

NO RELATIONSHIP BETWEEN ACETATE AND HYPOTENSION IN A STANDARD DIALYSIS SCHEDULE

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Summary

Plasma acetate (PA) kinetics was analysed in two groups of patients on regular dialysis treatment (RDT). The first group presented frequent symptomatic hypotension (SHY), the second did not experience SHY during RDT. The parameters examined showed no difference between the two groups.

Seven patients of the first group were then switched to bicarbonate dialysis. SHY rate and blood pressure changes did not significantly differ between the two methods of treatment.

Introduction

Hypotension is the most frequent side effect of regular dialysis (RDT). The relative importance of acetate in its multifactorial aetiology remains controversial [1]. A relationship between plasma acetate (PA) and hypotension has been observed by some authors [2, 3], but not by others [4].

In some studies bicarbonate has favourably replaced acetate when large surface area dialysers were used [5], or when dialysate sodium concentration was low [6].

The purpose of this study was to determine whether PA concentrations and kinetics, during a standard dialysis with normal surface area dialysers and dialysate sodium of 140mmol/L, were different in patients with or without SHY and to evaluate if switching to bicarbonate dialysis in patients with SHY could prevent hypotension.

Patients and methods

Twenty uraemic patients on RDT were studied. None had diabetes mellitus, systemic diseases or hepatic failure. They were subdivided into two groups:

Group 1: Nine patients who developed SHY with a frequency ranging from 20 to 95 per cent of dialyses;

Group 2: Eleven patients who did not develop SHY during RDT.

Table I shows clinical and dialysis details of the two groups. There was no difference between the groups except for the blood pressure change during dialysis. Both the groups were dialysed for three hours (two cases) or four hours (18 cases) thrice weekly with 0.9–1.2m² effective surface area flat plate or hollow fibre dialysers. Q_B, Q_D, dialysate sodium (140 ± 1mmol/L) and acetate (38mmol/L) were the same in both groups. During two dialyses in each patient heparinised blood samples were drawn from the arterial line before, each hour during, and 20 minutes after dialysis and 10 minutes after SHY (nine SHY occurred during the study).

TABLE I. Patient groups

Variable	Group 1	Group 2	p
Patients	9	11	
Male patients %	33	66	NS
Age (years)	58.1 ± 8.1	58.9 ± 7.7	NS
Months on dialysis	65.8 ± 29	48.2 ± 25	NS
RKF (Cl _{CR})	0.6 ± 0.9	0.9 ± 0.8	NS
MAP (mmHg)	115.2 ± 17.7	108.7 ± 11.3	NS
Dry body weight (kg)	61.5 ± 12.8	66.1 ± 8.2	NS
Weight loss per dialysis (% bw)	3.1 ± 1.6	3.3 ± 1.4	NS
UF (ml/min)	8.4 ± 3.8	9.1 ± 3.4	NS
Δ pulse rate (beats)	+5.8 ± 4.2	+2.6 ± 3.8	NS
Δ syst. AP (mmHg)	-35.3 ± 16.7	-2.6 ± 10.4	< 0.001
Δ diast. AP (mmHg)	-12.5 ± 9.5	-0.1 ± 5.8	< 0.01

Blood was immediately cooled, centrifuged at 3000rpm and plasma was frozen at -30°C.

For acetate analysis 1ml of the sample was acidified with 40μL of hydrochloric acid and analysed with a Perkin Elmer 3920 gas chromatograph. The analysis conditions were: FID detector, Poropak Q 80–100 mesh in glass column (0.6cm outlet diameter, 200cm length). Temperatures were: injection 175°C, column 160°C, detector 210°C. Injection volume was 2μL.

Seven patients of the first group were then switched to bicarbonate dialysis: each patient underwent about 70 bicarbonate dialyses. Q_B, Q_D, dialysis hours, dialysers, dialysate sodium concentration were the same as for acetate dialysis. Dialysate bicarbonate was 27–35mmol/L plus 10mmol/L acetate (Bellco System). Blood pressure was recorded before and after dialysis and every hour during dialysis. The SHY and hypotension treatment (2–4g bolus of 20% sodium chloride) were also recorded.

The last 40 acetate dialyses for each patient and the bicarbonate dialyses were compared.

Results

PA kinetics was sufficiently stable for each patient. The mean difference between the highest and the lowest value at each time was less than 1mmol/L.

In both groups PA increased quickly during the first hour, rose slowly during the second hour and then reached a plateau (Figure 1).

