

# South African Medical Journal

## Suid-Afrikaanse Tydskrif vir Geneeskunde

P.O. Box 643, Cape Town

Posbus 643, Kaapstad

Cape Town, 12 May 1956  
Weekly 2s. 6d.

Vol. 30 No. 19

Kaapstad, 12 Mei 1956  
Weekliks 2s. 6d.

### VAN DIE REDAKSIE

#### ANURIE EN UREMIE

Kolff<sup>1</sup> wat 'n spesiale studie van die onderwerp, en veral van die moontlikhede van dialise gemaak het, bespreek die moderne benadering tot hierdie ernstige siekte in 'n onlangse verhandeling oor die oorsake en behandeling van akute nierversaking. Spesiale geriewe is dikwels nodig om die beste behandeling te voorsien, en pasiënte moet binne die eerste paar dae, voordat hulle ernstig siek word, na geskikte hospitale verplaas word.

In die behandeling van gevestigde anurie moet die hoeveelheid liggaamsvloeistof konstant gehou word deur vergoeding vir die onwaarneembare verlies van water, en vir die vloeistof wat verlore gaan weens braking, diarree of dreinerings. Oor-hidrerings van die pasiënt moet met die grootste versigtigheid vermy word want dit mag longedeem of stuiptrekkings veroorsaak. Behalwe die water wat ingeneem word, moet die oksidasie-water ook in gedagte gehou word; die meeste siektes wat anurie veroorsaak, veroorsaak ook 'n toename in die stofwisseling. Parentale toediening is nie nodig solank die pasiënt vloeistowwe kan drink nie, tensy 'n hipertoniëse oplossing van dektrose of fruktose gegee moet word. As oor-hidrerings wel voorkom, is bloedlating die beste noodhulp om die bloedvolume te verminder; 300-500 ml. bloed kan afgetap word. Om edeem by urinelose pasiënte te verminder, word daar gebruik gemaak van hipertoniëse soutpurgasies, verwydering van die buikwater, die kunsnier, of uitspoel van die buikholte.

In die akute fase van anurie moet natrium met die grootste versigtigheid toegedien word want dit kan nie uitgeskei word nie. Gedurende die urine-afdrywingsfase, wat gewoonlik uiteindelik voorkom by akute nierversaking, moet natriumchloried gegee word om vir die verlies daarvan in die urine te vergoed—3 g. natriumchloried teen elke liter urine wat uitgeskei word.<sup>1</sup> Dikwels word die kaliumstand van die bloed by akute urineloseheid te hoog, veral by verbrysing en by ernstige besmetting en vergiftiging, en hierdie hiperkalemie kan ten beste met 'n vlam-ligmeter, en minder bevredigend met elektrokardiogramme, gemeet word. Daar word met die toediening van katioon wisseling-

### EDITORIAL

#### ANURIA AND URAEMIA

In a recent review of the causes and treatment of acute renal failure Kolff,<sup>1</sup> who has made a special study of the subject, particularly the possibilities of dialysis, discusses the modern approach to this serious condition. To provide the best treatment, specialized facilities are often required, and patients should be transported to appropriate centres during the first few days, before their condition has become grave.

In the treatment of established anuria, body fluid must be maintained by making good the insensible loss of water and the fluid lost by vomiting, diarrhoea, or drainage. Care is necessary to avoid over-hydrating the patient, which may produce pulmonary oedema or convulsions. Beside fluid intake, the water of oxidation must be borne in mind; most conditions that give rise to anuria also produce an increase in metabolism. As long as the patient can take fluids by the mouth, parenteral administration is not indicated, unless hypertonic dextrose or fructose is to be given. Should over-hydration occur, venesection is the best immediate means of reducing the blood volume; 300-500 ml. of blood may be withdrawn. To reduce oedema in anuric patients use has been made of hypertonic saline purgatives, the removal of ascitic fluid, the artificial kidney, or peritoneal lavage.

