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Clinical Uses of the Artificial Kidney

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I. CLINICAL USES OF THE ARTIFICIAL KIDNEY*

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Among the various types of acute renal failure are those in which recovery of renal function may be expected if the patient can be maintained through the acute episode. To aid in temporarily assuming some of the functions of the kidney during the period of acute failure a number of technics have been developed. The most promising of these have been peritoneal irrigation[7, 8, 9], intestinal lavage[17, 24], and the artificial kidney.

HISTORICAL REVIEW

While the development of a practical clinical model suitable for use in patients had to wait until 1944[14], it is of interest that the basic principles inherent in all later artificial kidneys were first set forth in 1914 at which time Abel, Rowntree, and Turner[1], working in the Department of Pharmacology at Johns Hopkins Medical School developed their process of "vivi-diffusion". They envisioned its ultimate clinical use in the patient with an accumulation of diffusable toxic substances. Their machine consisted of a series of celloidin tubes in a medium of saline with rubber tubes leading from the celloidin to cannulae in an artery and a vein. Hirudin, an extract of leech heads, was their anticoagulant. They mentioned survivals in dogs after three hours of dialysis, and one animal lived during a 16-hour period of dialysis but died later. They concluded that such non-protein substances as urea, sugar, phosphates and aspirin could be removed by the method. The work of these three men was monumental in establishing a base line for the future development of artificial kidneys.

Between 1914 and 1944 when Kolff[14] in the Netherlands reported the use of an artificial kidney in human patients, there was a gradual evolution toward a more practical machine. Heparin proved to be a much more reliable and safe anticoagulant than hirudin, and cellophane was shown in 1937 to be superior in both strength and uniformity to celloidin or other dialyzing membranes in use at the time. In 1944 Kolff introduced a new type of artificial kidney with cellophane tubing wrapped on a revolving drum providing a much greater dialyzing area than had been possible in earlier models. In one case report in which short dialyzing periods were used it succeeded in removing 20-40 grams of urea a day from a patient with chronic nephritis. This machine was criticized by Alwall[2] who in 1947 had developed another artificial kidney model. Alwall claimed first, that the Kolff kidney traumatized the red cells because it was necessary to use a pump, secondly, that the colloid osmotic pressure of the blood was not equalized resulting in a fluid loss and, finally, that the Kolff machine was excessively complicated. Alwall's machine consisted of a cellophane loop wrapped on a grooved cylinder, with another cylinder placed outside this at a distance calculated to keep the pressure on the blood in the cellophane tubing about equal to the colloid osmotic pressure.

Independently, Murray of Canada[22] developed and used clinically an artificial kidney consisting of small diameter cellophane tubing wrapped around a stationary verticle cylinder immersed in a bath. The blood was pumped from the inferior vena cava and allowed to run back into another vein.

A different type of artificial kidney was recently introduced by Skeggs and Leonard[27] in 1948. Their machine had rubber pads treated with silicone between which were sheets of cellophane. Blood flowed on one side of the cellophane and dialyzing solution on the other. They claimed full therapeutic potency with several of these "units" in tandem, but most of their work has been of an experimental nature.

*These researches were supported, in part, by a grant-in-aid to Doctor Lewis from The Graduate School, University of Minnesota.

A more extensive review of the literature pertinent to the development of the artificial kidney can be obtained from some of the articles listed in the bibliography, among which that of Norvitt [23] is of special merit.

TECHNICAL CONSIDERATIONS

The apparatus we have used is a modification of the Kolff artificial kidney* developed by Merrill[18]. It consists essentially of a large rotating wire mesh drum around which is wrapped in a spiral fashion 120 feet of cellophane tubing. The patient's blood leaves the cannulated radial artery in a plastic tube, passes through a rotating coupling in the axle of the drum, and enters one end of the cellophane tubing where rotation of the drum combined with the effect of gravity moves the blood through its length. At the far end of the drum the stream of blood leaves through a second rotating plastic coupling and is elevated by a pump to a burette from which it flows by gravity through a clot catcher and down into the patient's cannulated antecubital vein.

The rotating drum is partially immersed in a 100-liter bath wherein the dialysis takes place. This bath is tap water, usually containing the constituents listed in Table I.

TABLE I
Constituents of the Bath Fluid
Gm/Liter of Tap Water

Na Cl	6.60
Na HCO_3	2.25
K Cl	.30
Ca Cl_2	.40
Mg Cl_2	.10
Glucose	2.00
Total Na+ in Meq/L	140.00
Total Cl- in Meq/L	126.00

These may be varied in each case depending upon the chemical concentrations in the patient's blood and his state of hydration. To assure an adequate diffusion gradient, the entire bath is changed after 3 hours of the usual 6-hour dialysis. The bath water is kept at a temperature of 101° F with thermostatically controlled heating units, thus assuring return of blood to the patient at approximately body temperature.

Clotting of blood in the machine is prevented by heparinizing the patient and allowing the blood to come in contact with only plastic tubing, plastic connections, and silicone-treated[12] glassware. The average adult patients were given from 180 to 220 mgm. of heparin during a 6-hour period of dialysis. Oozing of blood from the wounds of the arterial and venous dissections was occasionally troublesome but in no case did a large hemorrhage occur. Nevertheless we have considered serious recent hemorrhage such as massive hematemesis or melena, a contraindication to use of the artificial kidney.

Hemolysis produced by the artificial kidney has been no problem in our experience nor in that of others who have used this apparatus. The same precautions and use of materials which lessen the likelihood of clotting also reduce the probability of hemolysis.