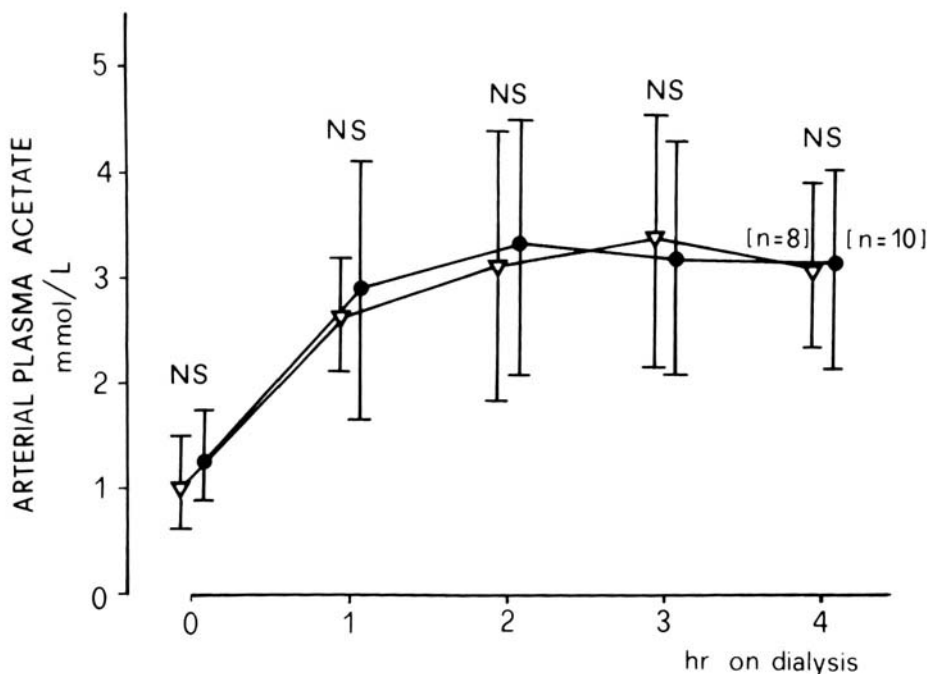


Figure 1. PA concentrations during haemodialysis. Mean \pm SD for nine patients with SHY (∇) and for 11 patients without SHY (\bullet)

At each hour we could not observe a significant difference between the two groups.

The PA peaks had a wide range from patient to patient (2.1–6.8mmol/L) but no significant differences were observed between the groups (group 1: 4.55 ± 1.28 mmol/L; group 2: 4.37 ± 1.22 mmol/L).

In group 1 no relationship was detected between PA peaks and systolic blood pressure Δ (r 0.0992), diastolic blood pressure Δ (r 0.2935) and frequency of SHY (r 0.074).

Sharp increases of PA were never observed before and seldom after SHY (before 2.98 ± 1.19 mmol/L; after 3.95 ± 1.37 mmol/L; $t = -1.47$).

Circulating acetate disappears at the same rate in both groups after dialysis (Figure 2).

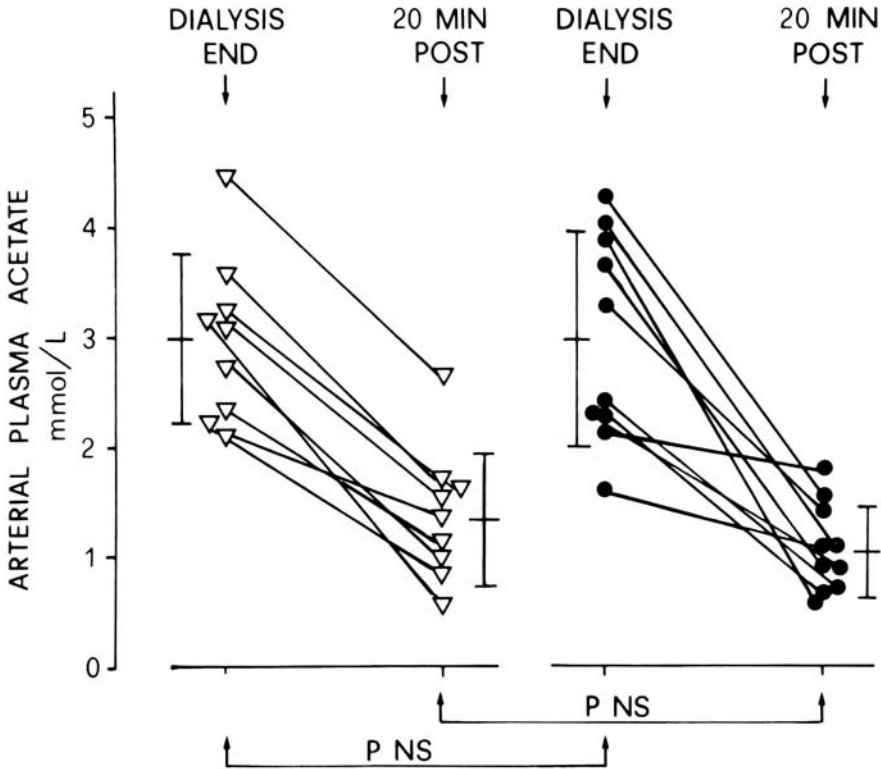


Figure 2. PA decrease in the immediate after-dialysis period in nine patients with SHY (∇) and in 10 patients without SHY (\bullet)

Blood pressure Δ and incidence of SHY did not change after switching seven patients of the group 1 to bicarbonate dialysis (Figure 3).

