SURVEY OF DIALYSES FOR ACUTE RENAL FAILURE AT CLEVELAND CLINIC HOSPITAL IN 1958

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THIS report presents the results of our most recent study of records concerning hemodialyses of acutely uremic patients. Our first study[1] was an analysis of the records of 38 patients dialyzed for acute renal failure in 1957; our current analysis concerns the records of the 27 patients dialyzed for acute renal failure in 1958. The correlation of the many variables has been facilitated by the McBee punch cards used in both studies.

Methods

Dialysis removes retention products and corrects electrolyte abnormalities; ultrafiltration at the same time may remove excess fluid. The twin-coil disposable artificial kidneys were used, as previously described.[1-4] For small children, only one coil was utilized[5] and dialysis was continued for four hours; for adults, dialysis usually lasted six hours. The radial artery or greater saphenous vein was used to draw blood from the patient, any other vein to return it. Usually polyvinyl chloride cannulae[1] were used. For the saphenous vein, plastic tubing of (French) sizes 12, 14, or 16 with multiple holes was inserted to the level of the inferior vena cava. The catheter was left in place for continuous administration of 40 per cent invert sugar (Travert†) if needed.[1] If risk of hemorrhage existed, regional heparinization[6] was used, and the patient's clotting time was maintained at normal values. While 200 mg. of heparin in 1,000 ml. of 5 per cent dextrose in water was being administered by constant drip under pressure to the blood inflow tube of the artificial kidney, 150 mg. of protamine sulfate in 700 ml. of 5 per cent dextrose in water was simultaneously administered to the outflow tube.

The fluid intake of the acutely uremic patient was restricted to 600 ml. per 24 hours.[7] Electrolytes were administered only when they were demonstrably lost.[8] A forced high-calorie, low-protein regimen was instituted insofar as was practical.[9]

Results and Comments

The analysis of the data concerning the 27 patients and 73 dialyses confirms the conclusions derived from the similar analysis[1] made for the preceding year. By summarizing the results of yearly analyses and perhaps also the experience of other centers now using an identical punch card system, statistically significant

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results may be obtained. One card summarizes the data for one patient or may be used to summarize the total data for one year *(Fig. 1)*.

