MEDICAL GRAND ROUNDS
PARKLAND MEMORIAL HOSPITAL
October 26, 1961

USE OF DIALYSIS IN DRUG INTOXICATION

Case #1 -- [redacted]

31-year-old woman ingested an undetermined amount of Seconal® and meprobamate about six hour prior to admission.

BP - 90/70; P - 120; R - 25, shallow. Patient was completely unresponsive. Pupils equal, not dilated, reacted to light. Corneal reflex absent. No gag reflex. Chest clear on admission. Hyperactive DTR's. No pathological reflexes. There was a sustained ankle clonus bilaterally. Gastric aspirate had 15.9 mg% barbiturate (results from another hospital).

In spite of Emivan® injections and continuous Emivan infusion, patient failed to maintain BP. Developed a massive right-sided pneumonia (aspiration ?).

At 24 hours, patient was dialyzed for six hours. Barbiturate blood level at that time was 5.9 mg%. At the end of six hours of dialysis, patient responded well to pain stimuli, but was not awake. BP was 120/80. Barbiturate blood level was 2.1 mg% at the end of dialysis.

Patient was fully awake at 48 hours, and had an uneventful course thereafter. Gas chromatography confirmed the drug as Seconal. No meprobamate was found.

Case #2 -- [redacted]

This 38 year-old woman was a [redacted] in an osteopathic hospital and apparently had access to many drugs. She was seen awake one hour prior to admission, but 30 minutes later was found unconscious and brought to this hospital. No objective history of drug intake could be obtained from her friends.

BP - 100/60; P - 96; T - 96; R - 16. She was unresponsive. Pupils reacted to light. Corneal reflex absent. Deep tendon reflexes and superficial reflexes were absent. No pathological reflexes.

In spite of Emivan therapy, which did stimulate respiration, BP decreased to 90/60. Patient was dialyzed for six hours with return of deep tendon reflexes at that time. BP was stable after dialysis at 130/80. She awoke 32 hours after admission.

Blood barbiturate levels: Before dialysis -- 5.18 mg%
After dialysis -- 3.05 mg%

Case #3 -- [redacted]

44 year-old woman arrived at hospital in comatose state. No history of drug intake could be obtained from husband or friends.

BP - 140/84; P - 112; T - 101 R; R - 36. Pupils 3 - 4 mm; Reacted to light. Corneal at first absent, but returned shortly after admission. DTR's present - no pathological reflexes.

Patient had uneventful course. Was entirely awake 20 hours after admission. Blood barbiturate level on admission was 5.98 mg%. (This value was not available until 13 hours after admission.) Blood alcohol - negative.

Case #4 -- [redacted]

The patient was a 15 year-old girl. Nine months prior to this admission she had been in this hospital with a diagnosis of encephalitis with aseptic meningitis. During that admission she had three grand mal convulsions, and at discharge phenobarbital grs. 1 1/2 was prescribed. About 7 hours prior to this admission she took about 150 1/2 gr. phenobarbital tablets.

BP - 80/0 - 90/60; P - 64; R - 15. Patiend did not respond to pain stimuli. Pupils small, but reacted to light. Corneal reflex absent. Deep tendon reflexes markedly depressed. No pathological reflexes were present.

The injection of 500 mg Emivan IV resulted in an increased rate and depth of respirations. The patient then reacted somewhat to painful stimuli. With a constant infusion of Emivan, the blood pressure was maintained between 90/60 - 110/70 for about 15 hours. Repeated stat injections of Emivan were given to stimulate respiration. During these injections, she frequently moved her extremities in an aimless manner.

The patient was dialyzed about 16 hours after admission because 1) she had taken a potentially lethal dose of drug; 2) the initial blood level supported the lethal dose concept (14.3 mg%); and 3) the blood pressure tended to fall more toward the 90/60 range.

Blood level before dialysis was 17.2 mg%. Within the first hour of dialysis, the patient was awake, but the dialysis was continued for an additional 4 1/2 hours. Blood level after dialysis was 7.8 mg%. The patient was oliguric for 24 hours (volume 550 cc) after dialysis. Her further recovery was uneventful, however.

The blood barbiturate level 57 hours after admission was still 6.6 mg%.

Case #5 -- [redacted]

When this 20 year-old girl was admitted in a comatose state, the history was obtained from her family that she had ingested at least 4.0 gms of glutethimide (Doriden®) a few hours prior to admission.

