Effects of Controlled Blood Cooling on Hemodynamic Stability and Urea Kinetics during High-Efficiency Hemodialysis

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Abstract. Although the use of cooled dialysate during hemodialysis is associated with stabilization of intradialytic BP, the effects of blood cooling on hemodynamics and urea kinetics in high-efficiency hemodialysis have not been completely studied. In particular, the effects of blood cooling have not been elucidated in very short-time, high K/V dialysis treatments, in which postdialysis urea rebound is maximized. In theory, blood cooling could increase urea compartmentalization during treatment and decrease dialysis efficacy. Measurements of cardiovascular hemodynamics and urea kinetics were performed in 15 patients (56 studies) during dialysis, using a blood temperature monitor with control of dialysate temperature. Dialysate temperature was adjusted to either lower the core temperature or raise the core temperature by, respectively, producing negative heat-energy exchange (cooled dialysis) or keeping heat-energy exchange in the extracorporeal circuit neutral (thermoneutral dialysis) so that energy was not transferred to or from the patient. Each subject was studied on both protocols, thereby allowing each individual to act as his own control. In cooled dialysis, heat-energy exchange in the extracorporeal circuit averaged $5 \pm 31$ kJ per treatment, and dialysate temperature averaged $37.1 \pm 0.02^\circ C$. Dialysate cooling resulted in a reduction in mean body temperature compared with thermoneural therapy ($-0.22 \pm 0.04$ versus $+0.31 \pm 0.05^\circ C$). Cooling resulted in a greater increase in peripheral vascular resistance index ($+515 \pm 160$ versus $+114 \pm 92$ dyn.sec/cm$^5$ per m$^2$), an increase in mean arterial pressure ($+4 \pm 3$ versus $-4 \pm 4$ mmHg), a reduction in the maximum intradialytic fall in mean arterial pressure ($-10 \pm 2$ versus $-18 \pm 3$, mmHg), and a reduction in staff interventions for hypotension or dialytic symptoms (6 of 28 versus 12 of 28 studies). These differences occurred without differences in the change in blood volume ($-14.3 \pm 1.8\%$ versus $-13.9 \pm 2.2\%$) or cardiac index ($-0.4 \pm 0.1$ versus $-0.4 \pm 0.2$, L/min per m$^2$). Urea rebound (37 \% versus 38 \% and effective $K_t/V$ (1.29 \% versus 1.32 \%) were not different between groups. Thus, body temperature cooling can be used to stabilize BP and reduce intradialytic events requiring staff intervention without compromising the efficacy of treatment in high-efficiency dialysis. (J Am Soc Nephrol 9: 877–883, 1998)

Advances in dialyzer design have resulted in more efficient dialyzers that permit delivery of an adequate dose of hemodialysis in a shorter period of time. More rapid hemodialysis, however, poses problems with hemodynamic stability as time available for removal of interdialytic fluid gain is concomitantly reduced. The development of volumetric fluid removal devices and of bicarbonate dialysate has made highly efficient, short-time hemodialysis a widely applicable therapy (1). However, episodes of symptomatic hypotension remain a problem in a significant minority of patients (2,3).

The beneficial role of cooled dialysate in promoting hemodynamic stability has been described previously for conventional (4) and high-efficiency hemodialysis treatment (5). However, the relationship of dialysate cooling to actual changes in body temperature has not been evaluated systematically. In addition, the potentially adverse effects of cooling-induced vasoconstriction on solute disequilibrium have not been fully explored. Multicompartment models have been developed to explain intradialytic disequilibrium and posthemodialysis urea rebound (6,7). In these models, differences in regional blood flow during hemodialysis (such as in skin and muscle) can predict posthemodialysis urea rebound (8). Compartmental disequilibrium, enhanced by high dialyzer clearance-to-body water ratios ($K_t/V$), is more likely to be significant in high-efficiency hemodialysis treatments (9–12). Blood cooling, although helpful in improving hemodynamic stability, could, in theory, produce a thermally induced decrease in
regional blood flow and further enhance compartmental disequilibrium (13). In addition, rapid fluid removal with very short-time dialysis could also enhance compartmental disequilibrium. Volume removal during hemodialysis can result in vasoconstriction of the skin and possibly other organs (14). Ultrafiltration-induced changes in blood volume during hemodialysis can produce episodes of hypotension (15), which may further increase compartmental disequilibrium. Thus, under the conditions of blood cooling during high K/V, short-time dialysis with rapid rates of ultrafiltration, posthemodialysis urea rebound would be expected to be maximized.

