TABLE II—Total Grades of 20 Placebo Patients

	Swelling	Pain	Disability	Total
Before treatment first day ..	38	37	41	116
After treatment third day ..	26	25	23	74
Percentage improvement ..	31.6	32.4	43.9	36.2

TABLE III—Total Grades of 20 Treated Patients

	Swelling	Pain	Disability	Total
Before treatment first day ..	38	43	46	127
After treatment third day ..	14	11	6	31
Percentage improvement ..	63.2	74.4	86.9	75.6

STATISTICAL ANALYSIS

With a non-sequential method of analysis the Diapulse treatment was found to be significantly preferable to the placebo treatment on all four counts—that is, swelling, disability, pain, and total scores.

A Bross plan B chart for sequential analysis of matched pairs was used and Diapulse was found to be a preferable treatment compared with placebo, statistically speaking, with respect of improvement in disability scores, pain scores, and total scores. No significant difference between the two groups was found as regards improvement in swelling scores, although the trend of results seen looked likely to produce a significant result, given more results.

The outcome of awarding a positive sign when the treated patient's progress was better than his or her matched pair and a

TABLE IV—Results of Treatment in 20 Matched Pairs of Patients

	No. Positive	No. Negative	P Value
Swelling	9	2	0.033
Disability	12	2	0.006
Pain	13	1	0.002
Total Scores	13	2	0.011

negative sign when the placebo patient made more progress is shown in Table IV. No sign was awarded when there was no clear preference between the two patients. The P values in all four categories are less than 0.05.

Discussion

This investigation shows that so far as sprained ankles are concerned pulsed, high-frequency electrical treatment has a biological effect on recently-injured soft tissues. This is particularly noticeable in the reduction of pain and also disability. The treatment is time consuming, however, both for the patient and for the physiotherapist and it is probably not justified in treating minor sprains where the patient can walk on the ankle after the application of an adhesive bandage, but for the patient who is more severely injured it has been shown to be a valuable method of treatment.

I wish to express my thanks to the Principal and Governors of Nonington College of Physical Education for making available the services of Miss V. Barclay, who gave the physiotherapy treatment.

The Diapulse machines were loaned by the Diapulse Corporation of America.

The statistical analysis of the results was prepared through the good offices of Messrs. Smith & Nephew Limited.

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MEDICAL MEMORANDA

Fatal Hypoglycaemia Occurring after Peritoneal Dialysis

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This report illustrates a potentially fatal complication of peritoneal dialysis which has received little attention in the medical literature.

Case History

A 52-year-old Negro man was first admitted to hospital on 29 December 1970 because of chest pain. A diagnosis of uraemic

pericarditis was made. He had been subjectively well until a few hours before admission and there was no personal or family history of diabetes, hypertension, or renal disease. A suprapubic prostatectomy had been performed seven years previously at another hospital.

On admission he was found to be acutely ill, cachectic, and dehydrated, and a loud pericardial friction rub was heard. Haematocrit reading 20%; white cell count 8,800/mm³; blood urea nitrogen (B.U.N.) 177 mg, creatinine 18.5 mg, glucose 140 mg, and uric acid 11.6 mg/100 ml; serum potassium 6.9 mEq/l, bicarbonate 20 mEq/l. Urine analysis showed proteinuria, pyuria, and many white blood cell casts. E.C.G. was consistent with pericarditis and hyperkalaemia.

He was immediately treated by peritoneal dialysis with 1.5% glucose dialysate. This produced noticeable subjective and objective improvement, with disappearance of the friction rub and chest pain, regression of the E.C.G. abnormalities, and reduction of the B.U.N. to 90 mg/100 ml. Subsequent isotope renography showed symmetrically shrunken kidneys with severely reduced function but no obstruction. Cystoscopy showed a stricture in the area of the prostatic urethra, which was dilated.

Ampicillin, allopurinol, polystyrene sulphonate resin, dietary restriction of protein and potassium, transfusion of packed erythrocytes, and two further peritoneal dialyses comprised the subsequent therapeutic regimen. Multiple blood glucose determinations, including the periods while dialyses were in progress and

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immediately thereafter, fell in the range 90-255 mg/100 ml except for a value of 70 mg/100 ml noted one hour after the final dialysis. After 46 days in hospital he was discharged on an intensive medical regimen, with a diagnosis of chronic renal failure secondary to chronic pyelonephritis. The B.U.N. at that time was 91 mg/100 ml.