In preparation for connecting the apparatus to the patient, the radial artery and a large antecubital vein are isolated through small incisions under local anesthesia, and at the same time the assembled machine is being irrigated with saline and tested for leakage. Then while silicone-treated glass cannulae are inserted into the artery and vein, the artificial kidney is charged with 500-600 cc. of crossmatched blood, finally the vein and artery are connected to the machine, and inflow and outflow are started simultaneously. Flow of blood through the apparatus is adjusted to a rate of 150-200 cc. a minute by tunnel clamps on the inflow and outflow tubes. The flow is measured by observing the rate of blood level rise in the burette when its outflow is clamped, and an un-

*Manufactured by Edward A. Olson, Ashland, Mass.

balanced arterial flow is detected by observing the amount of distention in the distal cellophane spirals.

RESULTS

1. Clinical Changes During the Dialysis:

During the period of dialysis, certain changes in the patients have been noted. Those not comatose were understandably apprehensive at first but even without sedation they soon became drowsy and frequently slept through most of the procedure. This sedative effect is unexplained at present. It occurred more rapidly than changes in the level of blood chemistries measured and it disappeared when the dialysis was terminated, though at this time changes produced in the blood chemistries were maximal.

A more lasting effect was an increase in mental alertness and sense of well-being noted in most patients when they were aroused near the end of the dialysis. Occasionally comatose patients became aware of their surroundings and were able to recognize and speak to their relatives. These evidences of clinical improvement roughly paralleled the changes produced in blood chemistries though a simple chemical explanation is unlikely, for it has been amply demonstrated that changes in individual blood electrolytes or metabolites do not cause the symptom complex of uremia[10, 25].

A temporary elevation of blood pressure during dialysis was occasionally observed, with the systolic pressure rising 40 mm. of mercury or more above the initial values during 9 out of 25 dialyzing procedures. The diastolic pressure did not rise correspondingly and there was little change in pulse rate in these cases. A similar hypertensive response was noted by Merrill, et al,[19] and cardiac output studies in one of their cases led them to believe that a peripheral arteriolar constriction was the mechanism responsible for the elevated blood pressure.

2. Patients Treated and Ultimate Effects:

To date the artificial kidney has been used here 25 times on 15 patients afflicted with various acute or chronic diseases. The results are summarized in Table II. In most cases the immediate results have been good but it has been seldom that the ultimate course of the disease has been distinctly modified. This is in large degree due to the type of patient we have treated, for those with chronic diseases were generally in the terminal stages of their illness and most of those with acute renal failure, which we have treated, suffered from a number of additional complications which frequently cause death even in the face of good renal function. We have not limited our use of this apparatus to the infrequent case in which recovery might fairly be expected but have attempted to explore its usefulness in a rather wide variety of desperate situations.

Six patients treated were suffering from acute renal failure of the type known as lower nephron-nephrosis[16] and characterized pathologically by tubular damage and occlusion. One of these (No. 14) had a reaction following a blood transfusion which was given for hemorrhage from uterine myomata, and recovered completely after a stormy course. It was felt that the artificial kidney was of distinct benefit in her case. A second patient (No. 13) developed anuria following a motorcycle accident and ultimately died with a traumatically destroyed right kidney and lower nephron-nephrosis of the left kidney. An interesting feature of his disease was the occurrence of massive hemorrhage from what appeared to be an acute peptic ulcer. Four patients (Nos. 1, 2, 5 and 10) developed anuria after extensive surgery. In two of these it was felt that life was prolonged by use of the artificial kidney but all ultimatley died with grave complications.

In addition to the 6 patients with acute renal failure described above, we treated 2 other patients with acute renal failure due to acute or subacute glomerular nephritis. One of these (No. 8) succumbed with a hemorrhagic pneumonia which was thought to be pulmonary edema

TABLE II

PATIENTS TREATED WITH ARTIFICIAL KIDNEY

No.	Pt.	Sex	Age	Diagnosis and Complications	Procedure Number	Results	Living 1 Mo. later	Days of Anuria*	Days of Oliguria**
1		F	70	Renal tubular and vascular disease; ca. of breast post-op.; mediastinitis; pulmonary embolis	1	Died 24 hours after dialysis, immediate clinical improvement	No	5	3
2		F	48	Lower nephron-nephrosis; post-op. resection of recurrent cancer; peritonitis; pancreatitis	2 3 4 5 6	Died 24 hours after last dialysis; life may have been prolonged	No	15	8
3		F	37	Malignant hypertension with uremia	7	Died 27 days after dialysis; immediate result was good	No	0	0
4		F	47	Polycystic disease of kidneys and liver	8	Immediate results good, died 4 months later	Yes	0	0
5		M	57	Lower nephron-nephrosis; post-op. bile duct exploration; peritonitis; liver necrosis	9 10 11	Immediate results good after each dialysis; diuresis did not occur	No	22	0
6		M	62	Vesicle neck obstruction; B.P.H.; bilateral pyohydronephrosis	12	Immediate result good; died later of uremia	Yes	0	0
7		F	44	Encephalitis, acute liver necrosis, diabetic acidosis	13	Died a few hours later	No	0	? (2+)

