7.2		(1) 46 L/64 hrs.
END 8-7	62.5	26	3.3	140	94	8.9	4.2		(2) 12 L. deficit

At the beginning of the 1st dialysis, the creatinine was 20, uric acid 9.28, cholesterol 220, and TSP 5.2 (albumin 3.3). The UA showed a 4^+ albumin and CBC a left shift with 10,200 WBC. During the dialysis, the patient developed acute hypertension (BP ↑ 300/190) and acute hypertensive encephalopathy.

Patient was treated with large doses of reserpine and went into a profound depression characterized by catatonia, crying, and staring. Following the dialysis and cessation of reserpine, the patient was markedly improved, the clinical signs of uremia disappeared. The second dialysis was performed due to deterioration. Following the second dialysis, the patient was clinically and subjectively improved and stable for approximately one month, following which clinical deterioration occurred requiring a 3rd peritoneal dialysis. This did not appear to help the patient and it was decided not to prolong her misery. Terminally the patient developed oliguria, a high fever and rising BUN. On the morning of her death, 8-28-63, potassium was 9.1 and sodium 160.

DISCUSSION: This patient represents a typical patient with chronic renal failure; however, the course is atypical due to the fact her life was prolonged some two months. The danger of using excessive quantities of reserpine is seen here. Many side effects may occur with reserpine (nasal congestion, increased appetite, nightmare, tranquilizer) and the most serious one may produce a true depression. This reaction cannot be differentiated from the depressed phase of manic depressive psychosis or of involutional melancholia (29).

A yellowish - brown - gray discoloration of the face and

hands is characteristic in uremia (55) and is due to retention of urinary chromogens. It may resemble the discoloration found in pernicious anemia but it is not generalized, being more limited to exposed portions of the body.

Case 2 #36631 UNH H.T.

History and Clinical Course: This is a 21 year old N M who had been followed in the UND. Past history revealed an attack of acute glomerulonephritis in the fall of 1959. Patient was hospitalized in November, 1959, for edema of fingers, hands, face, and legs, and was started on steroids. Repeated hospitalizations were required for above and then the patient was followed in the clinics until hospitalization on 5-22-61 at BCMH for persistent albuminaria. UA at this time showed numerous oval fat bodies, maltese crosses and WBC. A needle biopsy of kidney demonstrated subacute membraneous glomerulonephritis. Conservative management was continued and in December, 1961, patient was taken off of steroids. Reserpine was started May, 1962, for ↑BP and digitalization was instigated in August with improvement for progressive weight gain and ↑BP. Patient was admitted to UNH for the first time on 10-8-62 with history of nausea, vomiting and shakiness.

Physical Exam.: BP 240/120 P 88 RR 26 Patient appeared pale and exhibited generalized neuromuscular irritability

and uremic breath. Fundiscopic exam demonstrated multiple flamed shaped hemorrhages.

TABLE NO. 2 A

LAB ADMISSION	mgm% BUN	mEq/l CO_2CP	mEq/l K^+	Na^+	Cl^-	mgm % Ca^{++}	mgm % PO_4	creatinine	Hb	FACTS
1st 10-9 Baseline	230	11	4.4	130	84	6.3	8.3	28	6.7	(1) 58 L/48 hrs.
Dialysis Ended	105	22	4.0	138	93	9.8	6.7	14		(2) 4300 cc fluid removed.
2nd 11-26 Baseline	275	14	5.35	118	79	-	-		4.9	(1) 122 L/64 hours
Dialysis Ended	63	29	3.2	135	94	-	-			(2)10,000 cc net gain
3rd 12-7 Baseline	225	12	4.7	125	89.5	8.3	-	20	8.6	(1)100 L/48 hours
Dialysis Ended	53	25	3.5	135	94	-	-			----------
4th 12-26 Baseline	223	12	4.6	120	91	8.6	6.1	23.2		(1)47 L/58 hours
Dialysis Ended	70	33	4.3	135	100	8.8	4.0			(2)21,460 cc fluid removed
5th No Dialysis										Hospitalized 1-12-63 for bleeding diathesis.
6th 1-21 Baseline	273	-	-	-	-	-	-		6.6	(1)66 1/52 hrs.
Dialysis Ended	88	30	4.25	138	97	9.8	3.6			(2)3000 cc removed
7th 2-6 Baseline	260	11	4.8	120	81.5	9.5	7.8		6.5	(1) Dialyzed 42 hours
Dialysis Ended	60	31	-	-	-	9.6	-			(2)26,995 fluid removed

TABLE NO. 3 A

1st ADMISSION:	BUN Plasma	182.5
	Peritoneal fluid	
	30 min.	142.5
	60 min.	155

Admissions: TABLE NO. 4 A

Interval			1st Adm.	10-8-62	10-11-62	
between	46	days	2nd	11-26	11-30	Lethany, insom., nausea
Dialysis	7	"	3rd	12-7	12-13	(↑ dyspnea, nausea vomiting
	54	"	4th	12-26	12-30	(↑ weight
	↓		5th	1-12	1-14	
	22	"	6th	1-21	1-24	Nausea, hematemesis
	13	"	7th	2-6	2-9	Nausea, hemoptysis, emesis

Comment: This patient is an example of a chronic glomerulonephritis with chronic renal failure necessitating repeated attempts of dialysis to maintain life. The ability of peritoneal dialysis to lower the BUN to 60-80 mgm % is well demonstrated and the electrolytes are all in physiologic ranges. The increasing severity of the kidneys is seen above by the decreasing periods between dialysis. The important fact here is demonstrated in that intermittent periods of dialysis is capable of keeping this individual active as well as prolonging life.

Table No. 3 A represents the time for the BUN to approach equilibrium across the peritoneal membrane. By the end of 30 minutes most of the electrolytes have

exchanged (142/132) and little advantage is obtained by waiting longer (155/182).

