

RESPIRATORY RESPONSE OF ACETATE DIALYSIS AND BICARBONATE DIALYSIS

A Salvadeo, G Pezzagno*, S Segagni, F Galli, G Villa, F Poggio,
V Piazza, G Bovio, L Picardi, E Petrella†, L Bigi†

*Clinica del Lavoro, Pavia, *Ist Medicina del Lavoro, Pavia, and
†Bellco Laboratories, Mirandola, Italy*

Introduction

Acetate dialysis (HDA) hypoxaemia can be attributed to: a) pulmonary micro-embolisation by fragments of fibrin, thrombocytes and/or leucocytes [1]; b) pulmonary leucostasis induced by complement activation [2], with a subsequent reduction of pulmonary CO₂ diffusion [3]; c) hypoventilation caused by CO₂ loss in the filter [4–7]; d) increased O₂ consumption [4, 6]; e) CO₂ consumption for acetate metabolism [8]. Hypoxaemia is not observed during bicarbonate dialysis (HDB) [9].

To elucidate mechanisms responsible for the hypoxaemia we studied: ventilation, pulmonary gas exchange, blood gas pressures and exchanges of total CO₂ (CO₂t), dissolved CO₂ (CO₂d) (CO₂d = PCO₂ × 0.03) and HCO₃⁻ across the dialyser during HDA and HDB.

Materials and methods

Nine patients (seven males, two females), ranging in age from 23 to 67 years, undergoing dialysis for 13–149 months, and having no pulmonary or cardiovascular disease were examined. All patients were studied both during HDA (38mEq/L of acetate) and HDB (31mEq/L of HCO₃⁻ and 10mEq/L of acetate) (Unimat + BL 723, Bellco, Mirandola). The patients were dialysed with a 1m² cuprophan filter; blood flow 300ml/min, dialysate flow 500ml/min. Samples of blood and dialysate were taken at the filter inlet and outlet to measure the partial pressures of O₂ (PO₂), CO₂ (PCO₂), pH (BMS Acid Base Analyser Radiometer) and CO₂t (Corning 965 Kontron); exchanges of CO₂t, HCO₃⁻, CO₂d across the dialyser were evaluated both for blood and dialysate by multiplying the difference between inlet and outlet values by the blood or dialysate flow rate. External ventilation (\dot{V}_E) was measured with a ventilometer (Life System Div.). Expired air was collected for 5min in a small bag and analysed for O₂, CO₂ and N₂ by gas chromatography (Sigma 4B Perkin Elmer) employing a

charcoal column and molecular sieves.

Data on ventilation and respiratory gases were used to calculate $\dot{V}O_2$, $\dot{V}CO_2$ and respiratory quotient (RQ); the Rahn and Fenn equations [10] were used to calculate alveolar ventilation (\dot{V}_A) and alveolar partial pressure of O_2 (PAO_2). All measurements were carried out immediately prior to dialysis, after 30 minutes, each hour and 30 minutes after the termination of dialysis. Statistical analysis was performed using t-paired Student's test.

Results

Blood passing through the dialyser loses, at a constant rate, 2.86mM/min of CO_2 t during HDA (2.57mM/min as HCO_3^-), and gains 0.47mM/min of CO_2 t during HDB (0.36mM/min as HCO_3^-) (Table I). \dot{V}_A , \dot{V}_E , PAO_2 and PaO_2 decrease significantly in the first hour of HDA, while they do not vary during HDB. $PaCO_2$ also decreases in HDA but not significantly until the second hour