BP - 120/80; P -- 80; R - 22. Pupils equal - not dilated. Reacted to light. Could not be aroused by painful stimulus. Deep tendon reflexes present. No pathological reflexes.

Instead of awakening as expected, the patient manifested a deepening coma. Pupils became dilated. Deep tendon reflexes became hypoactive, and the BP at 18 hours post admission was 90/60.

At this point, hemodialysis was begun. Almost immediately the BP returned to 120/80. At 5 1/2 hours, it was noted that her previously clear yellow urine was now dark amber. It was ascertained that there was visible hemolysis in the plasma. No cause for this hemolysis was found. The dialysis was discontinued at this point. Deep tendon reflexes had become normally active. She responded to painful stimuli.

She was fully awake 12 hours after dialysis and remained so. Blood glutethimide was positive on admission.

Case #6 -- [redacted]

The patient, a 39-year-old woman, was known to have been asleep (comatose ?) for at least 28 hours prior to admission. Her husband found a bottle containing

methaminodiazepoxide (Librium®) tablets by her bedside, and thought that many fewer were in the bottle than he had previously seen. Also beside her was an empty bottle that had contained "sleeping pills", but the husband knew she had taken the last of these several days previously.

BP - 98/70; T - 102° R; P - 132; R - 60. Patient responded slightly to painful stimuli. Pupils were dilated, but reacted to light. Corneal reflex absent. A few rhonchi were heard in the right chest. Deep tendon reflexes were absent. No pathological reflexes were present. Superficial reflexes were absent.

Shortly after the start of an infusion of saline, the blood pressure rose to 130/85 and remained there. The pulse slowed to 100. Respirations also slowed, but remained 26 to 32 per minute.

About six hours after admission, a tracheostomy was performed because she became cyanotic. Thirteen hours after admission, the patient was responsive and went on to have an uneventful recovery.

Admission blood tests: Librium - negative; meprobamate - negative; alcohol - negative; Doriden - Positive - probably less than 1 mg%.

Case #7 --[redacted]

This 23 year-old woman was admitted in a comatose state after ingesting unknown amounts of codiene, nembutal®, and librium some hours before. She had been receiving insulin shock therapy in another hospital shortly before admission here.

BP - 110/70; P - 80; R - 16, shallow; T - 99°. Responded to painful stimuli. There was a bruised area over the right side of the skull. Pupils reacted to light. Corneal reflexes were absent. Deep tendon reflexes were present and equal. No pathological reflexes. Abdominal reflexes were absent.

8 hours post admission: Pupils were dilated - reacted sluggishly to light. Corneal reflexes still absent. Deep tendon reflexes in lower extremities hyperactive, perhaps more so on the right. Emivan used to increase respiratory rate. Tracheotomy performed.

12 hours post admission: Developed bilateral sustained ankle clonus. Still no pathological reflexes. Right carotid arteriogram then disclosed no pathology.

Patient slept for seven days. Emivan was used continuously for first 3 1/2 days.

Blood drug survey: 1) Darvon - negative; 2) Barbiturate - negative; 3) Doriden - negative; 4) Meprobamate - negative; 5) Librium - 0.3µgm%.

Case #8 -- [redacted]

Admitted in coma, this 17 year-old [redacted] was known to have taken just under 25 gms of Ethchlorvynol (Placidyl®) a few hours before admission. She had a long history of bronchial asthma.

BP 120/70; P - 104; R - 22; T - 100°. There was an unusual fruit-like odor to the breath which was presumed to be due to placidyl. Responded sluggishly to pain.

Pupils constricted but did react to light. Corneal reflexes present; deep tendon reflexes present; no pathological reflexes.

Shortly after admission, respirations slowed to 15 per minute, but could be increased with Emivan. Blood pressure, however, steadily fell. About 24 hours after admission it ranged 100/70 - 90/60.

At 24 hours post admission hemodialysis was begun. The Placidyl odor was imparted to the dialysis bath. During dialysis the blood pressure rose to 130/80 and remained there. She was dialyzed for 13 hours.

Blood Placidyl level prior to dialysis was 5 μgm% (not available at the time of dialysis.) Total drug recovered in dialysis bath was 50 μgm%.

The patient slept for just over 7 days.

