Dialysis-Induced Hypoxemia: 
Membrane Dependent and Membrane Independent Causes

George C. Francos, Anatole Besarab, James F. Burke, Jr, John Peters, 
Maria V. Tahamont, Marlys H. Gee, John T. Flynn, and Dan Gzesh

Hypoxemia during hemodialysis may result from several differing processes. We initially studied patients undergoing standard acetate hemodialysis. At 15 minutes of dialysis, leukopenia (primarily neutropenia), a decline of platelet count, and hypoxemia occurred, but without a significant change in mean minute ventilation. Complement activation (V/A ratios of Csa > 1.0) persisted throughout dialysis. Leukocyte count returned to baseline by one hour.

To separate the effects of solute and/or gas fluxes from those of blood-membrane interaction we studied changes in Po2, WBC, Csa, TxB2, and PGI2 during a period of blood membrane interaction without dialysis, and during subsequent acetate dialysis. Patients were studied with both polyacrylonitrile (PAN) and cuprophan membranes containing different priming solutions during membrane contact alone. Despite leukopenia and complement activation, hypoxemia failed to occur during membrane contact alone. At 15 minutes of subsequent acetate dialysis, significant hypoxemia occurred with both membranes. However, the degree of hypoxemia was twice as great with a cuprophan membrane primed with acetate (18.6 ± 3.3 mm Hg) compared with air or bicarbonate (9.1 ± 1.4 and 7.0 ± 2.0 mm Hg, respectively), or compared with PAN (8 ± 2.8 mm Hg). Changes in thromboxane B2, PGI2, and Csa did not correlate with changes in Po2.

We conclude that there are two major components to dialysis related hypoxemia. One is membrane independent, and may relate to the metabolic effects of acetate or to dialyzer CO2 loss. The remaining portion is membrane dependent, occurring with cuprophan, but not with PAN, and is conditioned by an acetate dependent interaction between blood and membrane.

© 1985 by The National Kidney Foundation, Inc.

INDEX WORDS: Hypoxemia; dialysis; leukocytes; complement.

Despite intensive study by many investigators, hemodialysis associated hypoxemia remains inadequately explained. Craddock et al1 have proposed that complement activation by the dialyzer membrane results in pulmonary vascular sequestration of leukocytes. This pulmonary vascular leukostasis presumably results in ventilation-perfusion mismatch and hypoxemia. Dumler and Levin,2 however, were able to dissociate leukopenia and hypoxemia with sequential ultrafiltration and hemodialysis, with reused cellulosic dialyzers, and with polyacrylonitrile dialyzers. They concluded that there is no relationship between the phenomena of dialysis-related leukopenia and hypoxemia.

Dolan et al compared the effects of acetate and bicarbonate hemodialysis on pulmonary function.3 They found that acetate dialysis produced a fall in CO2 output at the mouth (v CO2), with no change in arterial Pco2. Bicarbonate dialysis, with a dialysate Pco2 of 35 to 42 mm Hg, did not produce hypopnea or hypoxemia. Patterson and coworkers4 supported these findings by demonstrating a decreased respiratory quotient (RQ) during acetate dialysis. Both groups of investigators concluded that CO2 transfer from the patient to acetate dialysate produced hypopnea and hypoxemia, as respiratory chemoreceptors defended arterial Pco2 levels. The high Pco2 of bicarbonate dialysate, they reasoned, prevented this CO2 transfer and thus prevented hypopnea and hypoxemia. Oh et al has proposed that, rather than dialyzer CO2 loss, CO2 consumption in the metabolism of acetate underlies hypopnea.5 Hypopnea alone, however, fails to explain the differing degrees of hypoxemia between various dialyzer membrane materials during acetate dialysis noted by some observers.6,7

We sought to clarify the etiology of dialysis-related hypoxemia by first studying the effects of acetate hemodialysis with cuprophan membrane on arterial blood gases, minute ventilation, peripheral leukocytes, complement activation (Csa), and prostaglandins (thromboxane, prostacyclin). To separate the effects of solute and/or gas fluxes...
from those of blood-membrane interaction, we measured these same parameters during a period of blood-membrane contact without dialysis, and followed by acetate hemodialysis, using different dialyzer membranes.