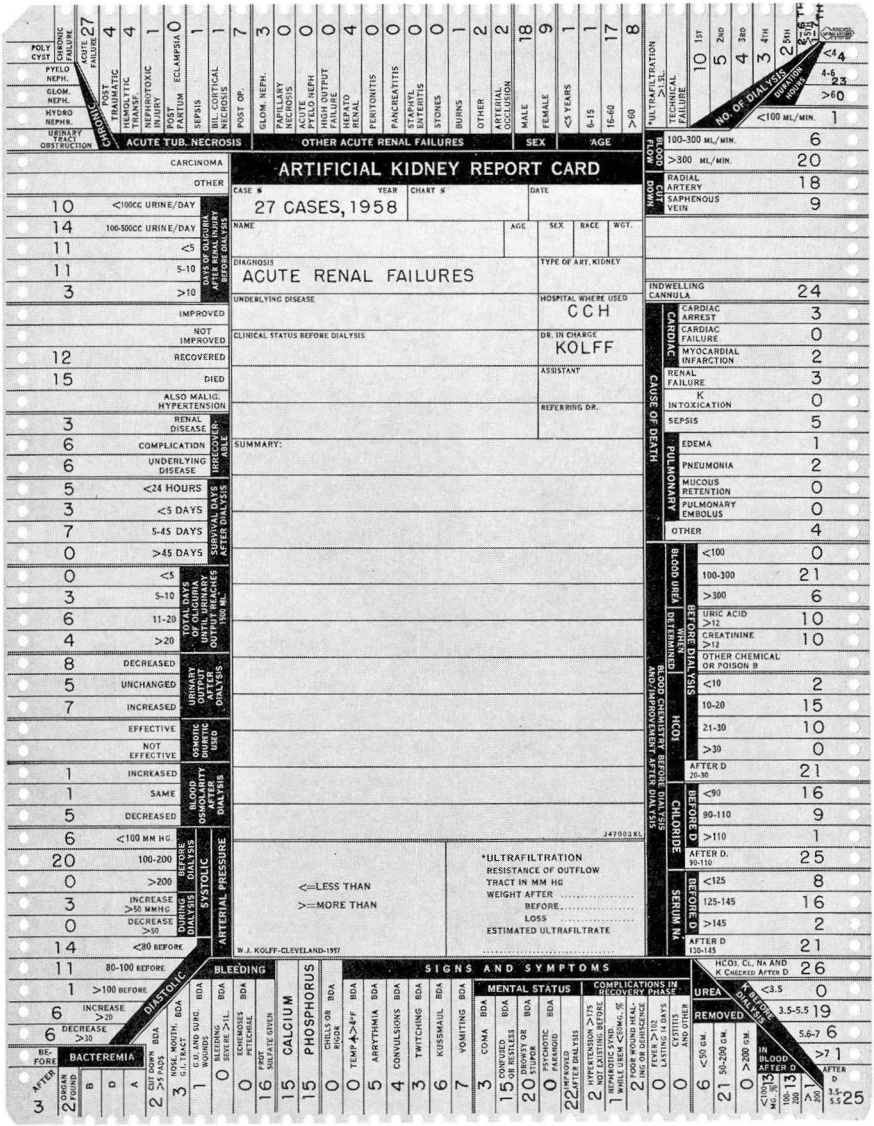

Fig. 1. The card shown in the photograph is used to summarize the data for 27 patients dialyzed in 1958. The data for each patient are filled in on his own card. The questions in the margin are answered with yes or no. For the positive answers the holes are cut open. A stack of cards is quickly analyzed with the help of an ice pick by "spearing" the particular item. The results of the summary can conveniently be entered on an extra card.

In 1958, 56 new patients were treated with a total of 142 dialyses. Twenty-seven patients (48 per cent) had acute renal failure and were treated with 73 dialyses; 12 (44 per cent) of those recovered; in 1957, 52 per cent of 38 patients recovered. Of the 15 patients (in 1958) who were dialyzed only once or twice, eight recovered. With more dialyses per patient, the percentage of mortality increased, but one patient recovered after five dialyses. Six of 18 men, and 6 of 9 women recovered. All six of the men more than 60 years of age died; only two women more than 60 years of age were dialyzed, one of whom survived.

Diagnoses and causes of acute renal failure. Table 1 presents the list of common causes of acute renal failure as it is printed on our punch cards. The greatest number of patients had renal failure associated with surgical procedures. However, the

Table 1.—*Summary of data on causes of acute renal failure in and survival rate for 27 patients treated with dialysis at Cleveland Clinic Hospital in 1958*

Diagnoses (order as on punch card)	Number of patients*		
	Total	Survived	Died
Acute tubular necrosis			
Post-traumatic	4	3	1
Hemolytic transfusion	4	4	0
Nephrotoxic injury	1	1	0
Postpartum—eclampsia	0	0	0
Sepsis	1	1	0
Bilateral cortical necrosis	1	0	1
Postoperative	7	2	5
Other acute renal failures			
Glomerulonephritis	3	2	1
Papillary necrosis	0	0	0
Acute pyelonephritis	0	0	0
High output failure	0	0	0
Hepatorenal syndrome	4	0	4
Peritonitis	0	0	0
Pancreatitis	0	0	0
Staphylococcal enteritis	0	0	0
Stones	0	0	0
Burns	1	0	1
Other	2	0	2
Arterial occlusion	2	0	2

*Three patients had two causes for acute renal failure.

high mortality, five of seven patients, was attributable to causes not immediately related to surgery. For example, three of the patients died of cardiac disease. One of the two patients in whom anuria developed after aortic grafting recovered.

Four patients with hemolytic transfusion reaction recovered and left the hospital. One reaction had occurred on the basis of sensitization to the Kell factor. This patient was readmitted because of severe serum hepatitis about six weeks later, and died. Three other patients had hepatorenal syndrome that was associated with hepatic necrosis, and all three died. It would be incorrect to conclude from the results in this small series that the artificial kidney has nothing to offer in cases of hepatorenal syndrome. Rather, the results confirm previous experience that when hepatic disease is progressive, the renal damage does not reverse either. However, a patient may be saved by dialysis from uremic death and may recover from the hepatorenal syndrome if the hepatic failure is reversible.