Because of increasing malaise and azotaemia the patient was re-admitted on 12 March 1971. The B.U.N. was 166 mg and the glucose 160 mg/100 ml. Mental confusion and twitching rapidly developed and peritoneal dialysis was performed on the third hospital day, with reduction of the B.U.N. to 131 mg/100 ml but with only slight subjective improvement.

At 5.40 p.m. on the sixth hospital day peritoneal dialysis was again instituted. Progressive hyperglycaemia developed during this dialysis (see Chart); three hours before the end of the procedure the blood glucose was 615 mg/100 ml. Dialysis was terminated

Blood glucose concentrations during and after final peritoneal dialysis.

at 3 p.m. on 19 March, at which time the B.U.N. was 141 mg/100 ml.

At 7 a.m. the next day he was found to be somewhat improved neurologically. At 8 a.m., however, the onset of lethargy, confusion, and diminished responsiveness was noted. These signs progressed rapidly and he became completely unresponsive and apnoeic. The trachea was intubated and assisted ventilation undertaken. There were no focal neurological signs and the E.C.G. was unchanged. At 11.30 a.m. the 7-a.m. blood glucose value was reported to be 33 mg/100 ml. The 11.30 a.m. value was 29 mg/100 ml. Intravenous glucose was immediately administered and there was transient improvement of the neurological status. Despite continuous infusion of 10% glucose solution, however, he failed to emerge from coma and died from cardiac arrest one day later.

Comment

Prolonged hypoglycaemia 16 to 20 hours after peritoneal dialysis was implicated as the immediate cause of death in this patient. Hyperglycaemia, sometimes of severe degree, commonly occurs during dialysis, but postdialysis hypoglycaemia has been infrequently reported. White and Kurtzman (1971) reported the case of a non-diabetic azotaemic patient who developed symptomatic hypoglycaemia on three different occasions after peritoneal dialysis. Two reports (Gutman *et al.*, 1967; Rigg and Bercu, 1967) described hypoglycaemia occurring after haemodialysis with high dialysis bath glucose concentrations.

Abnormal glucose tolerance is known to be associated with renal insufficiency. Unlike the true diabetic, in whom there is impaired insulin release in response to a glucose load, the azotaemic patient responds to carbohydrate loading with normal or supranormal plasma insulin levels (Horton *et al.*, 1968; Rabkin *et al.*, 1970). Peripheral insulin resistance has been implicated and confirmed as the cause of the metabolic defect (Westervelt, 1969).

The metabolism of insulin itself is also altered in renal insufficiency. In the healthy individual the kidney is the major site of degradation of insulin which is injected into the peripheral circulation and of endogenously-released insulin once it has passed through the liver (Rubenstein and Spitz, 1968). The importance of renal insulin metabolism increases at high levels of circulating insulin, and the kidney may act as a physiological buffer in preventing excessive or abnormal rises in plasma insulin levels.

When functioning renal tissue is destroyed to the degree that severe azotaemia occurs degradation of insulin is severely impaired. Horton *et al.* (1968) showed delayed disappearance of peripherally-injected insulin in uraemic subjects as well as an abnormally prolonged hypoglycaemic effect. Rabkin *et al.* (1970) measured renal arterial and venous insulin levels in both normal and azotaemic subjects. In those with normal renal function some 35% of insulin reaching the kidneys was removed, whereas in patients with glomerular filtration rates in the range of 3 ml/minute this was reduced to 10%. These workers calculated that normal kidneys will remove about 7 units of insulin in 24 hours, compared with less than 0.2 unit in their subjects with severe renal insufficiency. Such observations may explain the decreased insulin requirements seen in diabetics who develop renal failure. These patients may become highly insulin sensitive and have been reported to develop spontaneous and life-threatening hypoglycaemia even without exogenous insulin administration (Block and Rubenstein, 1970).

The fatal hypoglycaemic episode in the present case was most reasonably attributed to hyperinsulinism resulting from dialysis-induced hyperglycaemia, in which functioning renal tissue was not available to metabolize excess insulin. This example serves to emphasize the potential hazard of post-dialysis hypoglycaemia and the necessity of monitoring blood glucose concentrations in the period after termination of dialysis.

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