Acid Production in Chronic Hemodialysis Patients

JAIME URIBARRI,* MOHAMMAD ZIA,* JAFAR MAHMOOD,* ROY A. MARCUS,* and MAN S. OH†
*Departments of Medicine, Mount Sinai Medical Center, New York, New York, and †SUNY Health Science Center at Brooklyn, Brooklyn, New York.

Abstract. This study examines endogenous acid production in a group of stable chronic hemodialysis patients with no residual renal function who were admitted to the chronic hemodialysis unit of Mount Sinai Hospital. Net acid production was estimated by the change in body bicarbonate content, which occurs in the interdialytic period. The body bicarbonate content at any time was measured by multiplying the concentration of blood bicarbonate by the apparent bicarbonate space at that time. The apparent bicarbonate space was determined by the change in blood bicarbonate concentration induced by the intravenous administration of a known amount of sodium bicarbonate. Daily sulfuric acid production was also estimated from the reduction in body sulfate content during dialysis. The interdialytic net acid production was measured at approximately 28 mEq/d, a value much lower than that predicted from the protein catabolic rate. This reduced acid production can be explained by reduced sulfuric acid and organic acid production. The mechanism of reduced sulfuric acid production is unknown. Reduced organic acid production is explained partly by the absence of renal excretion of metabolizable organic anions, leaving only the nonmetabolizable fraction as the main source of acid in the interdialytic period. (J Am Soc Nephrol 9: 114–120, 1998)

Net endogenous acid production is measured as the sum of sulfuric acid and organic acids produced minus alkali absorbed from the gastrointestinal (GI) tract (net GI alkali absorption) (1). In healthy individuals, the production of sulfuric acid and organic acids is measured from urinary excretion of sulfate and organic anions (2). Traditionally, net GI alkali absorption has been measured by the analysis of food and stool noncombustible cations and anions (1), but the method is so highly tedious and technically demanding that it is rarely used even as a research tool. An equally accurate but much simpler method is now available, and it is based on the excretion of urinary noncombustible cations and anions (3).

Hence, all of the methods needed for the measurement of the various parameters of acid production are based on measurements in urine samples, which obviously cannot be done in dialysis patients with reduced urinary output. Consequently, there have been few data on the measurement of endogenous acid production in this population, and the available data are inadequate (4).

Realizing that the urinary data cannot be obtained in dialysis patients, some authors have attempted to estimate net acid production from the protein catabolic rate (PCR) (5). However, this method would not be suitable for dialysis patients for several reasons.

First, the method was developed with data obtained in subjects with normal renal function (5). Because we are trying to determine whether acid production in dialysis patients differs from healthy subjects, the measurement of acid production using PCR would not shed any light on the question. In fact, there is a theoretical reason to believe that patients on dialysis are likely to have lower endogenous acid production, in part because of reduced organic acid production. Urinary organic anions whose excretion rate represents the rate of organic acid production consist of nonmetabolizable and metabolizable anions (4). The production of nonmetabolizable organic anions, such as urate, would represent acid production regardless of whether they are excreted in urine or retained in the body. In contrast, production of metabolizable organic anions, such as citrate, represents acid production only when they are excreted. When they are retained in the body fluid in anuric patients, they would be metabolized back to bicarbonate and have no effect on acid generation.

Second, even in a healthy population, a positive correlation between PCR and net endogenous acid production rate might be largely fortuitous. Protein as the source of the sulfur-containing amino acids might be a reasonable indicator of the rate of sulfuric acid production (6), but there is no theoretical basis for considering protein intake as an indicator of the rate of organic acid production or net GI alkali absorption.

Third, the uremic state in a dialysis patient might affect endogenous acid production. For instance, metabolism of sulfur-containing amino acids results in acid production only if the final metabolite is inorganic sulfate (7). If sulfur is eliminated from the body in some other form, such as thios or organic conjugates, it does not result in acid production (4). Metabolic alterations in uremia might alter sulfur metabolism and thereby alter the contribution of sulfur-containing amino acids to acid production. Similarly, although the absence of renal function might reduce the rate of organic acid production...
by preventing urinary excretion of metabolizable organic anions, altered metabolism in uremia might lead to increased production of nonmetabolizable organic acids. For these reasons, direct measurement is the only accurate way to determine the rate of net endogenous acid production in dialysis patients.

