Original Paper

Hemodynamic Behavior During Hemodialysis: Effects of Dialysate Concentrations of Bicarbonate and Potassium

Bruno C. Silva^a Geraldo R.R. Freitas^a Vitor B. Silva^a Hugo Abensur^a
Claudio Luders^a Benedito J. Pereira^a Rodrigo B. de Oliveira^a
Manuel C.M. Castro^a Rosa M.A. Moysés^{a,b} Rosilene M. Elias^a

^aNephrology Division, University of São Paulo, School of Medicine; ^bMedicine Master Degree Program, Universidade Nove de Julho (UNINOVE), São Paulo, Brazil

Key Words

Hemodialysis • Bicarbonate • Potassium • Hemodynamic • Peripheral arterial resistance • Dialysate

Abstract

Background/Aims: Ultrafiltration that occurs during hemodialysis (HD) promotes profound alterations in a relatively short period of time. The dialysate content of bicarbonate (DBic) and potassium (DK) may have impact over intradialytic hemodynamics, which goes beyond ultrafiltration, and its impact was evaluated in a prospective cohort. **Methods:** 30 patients under HD were submitted to hemodynamic assessment (HA) at the beginning and at the end of HD sessions, through a non-invasive method. Serum minus dialysate potassium concentration was expressed as K-Gap. Cardiac index (CI) and peripheral arterial resistance (PAR) variation (post-HD minus pre-HD) were expressed as Δ CI and Δ PAR. Dialysate content of sodium and calcium were expressed as DNa and DCa, respectively. **Results:** Mean DNa, DK and DBic were, respectively, 136.4 ± 1.1 , 2.1 ± 0.6 and 38.2 ± 2.1 mEq/L. In 15 patients, DCa was >1.5 mmol/L and in the other 15 patients ≤ 1.5 mmol/L. The K-Gap ranged from 1.4 to 5.1 mEq/l (median 3.0 mEq/L). There was a reduction in post-HD CI and systolic blood pressure (Δ CI = -0.72 l/min/m² and -11.3 ± 15.1 mmHg, respectively, $p < 0.001$ for both). Conversely, PAR increased (Δ PAR = 272 dyn.s/cm⁵, $p < 0.001$). Lower post-HD CI was associated to higher DBic ($p = 0.0013$) and lower K-Gap ($p = 0.026$). In multivariate analysis, Δ CI was dependent on DBic and K-Gap, whereas Δ PAR was dependent on dialysate calcium during HD. **Conclusion:** We confirmed that Na and Ca dialysate content exerts an important role on hemodynamic during HD. In addition, our findings pointed out that higher dialysate concentrations of bicarbonate and potassium promote lower cardiac performance at the end of hemodialysis session.

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Introduction

Not only traditional cardiovascular (CV) risk factors, but also bone mineral disorders and chronic volume overload play major role in the high mortality observed in chronic kidney disease (CKD) population [1-3]. Individualized hemodialysis (HD) is prescribed by nephrologists in order to keep CKD patients close to optimal physiologic conditions, through reaching dry weight and keeping serum electrolytes, hemoglobin and bicarbonate concentrations within a normal range. Notably, volume overload leads heart and vascular bed to a stressful hemodynamic condition, in which both are submitted to an overfilling state during the interdialytic interval, while ultrafiltration (UF) during HD treatment promotes rapid volume removal. (2) This process may be accompanied by significant intradialytic hypotension and possibly, transient myocardial ischemia, which may impair left ventricular (LV) function even after restoration of myocardial blood flow, a phenomena known as myocardial stunning (MS), which is a cause of chronic LV dysfunction [4].

In maintenance HD treatment, dialysate prescription could exert significant impact over hemodynamic stability during the UF process [5-7]. It is already known that higher dialysate concentrations of sodium and calcium promote better UF tolerability, with lower hypotensive episodes during HD [8]. Nevertheless the role of other electrolytes on intradialytic hemodynamics remains subject of debate.

Individualizing dialysate content of bicarbonate and potassium may bring serum concentrations of these electrolytes close to normal range. Nevertheless, the impact of such prescription on intradialytic hemodynamics remains unknown. Therefore, the aim of the present study was to investigate, through a non-invasive methodology, the impact of dialysate content of bicarbonate and potassium on hemodynamic changes during HD treatment.