MATERIALS AND METHODS

A total of 11 subjects on maintenance hemodialysis (4 months to 7 years) participated in a total of 38 studies. Informed consent was obtained from each subject. The research protocol was reviewed and approved by the Human Research Committee of Thomas Jefferson University. Criteria for patient selection included a starting Po2 of > 90 mm Hg, no history or clinical evidence of pulmonary disease or of congestive heart failure, and minimal weight gain (less than 1.6 kg) between dialysis treatments. All subjects were undergoing routine hemodialysis. Studies of Group 3 used a polyacrylonitrile dialyzer (Hospal RP610, Berr, Switzerland) primed with air (5 patients), 230 mL of acetate dialysate, or 230 mL of dialysate consisting of acetate and glucose (5 patients). The initial dialysate concentrations of acetate and glucose were 36 mEq/L and 200 mg/dL, respectively. Blood flow was kept constant at 250 mL/min and the dialysate flow rate was 500 mL/min. Arterial blood gases (pre- and postdialyzer), and arterial leukocytes and platelet counts were measured prior to dialysis; at 15 minutes; at one, two, and four hours of dialysis; and at one hour after dialysis. Minute ventilation was measured prior to dialysis; at 15 minutes and two hours of dialysis; and one hour postdialysis. Four other patients (Group IB) were studied in an identical protocol but without measurement of minute ventilation. In this group, C50 and C50 were determined. Predialyzer (hereafter referred to as 'arterial') levels of TxB2, PGI2, and C50 were measured at 0, 5 minutes, one hour, and four hours of dialysis. Post dialyzer (hereafter referred to as 'venous') C50 was obtained at 15 minutes and at four hours of dialysis.

The next phase of the study was designed to separate the effect of solute and/or gas fluxes from those of blood-membrane interaction. Six of the above 10 patients (Group 2) were first studied during each of three protocols. Patients underwent 30 minutes of membrane contact without dialysis, in which the dialysate chamber of a cuprophan dialyzer was primed either with air, 230 mL of acetate dialysate, or 230 mL of a bicarbonate solution (Na138, K 2.0, HCO3 24, CI 116 mEq/L, Ca 6 mg/dL, glucose 200 mg/dL, gassed with 5% CO2 for a pH of 7.44). The dialysate ports were then tightly sealed and blood flow through the dialyzer was started. Thus, no dialysis or loss of bicarbonate or CO2 occurred during an initial 30 minutes of blood-membrane contact. The dialysate ports were opened after 30 minutes, allowing acetate hemodialysis to commence.

Six patients (5 of whom had participated in one or more of the above protocols) were then studied in an identical protocol (Group 3) but using a polycryliclonitrile dialyzer (Hospal RP610, Berr, Switzerland) primed either with air (5 patients), or acetate (5 patients).

In all patients, predialyzer blood gases, leukocyte, and platelet counts, TxB2, PGI2, and C50 were measured prior to, following 15 and 30 minutes of membrane contact alone, and after an additional 15 minutes of acetate dialysis. Venous C50 was measured at 15 minutes of membrane contact alone and after 15 minutes of true acetate dialysis.

Analytical Methods

Blood gases were performed on either an ABL 2 or ABL 3 (Radiometer, Copenhagen, Denmark) blood gas analyzer. Accuracy of Po2 measurements at 100 mm Hg was 2.9% and repeatability was ± 2 mm Hg. Accuracy and repeatability for Pco2 at 40 mm Hg were 1.2% and ± 1 mm Hg, respectively. Leukocyte and platelet counts were determined with a Coulter Plus cell counter. Differential counts were done on 100 leukocytes. Minute ventilation was measured using a Beckman metabolic cart (MMCI, Anaheim, Calif) with the subjects' breathing through an attached mouthpiece. Results were normalized to each individual's dry weight.