Previously (8), we measured acid production in patients on continuous ambulatory peritoneal dialysis (CAPD). Instead of normal urine flow, these patients excrete approximately 10 L of dialysate per day, and the measurement of sulfate and organic anions and noncombustible cations and anions in the dialysate enabled us to determine net endogenous acid production. We have shown that endogenous acid production was markedly diminished for a given PCR (8).

In this study, we wish to measure endogenous acid production in patients on maintenance hemodialysis. However, there is a technical problem that arises from the large volume of dialysate that is routinely used during hemodialysis, usually in excess of 100 L per dialysis. Because of the large volume of dialysate, the concentrations of sulfate and organic anions are very low, and a minor random error in measurement would result in a large error in the final result. For this reason, we have decided to measure net acid production by a different approach.

First, sulfuric acid production was measured by a reduction in the body sulfate content during dialysis, calculated as the difference between the body sulfate content before dialysis and that immediately after dialysis. The amount of organic acid production and net GI alkali absorption was not measured directly. Instead, we measured net acid production between dialysis by the change in body content of bicarbonate and assumed that net acid production between dialysis is equal to net bicarbonate gain during dialysis. Because predialysis serum bicarbonate concentration remained stable during the study period, the above assumption is probably valid. Thus, the difference between net bicarbonate gain during dialysis and sulfate loss during dialysis is considered equal to organic acid production minus net GI alkali absorption.

Materials and Methods

Patients

Fifteen adult hemodialysis patients at Mount Sinai Hospital participated in the study. All patients were stable and consuming ad libitum diets, and had a urinary output less than 50 mL/d. All patients had been previously dialyzed three times a week with 35 mEq/L bicarbonate dialysate for at least 4 mo before the study.

All of the studies were performed during the course of the patients regular hemodialysis treatment program without any changes in the type of dialyzer (Fresenius–8), blood flow (250 to 400 mL/min), dialysate flow (500 mL/min), ultrafiltration rate, or dialysis prescription made for the study.

Pre- and postdialysis plasma bicarbonate levels were monitored for four consecutive treatments. During one of the dialysis sessions, the arterial line was left in place at the end of dialysis, and a sample of blood was obtained 30 min later. Six patients agreed to return the day after dialysis to obtain a blood sample for measurement of bicarbonate concentration. Ten patients consented for measurement of the volume of distribution of bicarbonate, which was always done at the end of the study (9,10). The study protocol was approved by the Institutional Review Board at Mount Sinai Hospital.

Methods

Blood samples for blood gas determination were obtained anaerobically from the arterial end of the vascular access and analyzed within 5 min with a blood gas analyzer. Plasma bicarbonate concentration was calculated from pH and Pco2, using the Henderson–Hasselbalch equation.

For measurement of the volume of distribution of bicarbonate, patients were asked to come to the dialysis unit 2 h before their regular time, and the arterial segment of their vascular access was cannulated at that time. A solution of sodium bicarbonate (2 mEq/kg) was infused over 10 min via a pump. Blood was obtained at baseline and then at 15, 30, 60, and 100 min after finishing the intravenous infusion to measure the change in bicarbonate concentration. Usual hemodialysis was started as soon as the 100-min sample of blood was obtained. Blood was also obtained at the beginning and immediately at the end of this dialysis session for measurement of urea to calculate PCR and for measurement of sulfate to calculate dialysate sulfate removal.

The following calculations were made:

1. Apparent volume of distribution of bicarbonate or apparent bicarbonate space (ABS). ABS (liters) = administered HCO3/ΔHCO3, where administered HCO3 is the amount of bicarbonate administered in milliequivalents, and ΔHCO3 is the difference between bicarbonate concentration at baseline and the average concentration at 60 and 100 min after bicarbonate infusion (see Results).
2. Body content of bicarbonate was calculated by multiplying the concentration of blood bicarbonate by the ABS. Predialysis ABS was calculated as 53.2% of the predialysis weight expressed in kilograms (see Results). Postdialysis ABS was calculated from the predialysis volume of distribution minus the weight loss during dialysis expressed in kilograms.
3. Interdialytic net acid production was calculated from the difference between the body content of bicarbonate at the end of one dialysis session and at the beginning of the next dialysis session. Separate calculations were made for 2- and 3-d interdialytic intervals.
4. Daily net acid production = Interdialytic net acid production divided by 2 or 3 depending on the number of days in between dialysis.
5. Body content of sulfate was calculated by multiplying the concentration of serum sulfate by the space of distribution of sulfate (SDS). The SDS was assumed to equal the extracellular space, which in turn was calculated as 16% of the dry body weight (11). Postdialysis SDS was calculated as 16% of the body weight at the end of dialysis. Predialysis SDS was calculated from the postdialysis space of distribution plus the weight loss during dialysis expressed in kilograms.
6. Dialytic sulfate removal was calculated from the difference between the body sulfate content at the beginning and at the end of a dialysis session. This value should be identical to interdialytic sulfate production in stable chronic patients.
7. PCR was calculated by a computer program from the pre- and postdialysis concentrations of urea nitrogen (12). Normalized PCR (nPCR) was calculated by dividing PCR by the body weight in kilograms.