Two patients had renal arterial occlusion. In one patient occlusion occurred after an aortic graft; cholesterol-containing emboli of the small renal arteries led to focal cortical infarcts. The other patient had spontaneous renal arterial thrombosis related to severe arteriosclerosis and an aortic aneurysm. The diagnosis may be suspected on the basis of severe pain. Hypertension need not follow. Nothing short of arteriography can establish the diagnosis and the extent of the occlusion.

In *Table 1* two patients are listed under "Other." One had membranous glomerulopathy and acute tubular necrosis of unknown origin. The diagnosis can only be established by renal biopsy, and the treatment currently consists of large doses of corticosteroids. Sepsis at the end of a prolonged illness notwithstanding temporary improvement finally led to this patient's death. The other patient had a large myocardial infarction that resulted in shock, renal failure, and finally cardiac tamponade.

Immediate causes of death. If, in desperately ill patients in uremia, life is to be prolonged or perhaps saved, the currently prevailing causes of death should be recognized in the hope that they can be eliminated in the future. This is the purpose for the analysis in *Table 2.* There may be more than one contributory cause of death in a patient. Three patients died during dialysis, only one patient during the first dialysis; in 1957 there were no deaths during dialysis. These fatalities reflect the desperateness of the attempt to save life when the clinical outlook is almost hopeless. Constant supervision with a cardioscope during dialysis aims at earlier recognition of abnormalities in heart rate or in QRS pattern in the hope of avoiding sudden death. Some patients are indeed as much or more in need of a heart-lung machine as of an artificial kidney to prolong life. A combination of these services is being planned for the near future.

At the end of illness when all the data are available after a necropsy, it usually is possible to determine whether or not the patient was potentially curable when first examined. The records of the potentially recoverable patients can be traced

on our punch cards. It is in this category that future improvement of management and diagnosis should be rewarding. The necessity of profound diagnostic studies and alertness for new evidence is obvious; however, these patients should not be literally "studied to death," and often it is sound judgment to postpone diagnostic procedures in the hope that clinical improvement may be obtained first. Last year four patients died who could be classified as having had potentially recoverable disease. None of the patients who died this year could be so classified.

Of the four patients classified as having irrecoverable renal disease, one woman, aged 69 years had bilateral cortical necrosis and died 35 days after having virtually complete anuria. A 58-year-old man had an enlargement of the right kidney; biopsy revealed acute glomerulonephritis. Though the urinary output at one time attained 1,000 ml. per 24 hours, oliguria recurred and clinical evidence suggested progression to subacute glomerulonephritis. An aortogram revealed an obstructed left renal artery. At the present status of our knowledge, these diseases are irrecoverable.

Four patients had irrecoverable complications: overwhelming sepsis, septicemia, abscesses, and peritonitis. Three patients had irrecoverable underlying disease. One patient had a recognized myocardial infarction and unrecognized rupture of the left ventricle. Two patients were listed as having irrecoverable complications

Table 2.—*Immediate cause of death of 15 patients treated with dialysis for acute renal failure at Cleveland Clinic Hospital in 1958*

Immediate cause of death (order as on punch card)	Number of patients*		
	Total	Cause recognized before death	Cause not known until necropsy
Cardiac	5	5	0
Cardiac arrest	(3)	(3)	(0)
Cardiac failure	(0)	—	—
Myocardial infarction	(2)	(2)	(0)
Renal failure	3	2	1
Potassium intoxication	0	—	—
Sepsis	5	3	2
Pulmonary	3	1	2
Edema	(1)	(0)	(1)
Pneumonia	(2)	(1)	(1)
Mucus retention	(0)	—	—
Pulmonary embolus	(0)	—	—
Other	4	4	0

*In one patient there may be two causes of death.

and irrecoverable underlying disease. One patient had necrosis of the liver as the underlying disease and multiple acute occlusions of the hepatic, splenic, and mesenteric arteries, and terminal aorta. The other patient primarily had post-necrotic cirrhosis with severe generalized ischemic necrosis of the liver and peritonitis as complications