Plasma samples, collected from tubes containing heparin and meclofenamid, were assayed for PGI2 and TxA2 concentrations using a specific radioimmunoassay for their stable metabolites 6-keto PGF1 and thromboxane B2. To raise antibodies to these metabolites, the carbodiimide conjugate of either TxB2 or 6-keto PGF1, emulsified with complete Freund's adjuvant, was injected subcutaneously into the foot pads of rabbits. The antibody for TxB2 is used at a final dilution of 1:20,000, while that for 6-keto PGF1 is used at a final dilution of 1:5,000. The linear working range for both of these antibodies is between 20 and 2,500 pg of their specific eicosanoid. The cross-reactivities with other eicosanoids for both of these antibodies have been previously reported. Both radioimmunoassays are carried out at pH 7.3 in 0.1 mol/L Tris buffer containing 0.1% gelatin. After one hour at 20 °C, the incubation is terminated by adding dextran-coated charcoal and centrifuging the samples. Aliquots of the supernatant are dissolved in a suitable counting medium and counted to a 2.0% confidence limit in a liquid scintillation spectrometer (Beckman Model LS-230 Anaheim, Calif). Calculations of cpm, the ratio of binding constant counts to sample counts, and a linear regression equation for an 8-point standard curve are made by microcomputer. All samples are run in duplicate. Data for unknown samples are fit to the regression line by the RIA program, corrected for aliquot volume, and expressed as ng of prostanoid/mL of sample. The limits of detectability for both TxB2 and 6-keto-PGF1 were 0.20 ng/mL.

Plasma samples for C50 determinations were obtained from blood collected into NaEDTA tubes. The assay used was obtained from Upjohn Diagnostic (Kalamazoo, MI) and was based on the method of Chenoweth and Hugli.9 The linear working range was 8 to 200 ng/mL. All plasma samples were assayed in duplicate. Samples were kept at −27 °C till assayed. Duplicate values were within 6% ± 4% of each other. All data are expressed as mean ± SEM. Tests of significance utilized the paired or unpaired Student t test. A P value < 0.05 was considered to be significant.

RESULTS

Predialysis values for blood gases, leukocyte and platelet counts, minute ventilation, PGI2, and
DIALYSIS-INDUCED HYPOXEMIA 193

Table 1. Changes in Parameters During Acetate Hemodialysis Using Cuprophan Membrane

<table>
<thead>
<tr>
<th>Group 1A (n = 6)</th>
<th>Predialysis</th>
<th>Absolute Values</th>
<th>Change From Predialysis Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.37 ± 0.02</td>
<td>-0.02 ± 0.01</td>
<td>0.02 ± 0.02</td>
</tr>
<tr>
<td>P0₂ (mm Hg)</td>
<td>106 ± 5</td>
<td>-21 ± 4*</td>
<td>-20 ± 8*</td>
</tr>
<tr>
<td>PCO₂ (mm Hg)</td>
<td>31 ± 3</td>
<td>+2 ± 2</td>
<td>+2 ± 2</td>
</tr>
<tr>
<td>WBC (10³/mL)</td>
<td>5.7 ± 5</td>
<td>-2.8 ± 0.8*</td>
<td>+0.7 ± 0.8</td>
</tr>
<tr>
<td>Platelets (10³/mL)</td>
<td>205 ± 16</td>
<td>-8 ± 4*</td>
<td>-2 ± 5</td>
</tr>
<tr>
<td>Vₑ (mL/min/kg)</td>
<td>101 ± 8</td>
<td>-8 ± 10</td>
<td>+5 ± 10</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Group 1B (n = 4)</th>
<th>Predialysis</th>
<th>Absolute Values</th>
<th>Change From Predialysis Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.33 ± 0.01</td>
<td>-0.02 ± 0.01</td>
<td>+0.01 ± 0.01</td>
</tr>
<tr>
<td>P0₂ (mm Hg)</td>
<td>111 ± 5</td>
<td>-18 ± 3*</td>
<td>-20 ± 4*</td>
</tr>
<tr>
<td>PCO₂ (mm Hg)</td>
<td>31 ± 1</td>
<td>+1 ± 1</td>
<td>+1 ± 1</td>
</tr>
<tr>
<td>WBC (10³/mL)</td>
<td>5.1 ± 0.6</td>
<td>-2.5 ± 6*</td>
<td>-0.2 ± 0.7</td>
</tr>
<tr>
<td>TxB₂ (pg/mL)</td>
<td>1.7 ± 0.2</td>
<td>+0.45 ± 0.4</td>
<td>+1.49 ± 0.51</td>
</tr>
<tr>
<td>6-keto PGF₁₂ (pg/mL)</td>
<td>31 ± 10</td>
<td>+0.05 ± 0.05</td>
<td>-0.06 ± 0.06</td>
</tr>
</tbody>
</table>

All results ± SE.