Properties a Poison Must Have in Order for Hemodialysis to be an Effective Form of Therapy:*

1. The poison must diffuse through the cellophane membrane at a reasonable rate. Significant amounts of poison must be cleared from the blood at kidney blood flow rates as low as 100 cc/min.

2. The poison cannot be significantly bound to any plasma fraction, tissue cell, or water compartment. This refers to irreversible or near irreversible binding.

3. The clinical illness resulting from the poison must, for the most part, have its severity correlate directly with the blood concentration of the poison.

4. The amount of poison that can be removed in a reasonable length of time by hemodialysis must be a significant addition to the patient's metabolism and removal of the poison by the bowel and kidney.

Dialyzable Drugs and Poisons:

1. Barbiturates
   Barbital -- Excellent dialysance; rare intoxication nowadays.
   Phenobarbital -- Good dialysance.
   Secobarbital (Seconal) -- Fair dialysance
   Amobarbital (Amytal) -- Fair dialysance
   Pentobarbital (Nembutal®) -- Probably poor dialysance, but worth attempting.

2. Glutethimide (Doriden) -- Dialysance about equal to pentobarbital, but "physiologic re-circulation" impairs dialysis efficiency.

3. Diphenylhydantoin (Dilantin®) -- Dialysance good, but significant tissue reservoir has been suspected.

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* As modified from G. E. Schreiner.

4. Acetylsalicylate (Aspirin)
5. Methyl salicylate (Oil of Wintergreen)
   Dialysance excellent despite plasma protein binding.

6. Ethyl alcohol
7. Methyl alcohol
   Dialysance of these alcohols is about equal and is excellent. Little clinical experience at present.

8. Bromide -- Dialysance excellent.

The following list comprises drugs and toxins which can be dialyzed. Their clinical importance is based, for the most part, on single observations.

9. Meprobamate (Milltown® - Equinal®)
10. Ethymyl - cyclohexyl carbamate (Valmid®)
11. Thiocyanates
12. Borates
13. Calcium
14. Ethylene glycol
15. Penicillin
16. Chloromycetin®
17. Streptomycin
18. Sulfadiazine

Indications for Dialysis in Poisoning:

1. Coma with a history of ingestion of a lethal amount of the drug.
   Lethal Amounts:
   Phenobarbital -- 5.0 gms.
   Short acting barbiturates -- 3.0 gms.
   Glutethimide -- 10.0 gms.
   Aspirin -- over 20.0 gms.
   Lethal doses for other drugs and toxins are less certain.

2. Coma with a blood concentration for the poison above a lethal value.
   Lethal Blood Concentrations:
   Phenobarbital -- 8 mg.%
   Short-acting barbiturates -- 4 mg.%
   Glutethimide -- 3 mg.%
   Aspirin -- 50 mg.%

3. The presence or subsequent appearance of any of the following signs:
   a) Areflexia and apnea.
   b) Progressive fall in blood pressure not related to respiration.
   c) Variations in body temperature.
   d) Severe metabolic acidosis.
4. The existence of significant hepatic, renal, or pulmonary disease.
5. The development of pneumonia.

REFERENCES

1. Abe, J.J., Rowntree, L.G., and Turner, B.B. The removal of diffusible substances from the circulating blood by means of dialysis. Trans. Assoc. Am. Physicians 28:51, 1913.
   Showed that even with their primitive artificial kidney, significant amounts of aspirin could be removed from a dog previously given aspirin.
2. Schreiner, G.E. The role of hemodialysis (artificial kidney) in acute poisoning. Arch. Int. Med. 102:896, 1958.
   This is still the most complete review of the dialysis of poisons.
3. Wolf, A.V., Remp, D.G., Kiley, J.E., and Currie, G.D. Artificial kidney function: kinetics of hemodialysis. J. Clin. Invest. 30:1062, 1951.
4. Maher, J.F., Schreiner, G.E., and Marc-Aurele, J. Methodologic problems associated with the in vitro measurement of dialysance. Trans. Am. Soc. Artificial Internal Organs 5:120, 1959.
   These two papers deal with the methods of numerically stating the efficiency of dialysis.