Patients and Methods

Study population

Adult patients receiving HD for at least 6 months were prospectively enrolled in this observational study. Patients with *diabetes mellitus*, non-sinusal cardiac rhythm, recent parathyroidectomy (less than 6 months), and prior history of congestive heart failure were excluded.

Study protocol

The study was performed according to the guidelines of the Helsinki Declaration and was approved by the Research Ethics Boards of the Universidade de São Paulo (CAPPesq #235.350). All subjects provided written informed consent before participation.

During the study, there was no change in dialysate content of sodium (DNa), potassium (DK), calcium (DCa), glucose and bicarbonate (DBic).

All HD sessions were performed using Fresenius 4008S machines and high flux polysulfone membranes (Fresenius Medical Care™, Bad Homburg, Germany). Blood flow was set to 350 ml/minute and dialysate flow to 800 ml/min in all patients.

Laboratory Values

Blood tests were performed before and after each dialysis session. Biochemical concentrations of serum Ca, P, Na, K, creatinine, albumin and hemoglobin were measured. Serum intact parathyroid hormone (PTH) levels were measured using a chemiluminescence assay (RR = 15-65 pg/ml). HD adequacy was assessed by monthly Kt/V measurements using the Daugirdas' equation [9]. Additionally, the difference between serum and dialysate concentration of bicarbonate, potassium and sodium were calculated and named as Bic-Gap, K-Gap and Na-Gap, respectively.

Hemodynamic measurements

Cardiac output index (CI), stroke volume (SV - integrated mean of the flow waveform between the current upstroke and the dichotic notch), peripheral arterial resistance (PAR - ratio of mean arterial pressure to stroke volume multiplied by heart rate) and blood pressure (BP) were accessed by finger beat-

to-beat monitor Finometer® (Finapres Medical Systems BV, Arnhem, The Netherlands) within 15 minutes after starting HD session (pre-dialysis) and again, 15 minutes before its end (post-dialysis). If frequent extra systoles were observed, monitoring was interrupted and the entire protocol was repeated afterwards. Ectopic beats were excluded from analysis. Differences between post- and pre-HD CI and PAR were expressed as Δ CI and Δ PAR, respectively.

Statistical analyses

Categorical variables were presented as proportion, and continuous variables were presented as mean $\pm$ SD unless indicated otherwise. Differences between continuous variables were calculated using Student t test or Mann-Whitney U test as appropriate. The χ^2 or Fisher's exact test was used to compare categorical variables. Paired t test or Wilcoxon matched-pairs were used to compare variables pre- and post-hemodialysis, as appropriate. Relationships between single variables were examined by Spearman correlation coefficient. Multivariable relationships between Δ CI and independent variables, and between Δ PAR and independent variables were also examined by stepwise linear regression, with $P < 0.05$ to enter and $P > 0.1$ to remove. A two-tailed P value < 0.05 was considered significant. Analyses were performed with SPSS 21.0.1 (SPSS Inc., Chicago, USA) and GraphPad Prism 6 software (San Diego, USA).

Results

Baseline characteristics

As shown in Table 1, our population ($N = 30$) was relatively young, and with a high dialysis vintage. Sixty percent of them had arterial hypertension and also were taking hypotensive drugs. The dialysis dose (measured by standard Kt/V), serum albumin and hemoglobin levels were within recommended targets. The ultrafiltration rate was high.

Dialysate prescription

Mean DNa, DK and DBic were, respectively, 136.4 ± 1.1 , 2.1 ± 0.6 and 38.2 ± 2.1 mEq/L. In 15 patients, DCa was >1.5 mmol/L and in the other 15 patients ≤ 1.5 mmol/L. The K-Gap ranged from 1.4 to 5.1 mEq/l (median 3.0 mEq/L) and the Bic-Gap ranged from -24 to -8.7 mEq/L (median -17.8 mEq/L).