In a second phase, 10 patients were studied during the course of a regular dialysis session at the end of a 3-d interval, when blood samples were obtained at the beginning and end of dialysis from the arterial line and every hour during the treatment from both arterial and
venous lines. The net dialysis bicarbonate gain was calculated from the difference between the body content of bicarbonate at the beginning and at the end of the dialysis session. The bicarbonate transfer rate (BTR) into the patient across the dialyzer was calculated as follows: BTR (mEq/min) = HCO₃⁻ content in the dialyzer inlet − HCO₃⁻ content in the dialyzer outlet (see Appendix).

Total CO₂ and blood urea nitrogen (BUN) were measured in the routine hospital laboratory. Sulfate was measured by a precipitation method (13).

**Statistical Analyses**

Significant differences between the means were calculated by t test. Correlations between values were assessed by linear regression analysis. Statistically significant difference was defined as \( P < 0.05 \). All values were expressed as mean ± SEM.

**Results**

Fifteen patients participated in the study. Their ages ranged from 35 to 68 (48 ± 3 yr, mean ± SEM), and the mean time since the start of hemodialysis was 6 yr, with a range of 0.4 to 25 yr. There were eight men and seven women in this group; there were seven blacks, three Hispanics, three whites, and two Asians. Renal diagnosis revealed nephrosclerosis (5), diabetic nephropathy (4), chronic glomerulonephritis (2), HIV nephropathy (1), vasculitis (1), and idiopathic disease (2). All were dialyzed 3 times a week.

All of the patients were ingesting oral calcium carbonate or calcium acetate as phosphate binders; the dosage of calcium carbonate (14 patients) ranged from 1.95 to 5.85 g daily (3.1 ± 0.4 g, mean ± SEM), and one patient was ingesting calcium acetate at a dose of 4 g/d. Five patients were receiving oral calcitriol (0.29 ± 0.01 µg/d, mean ± SEM), and five were receiving intravenous calcitriol at a dose of 1 µg during each dialysis treatment. The doses of phosphate binders and calcitriol did not change during the duration of the study.

The dialysate bicarbonate concentration was 36 ± 0.5 mEq/L (mean ± SEM), which, added to the 4 mEq of acetate, will give a total alkali load of 40 mEq/L.

After the intravenous infusion of bicarbonate, the plasma bicarbonate concentration was the highest after 15 min and then gradually decreased, reaching a stable value by 60 min. The actual bicarbonate concentrations were 23.6 ± 1.4, 29.5 ± 1.5, 28.7 ± 1.7, 28 ± 1.8, and 27.8 ± 1.9 mEq/L (mean ± SEM) at baseline and 15, 30, 60, and 100 min after bicarbonate infusion, respectively. The average value of the concentration of bicarbonate at 60 and 100 min was compared with baseline to calculate the apparent volume of distribution of bicarbonate. The apparent bicarbonate space was 35.6 ± 3 L (mean ± SEM) and represented 53.2% of the body weight expressed in kilograms. The following blood parameters were also compared between baseline and the average at 60 and 100 min: \( \text{pH} = 7.38 \) to 7.42, anion gap = 13.2 ± 1 to 12 ± 1 mEq/L, and \( \text{Pco}_2 = 39.2 ± 2.5 \) to 41.9 ± 2.4 mmHg; only the difference between the \( \text{Pco}_2 \) values reached statistical significance (\( P < 0.001 \)).