Salicylates

5. Doolan, P.D., Walsh, W.P., Kyle, L.H., and Wishinsky, H. Acetylsalicylic acid intoxication: a proposed method of treatment. J.A.M.A. 146:105, 1951.
   This is the first report of a human treated by means of an artificial kidney for drug intoxication. Although the patient expired, the data presented show that the artificial kidney could remove far more aspirin than the normal kidney could excrete.
6. Leonards, J. The use of the artificial kidney for purposes other than treatment of uremia. Trans. Am. Soc. Artificial Internal Organs 1:46, 1955.
   Treated two patients with salicylate intoxication with excellent results. One patient took methyl salicylate; blood salicylate level 130 mg%. About 9.5 gms removed by dialysis. Blood level after dialysis 35mg%.
7. Schreiner, G.E., Berman, L.B., Griffen, J. and Feys, J. Specific therapy for salicylism. New Eng. J. of Med. 253:213, 1955.
   Two case reports. One patient ingested 210 gms of aspirin. Had blood level of 91 mg%. 9.4 gms were removed by dialysis.
8. Thomsen, A.C. and Dalgard, O.Z. Hemodialysis in acute acetylsalicylic acid poisoning. Am. J. Med. 25:484, 1958.
   Patient took 150 gms of aspirin and was successfully treated by dialysis.
9. Burns, R.O., et al. Salicylate intoxication treated with artificial kidney. West Virg. Med. J. 54:198, 1958.
   An adult with severe acidosis and a blood salicylate level of 56 mg% successfully treated.
10. Segar, W.E., Gibson, R.K., Rhamy, R. Peritoneal dialysis in infants and small children. Pediatrics 27:603, 1961.
   Treated a three-year-old with peritoneal lavage (19 liters in 48 hours). Blood salicylate lowered from 47.2 mg% to 7.3 mg%. 838.5 mg of salicylate removed in dialysate. Authors state this is 1/4 the rate of an artificial kidney. This is a mathematical misconception; it is closer to 1/50 the rate.

11. Etteldorf, J.N., Montalivo, J.M., Kaplan, S., Sheffield, J.A. Intermittent peritoneal dialysis in the treatment of experimental salicylate intoxication. J. Pediatrics. 56:1, 1960.
12. Etteldorf, J.N., Dobbins, W.T., Summitt, R.L., Rainwater, W.T., and Fischer, R. L. Intermittent peritoneal dialysis using 5% albumin in the treatment of salicylate intoxication in children. J. Pediatrics 58:226, 1961.
Etteldorf maintains that by the addition of albumin to the dialysis solution, the removal of aspirin by the dialysate is increased. From the data, this appears true.
13. Elliott, G.B. and Crichton, J.V. Peritoneal dialysis in salicylate intoxication. Lancet 2:840, 1960.
These authors recognize that peritoneal dialysis removes very little aspirin, but believe the rapid correction of the acidosis is an important adjunct to therapy.

Barbiturates:

14. Reed, C.E., Driggs, M.D., and Foote, C.C. Acute barbiturate intoxication: a study of 300 cases based on a physiologic system of classification of severity of intoxication. Ann. Int. Med. 37:290, 1952.
Classification:
0 - Sleeping patient who can be aroused.
I - Comatose patient that will withdraw from painful stimuli.
II - Patient showing no reaction to painful stimuli. Reflexes intact. No respiratory nor circulatory depression.
III - Areflexia -- no respiratory nor circulatory depression.
IV - Areflexia with respiratory depression or apnea and/or shock with an adequate airway.
15. Hahn, F. Analeptics. Pharmacol. Rev. 12:447, 1960.
Although the author recommends the use of some analeptics in barbiturate intoxication, he correctly emphasizes their dangers. One would clearly doubt that either pentylenetetrazol (Cardiozol® - Metrazol®) or Picrotoxin should ever be used.
16. Sonne, L.M. Megimide in the treatment of barbituric acid poisoning. Lancet 2:961, 1956.
Bemegride (Megimide®) was at first thought to be a specific barbiturate antidote, but now is thought to be in the same class as picrotoxin and Metrazol. Will cause convulsions. Does not appear to shorten the length of coma.
See also Reference 30.
17. Gardner, E.K. A new analeptic: preliminary communication (Corresp.). Brit. J. Anesthesiology 30:155, 1958.
18. Lochet, S. Vanillic acid diethylamide (Corresp.). Brit. Med. J. #5215, 1805, 1960.
19. Bernstine, M.L. and Moshal, J.P. Effect of vanillic diethylamid on arousal and awakening time following thiopental anesthesia (Abstract). Anesthesiology 21:90, 1960.
20. Lemere, F. and Baum, G.L. Barbiturate poisoning: management with a new respiratory stimulant. J.A.M.A. 174:896, 1960.
Emivan® clearly has a good therapeutic index and does stimulate respiration. It is useful in treatment of hypnotic drug intoxication.