*p < .05 by paired t test.

TxB₂ from all patients studied in Group 1 are shown in Table 1. The high starting P0₂ value (> 100 mm Hg) in these patients reflects their normal cardiopulmonary function and their normal hyperventilatory response to mild chronic metabolic acidosis.

The effects of acetate hemodialysis with a cuprophan dialyzer are depicted in Fig 1. Within 15 minutes from the onset of dialysis, there was a marked fall in P0₂ of 20 mm Hg in the initial 6 patients (Group 1A). P0₂ remained depressed throughout dialysis, returning to baseline by one hour posttreatment. PCO₂ did not change despite significant fluxes of CO₂ from patient to dialysate as demonstrated by the positive values for arteriovenous differences in CO₂ across the dialyzer. A significant decrease in white count occurred at 15 minutes, returning to baseline by one hour. The fall in white count resulted entirely from neutropenia. Platelet count also fell significantly at 15 minutes, returning to baseline at one hour. Only two of the 6 subjects had a fall in minute ventilation early in treatment (Table 2). Complement (C₅a), TxB₂, and PGI₂ determinations were made in four other patients (Group 1B). Changes in P0₂ and WBC were identical to those of the other six previously studied patients (Table 1). Ongoing complement activation, reflected as a step-up in C₅a across the dialyzer, occurred throughout dialysis (Fig 1). Despite this continuous complement activation, systemic C₅a levels never changed significantly from baseline. Arterial PGI₂ levels and TxB₂ did not change significantly during dialysis in the four patients studied (Table 1).

To separate the effects of solute and/or gas fluxes from those of blood membrane interaction, we studied changes in P0₂, WBC, C₅a, TxB₂, and PGI₂ during an initial 30 minute period of membrane contact alone and the subsequent 15 minute period of acetate dialysis (Group 2). Results utilizing a cuprophan dialyzer are shown in Fig 2. Six subjects were studied during each of three protocols in which the dialysis path of a cuprophan dialyzer was primed with air, acetate, or bicarbonate. The top panel shows that P0₂ did not change during membrane contact alone. There was, however, a marked leukopenic effect (comprised almost exclusively by neutropenia) by 15 minutes of membrane contact alone. Complement activation, reflected as significantly greater venous compared to arterial C₅a levels, was equivalent during membrane contact alone and at 15 minutes of acetate dialysis. Recovery of leukopenia and neutropenia began during membrane contact alone and continued during acetate dialysis. With the onset of acetate dialysis, there was a fall in P0₂, greatest with an acetate prime (18.6 mm Hg). This P0₂ fall in acetate prime patients was virtually identical to that seen during the conventional acetate dialysis protocol shown in Fig 1. In those patients whose dialyzers were primed with air or bicarbonate, the fall in P0₂ (-9.0 ± 1.4 and -7.0 ± 2.0 mm Hg, respectively) was significantly less than that observed with an acetate prime. Arterial TxB₂ levels did not change during membrane contact alone or...
during acetate dialysis, nor did levels of PGI₂ (Table 3).

Six patients subsequently were studied utilizing a polycrylonitrile dialyzer during 30 minutes of membrane contact alone followed by 15 minutes of acetate dialysis (Group 3). The dialysate path of the dialyzer was primed with acetate (5 patients), or air (5 patients). Results are depicted in Fig 3. Note that leukopenia did not develop nor did the percent neutrophils decrease during membrane contact alone, nor during subsequent acetate dialysis. There was no complement activation during
membrane contact alone. With the onset of acetate dialysis, there was slight activation of complement, as evidenced by a step-up in C₅a across the dialyzer. However, this arteriovenous increment in C₅a was markedly decreased compared to the results with a cuprophan membrane. Once again, Pₒ₂ did not change during membrane contact alone. At 15 minutes of acetate dialysis, a modest but significant fall in Pₒ₂ occurred in both the acetate and air prime groups. This change in Pₒ₂ was similar to that observed with a cuprophan membrane in the air and bicarbonate prime protocols, and was about half that observed with the cuprophan membrane and an acetate prime. Because there were no changes in TₓB₂ or PGI₂ during acetate/cuprophan dialysis (with or without prior membrane contact alone), these prostanoids were measured in the PAN/air prime, but not the PAN/acetate prime group. Furthermore, as mentioned above, Δ Pₒ₂ did not differ between the PAN/acetate prime and PAN/air prime groups. TₓB₂ and PGI₂ did not change during membrane contact alone or during acetate dialysis in the PAN/air prime group (Table 3).

