REGULATION OF ACID-BASE COMPOSITION BY DIALYSIS

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Both cellophane and peritoneum may be used effectively as dialysing membranes but as the former is an inert material and the latter a living tissue, it is reasonable to expect some differences in the result. It was decided to determine whether the method of dialysis had any differing effect on the acid-base composition of the blood.

METHOD

Blood was taken by arterial puncture at the beginning and end of 18 peritoneal dialyses performed on 6 patients, and from the arterial line in 35 dialyses on 5 patients. Both groups of patients had complete renal failure and were being maintained by chronic dialysis. Blood was taken anaerobically into a heparinized syringe. Estimations were carried out immediately, or if that was not possible the syringe containing the blood was plunged into ice water and estimations were carried out within four hours. The pH, pCO₂ and standard bicarbonate were estimated using the Astrup apparatus and Sigggaard Anderson nomogram. Corrections of pH for alteration in temperature were made when necessary but no corrections were made for haemoglobin level which was about 7.5 g% in all patients. All the figures for standard bicarbonate are therefore 0.5 mEq/litre too low.

RESULTS AND DISCUSSION

Figure 1 shows the changes which occurred. At the beginning of dialysis blood from both groups of patients had a similar acid-base composition, the mean values being pH 7.35, pCO₂ 29.5 mm Hg and standard bicarbonate 17.5 mEq/litre for the peritoneal dialysis group, and pH 7.34, pCO₂ 29.2 mm Hg and standard bicarbonate 17.4 mEq/litre for the haemodialysis group. With both methods the pH rose to the upper limit of normal, being 7.46 and 7.45 after peritoneal dialysis and haemodialysis respectively; with peritoneal dialysis the standard bicarbonate rose to normal—24.5 mEq/litre, and pCO₂ almost to normal—33.4 mm Hg, but with haemodialysis the rise in standard bicarbonate to 21.3 mEq/litre was less marked and pCO₂ at 29.8 mm Hg was unchanged. The results obtained by haemodialysis are similar to those reported by Weller et al. (1953) but differ from those of Cowie et al. (1962) and Lambie et al. (1965), who found a rise in pCO₂ although it did not reach normal. No comparable results for peritoneal dialysis have been published.

During haemodialysis (Fig. 2) the standard bicarbonate value shows an initial delay or fall; most of the increase occurs during the 2nd to 5th hours inclusive after which the curve tends to flatten. In two patients there was a second rise at the 9th hour but whether the curve for standard bicarbonate with time is truly biphasic cannot be determined from the present results. In haemodialysis the pH rises steadily and the pCO₂ varies. The changes with time occurring in peritoneal dialysis were not investigated as it was not considered justifiable to perform frequent arterial punctures or leave an arterial cannula in situ.
It was thought that the differences in the final values of $pCO_2$ and standard bicarbonate could be a consequence of the length of time of dialysis which was 30-36 hours for peritoneal dialysis and 10-12 hours for haemodialysis (Kiil dialyser), and that further equilibration between the E.C.F. and I.C.F. occurring after dialysis, especially the more rapid haemodialysis could result in equality. In three patients of each group arterial blood was collected 12 hours after the end of dialysis. Figure 3 shows that further changes do occur but in the same direction in both groups and are such as to lead to greater disparity in acid-base composition. In the peritoneal dialysis group pH fell 0.05, standard bicarbonate fell 1.0 mEq/litre and $pCO_2$ rose 4.6 mm Hg; in the haemodialysis group pH fell 0.9, standard bicarbonate fell 2.4 mEq/litre and $pCO_2$ rose 2.5 mm Hg (mean values). Thus whereas the former have
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Fig. 2. Change in standard bicarbonate during haemodialysis.

attained a normal blood acid-base composition the latter have a metabolic acidosis balanced by a respiratory alkalosis.

It is well known that for several hours or even days after correction of a metabolic acidosis, by any means, a respiratory alkalosis persists. Anderton et al. (1965) produced evidence that the cause is a lowered ventilatory threshold for $\text{pCO}_2$ and $\text{H}^+$, and Cowie et al. (1962) have shown that with haemodialysis the low pH of the C.S.F. shows a disproportionately small increase towards normal compared with that of the blood.

It is conceivable that peritoneal dialysis removes more of the acid products of catabolism, particularly organic acids, than does haemodialysis and that it is the continuing presence of such substances which accounts for persistent metabolic acidosis in patients treated by haemodialysis. Comparison of the serum electrolyte estimations in the two groups shows no difference in the total cation concentration before and after dialysis. The chloride concentration was slightly lower in the peritoneal dialysis group (94 mEq/litre before and after dialysis) than in the haemodialysis group (99 mEq/litre before, 98 mEq/litre after dialysis).

The patients maintained by peritoneal dialysis were treated once and those maintained by haemodialysis twice a week. Clinically the patients were equally well. At the beginning of dialysis the blood acid-base composition in both groups was the same. The speed with which the acid-base values return to the pre-dialysis level is unknown but the greater correction achieved by peritoneal dialysis is compatible with the longer interval of time between dialyses.

CONCLUSION

Measurement of arterial blood samples shows that with peritoneal dialysis the acid-base composition is corrected to normal, whereas with haemodialysis the correction achieved is
Fig. 3. Change of acid-base composition during and after dialysis.

less and normal values of standard bicarbonate are only present for a short time at the end of dialysis—there is virtually a persistent metabolic acidosis balanced by a respiratory alkalosis. At present this difference cannot be explained but one may postulate that it reveals that physiologically active transport across the peritoneal membrane rids the body more effectively of waste products than does exchange across cellophane.

REFERENCES