Biochemical and hemodynamic variations secondary to HD treatment

Extracellular fluid removal secondary to ultrafiltration caused an expected reduction in SV and CI and a compensatory PAR increase. All but three patients decreased post-HD CI. From pre- to post-HD, mean CI drop was -0.72 l/min/m² (range: -2.68 to 0.44 l/min/m²). Conversely, PAR increased in all but five patients (mean increase: 272 dyn.s/cm⁵, range: -522 to $2,231$ dyn.s/cm⁵). Systolic BP was significantly reduced (from 133.2 ± 23.8 to 121.9 ± 25.4 mmHg, $p=0.0003$), which was not observed for diastolic BP (from 74.5 ± 12.2 to 74.5 ± 13.6 mmHg, $p=0.990$). All these significant hemodynamic changes are shown in Figure 1. Diffusive process promoted an increase in serum bicarbonate (from 20.7 ± 3.4 to 29.6 ± 3.3 mEq/L, $p<0.0001$) and a decrease in serum potassium (from 5.2 ± 0.5 to 3.8 ± 0.5 mEq/l, $p<0.0001$).

Table1. Baseline characteristics of the studied population. (N=30)

Variable	Mean $\pm$ SD or %	Median (25 th , 75 th)
Age (years)	39.6 ± 11.3	28 (31,50)
Male Sex, %	57	
Dialysis vintage (months)	166 ± 108	176 (33.5, 245.0)
Body surface area (Kg/m ²)	1.72 ± 0.3	1.72 (1.57, 1.91)
Hypertensive subjects (%)	60	
Hemoglobin (g/dl)	11.5 ± 1.8	11.1 (10.3, 13.2)
PTH (pg/ml)	481 ± 373	524 (182, 663)
Standard Kt/V	2.54 ± 0.93	2.43 (2.16, 2.71)
Ultrafiltration (ml/Kg/h)	11.2 ± 4.4	11.41 (8.49, 14.87)
Serum albumin (g/dL)	4.0 ± 0.3	4.0 (3.9, 4.3)
Erythropoietin dose (U.I./Kg/week)	121.4 ± 114.1	74.7 (27.8, 203.7)

PTH, parathyroid hormone; Data are presented as mean $\pm$ SD or median and interquartile (25, 75), unless indicated otherwise.

Fig. 1. Changes from pre- to post-hemodialysis hemodynamics, expressed as % of change $\pm$ SEM. There was a significant decrease in cardiac index and in stroke volume, and peripheral arterial resistance increase.

Factors related to Δ CI and Δ PAR behaviour during hemodialysis sessions

a) Sodium and potassium Gap and its respective dialysate concentrations. Variables that correlated to a higher CI drop were: lower K-Gap ($r=0.405$, $p=0.026$), lower serum K levels ($r=0.430$, $p=0.018$), higher DBic levels ($r=-0.447$, $p=0.001$), higher serum Na levels ($r=-0.417$, $p=0.022$), and higher Na-Gap ($r=-0.405$, $p=0.026$). The relationships between Δ CI and Na- and K-Gap are better illustrated in Figure 2. No correlation was found between Δ PAR and any biochemical tested variables.

b) Bicarbonate dialysate content. DBic was categorized as low (<38 mEq/L) or high DBic (>38 mEq/L), according to its median, as shown in Table 2. Patients dialyzed with higher DBic presented a higher post-HD CI drop (-0.92 ± 0.59 vs. -0.09 ± 0.47 l/min/m², $p=0.002$). There was no difference in Δ PAR and UF rate.

A multivariable analysis was done to elucidate factors related to Δ CI and Δ PAR as a consequence of HD treatment, in which all significant biochemical variables and dialysate content profile were modeled together, adjusted to UF rate. All models are seen in Table 3. Δ CI was independently related to DBic and K-Gap, which accounted for 51.2% of its variation during HD. Higher DBic (>38 mEq/L) and lower K-Gap were associated to lower post-HD CI. Δ PAR was independently related to DCa, which explained 31.9% of its variation during HD.

Discussion

In this study, we found that Δ CI is strongly dependent on dialysate concentrations of bicarbonate and potassium. Lower post-HD CI (higher drop) was associated to higher DBic and lower K-Gap.

Fig. 2. Spearman correlations between cardiac index variation (Δ CI) and K-Gap (A), and Na-Gap (B). (A). Lower the K-Gap, higher the CI drop. This can be a consequence of higher pre-HD serum K or a lower DK concentration. (B). Higher Na-Gap (towards positive values), promotes higher CI drop. These patients are more likely to have negative Na balance during HD. On the opposite way, lower Na-GAP (towards negative values) leads to lower post-HD CI drop. These patients are more likely to develop positive Na balance during HD.