21. Alwall, N., Lindgren, P., and Lunderquist, P. On the artificial kidney: XX treatment of severe phenobarbital poisoning in rabbits by means of forced polyuria, exchange ultrafiltration, and dialysis and a preliminary report on dialytic treatment of barbiturate poisoning in patients. Acta med. Scand. 143:299, 1952.
First report of clinical use of dialysis in treatment of barbiturate intoxication.
22. Kyle, L.H., Jeghers, H., Walsh, W.P., Doolan, P.D., Wishinsky, H. and Pallota, A. The application of hemodialysis to the treatment of barbiturate poisoning. J. Clin. Invest. 32:364, 1953.
First report with adequate data to show the efficiency of removal of barbiturates from humans.
23. Sunshine, I., and Leonards, J.R. Use of the artificial kidney for removal of barbiturates in dogs. Proc. Soc. Exper. Biol. & Med. 86:638, 1954.
From dogs: 40 to 70% injected phenobarbital removed.
35% of injected amobarbital removed.
15 to 25% of injected pentobarbital removed.
These data have been borne out by clinical experience.

24. Brown, I.A., Ansell, J.S. and Schiele, B.C. The use of hemodialysis in the treatment of barbiturate intoxication. Minnesota Med. 37:650, 1954.
25. Berman, L.B., Jeghers, H.J., Schreiner, G.E., and Pallotta, A.J. Hemodialysis, an effective therapy for acute barbiturate poisoning.
26. Pender, J.C., Beebe, R.T., Garrett, J.J. and Kiley, J.E. Emergency treatment of barbiturate intoxication with hemodialysis. Ann. Int. Med. 46:997, 1957.
27. Honey, G.E. and Jackson, R.C., Artificial respiration and an artificial kidney for severe barbiturate poisoning. Brit. Med. J. #5160, 1134, 1959.
These four papers are representative of the many reports since 1953 on the use of dialysis in barbiturate poisoning. In general, an artificial kidney can remove the drug from the patient from six to thirty times faster than the patient's elimination of the drug by bowel and kidney. The rate of removal depends in large part on the specific barbiturate present.
28. Maxwell, M.H., Rochney, R.E., Kleeman, C.R., and Twiss, M.R. Peritoneal dialysis. 1. Technique and applications. J.A.M.A. 170:917, 1959.
The first report of the use of peritoneal dialysis in barbiturate intoxication; six grams of Tuinal® (secobarbital and amobarbital) were ingested. Dialyzed for 30 hours; 56 liters exchanged; 1620 mg. of drug removed. This compares well with drug removal by artificial kidney in six to twelve hours.

Glutethimide (Doriden)

29. McBay, A.J. and Katsas, G.G. Glutethimide poisoning; report of four fatal cases. New Eng. J. Med. 257:97, 1957.
The authors conclude that the fatal dose is in the range from 10 to 20 gms.
30. Schreiner, G.E., Berman, L.B., Kovach, R. and Bloomer, H.A. Acute glutethimide (Doriden) poisoning; the use of bemegride (megimide) and hemodialysis. Arch. Int. Med. 101:899, 1958.

31. Maher, J.F. and Schreiner, G.E. Acute glutethimide poisoning: II. The use of hemodialysis. Trans. Am. Soc. Artificial Internal Organs 7:100, 1961.

The authors of these two papers stress these points:

a) Doriden does not commonly depress respiration, but sudden Apnea is frequently seen.
b) Widely dilated, usually fixed pupils are commonly seen.
c) Sudden rapid fall in BP is also common.
d) Hemodialysis may be life-saving.

32. Kier, L.C., Whitehead, R.W., and White, W.C. Blood and urine levels in glutethimide intoxication. J.A.M.A. 166:1861, 1958.
33. Chandler, B.F., et al. Artificial hemodialysis in management of glutethimide intoxication. J.A.M.A. 170:914, 1959.
34. Nakamoto, S. and Kolff, W.J. The artificial kidney for acute glutethimide and barbiturate poisoning: report of four representative cases. Cleveland Clin. Quart. 27:58, 1960.