Warming of an extremity has decreased posthemodialysis urea rebound from the limb, suggesting temperature-dependent changes in compartment disequilibrium (16). However, Yu et al., using moderate-efficiency hemodialysis, did not demonstrate a significant change in posthemodialysis urea rebound or K/U after blood cooling, as evidenced by a fall in tympanic membrane temperature (17). However, in the study by Yu et al., dialysis was performed with a K/V 5.38 ml/min per L and a treatment time of 4 h, conditions that would not maximize postdialysis rebound.

The present study compares the effects of changes in body temperature and heat–energy exchange in the extracorporeal circuit on BP and posthemodialysis urea rebound at comparable rates of ultrafiltration and blood volume changes. The study was designed to determine whether hemodynamic stability achieved by body temperature cooling during rapid hemodialysis adversely affects solute clearance.

Materials and Methods

A total of 56 studies was performed in 15 patients receiving high-efficiency dialysis, using F80 dialyzers with a dialysate flow rate (Qs) of 800 ml/min, extracorporeal blood flow rate (Qh) > 400 ml/min, and equilibrated K/U > 1.0. The subjects did not consume food during treatment. Dialysate was bicarbonate-based with a sodium concentration of 142 mEq/L. Dialysate and blood flow rates and pre- and post-pump arterial line pressures were measured continuously on-line and recorded by a computerized data collection system that provided time-averaged values over a 5-min period (FDS08, Fresenius USA, Walnut Creek, CA). Fluid removal was constant throughout each dialysis treatment using a dialysis delivery machine equipped with a volumetric ultrafiltration control system (2008E, Fresenius USA). Weights were taken standing, pre- (Wt0, kg) and postdialysis (Wt, kg).

Studies were performed under conditions in which heat was removed to achieve controlled body temperature cooling (cooled, n = 28), or the heat–energy exchange in the extracorporeal circuit was kept neutral so that no energy was transferred to or from the patient (thermoneutral, n = 28). In the cooled studies, heat–energy exchange in the extracorporeal circuit was negative by several hundred kilocalories per minute (C±30).

Heat–energy exchange in the extracorporeal circuit (extracorporeal thermal balance [ThBal], kJ) was determined by a blood temperature monitor system (BTM) integrated into the dialysis machine (Fresenius AG, Bad Homburg, Germany). The BTM calculates both the arterial (Tsa, °C) and venous (Tsv, °C) fistula temperature from the measured tube temperature using sensor heads accurate to within <0.1°C and allows for control of body temperature by varying dialysate temperature. The temperature loss between the sensor heads and the fistula generally depends on blood and room temperature, air movement, flow rate, tube length, and the tube wall dimensions and material. A correction function derived from physical principles is used, which is valid for a length of 1.5 m between needle and sensor head and standard polyvinyl chloride tubing with an outer diameter of 6.5 mm and a wall thickness of 1 mm. The room temperature is assumed to be 23°C at low air movement. The blood flow rate is known to the BTM because of the data link to the dialysis machine.

