Keep your temper: how to avoid heat accumulation in haemodialysis

Daniel Schneditz¹ and Nathan W. Levin²

¹Department of Physiology, Karl-Franzens University, Graz, Austria and ²Renal Research Institute, New York, NY, USA

There is considerable interest in temperature balance during haemodialysis because of the well documented effect of warm dialysate on haemodynamic stability [1–4]. Even though haemodialysis does not lead to direct heat transfer from the extracorporeal circulation to the patient in most cases, patient temperatures tend to increase during treatments using conventional dialysate temperatures. What is the cause for the increase in body temperature often seen during haemodialysis?

Extracorporeal factors

Extracorporeal heat flow is determined by dialysate temperature, arterial blood temperature (a good representation of patient core temperature), blood flow, environmental temperature, length and insulation characteristics of the venous blood line, and ultrafiltration. Even though dialysate temperature governs the temperature of blood leaving the dialyser, it is the temperature of venous blood entering the access that determines the extracorporeal arterio-venous temperature gradient. Venous blood temperature entering the patient decreases with decreasing extracorporeal blood flow because of increased exposure to the cooler environment. Extracorporeal cooling decreases as extracorporeal blood flow increases [5]. The temperature drop in the venous bloodline can reach 1°C [6]. For example, a patient with a core temperature of 36°C dialysed at 37°C will be warmed at a blood flow of 450 ml/min, but cooled at a blood flow of 200 ml/min with a difference of 15 W in thermic flow rate. High blood flows are likely to contribute to heat accumulation using standard dialysis temperatures and the benefits of low dialysate temperatures will be more evident in high efficiency haemodialysis.

Patient temperature

Whereas the temperature of venous blood returning to the patient is largely determined by the dialysis procedure and to some degree by environmental factors, the temperature of arterial blood leaving the access is determined by patient core temperature. Due to the range in patient core temperatures the same dialysis temperature delivered under the same environmental conditions may not provide the same cooling to all patients. This explains why haemodynamic benefits of cool dialysate are most evident in patients with low core temperature [7]. Patients with low core temper-
atures are subject to increased heat accumulation and to an increased risk of heat-induced hypotension because of reduced extracorporeal heat losses.

Haemodynamic compensation

The volume hypothesis of heat accumulation refers to the physiologic mechanism to compensate for a drop in venous return and central venous pressure [8]. A physiologic example of this mechanism is the increase in core temperature with orthostasis [9]. This mechanism can be extended to the compensation of ultrafiltration-induced hypovolaemia. On one hand, hypovolaemia leads to sympathetichypovolaemia increases sympatho-metabolism such as by an increase in heart rate. On the other hand, compensatory redistribution of regional blood flow and blood volume in the cutaneous circulation leads to a reduction of heat transfer from the core to the body shell to the environment. Both factors cause the accumulation of heat and lead to an increase in body temperature. The rate of thermic energy accumulation during haemodialysis has been estimated as approximately 40% of resting energy expenditure [10]. The accumulation of heat and the increase in body temperature can be countered using cool dialysate [11,12].

Pyrogen hypothesis

An alternative explanation for intradialytic heat accumulation is offered by the pyrogen hypothesis, where it is assumed that exposure of blood to bioincompatible material and mechanical blood trauma triggers a fever response [13]. The pyrogenic reaction will also cause a physiologic increase in metabolic rate and a decrease in thermic losses.

The different mechanisms have different practical implications for everyday treatment. However, if haemodialysis-induced heat accumulation were related to ultrafiltration, dialysate temperature could be adjusted to compensate for ultrafiltration-induced effects.

Direct effect of ultrafiltration

Analysis of the physiologic mechanisms involved in ultrafiltration-induced heat accumulation requires quantification of direct thermic effects caused by ultrafiltration. Temperature balance with ultrafiltration is complicated because of the large energy content of ultrafiltration volume. In the presence of ultrafiltration, total extracorporeal heat flow can be separated into two flow components relating to constant temperature (isothemric flow) and to constant volume (isochoric flow) respectively. For example, if 1 litre of ultrafilterate is removed from the body within 1 h, approximately 1200 kJ of thermic energy are removed from the patient at a thermic flow rate of 330 W, a multiple of the average resting energy expenditure of 70 W. However, if this flow takes place without arterio-venous temperature gradients, the flow is entirely isothermic. Isothermic energy flow does not affect body temperature and there is no known direct effect on temperature regulation. The fraction of total extracorporeal heat flow to be considered for temperature regulation with ultrafiltration is isochoric heat flow ($Q_c$). $J_{ex}$ (in W) is determined from arterio-venous temperature gradients ($T_{art}$-$T_{ven}$, in °C), extracorporeal blood flow ($Q_b$, in ml/s), and ultrafiltration rate (UFR, in ml/s) according to the following equation [14]:

$$J_{ex} = -3.80 \cdot (T_{art} - T_{ven}) \cdot (Q_b - UFR).$$

Comparison with previous relationships that did not consider effects of ultrafiltration shows that isochoric heat flux is attenuated by ultrafiltration. The attenuation effect is small when UFR is only a small fraction of $Q_b$.

Indirect effect of ultrafiltration

If heat accumulation were related to haemodynamic compensation for ultrafiltration-induced hypovolaemia, it should be possible to observe either an increase in body temperature with constant extracorporeal heat flow, or an increase in extracorporeal heat flow with constant body temperature. The increase in either body temperature or extracorporeal heat flow would have to be related to ultrafiltration-induced haemodynamic compensation. In a recent study the amount of cooling provided by cool dialysate was adjusted to keep arterial blood temperatures constant within ±0.1°C [14]. Extracorporeal cooling requirements were observed to increase significantly with increased ultrafiltration in a linear relationship and to be independent of treatment time and extracorporeal blood flow. Dialysate was prepared from ultra-pure water. This result is in support of the volume hypothesis and at the same time rejects the pyrogen hypothesis as an explanation for intradialytic heat accumulation. In the attempt to keep body temperature constant during haemodialysis and ultrafiltration it was thus suggested to set extracorporeal cooling to 6% of estimated energy expenditure for every percent of ultrafiltration-induced change in body weight.

It is well known that the same change in body weight causes variable haemodynamic compensation in patients with different body hydration and cardiovascular reactivity [15]. Overhydrated patients who do not reach their true target weight will have sufficient vascular refilling, more stable blood volumes [16], and reduced blood volume shifts from compliant vascular beds to the central circulation. It is thus likely that variables related to volume status or cardiovascular reactivity provide a better prediction of intradialytic heat accumulation. An improved relationship has indeed been observed between cooling requirements and relative blood volume changes in a subsequent study [17].
Even though the relationship between weight loss and cooling to maintain constant body temperature may not hold for overhydrated patients, it makes sense to apply the same relationship for this patient group where the origin for overhydration is most likely related to haemodynamic instability. If extracorporeal cooling were based on blood volume, only insufficient removal of thermic energy would be prescribed for the overhydrated patient. However, with extracorporeal heat removal based on prescribed ultrafiltration, the overhydrated patient will be cooled, and body temperature will be lowered with the result of improving the patient’s haemodynamic stability. An improved haemodynamic stability will facilitate fluid removal and lead to adjustments in the prescription of target weight. Another advantage of ultrafiltration-based cooling relates to its prescription. The weight change is prescribed at the beginning of dialysis so that extracorporeal cooling, a slow process, can be set at the beginning of the treatment. Blood volume based cooling requires a feedback control from measurement of blood volume that is not routinely available yet.

**Blood temperature monitor**

The consideration of all factors involved in extracorporeal heat flow is greatly simplified by the blood temperature monitor (BTM, Fresenius Medical Care, Bad Homburg, Germany) discussed in a companion article [18]. With feedback control of extracorporeal heat flow [19] the removal of a fraction of estimated energy expenditure as a function of prescribed weight change is easily accomplished by the BTM.

**Conclusion**

The discussion of temperature balance during haemodialysis recently resurfaced [20,21] and the increase in body temperatures seems to play an important role in haemodialysis. Several factors have predictable effects on heat accumulation with everyday haemodialysis. Dialysate temperature is the single most important factor, with patient temperature. Low patient temperatures require lower dialysate temperatures to prevent heat accumulation. Technical factors such as environmental temperatures, bloodline characteristics, and extracorporeal blood flows have a significant effect on the amount and direction of extracorporeal heat flux. And last but not least, ultrafiltration elicits a predictable accumulation of thermic energy most likely because of compensatory reduction of heat losses from the body surface. These factors can now be quantitated and a rise in patient temperature predisposing to haemodialysis instability should be avoidable with an improved prescription of extracorporeal heat losses.

**References**

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