Table 1 shows the results of selected parameters in our group of patients. Most of the values are the usual ones observed in patients on maintenance hemodialysis. The Kt/V of 1.4 per dialysis and the nPCR of 1.13 g/kg per d are clearly within the recommended values for hemodialysis patients. The mean predialysis serum total carbon dioxide was 23.3 ± 0.6 (mean ± SEM), with values ranging from 19 to 27.8 mEq/L. Predialysis serum albumin and BUN values were very stable for the 4 consecutive months preceding the study (serum albumin: 4.1 ± 0.2, 4.08 ± 0.1, 4.1 ± 0.1, and 4.05 ± 0.1 mg/dl [mean ± SEM]; BUN: 62 ± 5, 61 ± 4, 65 ± 5, and 68 ± 5 mg/dl [mean ± SEM] at 4, 3, 2, and 1 mo before the study, respectively).

Figure 1 shows that there was no statistically significant difference among the values of pre- and postdialysis total carbon dioxide for the 5 consecutive months before the study. These values were obtained from the routine serum chemistries obtained monthly for the calculation of PCR.

The predialysis plasma bicarbonate concentration was smaller after the 3-d interdialytic period than after the 2-d interdialytic period, but the difference did not reach statistical significance (21.5 ± 0.2 versus 22.9 ± 0.2 mEq/L). The

<table>
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*All values are expressed as mean ± SEM and represent the average of four monthly consecutive tests before our study. BUN, blood urea nitrogen; nPCR, normalized PCR.

![Figure 1](https://example.com/figure1.jpg)

*Figure 1. Serum total CO₂ for 5 mo before study.*
postdialysis plasma bicarbonate concentration was virtually identical during both periods (26.5 ± 0.2 versus 26.6 ± 0.3 mEq/L). There was no significant difference between the plasma bicarbonate concentration measured immediately at the end of dialysis (26.7 ± 0.2 mEq/L) and that measured 30 min later (26.5 ± 0.2 mEq/L). Also, we found that the rate of titration of bicarbonate during the first 24 h after dialysis (plasma bicarbonate concentration 24 h after dialysis was measured in only six patients) was not different from that obtained dividing the total bicarbonate titrated during the interdialytic period by 2.

Table 2 shows the estimated daily acid production based on the change in body bicarbonate content in the 2-d interdialytic period. The estimated daily acid production of 28 ± 9 mEq/d is much lower than 61 mEq/d, the value predicted from PCR. (The assumption is that net endogenous daily acid production can be calculated from PCR by the formula: Daily acid production = PCR × 0.77 = 79 × 0.77 = 61 mEq/d.) Not shown in this table is the net daily acid production, which was calculated from the 3-d interdialytic period and was very similar at 31 ± 9 mEq/d. The predialysis body content of sulfate was 58 ± 8 mEq/L, the postdialysis value was 12 ± 3, and the dialytic removal rate was 46 ± 8 mEq/L. Because this measurement of dialytic sulfate removal was performed in the middle of the week, the daily sulfate appearance rate was 23 ± 4 mEq/d (all values are mean ± SEM).

In a separate group of 10 patients, the transfer or flux of bicarbonate across the dialyzer, calculated from the difference in bicarbonate content between the arterial and venous ends, was 48 mEq/dialysis, whereas the net dialysis bicarbonate gain, calculated from the difference in body content of bicarbonate at the beginning and end of the same dialysis session, was 96 mEq/dialysis. This last group of experiments was done at the end of a 3-d interdialytic period.

There was no correlation between the baseline plasma bicarbonate concentration and the apparent volume of distribution of bicarbonate. The change in bicarbonate concentration during dialysis correlated well with the predialysis plasma bicarbonate concentration (r = −0.82, P = 0.0006), but not with nPCR (r = 0.3, P = 0.2) or dialysate bicarbonate concentration (r = 0.2, P = 0.2). As shown in Figure 2, there was some correlation between predialysis serum total CO₂ concentration and nPCR (r = −0.5, P = 0.04). There was a good correlation between daily sulfate production and nPCR (r = 0.6, P = 0.04).

Discussion

Net daily acid production, estimated by the changes in body bicarbonate content in the interdialytic period, was 28 ± 9 mEq/d in our group of stable hemodialysis patients. This value is much less than the amount in healthy subjects, approximately 60 mEq/d. There are two possible explanations for the low net acid production in our patients: low net sulfuric acid production and low net organic acid production.