The error in calculation of the temperature drop is <0.1°C if the room temperature is between 20 and 26°C at blood flow 250 ml/min and between 17 and 29°C at blood flow 450 ml/min. Underlying assumptions include low air movement and an initial core temperature of 37°C. Body temperature is determined from the blood appearing in the arterial line, herein defined as core temperature (Tc, °C). The blood appearing in the arterial line is a mixture of blood that has passed through the capillary systems of the body (thermally equilibrated with the organ passed) and recirculated blood. Therefore, the basis of the calculation of core temperature is an equation that describes the mixing of two quantities of blood: Tc = (1 - R) × Ta + R × Tsv, where Ta and Tsv are arterial and venous fistula temperature, respectively; R is total recirculation (access and cardiopulmonary); and Tsv is core temperature. The fraction (1 - R) of blood with body temperature Tc is mixed with the fraction R of recirculated blood with temperature Tsv. Rearranging leads to the equation for calculating core temperature from the measured data: Tc = Ta × R + (Tsv - Ta) × R/(1 - R). Recirculation (R) was determined at least once during each treatment by a thermal method used by the BTM (19). Heat–energy exchange in the extracorporeal circuit was determined as follows: ThBal = \( JQ(t) \times dt \), where JQ (thermal flux) = c × ρ × Qh × \( (Tsa - Tsv) \), and where c is heat capacity of blood, 3.93 J/K × kg, with K = Kelvin = absolute temperature = 273.15 + °C, ρ is density of blood, 1050 kg/m³ at 37°C, Qh is extracorporeal blood flow in ml/min, and Tsa and Tsv are as defined previously. Representative cooled and thermoneutral dialysis profiles are depicted in Figure 1.

Blood volume changes were determined in 12 patients (40 studies) by on-line measurement of hematocrit (In-line Diagnostics, Ogden, UT). Blood volume changes were calculated using the change in hematocrit relative to a baseline hematocrit value determined during the first 3 min of the dialysis treatment. The percent change in blood volume at any time x during treatment (ΔBV) is defined by the relationship: ΔBV = (Hct(x)/Hct0) - 1, where Hct is initial, baseline hematocrit, %, and Hct0 is hematocrit at time x during treatment, %. The accuracy of the method is ±2% (20).

Mean arterial pressure (MAP, mmHg) was measured with an automated device (BPS08, Fresenius USA). Pressures were taken in the arm with the patient sitting in a lounge chair with feet parallel to the floor. The immediate predialysis pressure was taken after a 15-min period of equilibration and subsequent readings were taken every 30 min unless the clinical situation warranted more frequent measurements. Cardiac index (CI, L/min per m²) was derived from measurements of cardiac output, using an on-line bioimpedance method (CardioDynamics International, Irvine, CA) (21). Systemic vascular resistance index (SVRI, dyn.sec.cm²/m²) was determined from the mean arterial pressure and cardiac index. Skin temperature on the hand of the non-access arm was determined every 15 min by an infrared method (22).

Blood urea nitrogen (BUN) measurements for kinetic analysis were determined pretreatment (C0), at the immediate end of dialysis (C1), and at 30 min (C1.30) posttreatment. BUN was measured in triplicate.
Figure 1. Representative depictions of cooled dialysis (top panel) and thermoneutral dialysis (bottom panel). Time of dialysis (x axis) is in minutes, and arterial fistula line temperature, $T_a$ (bold solid line), venous fistula line temperature, $T_v$ (solid line), and dialysate temperature, $T_d$ (dashed line) are in °C. The periodic and transient large reductions in dialysate temperature and the subsequent reductions in venous and arterial fistula line blood temperature represent a recirculation measurement. Heat–energy exchange in the extracorporeal circuit was $-217$ kJ in the cooled dialysis study and $71$ kJ in the thermoneutral study.