*Anuria = less than 100 cc urine a day

**Oliguria = 100 - 300 cc urine a day

TABLE II (Cont.)
PATIENTS TREATED WITH ARTIFICIAL KIDNEY

No.	Pt.	Sex	Age	Diagnosis and Complications	Procedure Number	Results	Living 1 Mo. later	Days of Anuria	Days of Oliguria
8		M	22	Subacute glomerular nephritis; hemorrhagic pneumonia	14	Dialysis satisfactory; patient died 36 hours later	No	13	?
9		M	14	Aplasia of rt. kidney and hydronephrosis of left; pyelitis	15 16	Dialysis used twice in preparation for surgery; convulsions after 2nd run; intestinal loop made	Yes	0	0
10		M	52	Acute renal failure; post-op. liver resection for ca. of gallbladder; subphrenic abscess	17	Died 24 hours later	No	3	5
11		M	58	Cirrhosis of the liver; hepatic coma; ca.of the liver	18	Partially aroused from coma after dialysis	No	0	0
12		M	14	Chronic glomerular nephritis in late stage; pulmonary edema	19	Developed convulsions and died before dialysis was completed	No	0	0
13		M	19	Lower nephron-nephrosis; traumatic laceration of rt. kidney; retroperitoneal hemorrhage; bleeding peptic ulcer; peritonitis	20 21	Immediate response good, died 6 days after last dialysis	No	12	5
14		F	36	Lower nephron-nephrosis; transfusion following hemorrhage from uterus; pulmonary edema	22	Excellent; complete recovery	Yes	9	2
15		M	8	Acute glomerular nephritis	23 24 25	Immediate results good	?	25+	0

before autopsy. The other, an 8-year-old boy (No. 15), has survivied 24 days of anuria and three dialyses at this writing and is still in good clinical condition.

Five patients suffering from various types of chronic renal disease have been treated with the artificial kidney and in four of them (Nos. 3, 4, 6 and 9) some temporary benefit was gained. The fifth patient, a child who was in the terminal stages of chronic renal failure, expired following a series of convulsions while undergoing dialysis. Though he gained only 0.5 Kg. in body weight while attached to the artificial kidney, he was considered to be slightly overweight beforehand and we feel that more intensive efforts to dehydrate him during the dialysis were in order. Unfortunately autopsy was not granted and other mechanisms explaining his sudden death may remain hidden. Of the four patients who obtained a temporary benefit from use of the artificial kidney, one child, who had congenital renal aplasia and hydronephrosis, was twice prepared for surgery and at the second operation an intestinal loop was made for perfusion, as has been suggested by Kolff[15, 29]. Despite these efforts the patient finally died. It is possible that the other three patients with chronic uremia may have survived longer if the artificial kidney had been used repeatedly rather than just once, though of course the real value of such palliation may be held in question.

Two other patients have been treated with the artificial kidney (Nos. 7 and 11); one had cirrhosis with carcinoma of the liver and the other had encephalitis with diabetic acidosis. Though the patient with cirrhosis showed an increased mental alertness lasting a few hours after dialysis, both succumbed rapidly to their diseases.

3. Blood Urea Levels and Urea Recovery in the Bath:

Though it is recognized[10, 25] that high blood urea levels alone do not cause the clinical picture of uremia, fall in blood urea is directly correlated with clinical improvement and, in addition, is a good measure of the efficiency of an artificial kidney. In Table III the blood urea nitrogen drop and the amount of urea removed in the bath are shown for each case in which a satisfactory 6-hour dialysis was obtained. This Table includes all but two occasions when the artificial kidney was used. During each of these satisfactory treatments a gratifying reduction in blood urea nitrogen was obtained, with the removal of up to 197 grams of urea in the bath.

The blood urea nitrogen values returned to their former levels at variable rates after dialysis. In the patients with acute uremia having very low urine outputs, this usually took place within 5 to 7 days while, as might be expected, in those with chronic uremia and a good urine volume the rise in blood urea nitrogen occurred much more slowly and occasionally took several weeks.

4. Other Changes in the Blood Chemistries:

In renal failure, and especially when the urine output is very low and increased tissue break-down is taking place, the serum potassium levels may rise to high values[11]. In these cases death may be hastened by the toxic action of potassium on the heart[4, 5, 6, 13]. To avoid this eventuality in the uremic patient the artificial kidney provides the most efficient method of rapidly lowering the serum potassium concentration. Using the artificial kidney on dogs, it is possible to extract more potassium than would be expected in the total extracellular fluid at the beginning of the dialysis[26]. We have demonstrated this in patients as well. Yet, despite this relatively great extraction of potassium, the serum levels do not fall to dangerously low levels, doubtless because of a shift from the intracellular space.

On 12 occasions when the artificial kidney was used, potassium was omitted from the bath water during half or all of the dialyzing period in order to reduce high serum potassium levels. As is shown in Table IV, an adequate reduction in serum potassium was obtained in every

TABLE III
FALL IN BLOOD UREA NITROGEN AND UREA REMOVED
IN THE BATH WITH USE OF THE ARTIFICIAL KIDNEY

Patient	Initial B U N (mgm %)	Final B U N (mgm %)	Urea N removed (gm)	Urea removed (gm)
	120	85	33	71
(1)	119	46	33	71
(2)	148	43	36	77
(3)	105	19	28	60
(4)	151	64	24	51
	105	59	33	71
	130	46	36	77
(1)	93	44	33	71
(2)	154	58	38	82
(3)	227	66	41	88
	208	66	55	118
	88	33	23	49
	170	88	52	112
(1)	140	41	26	56
(2)	180	43	33	71
	125	45	37	79
(1)	278	92	92	197
(2)	232	75	73	157
	205	72	50	107
(1)	166	30	25	54
(2)	158	38	25	54
(3)	105	22	20	43

TABLE IV
REDUCTION IN SERUM POTASSIUM
WITH USE OF THE ARTIFICIAL KIDNEY

Patient	Serum Potassium before Dialysis	Serum Potassium after Dialysis
	6.7	4.3
(1)	6.9	4.0
(3)	7.8	4.7
(4)	5.9	5.3
	7.2	4.6
	5.6	3.7
	7.3	4.3
	5.6	3.6
	8.4	4.4
	6.0	4.8
	4.2	2.5
	7.6	3.6

instance. A return toward normal in the electrocardiographic tracings paralleled the fall in serum potassium levels.