Hemodynamic changes during hemodialysis

Ultrafiltration, a process that removes intravascular volume, promotes a controlled hypovolemic state during HD sessions. In this study, fluid removal led to stroke volume reduction, with consequent CI decrease, while PAR increased, with little changes in BP. These same hemodynamic findings were obtained by Riddez et al [10], in a study that performed a venosection of 900 ml in healthy individuals under invasive hemodynamic monitoring. However, HD is a much more complex procedure, in which serum electrolytes and osmolarity change throughout its duration, according to a myriad of factors, such as patients' clinical characteristics, HD efficiency and dialysate prescription, which also influence hemodynamic response to the induced hypovolemia.

Bicarbonate dialysate content

Metabolic acidosis is common in patients under maintenance HD and may have adverse consequences not only for bone metabolism [11, 12] but also on nitrogen balance, which might decrease skeletal muscle mass [13]. For such reasons, nephrologists struggle to reach KDIGO's goal of pre-HD serum bicarbonate concentration of 22 mEq/l, [14] which can be obtained by increasing dialysate bicarbonate concentration [15]. Nevertheless, a recent prospective cohort study from DOPPS linked prolonged exposure to high dialysate bicarbonate levels to increased mortality [16]. Possible mechanisms are more frequent cardiac arrhythmias [17] and a greater propensity for intradialytic hypotensive episodes [7, 18], which could be related to higher intracellular shift of both calcium and potassium. In the present study, higher DBic was associated to a more pronounced decrease in post-HD CI, regardless of pre-HD serum Bic concentration. Although we did not directly accessed intradialytic myocardial contraction/perfusion, one could hypothesize that higher DBic content might lead to MS and, therefore, to a worse cardiovascular outcome in this population.

Table 2. Baseline clinical, laboratorial, hemodynamic and dialysate content characteristics, according to higher or lower than median DBic content

	DBic ≤ 38 mEq/l (n=16)	DBic >38 mEq/l (N=14)	p
Men (%)	62	50	0.71
Age (years)	37.1 ± 11.3	42.6 ± 11.1	0.19
Dialysis Vintage (months)	185 ± 112	140 ± 105	0.38
Ejection Fraction (%)	65.4 ± 4.3	63.4 ± 6.0	0.38
LVMl (g/m ²)	103 (75, 132)	107 (71, 126)	0.91
ACEI/ARB (%)	31.2	28.6	1
β blockers (%)	25	43	0.44
Ca channel blockers (%)	25.0	18.7	1
DCa (mEq/L)	3 (3, 3.5)	3.5 (3, 3.5)	0.08
DK (mEq/L)	2.1 ± 0.5	2.0 ± 0.7	0.79
DNa (mEq/L)	136.6 ± 1.1	136.1 ± 1.0	0.24
Na (mEq/L)	137.3 ± 4.0	139.4 ± 3.7	0.15
K (mEq/L)	5.3 ± 0.5	5.1 ± 0.5	0.36
Bicarbonate (mEq/L)	21.25 (18.8, 22)	20.4 (18.9, 22.4)	1
Hb (g/dL)	11.45 ± 1.5	11.65 ± 2.1	0.77
Ionized Ca (mEq/L)	4.7 ± 0.5	4.8 ± 0.4	0.55
Baseline CI (L/min/m ²)	3.9 (3.2, 5.5)	3.7 (2.9, 4.4)	0.54
Baseline PAR (dyn.s/cm ⁵)	1215 ± 456	1347 ± 406	0.42

LVMl: left ventricular mass index, ACEI: angiotensin conversor enzyme inhibitor, ARB: angiotensin receptor blocker, DCa: dialysate content of Calcium, DK: dialysate content of potassium, DNa: dialysate content of sodium, Hb: hemoglobin, CI: cardiac index, PAR: peripheral arterial resistance.