by a standard urease method (BUN analyzer 2, Beckman Instruments, Fullerton, CA). The blood sample for determination of $C_0$ was obtained at the time of initial cannulation of the access. The blood sample for determination of $C_1$ was obtained from the arterial line port 20 s after the extracorporeal blood flow rate was reduced to 50 ml/min at the immediate end of treatment. Blood for $C_{+30}$ was obtained from the access needle after discarding the first 10 ml of blood sample. A two-point, variable-volume, single-pool urea kinetic model was used for determination of an index of the fractional clearance of urea (Kt/V) (23). Equilibrated BUN ($C_e$) was substituted for $C_1$ in the kinetic model to arrive at the equilibrated Kt/V (eKt/V). $C_e$ was determined by multiplying $C_{+30}$ by 1.039 to correct for continued equilibration (24). Urea rebound (UR, %) was calculated as follows: $\frac{[(C_e - C_0)/C_0] \times 100}{\text{The mean change in the core temperature ($\Delta T_{\text{skin}}$), systemic vascular resistance index ($\Delta \text{SVRI}$), cardiac index ($\Delta CI$), skin temperature}}$

$\Delta T_{\text{skin}}$, and mean arterial pressure ($\Delta MAP$) was determined from the difference between the weighted average value during treatment and the initial value. The maximum decrease ($-\Delta MAP_{\text{max}}$) or maximum increase ($+\Delta MAP_{\text{max}}$) in MAP was determined by comparing the maximal or minimal pressure reading during treatment with the predialysis measurement. If no decrease in MAP occurred, then $-\Delta MAP_{\text{max}}$ was considered to be zero. Similarly, if no increase in mean arterial pressure occurred, then $+\Delta MAP_{\text{max}}$ was considered to be zero.

Muscle cramps, nausea, vomiting, or episodes of hypotension requiring staff intervention occurring during hemodialysis were recorded. Patients' ages ranged from 19 to 85 yr, with a median age of 57 yr and a mean age of 56.1 ± 4.8 yr. There were eight women and seven men. The etiology of end-stage renal disease was diabetes in five patients, hypertension in six, and glomerulonephritis, nephrotoxicity, or collagen vascular disease in the remaining four. Eleven patients were on one or more antihypertensive medications. Procedures and protocols were approved by Beth Israel Medical Center Institutional Review Board. Informed consent was obtained from all experimental subjects.

Statistical Analyses

Statistical analyses were performed using paired $t$ tests unless otherwise specified. Data are reported as mean ± SEM unless otherwise specified.

Results

Results are described in Table 1 and Figures 2 and 3.

The basic dialysis prescription in terms of extracorporeal blood flow rate, dialysate flow rate, and treatment time was not different between groups. The prehemodialysis weight, pre- to posttreatment weight loss, and change in blood volume was not different between groups (Figure 2).

Dialysate cooling resulted in negative balance with respect to heat exchange in the extracorporeal circuit ($-266 ± 15$ versus $5 ± 31$ kJ per treatment) and a significant decrease in core temperature ($-0.22 ± 0.04$ versus $+0.31 ± 0.05$°C) relative to the thermoneutral studies in which core temperature actually increased during treatment. Cooling resulted in a decrease in skin temperature ($-1.1 ± 0.4$ versus $+1.1 ± 0.5$°C) and an increase in the systemic vascular resistance index ($+514 ± 160$ versus $+114 ± 119$ dyn·sec/cm² per m²), but no change in cardiac index relative to the thermoneutral treatment regimen. Shivering did not occur with blood cooling.

Blood cooling was associated with higher systolic pressure ($157 ± 8$ versus $144 ± 7$ mmHg) and MAP ($109 ± 5$ versus $101 ± 5$ mmHg) postdialysis and a higher average change in MAP throughout treatment ($+4 ± 4$ versus $-5 ± 3$ mmHg). Cooling also was associated with a lower maximum fall in MAP ($-11 ± 2$ versus $-19 ± 3$ mmHg), a higher maximum increase in MAP ($15 ± 3$ versus $8 ± 3$ mmHg) (Figure 3), and a reduction in staff interventions during hemodialysis (5 of 15 versus 10 of 15 patients). Prehemodialysis urea concentration, posthemodialysis equilibrated urea concentration, posthemodialysis urea rebound, and equilibrated Kt/V were not significantly different between groups.
Table 1. Physiologic effects of cooled versus thermoneutral dialysis treatments