The other blood metabolites and electrolytes, measured before and after use of the artificial kidney, all shifted toward their normal levels during dialysis. Decreases in creatinine and uric acid paralleled those of blood urea nitrogen. The low bicarbonate levels customarily found before dialysis as an indication of metabolic acidosis were elevated toward the normal value, but usually some acidosis remained. Blood chloride concentrations were easily adjusted, and the hypochloremia found almost uniformly beforehand was completely corrected or overcorrected in every case without danger of the overhydration which may follow attempts to correct hypochloremia by use of intravenous salt solutions

in oliguric patients. The low serum sodium values found in most of the patients treated were elevated to the normal range regularly and the one patient with a hypernatremia had a fall in his serum sodium from 185 to 161 milliequivalents per liter. It is important to note that this fall in sodium concentration was accompanied by a decrease in body weight. Blood calcium concentrations, which were usually low before dialysis, were raised to normal or above normal by the procedure and phosphorus levels fell toward but never quite to normal.

5. Overhydration:

Overhydration is a common complication of acute renal failure, usually due to the overzealous administration of parenteral fluids. A number of our patients had both peripheral and pulmonary edema before they were treated with the artificial kidney. This complication is so serious that it has been indicted as a more important cause of death in acute renal insufficiency than nitrogen retention or acidosis[28].

It is possible, with proper use of the artificial kidney, to effect a rapid dehydration in these patients. This is done by making the bath hypertonic through the addition of glucose in amounts sufficient to make a concentration up to 2 or 2½ per cent. When these high concentrations of glucose were used, small doses of insulin were given during the procedure to lower the blood sugar and thus keep a high differential gradient between the bath and the patient's blood. Incidentally, the glycogenesis brought about by this technic may have been helpful to the patient's liver. In controlling dehydration during dialysis we have found frequent determinations of body weight of some value.

Listed in Table V are body weights before and after dialysis taken on those occasions when dehydration was attempted. All of these patients lost weight, the largest amount being 4 kilograms during a 6-hour dialysis in a grossly over-hydrated, anuric, comatose man who showed a striking diminution in his mental stupor during and after use of the artificial kidney. Some of the other patients in this group might have benefited more if even greater efforts had been made to bring about a rapid dehydration with the artificial kidney and, it is doubtless true, that efforts at dehydration should have been made in a few additional patients on whom overhydration before dialysis was not easily detected.

TABLE V
BODY WEIGHTS BEFORE AND AFTER DIALYSIS
DEHYDRATION ATTEMPTED

Patient	Body Weight Before (K9)	Body Weight After (K9)
(1)	62.4	61.0
(2)	60.2	57.5
	61.8	58.8
	50.2	49.6
	67.3	63.2
	67.5	64.3
	57.2	56.7
	25.9	25.4

DISCUSSION

The importance of good conservative management for patients treated with the artificial kidney cannot be overemphasized. Correction of anemia and adequate nutrition are both important aspects of the treatment, but of even greater importance is the careful avoidance of overhydration during the anuric phase of the disease. In fact, it is probably true that development of the so-called "three-phase" conservative management of patients with acute renal insufficiency[3, 20, 21, 28], has been more important in lowering the fatality rate of lower nephron-nephrosis than perfection of the artificial kidney, peritoneal dialysis, or intestinal lavage.

It is interesting, as pointed out by Strauss[28], that during the early part of the century there were a number of case reports of individuals who lived three or more weeks while anuric, yet in recent

years the fatality rate of lower nephron-nephrosis was considered to be 90 per cent and in Lucke's series none survived more than 20 days of oliguria[16]. Strauss attributed this difference to the fact that intravenous fluids were not used by the earlier reporters and were used too liberally in later years. At the present time, with judicious management and without additional complicating factors, the prognosis for acute renal insufficiency due to lower nephron-nephrosis should be good even without use of the artificial kidney.

The question then is: What cases of acute renal insufficiency, if any, should be subjected to use of the artificial kidney? No doubt its use will continue to be suggested as a desperate last chance measure in the terminally ill patient with anuria, and we shall still accept such candidates with perhaps little anticipation of improving upon our present poor record. However, there is hope of benefiting many patients if they are treated earlier in the course of their disease. Certainly it would appear that the development of hyperkalemia or definite overhydration in cases of acute renal insufficiency are valid indications for treatment with the artificial kidney, for dialysis offers the most rapid and efficient method of correcting these dangerous complications.

Further, we feel that oliguria or anuria of over 5 and 6 day's duration should suggest treatment with the artificial kidney. Most of the reported cases of lower nephron-nephrosis which were successfully treated by conservative methods alone began to have diuresis after 4 to 10 days of low urine output[3,21], which fact implies that patients showing no indication of an increase in urine output after 6 days may need the help of the artificial kidney. In fact, in our own experience with patients who developed postoperative oliguria, none who received only conservative treatment during the past 2 years has survived more than 10 days of anuria or oliguria whereas, as may be seen in Table II, with use of the artificial kidney two similar patients survived over 20 days of low urine output.

In patients with chronic uremia the artificial kidney has a limited usefulness. It might be an aid in preparing some of these for operations which may be needed to correct the defects which have led to their uremia. Some temporary benefit has also been obtained for patients suffering from non-surgical types of chronic uremia but the ultimate course of their illness has not been changed.

We have had no success with use of the artificial kidney in treating one case with cirrhosis and hepatic coma and have, as yet, gained no experience in treating some of the other clinical conditions for which its use has been suggested, such as barbiturate poisoning and certain shock-like states[19].