UF rate did not differ between acetate and bicarbonate dialysis (9.53 ± 2.93 versus 9.78 ± 3.83 ml/min). The frequency of sodium chloride bolus infusion required to treat hypotension did not change (required in 77% of acetate dialyses versus 70% of bicarbonate dialyses).

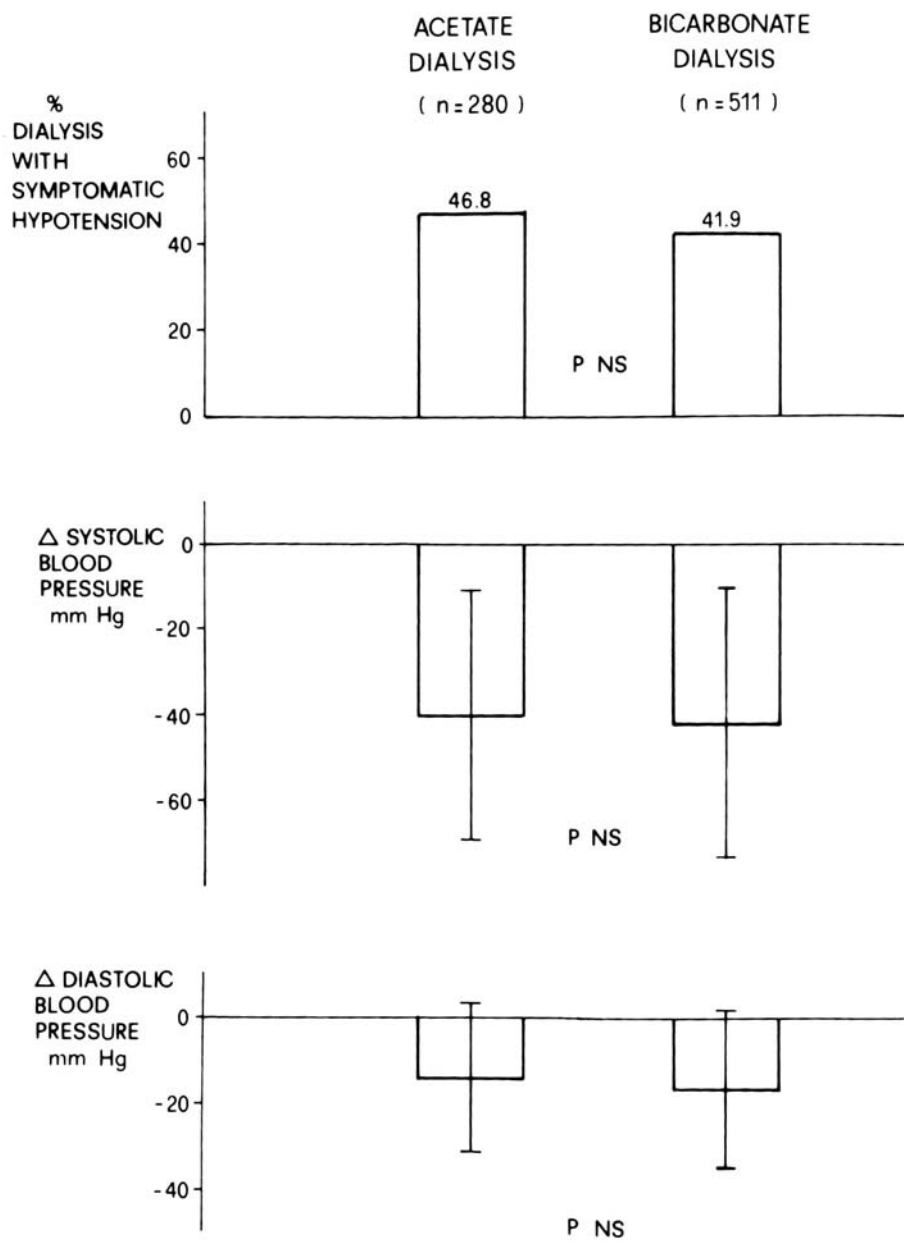


Figure 3. Frequency of dialysis with SHY and systolic and diastolic blood pressure Δ in seven patients during acetate dialysis (left) and bicarbonate dialysis (right)

Discussion

No significant differences in PA kinetics during and after dialysis nor in PA peaks were detected between the two groups. PA did not significantly increase before or after SHY. No evidence of defective acetate metabolism in patients of group 1 was therefore detected.

The possibility that patients with SHY are more sensitive than the patients of the second group to the same acetate concentration seems unlikely. In fact switching to bicarbonate dialysis did not lead to any improvement. It can be concluded that, in our standard dialysis schedule with a dialysate sodium of 140mmol/L, other factors, individual, due to haemodialysis [7], or both, seem to prevail in the genesis of hypotension.

References

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