<table>
<thead>
<tr>
<th>Panel A</th>
<th>$Q_b$</th>
<th>$Q_d$</th>
<th>$t$</th>
<th>$Wt_0$</th>
<th>$Wt_1$</th>
<th>$\Delta Wt$</th>
<th>$\Delta BV$</th>
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<tr>
<td>cooled</td>
<td>414±9</td>
<td>800</td>
<td>181±6</td>
<td>67.3±3.7</td>
<td>64.3±3.6</td>
<td>-3.1±0.3</td>
<td>-14.8%±2%</td>
</tr>
<tr>
<td>thermoneural</td>
<td>414±9</td>
<td>800</td>
<td>180±6</td>
<td>67.60±3.7</td>
<td>64.2±3.7</td>
<td>-3.4±0.3</td>
<td>-13.9%±2%</td>
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<tr>
<th>Panel B</th>
<th>ThBal</th>
<th>$T_d$</th>
<th>$Tc_0$</th>
<th>$\Delta T_c$</th>
<th>$\Delta T_{skin}$</th>
<th>$\Delta SVRI$</th>
<th>$\Delta CI$</th>
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<tr>
<td>cooled</td>
<td>$-266±15^b$</td>
<td>35.7±0.02</td>
<td>36.65±0.08</td>
<td>-0.22±0.04$^{b}$</td>
<td>-1.1±0.4$^{b}$</td>
<td>+514±160$^{b}$</td>
<td>-0.4±0.2</td>
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<tr>
<td>thermoneural</td>
<td>5±31</td>
<td>37.1±0.02</td>
<td>36.66±0.10</td>
<td>+0.31±0.05</td>
<td>+1.1±0.5</td>
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<th>Panel C</th>
<th>Systolic₀</th>
<th>Diastolic₀</th>
<th>MAP₀</th>
<th>Systolic₁</th>
<th>Diastolic₁</th>
<th>MAP₁</th>
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<tr>
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<td>157±8$^b$</td>
<td>84±5</td>
<td>109±5$^c$</td>
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<tr>
<td>thermoneural</td>
<td>157±5</td>
<td>86±4</td>
<td>110±4</td>
<td>144±7</td>
<td>80±5</td>
<td>101±5</td>
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<th>Panel D</th>
<th>avg ΔMAP</th>
<th>$-\Delta MAP_{max}$</th>
<th>$+\Delta MAP_{max}$</th>
<th>Staff interventions</th>
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<tr>
<td>cooled</td>
<td>+4±3$^b$</td>
<td>-11±2$^b$</td>
<td>15±3$^b$</td>
<td>5 of 15 pts (6 of 26 studies)</td>
</tr>
<tr>
<td>thermoneural</td>
<td>-5±3</td>
<td>-19±3</td>
<td>8±3</td>
<td>10 of 15 pts (12 of 28 studies)</td>
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<th>Panel E</th>
<th>$C_0$</th>
<th>$C_i$</th>
<th>$C_e$</th>
<th>$UR$</th>
<th>eKu/V</th>
<th>$V$</th>
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<tr>
<td>cooled</td>
<td>63.1±3.7</td>
<td>15.9±1.5</td>
<td>21.6±1.8</td>
<td>38±3</td>
<td>1.29±0.05</td>
<td>37.1±2.8</td>
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<tr>
<td>thermoneural</td>
<td>64.9±3.4</td>
<td>16.4±1.5</td>
<td>22.0±1.7</td>
<td>37±4</td>
<td>1.32±0.06</td>
<td>37.1±2.5</td>
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* Panel A: Treatment parameters and blood volume. Panel B: Core temperature, skin temperature, vascular resistance, and cardiac output. Panels C and D: Blood pressure parameters and staff interventions. Panel E: Urea kinetic parameters. Data are presented as mean ± SEM. Significant differences by paired t test analysis are indicated.