SUMMARY AND CONCLUSIONS

1. An artificial kidney used at the University of Minnesota Hospitals during the past year and the technical aspects of its application have been described.

2. This apparatus has been used 25 times in the treatment of 15 patients. Eight of these patients had acute renal failure of various types, 5 had chronic renal failure, 1 had hepatic cirrhosis, and 1 had encephalitis complicated by diabetic acidosis.

3. Most of these patients were temporarily benefited by treatment with the artificial kidney. A prompt lowering of the protein metabolite levels in the blood and an adjustment of the serum electrolyte levels toward normal was obtained in every case. It was possible to reduce the body weight when overhydration was present.

4. It is suggested that the artificial kidney be used in those cases of acute renal failure where hyperkalemia or overhydration has developed and when oliguria has persisted for over 6 days. In patients with chronic uremia the artificial kidney may be helpful in preparing suitable candidates for surgery.

REFERENCES

1. Abel, J. J., Rowntree, L.G., and Turner, B.B:
On the removal of diffusible substances from the circulating blood of living animals by dialysis.
J. Pharmacol. & Exper. Therap., 5:275-316, 1914.

2. Alwall, N., and Norviit, L.:
On artificial kidney; the effectivity of the apparatus.
Acta med. Scandinav., (Suppl. 196) 128: 250-258, 1947.

3. Coller, F., Campbell, K.M., and Icb, V.:
The treatment of renal insufficiency in the surgical patient.
Ann. Surg., 128: 379-390, 1948.

4. Crismon, J.M., Crismon, C.S., Calabresi, M., and Darrow, D.C.:
Electrolyte redistribution in cat heart and skeletal muscle in potassium poisoning.
Am. J. Physiol., 139: 667-674, 1943.

5. Currens, J.H., and Crawford, J.D.:
The electrocardiogram and disturbance of potassium metabolism,
New England J. Med., 243: 843-850, 1950.

6. Finch, C.A., Sawyer, C.G., and Flynn, M.M.:
Clinical syndrome of potassium intoxication.
Am. J. Med., 1: 337-352, 1946.

7. Fine, J., Frank, H.A., and Seligman, A.M.:
The treatment of acute renal failure by peritoneal irrigation.
Ann. Surg., 124: 857-870, 1946.

8. Frank, H.A., Seligman, A.M., and Fine, J.:
Further experiences with peritoneal irrigation for acute renal failure, including description of modifications in method.
Ann. Surg., 128: 561-608, 1948.

9. Grollman, A., Turner, L.B., and McLean, J.A.:
Intermittent peritoneal lavage in nephrectomized dogs and its application to the human being. A.M.A.
Arch. Int. Med., 87: 379-390, 1951.

10. Harrison, T.R., and Mason, M.F.:
The pathogenesis of the uremic syndrome.
Medicine, 16: 1-44, 1937.

11. Hoffman, W.S.:
Clinical physiology of potassium.
J.A.M.A., 144: 1157-1161, 1950.

12. Jaques, L.B., Fidler, E., Feldsted, E.T., and MacDonald, A.G.:
Silicones and blood coagulation.
Canad. M. A. J., 55: 26, 1946.

13. Keith, N.M., and Burchell, H.B.:
Clinical intoxication with potassium, its occurrence in severe renal insufficiency.
Am. J. M. Sc., 217: 1-12, 1949.

14. Kolff, W.J., and Berk, H.:
The artificial kidney, a dialyser with great area.
Acta med. Scandinav., 117: 123, 1944.

15. Kolff, W.J.:
New ways of treating uraemia.
J. & A. Churchill, Ltd., London, pp. 112, 1947.

16. Lucke, B.:
Lower nephron-nephrosis (the renal lesions of the crush syndrome, of burns, transfusions, and other conditions affecting the lower segments of the nephrons).
Mil. Surgeon, 99: 371-396, 1946.

17. Maluf, N.:
Urea clearance by perfusion of the entire intact small intestine in man.
Federation Proc., 7: 77, 1948.

18. Merrill, J.P., Thorn, G.W., Walter, C.W., Callahan, E.J., and Smith, L.H., Jr.:
The use of an artificial kidney: I Technique.
J. Clin. Investigation, 29: 412-424, 1950.

19. Merrill, J.P., Smith, S., Callahan, E.J., and Thorn, G.W.:
The use of an artificial kidney: II Clinical experience.
J. Clin. Investigation, 29: 425-438, 1950.

20. Muirhead, E.E., Halley, A.E., Haberman, S., and Hill, J.M.:
Acute renal insufficiency due to incompatible transfusion and other causes, with particular emphasis on management.
Blood, Spec. Issue, (Suppl. 2), 101-138, 1948.

21. Muirhead, E.E.:
Incompatible blood transfusion with emphasis on acute renal failure.
Surg., Gynec. & Obst., 92: 734-746, 1951.

22. Murray, G., Delorme, E., and Thomas, N.:
Development of an artificial kidney experimental and clinical experiences.
Arch. Surg., 55: 505-522, 1947.

23. Norviit, L.:
On the artificial kidney; XVI Experimental studies on some methodological problems in extra-corporeal dialysis of blood in vivo together with the therapeutical applications of this method to uremic rabbits.
Acta med. Scandinav., (Suppl. 245) 138: 1-82, 1950.

24. Odel, H. M., and Ferris, D.O.:
Continuous lavage of the small intestine as a means of treating renal insufficiency: Report of a case.
Proc. Staff Meet., Mayo Clin., 23: 201-207, 1948.