In the acute phase of anuria the administration of sodium calls for great care, because it cannot be excreted. During the diuretic phase, which usually occurs eventually in acute renal failure, sodium chloride needs to be given to replace the urinary loss—3 g. of sodium chloride for each litre of urine excreted.<sup>1</sup> Hypertotassaemia, which is best determined with the flame photometer, and less satisfactorily by means of electrocardiograms, often occurs during acute anuria, especially in crush injury and in severe infections and intoxications. Attempts to reduce the blood potassium are made by

harse (in die natriumsiklus), en met glukose, met of sonder insulien, gepoog om die bloedkaliumstand te verminder. Hipertoniese natriumbikarbonaat of kalsium glukonaat mag toegedien word, maar dialise met die kunstnier is die beproefde metode om hiperkalemie binne 'n paar uur te verbeter.<sup>1</sup> 'n Tekort aan bloedkalium, wat dieselfde kliniese simptome van spierverlamming en hartaantasting mag veroorsaak, kan in die diuretiese fase voorkom as baie urine uitgeskei word. In hierdie geval moet kalium toegedien word, maar dit moet nie in die vorm van binnearese kaliumchloried gegee word nie—hierdie behandeling is gevaarlik en het al die dood veroorsaak. Te min kalsium in die bloed kom soms by akute nierversaking voor maar veroorsaak selde enige simptome; kalsium moet toegedien word as rukkramp wel voorkom, en ook wanneer suurvergiftiging behandel word.

'n Poging moet aangewend word om 'n dieet wat so ryk as moontlik aan kalorieë is te forseer. Dit is nie altyd 'n maklike taak nie, want die pasiënte moet nie te veel vloeistof kry nie, en baie van hulle kan ook nie veel eet of drink nie. Kolff<sup>1</sup> gebruik die kalorie-ryke, proteïen-arm dieet wat Borst (Amsterdam) aanbeveel het, en gee 'n paar resepte vir die voorberei van die voeding aan, met besonderhede aangaande toediening.

Die simptomatiese behandeling van hartversaking, hoë bloeddruk, longedeem, abnormale asemhaling, uremiese verskynsels en ander aspekte van die behandeling van akute nierversaking word breedvoerig bespreek. Hier kom die moderne middels soos chlorpromazine (braak-teenmiddel), middels wat die senuweeknoppes versper (bloeddrukverlagend), en nog baie ander ter sprake. Waar 'n opgeleide personeel en die nodige toestelle beskikbaar is (klein hospitale moet nie hierdie duur toestelle aankoop nie), kan die produkte van terughouding, en die sure van die stofwisseling, sowel as edeem, deur middel van dialise en deursyfering verwyder word. Dit is duidelik dat die metodes betrokke by die gebruik van die kunstnier, en die uitspoel van die buikholte en derm, in baie min inrigtings toegepas kan word.

giving cation exchange resins (in the sodium cycle), and glucose with or without insulin. For temporary effect hypertonic sodium bicarbonate, or calcium gluconate, may be administered, but dialysis with the artificial kidney is the certain way to reduce hyperpotassaemia in a few hours.<sup>1</sup> Hypopotassaemia, which may cause the same clinical symptoms of muscular paralysis and cardiac arrest, may occur in the diuretic phase, when the urine volume is large, and in this condition potassium should be administered, but not in the form of potassium chloride given intravenously, which is dangerous, deaths having resulted from this treatment. Hypocalcaemia occurs frequently in acute renal failure, but seldom causes symptoms; however, if tetany occurs, and whenever acidosis is corrected, calcium should be administered.

An attempt should be made to force a diet as high in calories as possible. This is not always easy, since overloading with fluid is to be avoided and since many patients cannot take much by mouth. Kolff<sup>1</sup> uses the high-calorie low-protein regimen introduced by Borst (Amsterdam) and gives some recipes for the preparation of the recommended diet, and details for its administration.

The symptomatic treatment of cardiac failure, hypertension, pulmonary oedema, abnormal respiration, uraemic manifestations, and other aspects of the management of acute renal failure, are considered in detail; modern drugs have their place here, such as chlorpromazine (anti-emetic), ganglion-blocking agents (hypotensive), and many others. For those who have a trained team and the apparatus (small hospitals should not invest in the expensive equipment) the removal or retention products, acid metabolites and oedema can be achieved by dialysis and filtration. The techniques involved in the use of the artificial kidney, peritoneal lavage, and intestinal lavage can obviously be carried out in very few institutions.

1. Kolff, W. J. (1955): *Med. Clin. N. Amer.*, **39**, 1041.

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