Our subjects had an estimated daily protein intake of 79 g calculated from the PCR (5). On the basis of the information available for healthy subjects on a mixed-protein diet (6), one can predict a sulfate appearance rate of approximately 39

### Table 2. Daily net acid and sulfate production rates in hemodialysis patients

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<th>Pre BIC</th>
<th>Post BIC cont.</th>
<th>Pre BIC cont.</th>
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mean ± SEM 64.6 ± 4 67.2 ± 4 27 ± 0.5 23.5 ± 0.7 902 ± 66 846 ± 68 28 ± 9 58 ± 8 12 ± 3 23 ± 4 1.18 ± 0.01

* Post HDW, postdialysis weight in kilograms; Pre HDW, predialysis weight in kilograms; Post BIC, postdialysis bicarbonate concentration in mEq/L; Pre BIC, predialysis bicarbonate concentration in mEq/L; Post BIC cont., postdialysis body bicarbonate content in mEq; Pre BIC cont., predialysis body bicarbonate content in mEq; Daily Acid, daily net acid production in mEq/d obtained by dividing the interdialytic change in body bicarbonate content by 2; Pre HDS, predialysis body sulfate content in mEq; Post HDS, postdialysis body sulfate content in mEq; Daily Sulfate, daily sulfate production in mEq/d obtained by dividing the intradialytic sulfate removal by 2; nPCR, normalized protein catabolic rate in g/kg per d; ND, not determined. All values are expressed as mean ± SEM.
mEq/d. The actual sulfate appearance rate measured in our study was only 23 mEq/d. However, this appearance rate was estimated by assuming that dialysis patients have an SDS equivalent to healthy subjects, i.e., 16% of body weight. Additional studies with actual measurement of SDS in dialysis patients will be necessary to confirm this impression of a sulfate deficit. A sulfate deficit, however, has been described previously in both CAPD (8,13) and hemodialysis patients (14), and the cause is unknown, although postulated explanations have included increased loss of sulfate in the stool and loss of sulfur in some other forms, such as taurine, phenol sulfate, etc.

As predicted, organic acid production was also reduced in hemodialysis patients. Normally, two types of organic anions are excreted in the urine: metabolizable and nonmetabolizable (4). An example of the former is citrate and an example of the latter, urate. In the absence of renal function, the net production of nonmetabolizable organic acids probably continues at the same and constant rate, resulting in consumption of body alkali buffers and retention of organic anions during the interdialytic period. On the other hand, metabolizable organic anions would not accumulate, but instead will be converted back to bicarbonate without any significant increase in their plasma level and without any contribution to net acid production. Because net production of metabolizable organic anions ceases in patients with no residual function, total organic acid production will decrease in these patients. Our findings are consistent with this prediction.

Estimation of net GI alkali absorption requires measurement of noncombustible electrolytes (Na, K, Ca, Mg, P, and Cl) in food and stool (1) or in urine samples (3). The urine method could not be undertaken because the patients were anuric. The food and stool electrolyte method was not done in the current study because this method requires a special diet and admission of patients to a clinical research center. However, we have previously calculated this parameter in a group of stable CAPD patients by a simplified method based on the measurement of noncombustible electrolytes in urine and peritoneal dialysis effluent. The average net GI alkali absorption was 23 mEq/d in that study (8), and we assume similar values in hemodialysis patients. If we assume a net GI alkali absorption of 23 mEq/d in our patients, this value would negate a sulfate production of 23 mEq/d, and the net acid production of 28 mEq/d found in the current study can be explained entirely by the daily production of noncombustible organic anions.

In healthy people, urinary organic anion loss appears to be the main component of endogenous acid production (15). The number of organic anions is very large and most of them are excreted in minute amounts, making it virtually impossible to measure all of them individually (16). Thus, it would seem that, at this moment, the only available method to measure the total organic anion output would be a titration technique (4). The amount of organic anions lost during hemodialysis has been estimated at 101 mEq/session by Vreman et al. (17) and at 31 mEq/session for bicarbonate and 37 mEq/session for acetate hemodialysis by Gotch et al. (5). However, in both of these studies only a few organic anions were measured, and hence they cannot be trusted as the source of the information on the rate of organic anion loss during hemodialysis.

