THE CAUSE OF INADEQUATE HAEMODYNAMIC REACTIONS DURING ULTRADIFFUSION

H Pogglitsch, H Holzer, J Waller, H Pristautz, H Leopold*, H Katschnigg

Med. Univ. Clinic, Graz and *Institute of Physical Chemistry, Graz, Austria

Introduction

Following the reports of Silverstein [1] and Bergström [2] several authors have confirmed that the withdrawal of body fluid by ultrafiltration (UF) alone is tolerated better haemodynamically than ultrafiltration together with haemodialysis (UD). As already known, ultrafiltration rates of 16 to 20 cm³ per minute, which are usually necessary during a 4 hours’ routine haemodialysis, can lead to unexpected drops of blood pressure, while the same or even higher ultrafiltration rates are tolerated in UF without any complications.

The unexplained difference between tolerance to UF and UD motivated us to study the relations between haemodynamics and volume regulation in a way similar to previous workers [3] but with special respect to the possible disturbing influences during UD.

Patients and Methods

In 40 patients, continuous measurements of body weight with an electronic bed balance and of blood density using the bending-oscillator [4] were performed. Blood pressure, pulse rate, pulse wave velocity, the serum sodium, potassium and calcium, erythrocyte potassium content and serum osmolality were measured at intervals of half-an-hour and one hour. From the pulse wave velocity and the other haemodynamic parameters, the minute volume and peripheral vascular resistance were calculated.

In 10 patients we repeatedly measured the blood volume by erythrocyte labelling with 51 Cr during UF and UD.

The dialysate contained 135 mEq sodium and 1.5 mEs potassium per litre.

Results

Blood Volume and Blood Density

Within the physiological range there are close relationships between blood
density and blood volume. In Figure 1, the blood volumes of 10 patients are plotted against the corresponding blood density values during haemodialysis. There is a good correlation between these parameters (correlation coefficient between 0.83 and 0.99). The variation in the position of the regression lines depends on the individual differences of the haematocrit and plasma protein concentration.

If the differences between the blood volumes and the corresponding blood
density values are plotted against each other in patients with approximately the same haematocrit (Figure 2), a line can be computed which allows changes in the blood volume in cm$^3$ to be read directly from a given change in blood density.

The great advantage of blood densitometry measurements lies in the fact that the behaviour of the blood volume can be registered continuously, and the accuracy of the method permits recognition of blood volume changes as small as ± 10 cm$^3$.

Changes in Blood Volume During UF and UD

UF and UD lead to haemoconcentration as soon as the ultrafiltration rate exceeds the fluid influx from the interstitial and the intracellular spaces. The difference between the lowest ultrafiltration rate without significant haemoconcentration and the ultrafiltration rate which will certainly cause a sudden drop of blood pressure, varies from case to case and from dialysis to dialysis. It depends not only on the degree of overhydration of the patient but also on plasma protein concentration, cardiac efficiency and last but not least on other disturbing factors to be mentioned later.

At a constant transmembrane pressure the reduction in blood volume — expressed by the change in blood density — has a linear correlation with the amount of the ultrafiltrate, as demonstrated in the two patients in Figure 3: one patient was ultrafiltered at an ultrafiltration rate of 23 cm$^3$ while the other was dialysed at an ultrafiltration rate of 20 cm$^3$ per minute.
In routine treatment the correlation between ultrafiltration and haemoconcentration was not so close, because the transmembrane pressure was altered occasionally during the investigation. With UF, an average of $1507 \pm 950 \text{ cm}^3$ and with UD $1663 \pm 874 \text{ cm}^3$ body fluid were withdrawn. The blood volume
decrease after UF, however, was more pronounced (-488 ± 350 cm³) than after UD (-270 ± 220 cm³).

Relationships between UF, UD and Haemodynamics

To find out the relationship between UF, UD and haemodynamics, 7 patients with constant weight gain during intervals between dialyses were subjected first to a controlled sequential ultrafiltration-haemodialysis (CSU): the average fluid loss during this procedure was 3902 ± 1069 cm³. One week later approxi-
mately the same amount of fluid (4000 ± 915 cm³) was withdrawn by UD. (Figure 4: the dotted lines represent the changes during UF, the continuous lines show the changes during UD).

At the end of the UF period the blood pressure was only slightly lower than the initial value, the minute volume had moderately decreased, the peripheral resistance had risen corresponding to a blood volume loss of about 890 cm³. At the end of the subsequent haemodialysis the blood pressure fell significantly by 35 mmHg, although no additional fluid loss had occurred and although blood volume and minute volume had tended to go up. The same haemodynamic reactions as after CSU could also be noted after UD: a relatively slight haemoconcentration (up to 335 cm³ blood volume loss), a slight decrease of the minute volume but a marked diminution of the vascular resistance. As a consequence the blood pressure decreased significantly, by 41 mmHg. Compared with UD, controlled sequential ultrafiltration-haemodialysis obviously does not offer any haemodynamic advantages.

Changes of Osmolality and Electrolyte Concentrations and their Influence on Haemodynamics during UF and UD

Analysing the conditions which could cause the inadequate vascular resistance during UD, one will first of all have to take into consideration the changes of serum osmolality and electrolyte concentrations.

In our patients the serum osmolality fell by an average of 30 mOsm (Figure 5). This decrease could be of importance for the haemodynamics if thereby
an osmotic gradient developed between intracellular and extracellular space and if fluid shifted into the intracellular space along this gradient. In that case the linear correlation between the amount of ultrafiltrate and the diminution of blood volume during UD would disappear: we have, however, not noticed this phenomenon in any of our investigations. Furthermore, it became evident that the serum potassium level decreases significantly during the first hour, while the serum calcium concentration increases significantly and the serum sodium level remains constant.

To the same extent as hypokalaemia develops extracellularly, the intracellular/extracellular potassium quotient increases, as measured by the simultaneous determination of erythrocyte potassium content. If in this period potassium is infused (about 10 to 20 mEq within 20 to 30 minutes), stabilisation of blood pressure can be observed in the majority of patients with hypotensive symptoms (Figure 6) and occasionally even a return to normal.

![Figure 6](image)

We confirmed the effect of potassium substitution in 6 patients suffering from hypotensive reactions during UD. Whilst blood pressure drops occurred in 18 of 100 haemodialysis sessions without potassium infusion, there were only 3 episodes of hypotension in 56 treatments with potassium infusion.

**Discussion**

The effect of potassium substitution during UD may be explained as follows (Figure 7): any loss of blood volume by UF or UD leads to an inhibition of the sympathetic circulatory centre via stimulation of volume receptors [5].

251
The result of this inhibition is a controlled decrease of blood pressure, especially in the precapillary region, where the relationship between filtration and reabsorption is newly adjusted in favour of better fluid absorption. In the case of UD this physiological regulation becomes inadequate. Both hypokalaemia with an elevation of the intra/extracellular potassium quotient, and hypercalcaemia lead to an elevation of the threshold for sympathetic vasoconstrictor stimuli. In the course of UD a critical point may be reached where the balance cannot be maintained between the counteracting influences on blood pressure regulation.

References