TABLE I

mM/min	ACETATE		BICARBONATE	
	Blood	Dialysate	Blood	Dialysate
Dissolved CO_2	-0.14 ± 0.04	$+0.21 \pm 0.03$	$+0.041 \pm 0.03$	-0.38 ± 0.1
HCO_3^-	-2.57 ± 0.7	$+2.93 \pm 0.5$	$+0.36 \pm 0.2$	-0.62 ± 0.3
Total CO_2	-2.86 ± 0.4	$+2.97 \pm 0.4$	$+0.47 \pm 0.3$	-1.34 ± 0.37

of dialysis; $PaCO_2$ does not change during HDB (Figure 1). $\dot{V}O_2$ does not vary with either HDA or HDB. $\dot{V}CO_2$ and RQ decrease significantly throughout HDA; they do not change during HDB. During HDA (when $\dot{V}CO_2$ is corrected for CO_2 lost through the dialyser), overall CO_2 loss (ventilated + filtered) remains constant throughout dialysis. RQ, corrected in the same manner, also remains at baseline (Figure 2). During HDA, $\dot{V}O_2/\dot{V}_E$ ratio increases significantly in the first hour and then returns to baseline, while $\dot{V}CO_2/\dot{V}_E$ ratio decreases significantly during the entire course of dialysis. During HDB, $\dot{V}O_2/\dot{V}_E$ and $\dot{V}CO_2/\dot{V}_E$ ratios do not vary (Figure 3).

Discussion

During HDA, a moderate quantity of CO_2 t (about 3mM/min) is lost through the dialyser; blood at the outlet has a HCO_3^- of 9.2 ± 1.8 mM/L and a PCO_2 of 16.2 ± 3.1 mmHg. Provided that mixed venous blood is a mixture of about 95 per cent venous blood returning from the body and five per cent venous blood from