The authors of these three papers consider hemodialysis an important adjunct to therapy in severe Doriden intoxication. Dialysis appears to be 100 to 400 times more effective than renal intoxication.

Bromism

35. Merrill, J.P. and Weller, J.M. Treatment of bromism with the artificial kidney. Ann. Int. Med. 37:186, 1952.

Bromism is the only important chronic intoxication treated by dialysis at present. This report is the second clinical attempt to treat intoxications by means of hemodialysis (See Ref. 5).

36. Costa, J.S., Nenno, R.P., Schreiner, G.E. and Berman, L. Some observations of a case of bromide intoxication undergoing hemodialysis. Am. J. Psych. 113:1030, 1957.

Because bromide is so effectively removed by dialysis, it should be considered as therapy in severely ill patients whose serum Br concentration is 30.0 mEq/L (300 mg%) or over.

Dilantin (See Ref. 2 and 25)

It is somewhat difficult to evaluate dialysis therapy on the basis of the two presented cases. Apparently Dilantin can be dialyzed well, and since the severe intoxication is dangerous, dialysis should be considered.

Other Drugs and Toxins

37. Marc-Aurele, J. and Schreiner, G.E. The dialysance of ethanol and methanol: a proposed method for the treatment of massive intoxication by ethyl or methyl alcohol. J. Clin. Invest. 39:802, 1960.

A study in dogs given ethyl and methyl alcohol. Dialysance of both these alcohols is high. Certainly, if the methanol intoxication is recent, dialysis would be expected to benefit the patient. In addition to removing alcohol, correcting the acidosis, formate may be removed.

38. Stinebaugh, B.J. The use of peritoneal dialysis in acute methyl alcohol poisoning. Arch. Int. Med. 105:613, 1960
Report of three patients with methanol intoxication. Two survived. Blood alcohol concentration was rapidly lowered by peritoneal dialysis.
39. Davis, R.P., Blythe, W.B., Newton, M., Welt, L.G. The treatment of intoxicatic with ethymyl-cyclohexyl carbamate (Valmid) by extracorporeal hemodialysis. Yale J. Biol. & Med. 32:192, 1959.
This hypnotic can apparently be adequately dialyzed.
40. Schreiner, G.E., et al. Ethylene glycol: two indications for hemodialysis. Trans. Am. Soc. Artificial Internal Organs 5:81, 1959.
This toxin is over 90% fatal. In this case report, the presumption is that both ethylene glycol and its metabolic products were removed during the acute stage of illness by hemodialysis. See Levy, A.I. Renal failure secondary to ethylene glycol intoxication. J.A.M.A. 173:1210, 1960.
41. Segar, W.E. Peritoneal dialysis in the treatment of boric acid poisoning. New Eng. J. Med. 282:798, 1960.
Report of three small children. Author concludes that dialysis is a more efficient form of treatment than exchange transfusion. See Ref. 10.
42. Merrill, J.P. The artificial kidney. Arch. Int. Med. 106:143, 1960.
The author mentions the use of the artificial kidney to correct hypercalcemia in patients with calcium intoxication occurring during steroid therapy of metastatic carcinoma.
43. Danzig, L.E. and Kringel, A.J. Use of the artificial kidney in treatment of thiocyanate psychosis. J.A.M.A. 158:560, 1955.
44. Danzig, L.E. Dynamics of thiocyanate dialysis: the artificial kidney in the therapy of thiocyanate intoxication. New Eng. J. Med. 252:49, 1955.
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45. Edwards, K.D.G. and Whyte, H.M. Streptomycin poisoning in renal failure; an indication for treatment with an artificial kidney. Brit. Med. J. 1:752, 1959.
Hemodialysis was said to alleviate ototoxicity.
46. Goldsmith, H.J., Nakamotos, S., and Kolff, W.J. Expanding the indications for treatment with the artificial kidney: burns, cerebral edema, sulphonamide intoxication, and congestive heart failure. Lancet 2:111, 1960.
Hemodialysis altered the serum level of sulphadiazine in an anuric, overdosed patient from 26 mg% to 11 mg% presumably with improvement to the patient.