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VAN DIE REDAKSIE : EDITORIAL

DIE BEHANDELING VAN AKUTE NIERVERSAKING

Gedurende die Tweede Wêreldoorlog het beserings as gevolg van samepersing gelei tot die besef dat akute nierversaking kan voorkom op die basis van 'n omkeerbare letsel. Kort na die oorlog het die volgende konserwatiewe behandeling van dié toestand ontstaan: streng beheer van die inneem van voedsel met 'n hoë kaloriemengsel van glukose en vet wat vry is van elektroliete en proteïene.[1] Dit word nou aangeneem dat glukose in 40-50% sterkte genoeg is om die kalorieë te verskaf en dat voedings wat baie vet bevat nie hoef gegee te word nie. Hierdie vloeistof kan per mond gegee word of binneaars in die superior of inferior vena cava. Kleiner are toon die neiging tot trombose by die gebruik van hipertoniese oplossings. Die inneem van vloeistof behoort beperk te word tot omtrent 400 ml. per 24 uur en die daaglikse gewig is 'n goeie aanduiding van die vloeistof-behoeftes. Die ideaal is dat die pasiënt ongeveer 0·2 kg. in gewig elke 24 uur moet verloor. Dit mag nodig wees om die verlies van vloeistof deur braking te vervang, wat dit ook mag nodig maak om soutoplossings in halfsterkte per mond te gee—egter nie meer as wat nodig is om die verlies deur braking te vervang nie. Hierdie lastige braking kan dikwels deur chlorpromasien verhoed word as die toestand van die lewer bevredigend is.

Kalsiumglukonaat behoort gegee te word om te verhoed dat die serum-kalsium daal. Natrium-wisselharse en die toediening van glukose en insulien kan help om die styging van kalium in die serum teen te gaan. Die gebruik van anaboliese steroïede is onlangs bepleit en hierdeur kon die bloedureum aansienlik verminder word by sommige pasiënte.[2,3] Beskermende verpleging kan help om hierdie pasiënte teen die soort infeksies te vrywaar waaraan hulle besonderlik blootgestel is. Antibiotiese middels kan gegee word indien nodig, maar die dosisse moet versigtig bepaal word omrede van die stadige uitskeiding van hierdie stowwe by die oliguriese pasiënt. Hierdie pasiënte kry dikwels 'n anemie, maar oortapping moet vermy word indien moontlik.

Baie pasiënte kan gehelp word gedurende die oliguriese fase met hierdie konserwatiewe regime. Sommige pasiënte gaan egter progressief agteruit ten spyte van al hierdie maatreëls. Dit is in hierdie gevalle dat die kunsnier gebruik moet word. Soos Merrill[4] aangetoon het, is dit nie die *soort* dialiese wat van die grootste belang is nie, maar die *ondervinding* van die terapeutiese span, en ons is bly om te kan meld dat daar bedrewe spanne in hierdie land is.* Pasiënte wat tekens toon van kliniese of biochemiese agteruitgang onder die konserwatiewe regime gedurende die oliguriese fase, behoort sonder verwyl na 'n erkende sentrum verwys te word.

As die volume van die urine eers 1 liter per 24 uur bereik het, kan hierdie strawwe regime verslap word. 'n Groot deel van die poliurie van die diuretiese fase bestaan slegs uit lediging van oortollige ekstrasellulêre vloeistof en pogings tot vervanging hiervan mag lei tot oorhidrasie en 'n verlengde tydperk van diurese. Die pasiënt se algemene toestand, toestand van hidrasie, dors, en die vlak van die serumelektroliete behoort die maatstaf te wees liewers as die hoeveelheid en bestanddele van die urine. Gedurende die diuretiese fase behoort die pasiënt vrye toegang tot water te hê en 'n dieet wat ryk is aan kalium en sout.

* Kyk na die artikel oor die kunsnier wat op bladsy 374 van hierdie uitgawe van die *Tydskrif* verskyn.

1. Bull, G. M., Joekes, A. M. en Lowe, K. G. (1949): Lancet, 2, 229.
2. McCracken, B. H. en Parsons, F. M. (1959): *Ibid.*, 2, 885.
3. Gjorup, S. en Thaysen, J. H. (1958): *Ibid.*, 2, 886.
4. Merrill, J. P. (1955): *Treatment of Renal Failure.* New York: Grune & Stratton, Inc.

THE TREATMENT OF ACUTE RENAL FAILURE

Crush injuries during the Second World War resulted in the recognition of acute renal failure as a reversible lesion. Shortly after the war the conservative treatment of this malady was developed on the basis of strict control of fluid intake with a high-calorie mixture of glucose and fat, free from electrolytes and protein.[1] It is now thought that 40-50% glucose is all that should be given in the way of calories and that diets rich in fat need not be used. This fluid may be given either by mouth or intravenously into the superior or inferior vena cava; smaller veins are apt to be thrombosed by the hypertonic solutions. Fluid intake should be restricted to about 400 ml. per 24 hours, and a useful guide to fluid requirements is the daily weight. Ideally the patient should lose about 0·2 kg. in weight every 24 hours. Fluid loss through vomiting may have to be replaced, and this may also necessitate giving the patient some half-strength saline by mouth, but not more than would replace that lost by vomiting. Chlorpromazine is often able to prevent this troublesome vomiting if the condition of the liver is satisfactory.

Calcium gluconate should be given to counteract the tendency of the serum calcium to fall. Sodium exchange resins and the administration of glucose and insulin may help to lower a mounting serum potassium. Recently the use of anabolic steroids has been advocated and these have been shown to lower the blood urea considerably in some patients.[2,3] Barrier nursing helps to prevent the infections to which these patients are particularly liable. Antibiotics are given as needed, care being taken regarding dosage because of the slow excretion of these substances by the oliguric patient. These patients often become anaemic but transfusion should be avoided if possible.

While many can be saved during the oliguric phase by this conservative regime, there are some patients who

deteriorate steadily despite all these measures. It is in these cases that an artificial kidney should be used. As Merrill[4] has pointed out, it is not so much the *type* of dialysis used but the *experience* of the team that is important, and we are pleased to note the presence of experienced teams in this country.* Patients deteriorating clinically or biochemically on the conservative regime during the oliguric phase should be referred to a recognized centre without delay.

Once the urine volume has reached 1 litre per 24 hours this rigid regime can be relaxed. Much of the polyuria of the diuretic phase is only evacuation of excess extracellular fluid, and efforts to replace it may lead to overhydration and prolong the period of diuresis. One should be guided by the patient's general condition, state of hydration, thirst, and the serum-electrolyte levels rather than by the urine volume and its contents. During the diuretic phase the patient should have easy access to water and a diet rich in potassium and salt.

* See article 'Die Kunsnier (The Artificial Kidney)' published on this page of this issue of the *Journal*.

1. Bull, G. M., Joekes, A. M. and Lowe, K. G. (1949): Lancet 2, 229.
2. McCracken, B. H. and Parsons, F. M. (1958): *Ibid.*, 2, 885.
3. Gjorup, S. and Thaysen, J. H. (1958): *Ibid.*, 2, 886.
4. Merrill, J. P. (1955): *Treatment of Renal Failure.* New York: Grune & Stratton, Inc.