Table 3. Stepwise multiple linear regression analysis between the delta in cardiac index (ΔCI) and the delta in peripheral arterial resistance (ΔPAR) and independent variables

Independent Variable	Beta coefficient	Partial correlation	P
Model 1- Dependent ΔCI			
DBic ≤ or > than 38 mEq/L	-0.524	-0.614	0.0001
K-Gap	0.510	0.603	0.001
Model 2- Dependent ΔPVR			
DCa < or ≥ than 1.5 mmol/l	0.492	0.510	0.005

DBic, dialysate bicarbonate; K-Gap, serum minus dialysate K; PTx, parathyroidectomy, DCa, dialysate calcium. Entire model 1: r=0.739, adjusted r²=0.512, p=0.0001. Other variables in this model: ultrafiltration rate and Na-Gap; Entire model 2: r=0.605, adjusted r²=0.319, p=0.002. Other variables in this model: ultrafiltration rate.

Potassium dialysate content

Reducing DK content is an efficient measure for removing more potassium in a single dialysis session [19]. However, potassium's role on intradialytic hemodynamics remains elusive. *In vitro* studies have demonstrated increased myocardial contractility after acute exposure to potassium-deficient solutions [20, 21]. Conversely, chronic hypokalemia has an opposite effect, leading to inotropic response impairment after epinephrine or volume infusion [22]. Hypokalemia also affects vascular tonus, by inducing systemic vasoconstriction through a complex mechanism, which involves Na⁺-K⁺-ATPase inhibition, decreased uptake of norepinephrine into sympathetic nerve endings and increased calcium influx into vascular smooth muscle cell [23].

In this study, higher K-Gap (and, therefore, lower DK concentration) was associated to better post-HD CI. None of our patients were hypokalemic before HD. Our findings are consistent with increased myocardial contractility after acute reduction in serum potassium concentration, which are the same conclusions obtained by Brace et al. in a study performed with dogs treated with HD [24]. Such effect may be attributed to an ouabain-like effect of hypokalemia on sodium-potassium adenosine triphosphate of the myocytes [24]. Possibly, higher K-Gap promoted a more pronounced potassium removal, leading to a better myocardial performance at the end of HD session. Despite this favorable hemodynamic effect, it must be highlighted the risks associated to acute reductions in serum potassium on the development of arrhythmias and cardiac arrest during HD [25].

This study has some limitations: it was an observational study and the sample size was relatively small. We also did not directly measure effluent electrolyte concentrations in order to determine electrolyte balance during HD, which could explain some of our findings.

This study has several strengths: first of all, it describes how CI and PAR behave in response to ultrafiltration through a non-invasive assessment. Such behavior, although predictable, had never been described before through this methodology. Another strength was the demonstration that DBic and DK have independent impact over intradialytic hemodynamics. Previous studies only addressed the individual impact of DBic on hemodynamics. Additionally, as far as we are concerned, this is the first study to actually show the impact of acute hypokalemia on cardiac performance in humans under HD.

Conclusion

HD promotes a controlled hypovolemic state, with stroke volume and CI reduction, along with PAR increase in order to sustain BP. Dialysate content directly alters this hemodynamic balance: higher DBic and lower K-Gap lead to more profound post-HD CI reduction.

Disclosure Statement

The authors of this manuscript state that they do not have any conflict of interests and nothing to disclose.

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References

- 1 Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie EG, Chertow GM: Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. *J Am Soc Nephrol* 2004;15:2208-2218.