25. Olsen, N.S., and Bassett, J. W.:
Blood levels of urea nitrogen, phenol, guanidine and creatinine in uremia.
Am. J. Med., 10: 52-59, 1951.

26. Reinecke, R.M., Holland, C.R., and Stutzman, F.L.:
Homeostasis of potassium in the extracellular fluid of the dog during removal by vivodialysis,-
Am. J. Physiol., 156: 290-297, 1949.

27. Skeggs, L.T., Leonards, J.R., and Heisler, C.R.:
Artificial kidney II., construction and operation of an improved continuous dialyzer.
Proc. Soc. Exper. Biol. & Med., 72: 539, 1949.

28. Strauss, M.B.:
Acute renal insufficiency due to lower-nephron nephrosis.
New England J. Med., 239: 693-700, 1948.

29. Twiss, E.E., and Kolff, W.J.:
Treatment of uremia by perfusion of an isolated intestinal loop.
J.A.M.A., 146: 1019-1022, 1951.

III. MEDICAL SCHOOL NEWS

Coming Events

November 1 - The Annual Leo G. Rigler Lecture; "Recent Advances in Equipment for Fluoroscopy," Dr. W. Edward Chamberlain, Professor of Radiology, Temple University Medical School, Philadelphia; Museum of Natural History Auditorium; 8:15 p.m.

Oct. 29 - Nov. 3 - Continuation Course in Roentgenology of Chest Diseases for Radiologists

Nov. 8 - 10 - Continuation Course in Fractures and Traumatic Surgery for General Physicians

Nov. 26 - Dec. 1 - Continuation Course in Child Psychiatry for Pediatricians and General Physicians

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Roentgenology of Chest Diseases Course

Radiologists from all sections of the United States will attend a continuation course in Roentgenology of Chest Diseases to be presented October 29 to November 1 at the Center for Continuation Study. Dr. W. Edward Chamberlain, Professor and Head, Department of Radiology, Temple University Medical School, Philadelphia, will be one of the visiting faculty members for the course and will deliver the Annual Leo G. Rigler Lecture on the evening of Thursday, November 1. Other visiting faculty members for the course include: Dr. Benjamin Felson, University of Cincinnati Medical School, Dr. L. H. Garland, Stanford University Medical School, San Francisco, Dr. George R. Krause, Mount Sinai Hospital, Cleveland, Dr. Averill A. Liebow, Yale University Medical School, Dr. Laurence L. Robbins, Harvard University Medical School, and Dr. Erik Poppe, University Hospital, Oslo, Norway.

Faculty News

Dr. Ancel Keys, Director of the Laboratory of Physiological Hygiene, is spending the present academic year on sabbatical leave. Dr. Keys' headquarters will be in the Nutrition Laboratory of Dr. Hugh Sinclair at Oxford, England. Primary subject of study for Dr. Keys during this period will be cholesterol metabolism, a field of investigation in which he and his colleagues have already made many important contributions. During Dr. Keys' absence Dr. Henry L. Taylor is serving as Acting Director of the Laboratory.

Dr. Henry Michelson, Director of the Division of Dermatology, was elected a corresponding member of the British Dermatological Association at the Annual meeting of that organization.

Dr. David Glick spoke at a conference on Microspectrophotometry of Cells on September 28 and 29, 1951, in Boston, Massachusetts. The conference was sponsored by the National Institute of Health and was held at the Massachusetts Institute of Technology.

Dr. Allan Hemingway, former Professor of Physiology on our faculty, left the University of Minnesota during this past summer to begin his activities as Professor and Head of the Department of Physiology at the University of California Medical School in Los Angeles.

Dr. Charles Lowe, Instructor in the Department of Pediatrics, accepted an appointment as Associate Professor of Pediatrics at the University of Buffalo Medical School in Buffalo, New York. Dr. Lowe left this campus in August and began work at the University of Buffalo in September.

New Minnesota Medical Foundation Members

W. E. Rutherford, M.D., Nisswa
M. E. Lenander, M.D., St. Peter
Jules D. Levin, M.D., Milwaukee, Wisc.
M. C. O. Lindert, M.D., Milwaukee, Wisc.
Gerald T. Evans, M.D., Minneapolis
L. B. Kucera, M.D., Lonsdale
Helen M. Lerschen, Minneapolis
Lillian S. Edwards, Minneapolis
D. C. Edwards, Minneapolis
Cyrus O. Hansen, M.D., Minneapolis

II. UNIVERSITY OF MINNESOTA MEDICAL SCHOOL
WEEKLY CALENDAR OF EVENTS

Physicians Welcome

October 29 - November 3, 1951

Monday, October 29

Medical School and University Hospitals

9:00 - 9:50 Roentgenology-Medicine Conference; L. G. Rigler, C. J. Watson and Staff; Todd Amphitheater, U. H.

9:00 - 10:50 Obstetrics and Gynecology Conference; J. L. McKelvey and Staff; M-109, U. H..

10:00 - 12:00 Neurology Rounds; A. B. Baker and Staff; Station 50, U. H.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Eustis Amphitheater, U. H.

12:15 - 1:20 Obstetrics and Gynecology Journal Club; Staff Dining Room, U. H.

12:30 - Physiology Seminar; Some Relationships Between Steroidol Hormones and Tissue Glucuronidose and Esterose; Saul L. Cohen; 214 Millard Hall.

1:30 - 2:30 Pediatric-Neurological Rounds; R. Jensen, A. B. Baker and Staff; U. H.

4:00 - Pediatric Seminar; A Scoring System for the Bender-Gestalt Test; Wentworth Quast; Sixth Floor West, U. H.

4:30 - 5:30 Dermatological Seminar; M-346, U. H.