Case 3 #10611 (BCMH) U.F.

History: A 52 year old W F was admitted to BCMH on 9-26-61 with a history of rheumatic heart disease since infancy. Even as a child, the patient was weak, tired easy, and for the previous several years had done very little activity.

Physical: BP 150/90 P 66 RR 18/min.
Auscultation of the heart revealed an irregular irregularity, a coarse thrill overlying the precordium, a grade 4/6 systolic murmur, and the PMI was located in 6th ICS. A diastolic murmur was also present and the liver was palpable at 4 cm.

Clinical Course: On 10-3-61, a mitral valvuloplasty was performed, using extracorpeal circulation for 61 minutes. Following surgery, the patient went into acute renal shutdown, excreting only 25-50 cc dark amber urine between 10-4-61 and 10-6-61.

TABLE NO. 4 A

	BUN	CO_2CP	K^+	Na^+	Cl^-	PLASMA Hb
Following Surgery 10-6-61		23	3.4	140	104	114 mgm %
Begin P.D. 10-8-61	70	24	7.1	136		
End P.D.	91		5	136		
10-9	136					

Peritoneal dialysis was recommended on 10-6-61 due to pulmonary edema and hyperkalemia. The EKG demonstrated hyperkalemia. See

table No. 4 A. Peritoneal dialysis (P.D.) was continued from 10-6 to 10-8 (third to fifth post - operative days). Twenty-nine liters of dialysate was used over a 49 hour period and 5990 cc were removed. On 10-9, the patient was noted to be icteric (total bilirubin 6.7 3.2/4.7) The following day, the insertion of an A-V fistula for renal exchange was performed.

Hemodialysis was performed on 10-11-61 lowering the BUN from 180 to 92 mgm % and the K^+ to 5.3. The patient expired that evening due to peripheral vascular collapse and pulmonary edema.

Autopsy: Edema and congestion of lungs and congestion of liver, spleen and kidneys. Dilatation of L atrium, LVH and old rheumatic heart disease were present.

Microscopic exam of kidneys revealed epithelial degeneration of tubules and hemoglobin cast and protein percipitate in proximal and distal convuluted tubes.

Comment: Acute tubular necrosis and failure were related to hemolytic episode following surgery - see table No. 4 A for high plasma hemoglobin. The failure of the peritoneal dialysis to further lower the BUN is related to the small amount of dialysate used. Peritoneal dialysis is seen to correct the abnormal electrolyte picture and BUN as efficiently as hemodialysis does.

Case 4 #1178 (BCMH) A.W.

History: A 37 year old white male was transferred to BCMH (Hospital) on 1-18-63 with a history of acute oliguria, progressive

weight gain and uremia of 4 days duration. Five days prior, the patient had been discovered unconscious after being exposed to carbon monoxide gas for 11 hours.

Physical Examination : Patient was a large, obese white male, unresponsive except to noxious stimuli. Wt. equals 241 lb.

BP 208/130 P 108/min RR 28

Lab: See table No. 5 A

UA - Two to 3+ WBC, RBC and epithelial cells.

TABLE NO. 5 A

	DATE	mgm % BUN	mEq/l CO_2CP	mEq/l K^+	mEq/l Na^+	mEq/l Cl^-
	admission					
Hemodialysis	1-18-63	204	13	8.0	123	41
	end of Hemodialysis	108	15.5	4.6	134	93
	1-19 9$^{00}_{AM}$	176	12	8.0	132	91
Peritoneal	1-25	215	18.5	6.6	151	--
Dialysis	1-30	87	27	4.0	145	96
13 days 7 hours	2-2 1$^{45}_{AM}$	113	22	4.2	134	--
	2-7					
Hemodialysis	2-14					

Clinical Diagnosis: Acute Tubular Necrosis

Clinical Course: On admission, immediate hemodialysis was recommended due to severe hyperkalemia. However, on the day following hemodialysis, the laboratory values were approaching the values prior to hemodialysis and peritoneal dialysis was recommended. Impersol and Peridial solution was used, following the technic of Maxwell et al. (48),

over a period of 13 days 7 hours, during which time 292 liters of dialysate were used.

Patient was essentially anuric 1-18 to 1-31 15-36 cc/24 hrs. Following this urinary output began to rise until 2-8-63 and since then the urinary volume 600-1000 cc/day.

During the period of dialysis (11 days) the patient gradually became more responsive but absence of movement was noted in the upper extremity and loss of sensation in various areas of the body. The neurologic consult diagnosis was that of spotty cord involvement due to carbon monoxide intoxication.

On 1-26-63, the patient's temperature started to rise (see chart 1 A) and on 2-2-63 dialysis was discontinued due to peritonitis. Psuedomonas auerogenes, B-hemolytic streptococcus, and coaguluse positive staphylococcus was cultured from the dialysate.

CHART NO. 1 A	
1-25	OF
26	↑temp
27	101.4
28	103
29	102.8
31	103
24	101
2-3	100
7	99.4
11	97
12	100.4
13-14	101
15	102.2

Hemodialysis was again performed on 2-7-63 and 2-14-63 due to rising BUN and Clinical deterioration of the patient.

At this time, the patient is still oliguric and arterial cannulas are in place for future dialysis.

Comment: No attempt was undertaken to add additional antibiotics to the dialysate; therefore, peritoneal dialysis may not have been needed to be discontinued. Throughout the dialysis the clearance of the BUN, and electrolytes was maximal and therefore attaining physiologic values - even with the increased catabolism resulting from the increased temperatures. Here again, peritoneal dialysis was found to correct the electrolyte picture and BUN as efficiently as hemodialysis does.

Further peritoneal dialysis could be attempted in such an individual if the need arises.

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