$^b$ P < 0.01.
$^c$ P < 0.05.
$^d$ Significant differences by McNemar’s test for sample proportions, which are not independent.

Measurement Units: Panel A: Extracorporeal blood flow ($Q_b$, ml/min), dialysate flow ($Q_d$, ml/min), body weight pre- ($Wt_0$, kg) and postdialysis ($Wt_1$, kg), change in body weight ($\Delta Wt$, kg), and change in blood volume pre- to postdialysis ($\Delta BV$, %). Panel B: Extracorporeal thermal balance ($ThBal$, kJ/treatment), mean dialysate temperature ($T_d$, °C), initial core temperature ($Tc_0$, °C), mean change in core temperature ($\Delta T_c$, °C), mean change in skin temperature ($\Delta T_{skin}$, °C), mean change in systemic vascular resistance index ($\Delta SVRI$, dyn.sec/cm² per m²), and mean change in cardiac index ($\Delta CI$, L/min per m²). Panel C: Predialysis mean systolic (Systolic₀, mmHg), diastolic (Diastolic₀, mmHg), and mean arterial pressure (MAP₀, mmHg). Postdialysis mean systolic (Systolic₁, mmHg), diastolic (Diastolic₁, mmHg), and MAP (MAP₁, mmHg). Panel D: Time-averaged change in MAP throughout treatment (avg $\Delta MAP$, mmHg), maximum fall in MAP during dialysis ($-\Delta MAP_{max}$, mmHg), maximum increase in MAP during dialysis ($+\Delta MAP_{max}$, mmHg), staff interventions for hypotension or symptoms (number/patients studied or number/studies performed). Panel E: Predialysis blood urea nitrogen concentration ($C_o$, mg/dl), postdialysis blood urea nitrogen concentration ($C_i$, mg/dl), equilibrated postdialysis blood urea nitrogen concentration ($C_e$, mg/dl), urea rebound ($UR$, %), and kinetically determined urea distribution volume ($V$, liters).

Discussion

Hemodynamic Stability

In these studies, dialysate cooling resulted in blood cooling. When extracorporeal thermal energy exchange was kept neutral, core temperature increased. An increase in thermal energy production relative to the cooled patients could increase core temperature but, more likely, the increase in body temperature in the thermoneutral group can be explained by ultrafiltration-induced vasoconstriction of skin vessels (25) and a concomitant reduction in convective heat loss from the skin.

During hemodialysis, blood cooling resulted in improved hemodynamic stability as defined by maintenance of arterial pressure and reduction in symptomatic events. Because blood cooling did not result in a difference in the intravascular volume or cardiac index relative to the thermoneutral studies, differences in cardiac output or vascular refilling (26) could not explain the hemodynamic findings. However, blood cooling was associated with a decrease in skin temperature relative to the thermoneutral studies. Skin temperature is a function of heat transport from the body core to the skin by blood and by thermal dissipation (mostly radiation) to the environment (27). During unchanged environmental conditions, changes in skin temperature reflect changes in skin blood flow. Thus, the lower skin temperature with blood cooling suggests a state of enhanced peripheral vasoconstriction relative to the thermoneutral studies. This conclusion is supported by the significantly greater increase in peripheral vascular resistance with blood cooling relative to the thermoneutral studies. Thus, blood cooling induces enhanced vasoconstriction that, in addition to the skin, involves the small vessels responsible for BP determination.

Changes in blood volume are claimed to predict hypotensive episodes on hemodialysis in that a rise to a specific hematocrit is reproducibly associated with a substantial fall in BP (20).
the current study, differences in episodic falls in arterial pressure with similar blood volume changes suggest that blood volume changes may not predict hypotensive episodes during hemodialysis when viewed independently of temperature effects. It is likely that core temperature cooling allows a greater reduction in blood volume to occur without hypotension. The increase in peripheral resistance after cooling shifts the nonlinear curve relating blood volume change to BP to the right (28) so that greater loss in volume is required before the BP falls (Robert R. Steuer, personal communication).

