

Hypoventilation during Hemodialysis

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Dear Sir, Methods

In a recent article in this journal [1], Faro et al. studied the influence of CO₂ changes, due to CO₂ extraction during acetate dialysis, on the central venous blood composition and their effect on the pulmonary ventilation. They postulated that the decrease in CO₂ in the venous line blood has influence on the central venous blood composition, and that this plays a role in the control of the pulmonary ventilation in these patients.

In this communication we present our results on the influence of CO₂ loss through the dialyzer on the carbon dioxide tension measured in the pulmonary artery (mixed venous blood).

Ten patients, mean age 54,7 years (24–72 years), 6 male and 4 female, undergoing 4-hour maintenance hemodialysis sessions three times weekly, were studied. Nine patients were studied during hemodynamic monitoring for evaluation of different cardiac problems. They were all critically ill at the time of the study and treated with oxygen through a nasal cannula. One male patient volunteered to participate in the study. All patients gave informed consent. The patients were dialyzed with acetate-containing dialysate and a single-needle, double-headed pump system.

A Swan-Ganz thermodilution catheter (93A-131H-7F; Edwards Laboratory, Santa Anna, Calif., USA) was introduced percutaneously through the internal jugular vein into the pulmonary artery, at least 15 min before the start of the first measurement. Blood samples were drawn from the pulmonary artery and from the arterial side of

Table I. Mixed venous carbon dioxide tension (PvCO₂, expressed in mm Hg) in individual patients during the first hour of the hemodialysis session

Table II. Arterial carbon dioxide (PaCO₂, expressed in mm Hg) in individual patients during the first hour of the hemodialysis session (mean mm Hg values are shown in parentheses)

Mean	39.1(5.1)	37.9(4.9)	36.8(4.8)	37.7(4.9)
SD	4.8	5.2	2.7	3.7
SEM	1.5	1.8	0.9	1.2

p	NS	NS	NS		
Mean	32.7(4.3)		25.4(3.3)	25.3(3.3)	26.6(3.5)
SD	7.5	7.1	7.8	6.1	
SEM	2.4	2.4	2.5	1.0	
p	-	0.05	0.05		

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the fistula into heparinized cooled (4CC) plastic syringes and immediately assayed for pH, mixed venous carbon dioxide tension (PvCO₂), HCO_i, oxygen tension (PvO₂), and oxygen saturation (AVL 940; AVL, Graz, Austria). All parameters were analyzed at 0, 15, 30 and 60 min.

Statistics

Differences from control values at time 0 were evaluated using the Wilcoxon signed ranks test for paired values. The degree of significance was set at the 0.05 level.

Results

The average PvCO₂ did not decrease significantly during the study period (table I). In table II, where the concomitant arterial PCO₂ values are given, a much more pronounced decrease in PaCO₂ can be seen in some patients. The average values were statistically different from zero values at time 15 min, and returned to values at time 0 after 60 min. Arterial P_O₂ (PaO₂) did not decrease except in the 1 male volunteer patient. No difference in the parameters studied was observed between patients treated with cuprophane membrane (n = 6) and other membranes. bath, are confirmed in our study, measuring true mixed venous blood. However, we disagree with their conclusion that venous line blood plays a role in the control of pulmonary ventilation during routine hemodialysis. Indeed these authors based their conclusions mainly on the observed hyperventilation during dialysis with addition of CO₂. There is no doubt that any change in CO₂ tension in the pulmonary circulation can lead to a change in minute ventilation due to the presence of slowly adapting pulmonary chemoreceptors [2].

We therefore propose that the same conclusions can be drawn from both studies, i.e. there are until now no arguments that mixed venous blood composition plays any role in the hypoventilation during hemodialysis. The possibility of a direct effect of acetate on ventilation, as already stressed previously [3–5] has still to be questioned. Other methods, using continuous, highly accurate, measurements of blood gases are necessary.

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Conclusions

From these results it is clear that the loss of CO₂ in acetate-containing dialysate, although leading to very low PCO₂ in the blood returning to the patient (8 mm Hg), is not sufficient to cause a decrease in mixed venous CO₂ tension measured 4 times during the first hour of hemodialysis. This is even more striking in this particular group of patients since hyperventilation, presumably due to additional respiratory drive from worsening cardiac status and severe metabolic acidosis, was observed.

From our limited number of observations of constant PvCO₂ tension, one could conclude that a change in the activity of the slowly adapting CO₂-sensitive chemoreceptors is not apparent.

However, it remains possible that nondetectable oscillations in arterial or mixed CO₂ tension are the most important triggers for controlling ventilation during CO₂ unloading. Furthermore, others have postulated that total CO₂ load to the pulmonary circulation may be the mechanism involved. The results in the article by Faro et al. [1], regarding acetate dialysis without addition of CO₂ into the dialysis

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