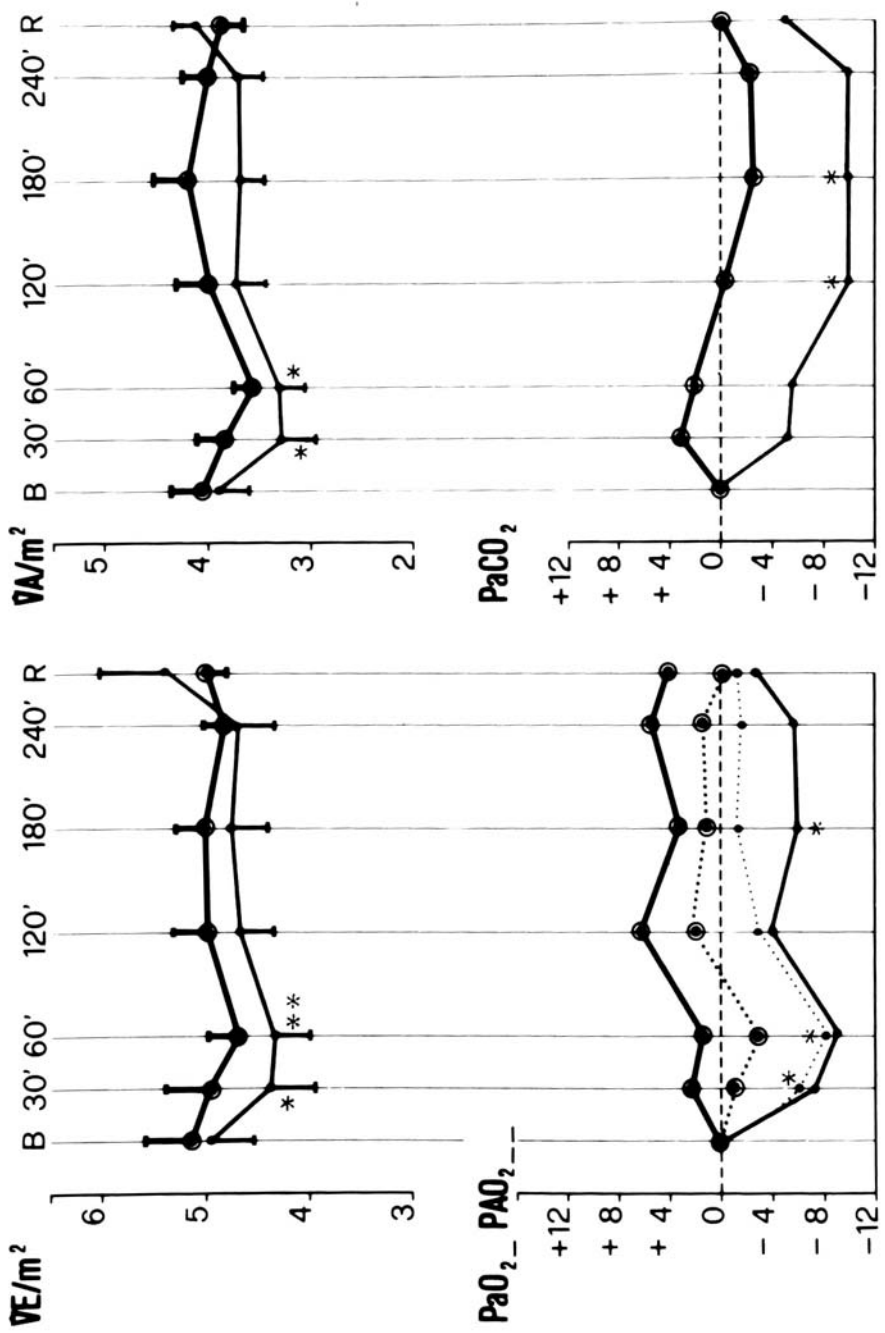
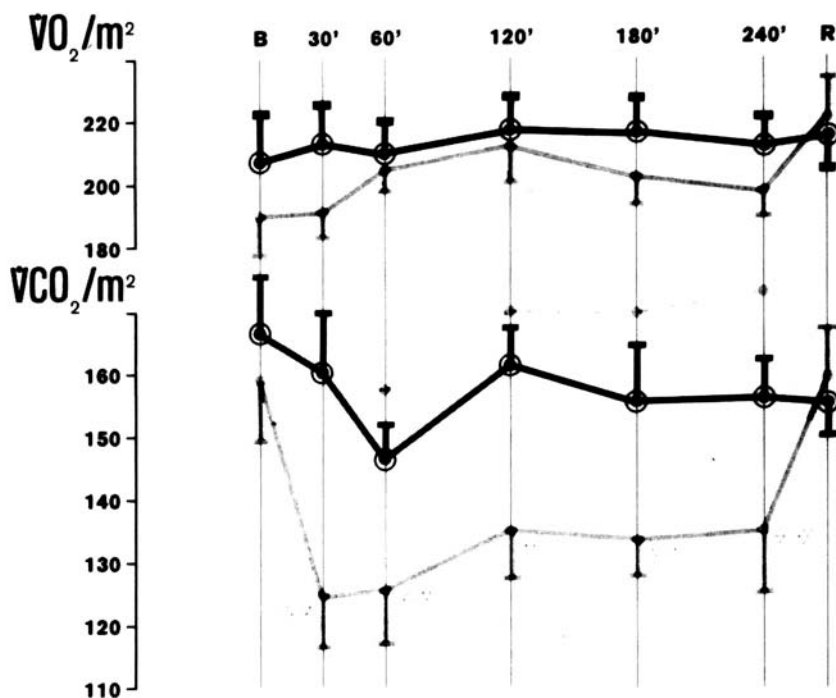


Figure 1. ●.....● and ●.....● = acetate dialysis; ○.....○ and ○.....○ = bicarbonate dialysis. * p < 0.05; ** p < 0.01



CO ₂ lost through filter ml/min/m ²	35.2	31.9	33.2	35.5	38
	+	+	+	+	+
	1.8	2.2	1.5	2.4	1.6