Previous measurements of plasma concentration of lactate and citrate (18) have shown that these levels do not change between the beginning and end of dialysis, suggesting an ongoing production of organic anions during hemodialysis. The alkalinization that takes place during hemodialysis may stimulate the production of these organic acids (19). Nevertheless, serum bicarbonate concentration rose during dialysis because bicarbonate gained during dialysis exceeded the amount titrated by these organic acids. The increase in body bicarbon-
Acid Production in Hemodialysis Patients

HCO₃⁻ content during dialysis (96 mEq/dialysis) was greater than the actual input of bicarbonate through the dialyzer (48 mEq/dialysis), suggesting a second source of bicarbonate, most likely the result of the net gain of organic anions during the treatment session. Current concentration of acetate in bicarbonate dialysate is the result of the addition of acetic acid for the purpose of decreasing the pH of the bicarbonate solution; however, even these low concentrations of acetate seem to have a significant effect as a source of alkali during hemodialysis treatment.

Direct measurement of organic anion excretion rates during hemodialysis would be more accurate, but this is hampered by the large output of dialysate. For example, a total excretion of 100 mEq of organic anions during each dialysis will be dissolved in 200 L of dialysate; this will produce an average dialysate concentration of 0.5 mEq/L of organic anions. An error of measurement of ± 0.2 mEq/L, which is not unlikely with our current analytical techniques, would lead to an under- or overestimation of organic anion excretion of as much as 40 mEq.

Two other observations in this study are also of interest. First, the average predialysis serum total CO₂ of 23.3 mEq/L is much better than values observed in the past with acetate hemodialysis, but still below the levels observed in CAPD patients (8). Second, there was some correlation between PCR and average predialysis serum total CO₂. The current study does not allow us to determine the primary event: either high protein intake leading to increased acid production or acidosis leading to increased protein breakdown.

In conclusion, we have estimated net acid production in a group of chronic hemodialysis patients by measuring the decline in body bicarbonate content in the interdialytic period. This value was much lower than predicted, because net acid production is much lower than in healthy individuals with similar amounts of PCR. The reduced net endogenous acid production can be explained by reduced sulfuric acid and organic acid production. The mechanism of reduced sulfuric acid production is not known. Reduced organic acid production can be explained partly by the absence of renal excretion of metabolizable organic anions; among organic acids, only non-metabolizable fractions contribute to acid production.

Appendix

BTR (mEq/min) = HCO₃⁻ content into the dialyzer - HCO₃⁻ content leaving the dialyzer into the patient.

HCO₃⁻ content into the dialyzer was calculated as (Qₚ × \[\text{[HCO}_3\text{]}\]) + (Qₒ × 0.69 × 0.72[HCO₃⁻]), where Qₚ is inlet plasma flow (in L/min and calculated by Total blood flow - Red blood cell flow), [HCO₃⁻] is inlet plasma bicarbonate concentration (mEq/L), Qₒ is Inlet red cell blood flow (calculated by multiplying total blood flow by hematocrit), 0.69 is the ratio of bicarbonate concentration in red cells to that in plasma as the result of Donnan equilibrium (5), and 0.72 is red cell water fraction (5).

HCO₃⁻ content leaving the dialyzer was calculated as (Qₒ × \[\text{[HCO}_3\text{]}\]) + (Qₚ × 0.69 × 0.72[HCO₃⁻]), where Qₒ is outlet plasma flow (calculated as Inlet plasma flow - Ultrafiltration rate, both in L/min), Qₚ is outlet red cell flow (in L/min and assumed to be the same as inlet red cell flow), and [HCO₃⁻] is outlet plasma bicarbonate concentration, which was calculated as measured outlet bicarbonate concentration minus the change in bicarbonate concentration due to buffering of CO₂ by blood. Buffering of CO₂ by blood was determined empirically by measurement of the in vitro titration curve of CO₂ in 13 blood samples from chronic hemodialysis patients (20).

The average values were:

Dialysis duration = 194 min
Inlet total blood flow = 0.35 L/min
Ultrafiltration rate = 0.01144 L/min
Inlet hematocrit = 38.8%
Inlet HCO₃⁻ concentration = 23.9 mEq/L
Outlet HCO₃⁻ concentration = 28.22 mEq/L

Change in blood pH between inlet and outlet from dialyzer = -0.087. (According to our in vitro titration curve of CO₂ by blood, this change in pH produced a change in HCO₃⁻ concentration of 2.4 mEq/L. Thus, the calculated outlet plasma HCO₃⁻ concentration was 25.82 mEq/L.)

Inlet HCO₃⁻ content = 6.7318 mEq/min
Outlet HCO₃⁻ content = 6.9772 mEq/min
ΔHCO₃⁻ content = 0.245 mEq/min = 48 mEq/session

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References

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