- 2 Wizemann V, Wabel P, Chamney P, Zaluska W, Moissl U, Rode C, Malecka-Masalska T, Marcelli D: The mortality risk of overhydration in haemodialysis patients. *Nephrol Dial Transplant* 2009;24:1574-1579.
- 3 Chazot C, Wabel P, Chamney P, Moissl U, Wieskotten S, Wizemann V: Importance of normohydration for the long-term survival of haemodialysis patients. *Nephrol Dial Transplant* 2012;27:2404-2410.
- 4 McIntyre CW: Haemodialysis-induced myocardial stunning in chronic kidney disease - a new aspect of cardiovascular disease. *Blood Purif* 2010;29:105-110.
- 5 Gabutti L, Salvadé I, Lucchini B, Soldini D, Burnier M: Haemodynamic consequences of changing potassium concentrations in haemodialysis fluids: BMC Nephrol 2011;12:14.
- 6 Gabutti L, Lucchini B, Marone C, Alberio L, Burnier M: Citrate- vs. acetate-based dialysate in bicarbonate haemodialysis: consequences on haemodynamics, coagulation, acid-base status, and electrolytes: BMC Nephrol 2009;10:7.
- 7 Gabutti L, Bianchi G, Soldini D, Marone C, Burnier M: Haemodynamic consequences of changing bicarbonate and calcium concentrations in haemodialysis fluids. *Nephrol Dial Transplant* 2009;24:973-981.
- 8 Locatelli F, Covic A, Chazot C, Leunissen K, Luño J, Yaqoob M: Optimal composition of the dialysate, with emphasis on its influence on blood pressure. *Nephrol Dial Transplant* 2004;19:785-796.
- 9 Daugirdas JT: Simplified equations for monitoring Kt/V, PCRn, eKt/V, and ePCRn. *Adv Ren Replace Ther* 1995;2:295-304.
- 10 Riddez L, Hahn RG, Brismar B, Strandberg A, Svensén C, Hedenstierna G: Central and regional hemodynamics during acute hypovolemia and volume substitution in volunteers. *Crit Care Med* 1997;25:635-640.
- 11 Kraut JA, Mishler DR, Singer FR, Goodman WG: The effects of metabolic acidosis on bone formation and bone resorption in the rat. *Kidney Int* 1986;30:694-700.
- 12 Gasser JA, Hulter HN, Imboden P, Krapf R: Effect of chronic metabolic acidosis on bone density and bone architecture in vivo in rats. *Am J Physiol Renal Physiol* 2014;306:F517-F524.
- 13 Workeneh BT, Mitch WE: Review of muscle wasting associated with chronic kidney disease. *Am J Clin Nutr* 2010;91:1128S-1132S.
- 14 Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. *Kidney Int* 2013;3:S1-150.
- 15 Oettinger CW, Oliver JC: Normalization of uremic acidosis in hemodialysis patients with a high bicarbonate dialysate. *J Am Soc Nephrol* 1993;3:1804-1807.
- 16 Tentori F, Karaboyas A, Robinson BM, Morgenstern H, Zhang J, Sen A, Ikizler TA, Rayner H, Fissell RB, Vanholder R, Tomo T, Port FK: Association of dialysate bicarbonate concentration with mortality in the Dialysis Outcomes and Practice Patterns Study (DOPPS). *Am J Kidney Dis* 2013;62:738-746.
- 17 Heguilen RM, Sciarano C, Bellusci AD, Fried P, Mittelman G, Rosa Diez G, Bernasconi AR: The faster potassium-lowering effect of high dialysate bicarbonate concentrations in chronic haemodialysis patients. *Nephrol Dial Transplant* 2005;20:591-597.
- 18 Gabutti L, Ferrari N, Giudici G, Mombelli G, Marone C: Unexpected haemodynamic instability associated with standard bicarbonate haemodialysis. *Nephrol Dial Transplant* 2003;18:2369-2376.
- 19 Hou S, McElroy PA, Nootens J, Beach M: Safety and efficacy of low-potassium dialysate. *Am J Kidney Dis* 1989;13:137-143.
- 20 Eisner DA, Lederer WJ: Inotropic and arrhythmogenic effects of potassium-depleted solutions on mammalian cardiac muscle. *J Physiol* 1979;294:255-277.
- 21 Ng YC, Hume JR, Akera T: Paradoxical positive inotropic effect of K⁺ in the rat heart. *Am J Physiol* 1987;252:H1005-H1015.
- 22 Fitzovich DE, Hamaguchi M, Tull WB, Young DB: Chronic hypokalemia and the left ventricular responses to epinephrine and preload. *J Am Coll Cardiol* 1991;18:1105-1111.
- 23 Haddy FJ, Vanhoutte PM, Feletou M: Role of potassium in regulating blood flow and blood pressure. *Am J Physiol Regul Integr Comp Physiol* 2006;290:R546-R552.
- 24 Brace RA, Anderson DK, Chen WT, Scott JB, Haddy FJ: Local effects of hypokalemia on coronary resistance and myocardial contractile force. *Am J Physiol* 1974;227:590-597.
- 25 Santoro A, Mancini E, London G, Mercadal L, Fessy H, Perrone B, Cagnoli L, Grandi E, Severi S, Cavalcanti S: Patients with complex arrhythmias during and after haemodialysis suffer from different regimens of potassium removal. *Nephrol Dial Transplant* 2008;23:1415-1421.