4:30 - Public Health Seminar; 15 Owre (Medical Sciences) Hall.

5:00 - 5:50 Clinical Medical Pathologic Conference; Todd Amphitheater, U. H.

5:00 - 6:00 Urology-Roentgenology Conference; C. D. Creevy, O. J. Baggenstoss, and Staff; Eustis Amphitheater.

Minneapolis General Hospital

7:30 a.m. Fracture Grand Rounds; Dr. Zierold, Station A.

11:00 - Pediatric Rounds; Dr. Top; 7th Floor.

12:30 p.m. Surgery Grand Rounds; Dr. Zierold; Station E.

1:00 - 2:00 X-ray Conference; Classroom, 4th Floor.

1:30 - Pediatric Rounds; Dr. Ulstrom; 4th Floor.

Veterans Administration Hospital

9:00 - G. I. Rounds; R. V. Ebert, J. A. Wilson, Norman Shrifter; Bldg. I.

Monday, October 29 (Cont.)

Veterans Administration Hospital (Cont.)

11:30 - X-ray Conference; Conference Room; Bldg. I.

1:00 - Metabolic Disease Rounds; N. E. Jacobson and G. V. Loomis. Bldg. I.

Tuesday, October 30

Medical School and University Hospitals

8:30 - Conference on Diet Endocrines and Cancer; M. B. Visscher; Physiology Library.

9:00 - 9:50 Roentgenology-Pediatric Conference; L. G. Rigler, I. McQuarrie and Staffs; Eustis Amphitheater, U. H.

9:00 - 12:00 Cardiovascular Rounds; Station 30, U. H.

12:30 - 1:20 Pathology Conference; Autopsies; J. R. Dawson and Staff; 102 I. A.

12:30 - Selected Topics, Permeability and Metabolism; Nathan Lifson; Physiology Library.

3:15 - 4:20 Gynecology Chart Conference; J. L. McKelvey and Staff; Station 54, U. H.

4:00 - 5:00 Pediatric Rounds on Wards; I. McQuarrie and Staff; U. H.

4:00 - 5:00 Physiology-Surgery Conference; Todd Amphitheater, U. H.

Ancker Hospital

1:00 - 2:30 X-ray Surgery Conference; Auditorium.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Dr. Gibbs; 5th Floor Annex.

10:00 - Psychiatric Grand Rounds; J. C. Michael and Staff; 3rd Floor Annex.

11:00 - Pediatric Rounds; Dr. Platou; 7th Floor.

Veterans Administration Hospital

8:30 - Surgery Staff Seminar; Conference Room, Bldg. I.

9:30 - Surgery-Pathology Conference; Conference Room, Bldg.I.

1:00 - Surgery Chest Conference; T. Kinsella and Wm. Tucker; Conference Room, Bldg. I.

1:30 - Liver Rounds; Samuel Nesbitt.

2:00 - 2:50 Dermatology and Syphilology Conference; H. E. Michelson and Staff; Bldg. III.

Tuesday, October 30 (Cont.)

Veterans Administration Hospital (Cont.)

3:30 - 4:20 Clinical Pathological Conference; Conference Room, Bldg. I.

Wednesday, October 31

Medical School and University Hospitals

8:00 - 8:50 Surgery Journal Club; O. H. Wangensteen and Staff; M-109, U. H.

8:00 - 9:00 Roentgenology-Surgical-Pathological Conference; Allen Judd and L. G. Rigler, Todd Amphitheater, U. H.

11:00 - 12:00 Pathology-Medicine-Surgery Conference; Surgery Case; O. H. Wangensteen, C. J. Watson and Staffs; Todd Amphitheater, U. H.

1:30 - Conference on Circulatory and Renal Systems Problems; M. B. Visscher; Physiology Library.

5:00 - 5:50 Urology-Pathological Conference: C. D. Creevy and Staff; Eustis Amphitheater, U. H.

5:00 - 7:00 Dermatology Clinical Seminar; Dining Room, U. H.

7:00 - 8:00 Dermatology Journal Club; Dining Room, U. H.

8:00 - 10:00 Dermatological-Pathology Conference; Review of Histopathology Section; Robert Goltz; Todd Amphitheater, U. H.

Ancker Hospital

8:30 - 9:30 Clinico-Pathological Conference; Auditorium.

3:30 - 4:30 Journal Club; Surgery Office.

Minneapolis General Hospital

9:30 - Pediatric Rounds; Dr. Platou; 7th Floor Annex.

11:00 - Pediatric Rounds; Dr. Top, 7th Floor.

12:15 - Pediatric Conference; Emotional Problems in Pediatrics; Lewis Flynn; Classroom, 4th Floor Annex.

1:30 - Pediatric Rounds; Dr. Huenekens and Dr. Ulstrom; 4th Floor Annex.

2:00 - 4:00 Infectious Disease Rounds; 8th Floor.

4:00 - 5:00 Infectious Disease Conference; Classroom, 8th Floor.

Veterans Administration Hospital

8:30 - 10:00 Orthopedic X-ray Conference; Conference Room, Bldg. I.

8:30 - 12:00 Neurology Rehabilitation and Case Conference; A. B. Baker.

Wednesday, October 31 (Cont.)

Veterans Administration Hospital (Cont.)

7:00 p.m. Lectures in Basic Science of Orthopedics; Conference Room, Bldg. I.

Thursday, November 1

Medical School and University Hospitals

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; M-109, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

12:30 - Physiological Chemistry Seminar; 214 Millard Hall.

11:00 - 12:00 Cancer Clinic; K. Stenstrom and A. Kremen; Todd Amphitheater, U. H.

1:30 - 4:00 Cardiology X-ray Conference; Heart Hospital Theater.