Survival time. Five desperately ill patients died within 24 hours of the first dialysis. Four of these had to be dialyzed less than five days after the onset of renal injury, indicating the severity of their disease. Three patients lived 32, 34, and 36 days, respectively, after the first dialysis, although they later were proved to have irrecoverable disease. This is a testimony to the effectiveness of dialysis to maintain life even in the absence of renal function.*

Degree and duration of oliguria. Four of the six survivors with an initial daily output of less than 100 ml. required more than 20 days (24, 28, 30, 34 days respectively) for diuresis to occur. This confirms last year's observation that the more severe oliguria takes longer to recover from; however, the severity of oliguria does not predetermine the final outcome. Indeed, 6 of the 10 severely oliguric patients (but only 5 of the 14 mildly oliguric patients) recovered. This was the same ratio as the year before. One patient entered a diuretic phase after seven days of oliguria, but died in a late diuretic phase of complicating peritonitis. The occurrence of diuresis does not guarantee recovery.

Chemical efficiency of dialysis. Tabulations of chemical results show that although each dialysis led to improvement, the chemical pattern deteriorated again before the next dialysis. A continuous system of dialysis would be preferable though it is not practical. The next best solution is to dialyze repeatedly at short intervals; for example, every two or three days. We are doing this with gratifying clinical results. Five of the six patients with initial blood urea concentrations greater than 300 mg. per 100 ml. recovered. Serum uric acid and creatinine concentrations higher than 12 mg. per 100 ml. were followed by approximately 50 per cent survival, which is roughly identical to the survival percentage of the entire group.

Prognosis was not related to initial values of blood urea nor to electrolyte abnormality in this series; neither factor determines the patient's recovery. No difference in the extent of electrolyte derangement was found between those who survived and those who died. Dialysis corrects the initial chemical abnormalities so effectively that they play no role in the eventual occurrence of death, rather, the outcome is determined by the presence or absence of irrecoverable disease.

There were only two patients who had a carbon dioxide content reserve of less than 10 mEq. per liter. Both survived; one of them also had a blood urea concentration of 430 mg. per 100 ml. Both of those patients had normal serum sodium concentrations respectively of 145 and 143 mEq. per liter.

A blood urea concentration greater than 300 mg. per 100 ml. did not preclude

*One patient not described here because her renal disease was chronic, survived for seven days in the absence of effective renal function, thanks to dialysis.

rapid recovery in one patient, but three of five patients with blood urea values higher than 300 mg. per 100 ml. needed more than 20 days to achieve diuresis. Again, as in the analysis of 1957,[1] there was no correlation between the severity of oliguria and the survival rate; between the degree of oliguria and the concentration of the blood urea; nor between the duration of oliguria prior to dialysis and the predialytic concentration of the blood urea.

In seven of the eight survivors with a blood urea concentration of more than 300 mg. per 100 ml., the serum creatinine content was more than 12 mg. and the serum uric acid more than 12 mg. per 100 ml. Only once was a uric acid value higher than 12 mg. associated with a blood urea of less than 100 mg. per 100 ml. These exceptions should be kept in mind. The blood urea concentration generally is an excellent though not infallible guide as to the severity of the renal failure.

Summary

Twenty-seven patients in acute renal failure were treated at the Cleveland Clinic Hospital with 73 hemodialyses in 1958. The over-all recovery rate was 44 per cent as compared to 52 per cent in 1957. Eight patients were older than 60 years of age, of whom only one recovered. At the present status of our knowledge, 15 patients who died were considered hopeless because the renal disease (three patients), the complications (six patients), or the underlying disease (six patients) was irrecoverable.

The degree of oliguria again gave an indication for the duration of the renal failure. While the exact time of diuresis was unpredictable, more than 10 days was always required if the initial urinary volume after the insult (at the onset of anuria or oliguria) was less than 100 ml. per 24 hours. The duration of oliguria gave no clue as to the eventual outcome, since two patients who required more than 30 days for the onset of diuresis recovered. The efficiency of dialysis in correcting water and electrolyte disturbances and in removing nitrogenous products was again demonstrated. Not one patient in acute renal failure with potentially recoverable disease died.

Data: The original tables upon which this study is based and from which the conclusions are drawn are deposited at The Frank E. Bunts Educational Institute and are available for consultation upon request to the Director of Education of the Institute.

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