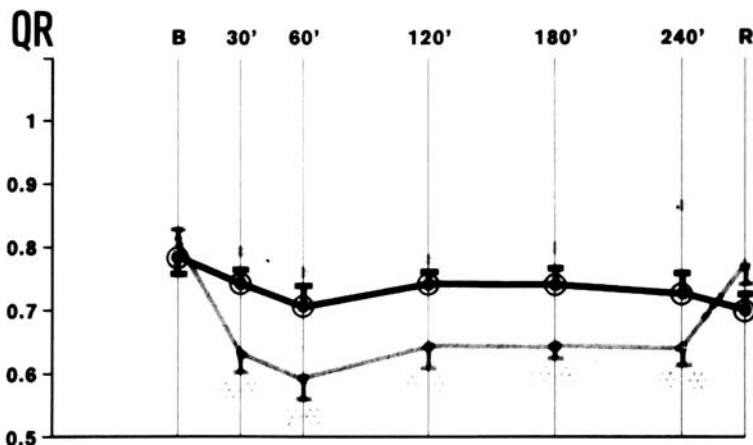


Figure 2. ●—● = acetate dialysis; ○—○ = bicarbonate dialysis; ●- - - ● = acetate dialysis corrected for CO₂ losses. * p < 0.05; ** p < 0.01