Although there were no differences between groups in the reduction in blood volume during treatment, greater reductions in blood volume in treatments with core temperature cooling would, in theory, be possible. Thus, the vascular refilling rate would be expected to decrease as a result of cooling-induced vasoconstriction and sequestration of blood volume in the splanchnic, and other, reservoirs. However, cooling effects on vascular volume can be variable (29). In the current studies, perhaps greater reductions in blood volume during ultrafiltration would have been observed with greater degrees of core temperature cooling.

In clinical practice when dialysate temperature is held constant, it is difficult, if not impossible, to predict the changes in heat–energy exchange in the extracorporeal circuit or the changes in core temperature. This is because thermal flow is variable not only in the extracorporeal circuit but also between the patient and the general environment. The effect of dialysis on core temperature is a result of the thermal flow relating to the extracorporeal circuit, as well as the variable factors of internal generation and external radiation and convection of heat. Thus, extracorporeal thermal flow is affected by extracorporeal blood flow rates that can vary from 200 to 500 ml/min and by the patient’s baseline core temperature, which varies from individual to individual by as much as 2°C. Therefore, the preferred approach to hemodynamic stabilization by dialysate temperature manipulation is to use a feedback control system that is responsive directly to changes in core temperature measured on-line.

**Effect on Urea Kinetics**

During hemodialysis, solute is removed from multiple compartments. Part of the steady increase in posthemodialysis blood urea levels over 30 to 60 min after dialysis may be related to physiologic blood flow differences to regional body compartments and can be modeled as such (7). In this parallel flow model, skin, muscle, and bone comprise slow-flow compartments containing the greatest proportion (80%) of body...
water and solute, including urea, in which urea transport is flow limited. Visceral organs comprise rapid-flow compartments in which urea transport is not flow limited. Differences in slow-flow compartment perfusion and recovery in response to hemodialysis have been hypothesized to contribute to differences between posthemodialysis urea rebound between different subjects. Thus, reduction of flow to muscle, for example, results in sequestration of solute and consequently less effective hemodialysis. This is manifested by an increase in posthemodialysis rebound when the reestablishment of regional blood flow posthemodialysis results in equilibrium with systemic blood. In the current study, there were no differences between the two groups in posthemodialysis urea rebound or equilibrated Kt/V, the standard by which hemodialysis efficacy can be measured (30).

The current study suggests that cold-induced vasoconstriction sufficient to result in hemodynamic stabilization occurs in a relatively small compartment. Most likely this occurs in skin with little effect on muscle flow. This is consistent with the large contribution that skin vessels make with respect to determining peripheral vascular resistance (31).

Yu et al. also did not demonstrate an alteration in dialysis kinetics with core temperature cooling as measured by changes in the tympanic membrane temperature (17). Their studies were performed in subjects undergoing dialysis with significantly lower K/V than the studies presented here (5.38 versus 8.89 ml/min per L). With lower K/V, the postdialysis rebound would be predicted to be less than in the current study, and in fact was (12% versus 37%). Although our findings also showed that eKt/V was not altered by core temperature cooling in the range studied, this conclusion would not, a priori, follow from the earlier study. It was reasonable to expect that changes in rebound (and hence eKt/V) would be more likely to be found during dialysis performed at the highest K/V.

Conclusion

When blood cooling is used to stabilize BP during very high efficiency hemodialysis with high ultrafiltration rates, the efficacy of treatment with respect to low molecular weight molecules such as urea is not compromised. Additional modifications in the prescription, beyond that which are necessary to adjust for compartment disequilibrium accompanying rapid hemodialysis (32), do not appear necessary. Blood cooling helps to maintain BP without compromising the efficacy of hemodialysis. Thus, techniques used to determine eKt/V, such as the Daugirdas–Schneditz rate adjustment method, the Smye method, or the BioStat method, would all be expected to remain useful with respect to monitoring the dialysis dose (33).

References