**DISCUSSION**

There probably exist both membrane dependent and membrane independent components of dialysis associated hypoxemia.

There was a tendency in our patients for minute ventilation to fall at 15 minutes of dialysis (the time of maximum Pₒ₂ change). This change in minute ventilation probably failed to reach significance because of the small number of patients studied and marked interpatient variability resulting from differences in predialytic metabolic acidosis. Several observers, however, have demonstrated a fall in minute ventilation during acetate hemodialysis.³⁴.¹⁰⁻¹² These studies suggest that CO₂ transfer from patient to dialysate produces hypoventilation and hypoxemia, as respiratory chemoreceptors defend arterial Pco₂ levels. Our study demonstrated CO₂ transfer into dialysate. Oh et al has suggested that, rather than dialyzer
Table 3. Changes in Prostanoids During Initial Blood Membrane Contact and Following Acetate Dialysis

<table>
<thead>
<tr>
<th>Time of Measurement</th>
<th>Acetate</th>
<th>Air</th>
<th>HCO₃</th>
<th>Air</th>
</tr>
</thead>
<tbody>
<tr>
<td>T₄ₓB₂ Predialysis</td>
<td>0.61 ± 24</td>
<td>1.04 ± 33</td>
<td>0.58 ± 17</td>
<td>0.30 ± 04</td>
</tr>
<tr>
<td>15-min contact</td>
<td>0.52 ± 16</td>
<td>1.33 ± 61</td>
<td>0.63 ± 20</td>
<td>0.33 ± 05</td>
</tr>
<tr>
<td>30-min contact</td>
<td>0.47 ± 12</td>
<td>1.26 ± 49</td>
<td>0.51 ± 19</td>
<td>0.37 ± 09</td>
</tr>
<tr>
<td>15-min dialysis</td>
<td>0.63 ± 21</td>
<td>1.19 ± 51</td>
<td>0.49 ± 15</td>
<td>0.29 ± 04</td>
</tr>
<tr>
<td>6-keto-PGF₁ Predialysis</td>
<td>0.2</td>
<td>0.35 ± 10</td>
<td>0.22 ± 02</td>
<td>0.2</td>
</tr>
<tr>
<td>15-min contact</td>
<td>0.23 ± 02</td>
<td>0.53 ± 14</td>
<td>0.2</td>
<td>0.29 ± 08</td>
</tr>
<tr>
<td>30-min contact</td>
<td>0.21 ± 01</td>
<td>0.21 ± 01</td>
<td>0.22 ± 01</td>
<td>0.2</td>
</tr>
<tr>
<td>15-min dialysis</td>
<td>0.21 ± 01</td>
<td>0.39 ± 09</td>
<td>0.2</td>
<td>0.01</td>
</tr>
</tbody>
</table>

All results mean ± SE.

CO₂ loss, hypopnea and hypoxemia are a result of increased CO₂ consumption in the metabolism of acetate. In all likelihood, both mechanisms contribute to a membrane independent component of dialysis associated hypoxemia that can attenuated by substituting bicarbonate for acetate as a dialysate buffer. This membrane independent component of dialysis hypoxemia probably accounts for the modest fall in PO₂ (7 to 9 mm Hg) noted during acetate dialysis following a period of membrane contact alone with a cuprophan dialyzer, primed either with air or bicarbonate, and that seen with the PAN membrane (8 mm Hg) during dialysis regardless of the prime used during membrane contact alone.