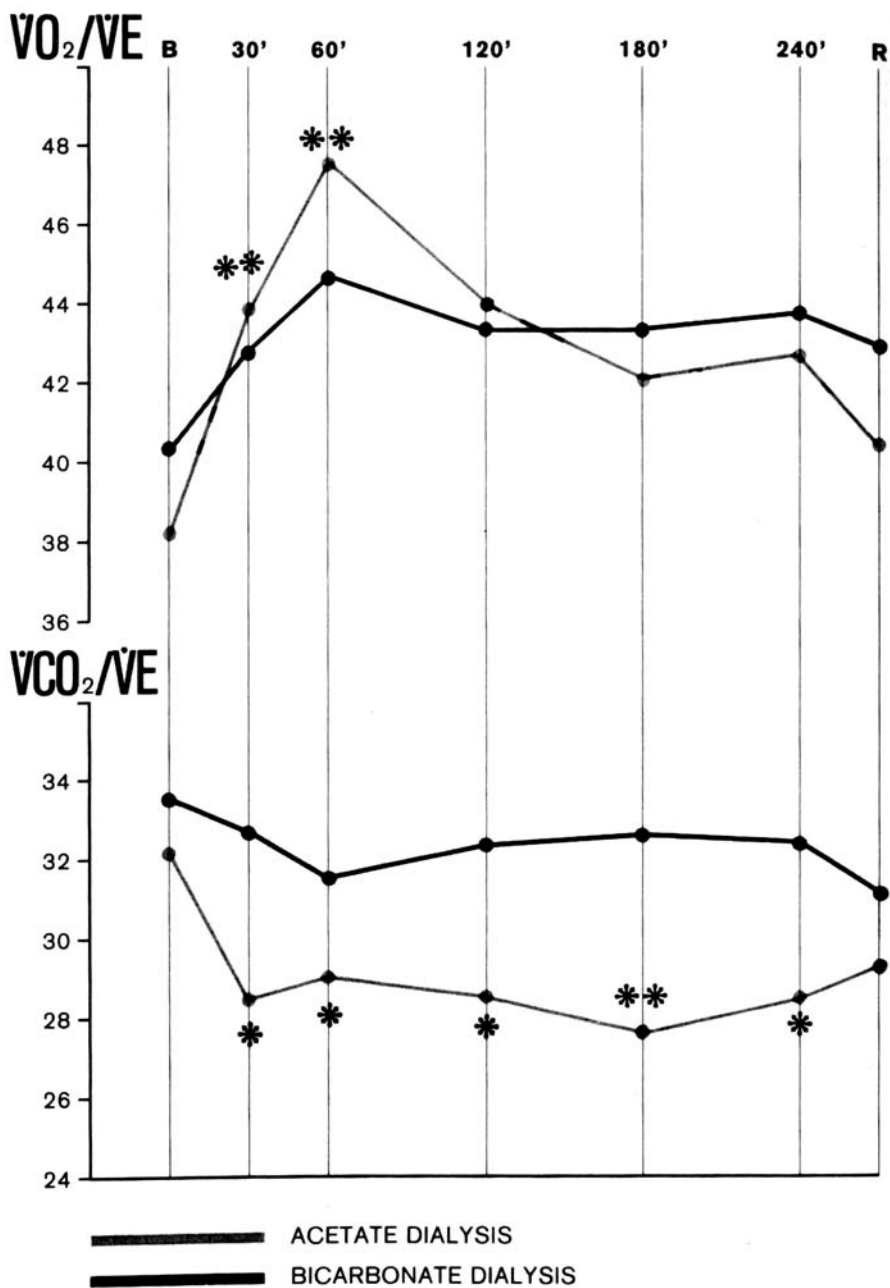


Figure 3. * p < 0.05; ** p < 0.01

the dialyser, one can calculate that HCO_3^- in the mixed venous blood is reduced by about 1mm/L and PCO_2 by about 2mmHg. Reduction of one or both of these values could explain the reduction of \dot{V}_E , \dot{V}_A , PAO_2 and PaO_2 observed in the first hour of HDA. In fact, pre-pulmonary CO_2 chemoreceptors have been described [11, 12]. In the first hour, hypoventilation reduces the fall of the PaCO_2 . In the second hour, \dot{V}_E , \dot{V}_A , PAO_2 and PaO_2 return to the baseline even if CO_2 loss in the dialyser remains constant. Then, once hypoventilation disappears, PaCO_2 decreases significantly. Hyperventilatory stimuli, hypoxaemia itself and/or worsening of acidosis by HCO_3^- loss through the dialyser, may therefore prevail over hypoventilatory stimuli after the second hour of dialysis. CO_2 retention by the lungs persists to meet dialyser CO_2 loss, in spite of normalisation of \dot{V}_E and \dot{V}_A . This saving of CO_2 is reflected in the persistently reduced $\dot{V}\text{CO}_2$ values and it results from reduction of the $\dot{V}\text{CO}_2/\dot{V}_E$ ratio.

Reduced $\dot{V}\text{CO}_2$ per litre of ventilated air might be consequent upon the hypothesised altered composition of mixed venous blood, i.e. 1) reduced PCO_2 which decreases the alveolar-capillary gradient, thereby slowing down CO_2 diffusion; 2) reduced HCO_3^- which slows down the reaction generating CO_2 to be eliminated in the lungs:



In HDB, there is no loss of CO_2 through the dialyser, and therefore, none of the above described phenomena take place.

References

- 1 Bische MD, Scoles BG, Mohler TG. *Chest* 1975; 67: 335
- 2 Craddock PR, Fehr J, Dalmaso AP et al. *J Clin Invest* 1977; 59: 879
- 3 Mahajan S, Gardiner H, Detar B et al. *Trans ASAIO* 1977; XXIII: 411
- 4 Sherlock J, Ledwith J, Letteri J. *Trans ASAIO* 1977; XXIII: 406
- 5 Aurigemma NM, Feldman NT, Gottlieb M et al. *N Engl J Med* 1977; 297: 871
- 6 Patterson RW, Nissensson AR, Miller J et al. *J Appl Physiol* 1981; 50: 259
- 7 Dolan MJ, Whipp BJ, Davidson WD et al. *N Engl J Med* 1981; 305: 72
- 8 Oh MS, Uribarri JV, Del Monte ML et al. *Proc Dial Transpl Forum* 1979; 18: 226
- 9 Nissensson AR. *Trans ASAIO* 1980; XXVI: 339
- 10 Rahn H, Fenn WO. *Washington DC Am Physiol Soc* 1955
- 11 Kollmeyer KR, Kleinman LI. *J Appl Physiol* 1975; 38: 819
- 12 Wasserman KB, Whipp J, Casaburi R et al. *J Appl Physiol* 1975; 38: 651

Address for correspondence: A Salvadeo, Div. Nefrologia e Dialisi, Fondaz., Clinica del Lavoro, Pavia, Italy