4:30 - 5:20 Ophthalmology Ward Rounds; Erling W. Hansen and Staff; E-534, U. H.

7:30 - 9:30 Pediatric Cardiology Conference and Journal Club; Review of Current Literature 1st hour and Review of Patients 2nd hour; 206 Temporary West Hospital.

* 8:15 p.m. Annual Leo G. Rigler Lecture; Recent Advances in Equipment for Fluoroscopy; Dr. W. Edward Chamberlain, Professor of Radiology, Temple University Medical School, Philadelphia; Museum of Natural History Auditorium.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Dr. Gibbs; 5th Floor.

8:30 - Neurology Rounds; Dr. Heilig, 4th Floor Annex.

9:00 - Neurology Grand Rounds; J. C. Michael and Staff; Station A.

11:00 - Pediatric Rounds; Dr. Platou; 7th Floor.

11:30 - Pathology Conference; Main Classroom.

1:00 - 2:00 Fracture - X-ray Conference; Dr. Zierold; Classroom, 4th Floor Annex.

2:00 - Psychiatry Rounds; Dr. Benton; 4th Floor Annex.

Veterans Administration Hospital

8:00 - Surgery Ward Rounds; Lyle Hay and Staff; Ward 11.

9:15 - Surgery Grand Rounds; Conference Room, Bldg. I.

11:00 - Surgery Roentgen Conference; Conference Room, Bldg. I.

Friday, November 2

Medical School and University Hospitals

8:30 - 10:00 Neurology Grand Rounds; A. B. Baker and Staff; Station 50, U. H.

9:00 - 9:50 Medicine Grand Rounds; C. J. Watson and Staff; Todd Amphitheater, U.H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:30 - 11:50 Otolaryngology Case Studies; L. R. Boiss and Staff; Out-Patient Department, U. H.

11:45 - 12:50 University of Minnesota Hospitals Staff Meeting; Studies and Therapy of Carcinoma of the Vulva; John L. McKelvey; Powell Hall Amphitheater.

1:00 - 2:50 Neurosurgery-Roentgenology Conference; W. T. Peyton, Harold O. Peterson and Staff; Todd Amphitheater, U. H.

2:00 - 3:00 Dermatology and Syphilology Conference; Presentation of Selected Cases of the Week; H. E. Michelson and Staff; W-312, U. H.

3:00 - 4:00 Neuropathological Conference; F. Tichy; Todd Amphitheater, U. H.

4:00 - 5:00 Dermatology Seminar; W-312, U. H.

4:00 - Neurophysiology Seminar; 113 Owre Hall, Medical Science Bldg.

5:00 - Urology Seminar and X-ray Conference; Eustis Amphitheater, U. H.

Ancker Hospital

1:00 - 3:00 Pathology-Surgery Conference; Auditorium.

11:00 - Pediatric-Surgery Conference; Dr. Wyatt, Dr. F. Adams; Classroom, Station I.

12:00 p.m. Surgery-Pathology Conference; Dr. Zierold, Dr. Coe; Classroom.

Minneapolis General Hospital

8:00 - Pediatric Allergy Rounds; Dr. Nelson; 4th Floor.

11:00 - Pediatric Rounds; Dr. Top; 7th Floor.

11:00 - Pediatric-Surgery Conference; Drs. Wyatt and F. Adams; Classroom, Sta. I.

12:00 - Surgery-Pathology Conference; Drs. Zierold and Coe; Classroom.

1:30 - Pediatric Rounds; Dr. Ulstrom, 4th Floor.

Veterans Administration Hospital

10:30 - 11:20 Medicine Grand Rounds; Conference Room, Bldg. I.

1:00 - Microscopic-Pathology Conference; E. T. Bell; Conference Room, Bldg.I

Friday, November 2 (Cont.)

Veterans Administration Hospital (Cont.)

1:30 - Chest Conference; Wm. Tucker and J. A. Meyers; Ward 62, Day Room.

3:00 - Renal Pathology; E. T. Bell; Conference Room, Bldg. I.

Saturday, November 3

Medical School and University Hospitals

7:45 - 8:50 Orthopedic X-ray Conference; Wallace H. Cole and Staff; M-109, U. H.

9:00 - 9:50 Medicine Case Presentation; C. J. Watson and Staff; E-221, U. H.

9:00 - 10:30 Pediatric Grand Rounds; I. McQuarrie and Staff; Eustic Amphitheater, U. H.

9:15 - 10:00 Surgery-Roentgenology Conference; J. Friedman, O. H. Wangensteen and Staff; Todd Amphitheater, U. H.

10:00 - 11:30 Surgery Conference; Todd Amphitheater, U. H.

10:00 - 11:50 Medicine Ward Rounds; C. J. Watson and Staff; E-221, U. H.

10:00 - 12:50 Obstetrics and Gynecology Grand Rounds; J. L. McKelvey and Staff; Station 44, U. H.

11:30 - 12:30 Anatomy Seminar; Hypophysis-adrenal System in the Fetus; Ralph L. Kitchell and L. J. Wells; 226 Institute of Anatomy.

Minneapolis General Hospital

8:00 - Pediatric Rounds; Dr. Gibbs; 5th Floor.

11:00 - 12:00 Pediatric Clinic; Dr. Thomas and Dr. May; Classroom, 4th Floor Annex.

Veterans Administration Hospital

8:00 - Proctology Rounds; W. C. Bernstein and Staff; Bldg. III.

8:30 - Hematology Rounds; P. Hagen and E. F. Englund.

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* Indicates special meeting. All other meetings occur regularly each week at the same time on the same day. Meeting place may vary from week to week for some conferences.