The degree of hypoxemia during acetate hemodialysis was twice as great when the dialysate compartment of a cuprophan dialyzer contained acetate, as compared with air or bicarbonate, during the prior 30 minutes of membrane contact alone. This marked fall in PO₂ was virtually identical to that observed in two separate patient groups during conventional acetate/cuprophan hemodialysis. During conventional dialysis, the cuprophan membrane comes into contact with acetate prior to blood, a situation analogous to the acetate prime during membrane contact alone with cuprophan. Reused dialyzers are coated with deposits of various protein components present in blood. There would, then, be less interaction between cuprophan and acetate with a reused dialyzer. Some observers have noted less hypoxemia with reused cuprophan dialyzers. Thus, there appears to be a component of dialysis associated hypoxemia that is conditioned by an acetate-dependent interaction between blood and membrane. Others have suggested that this interaction produces a systemic effect that results in (1) ventilation-perfusion mismatching as evidenced by the increased alveolar-arterial oxygen tension difference during cuprophan dialysis, and (2) the rise in pulmonary artery pressure noted in sheep and in humans upon exposure to cuprophan. This systemic effect is as yet undefined. Craddock et al has demonstrated that exposure to cuprophan...
causes activation of complement by the alternative pathway, with resulting pulmonary vascular leukostasis. In the present study, however, there was no correlation between the degree of complement activation and the ultimate magnitude of hypoxemia following prior priming with air, acetate, or bicarbonate of a cuprophan dialyzer during membrane contact alone. Like Dumler and Levin, we were able to dissociate leukopenia from hypoxemia with sequential membrane exposure followed by hemodialysis. Furthermore, we observed that patients dialyzed with cuprophan following a period of dialyzer exposure to air or bicarbonate developed the same degree of leukopenia as those whose membranes were primed with acetate, but developed significantly less hypoxemia. One can then reasonably conclude that, while leukopenia and hypoxemia both result from cuprophan exposure, there is no cause and effect relationship between these events.

The degree of complement activation was underestimated by our assay since $C_{5a}$ rapidly binds to leukocytes, the assay thus measuring only free $C_{5a}$. Nevertheless, increases in dialyzer venous $C_{5a}$ were consistently found. While complement was activated by both the PAN and the cuprophan membrane, the degree of complement activation was much greater with cuprophan. Because cuprophan produced more hypoxia than did PAN, one might conclude that complement activation mediates the extent of dialysis induced hypoxemia. However, the extent of complement activation by cuprophan was the same, regardless of whether the dialyzer contained acetate, air, or bicarbonate during membrane contact alone. As mentioned above, acetate contact during membrane contact alone ultimately produced more hypoxemia during subsequent acetate dialysis. Furthermore, the degree of complement activation during membrane contact alone (when hypoxia did not occur) was the same as during subsequent acetate dialysis (when hypoxia was significant). Thus, hypoxemia does not appear to be directly dependent upon complement activation by the dialyzer membrane.

Wonders et al have demonstrated a rise in pulmonary artery pressure associated with an increase in TxB$_2$ during extracorporeal membrane oxygenation in sheep. We measured arterial TxB$_2$ and PGI$_2$ levels during acetate hemodialysis, and during membrane contact alone followed by dialysis with cuprophan and PAN dialyzers and found no change in either substance. The absence of increases in these substances may indicate one of two possibilities; either they do not participate in the induction of dialysis induced hypoxemia, or the degree of stimulation which occurs is too small to be measured in the arterial blood entering the dialyzer. Sampling the blood going into, or leaving, the lung may have detected some changes, but invasive studies were not justified in these subjects.

In summary, dialysis related hypoxemia appears to be multifactorial in origin. A portion of the decrease in $P_O_2$ is membrane dependent and may be due to the metabolic effects of acetate or to dialyzer $CO_2$ loss. The remaining portion of the $P_O_2$ decrease is membrane dependent, occurring with cuprophan, but not with PAN. This portion is conditioned by an acetate-dependent interaction between blood and membrane, but is not mediated by changes in leukocyte counts, nor by $C_{5a}$ activation within the dialyzer. We were unable to detect changes in systemic levels of TxB$_2$ and PGI$_2$. However, the possibility exists that changes in pulmonary function are mediated by changes in these substances too subtle for detection in systemic blood. They could perhaps be detected by sampling pulmonary venous blood, but invasive studies were not indicated in our patients.

REFERENCES

9. Chenoweth DE, Hagi TE: Techniques and significance of
C₃a and C₅a measurement, in Nakamura RM (ed): Future Perspectives in Clinical Laboratory Immunoassays. New York, Alan R. Liss Inc, 1980


14. Eiser AR, Jayamanne D, Koksong E, et al: Contrasting alteration in oxygen consumption (VO₂) and respiratory (RQ) during acetate (A) and bicarbonate (B) hemodialysis (HD). Kidney Int 19:145, 1981 (abstr)


