Preventing complications: choosing the optimal dialysate composition

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Goals of acute dialysis

• Removal of uraemic toxins

• Fluid removal

• Electrolyte removal

• Correction of acid-base

• Avoid complications
Overview

• Elements:
  ▪ Sodium
  ▪ Potassium
  ▪ Calcium
  ▪ Magnesium
  ▪ Temperature
Clinical scenarios: sodium

- Decompensated liver disease
  - Salt and water overload
  - Hyponatraemic
  - Hypotension
  - Bleeding risk

- Hypo/hypernatramia

- Neurosurgery or neurotrauma

The sodium gradient between plasma and dialysate is key

Dialysate sodium

1. Stability during HD
   Higher D.Na$^+$ limits reduction in plasma osmolality

2. Thirst and fluid gains
   Higher D.Na$^+$ increases thirst and IDWG

3. Alteration of plasma Na$^+$ concentration
Hyponatremia

- Risk of over-rapid correction of hyponatremia
  - Central pontine myelinosis

Change D.Na$^+$ (max gradient 10mmol/l)

Change dialysis time

Hypotonic infusion

Continuous RRT?

Fig. 1. $T \times 1 =$ first hemodialysis treatment: $Kt/V$: 0.52, urea reduction ratio (URR): 36%; $T \times 2 =$ second hemodialysis treatment: $Kt/V$: 0.4, URR: 29%. Treatment time: 3 hours; blood flow: 50 ml/minute; dialysate sodium: 130 mEq/l for both treatments.

Wendland EM, Kaplan A. Semin Dial 2012; 25(1):
Monitoring conductivity

Petitclerc T. NDT 1999; 14:2607-2613
Diacontrol delivers prescribed plasma CDn

Plasma CDn at start of dialysis

Plasma CDn at end of dialysis

Selby NM et al. ASAIO J 2006
Fixed but individualised dialysate Na

Cross-over study

- Fixed dialysate sodium (138mmol/l)
- Individualised dialysate sodium (matched to pre-HD Na)

- Aim of individualised: No gradient between plasma and dialysate Na

- Delta plasma Na:
  - 1.9mmol/l (fixed)
  - 0.9mmol/l (Individ)

De Paula et al. KI 2004; 66: 1232
Stability: sodium profiling

Impact of Sodium and Ultrafiltration Profiling on Hemodialysis-Related Symptoms

MATTHEW J. OLIVER,* LLOYD J. EDWARDS,1 and DAVID N. CHURCHILL1
*McGill University Health Centre, Toronto, Ontario, Canada; 1School of Public Health, The University of North Carolina at Chapel Hill, Chapel Hill, North Carolina; and 2St. Joseph’s

Table 2. Hypotension, symptomatic events, and interventions during dialysis treatments according to dialysis profile

<table>
<thead>
<tr>
<th>Event</th>
<th>Standard</th>
<th>Profiled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms† [No. (%)]</td>
<td>59 (31)</td>
<td>37 (20)</td>
</tr>
<tr>
<td>Hypotension</td>
<td>3.7 (20)</td>
<td>2.4 (13)</td>
</tr>
</tbody>
</table>
†The odds of experiencing hypotension or a symptomatic event during profiled treatments was 0.61 (95% CI, 0.39 to 0.96) compared with standard treatments.
‡The odds of receiving an intervention during a profiled treatment was 0.62 (95% CI, 0.38 to 0.99) compared with standard dialysis.

Variable sodium dialysate in ARF

- Sodium profiling in dialysis in critically ill patients

Oliver MJ et al. JASN 2001 Jan;12(1):151

Paganini EP NDT 199
Sodium profiling

**Impact of Sodium and Ultrafiltration Profiling on Hemodialysis-Related Symptoms**

MATTHEW J. OLIVER,* LLOYD J. EDWARDS,† and DAVID N. CHURCHILL‡

*Sunnybrook & Women’s College Health Sciences Centre, Toronto, Ontario, Canada; †School of Public Health, The University of North Carolina at Chapel Hill, Chapel Hill, North Carolina; and ‡St. Joseph’s Health Care, Hamilton, Ontario, Canada.

**Table 4.** Patient weights, ultrafiltration volume, serum sodium, and urea reduction ratios according to dialysis treatment profile

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Standard</th>
<th>Profiled</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predialysis weight (kg)</td>
<td>71.7</td>
<td>72.1</td>
</tr>
<tr>
<td>Postdialysis weight (kg)</td>
<td>69.1</td>
<td>69.2</td>
</tr>
<tr>
<td>Net ultrafiltration (L)</td>
<td>2.6</td>
<td>2.9</td>
</tr>
<tr>
<td>Predialysis supine BP (mmHg)</td>
<td>151/79</td>
<td>149/78</td>
</tr>
<tr>
<td>Postdialysis supine BP (mmHg)</td>
<td>134/74</td>
<td>137/76</td>
</tr>
<tr>
<td>Predialysis sodium (mmol/L)</td>
<td>137.0</td>
<td>137.3</td>
</tr>
<tr>
<td>Postdialysis sodium (mmol/L)</td>
<td>139.7</td>
<td>141.0</td>
</tr>
<tr>
<td>Urea reduction ratio (%)</td>
<td>69.9</td>
<td>69.9</td>
</tr>
</tbody>
</table>

*P = 0.008.

†P = 0.002.

‡P = 0.001.

Mortality risk and fluid gains

Important scenarios - dialysate

\[ K^+ \]

- Increased K generation
- Nutrition
- Cardiovascular disease
- Digoxin (use or poisoning)
Digoxin and potassium

- Cohort study of 120,864 patients
- Digoxin use was associated with increase in crude mortality
- Serum levels (not dose) were the important predictor

Analysis included:
- Pt characteristics
- Propensity for treatment
- Disease severity (NYHA class)
- Time

Chan KE et al. JASN 2010;21:1550-1559
Dialysate potassium

- Normalise serum $K^+$
  → Depends on pre-dialysis $K^+$

- Avoid arrhythmias

- Take account of inter-dialytic $K^+$ generation

- Remember effects of dialysate glucose

Pun PH et al. KI 2011; 79: 218
Factors associated with SCD

<table>
<thead>
<tr>
<th>Patient factors</th>
<th>Dialysis related factors</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Recent hospitalisation</td>
<td>• Fall in BP</td>
</tr>
<tr>
<td>• Age</td>
<td>• Myocardial ischaemia</td>
</tr>
<tr>
<td>• Diabetes</td>
<td>• Dialysate potassium</td>
</tr>
<tr>
<td>• CV disease</td>
<td>• Serum potassium</td>
</tr>
<tr>
<td>• Medication - digoxin</td>
<td>• Dialysate calcium</td>
</tr>
<tr>
<td>• Venous catheter use</td>
<td>(&lt;1.25mmol/l)</td>
</tr>
</tbody>
</table>

Pun PH et al. KI 2011; 79: 218
Karnik JA et al. KI 2001; 60: 35
Myocardial ischaemia and arrhythmias

Munger MA et al. AJKD 2000; 36(1): 130
Potassium and sudden cardiac death

<table>
<thead>
<tr>
<th>Dialysate $K^+$</th>
<th>0-1mmol/l</th>
<th>≥2mmol/l</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence SCD</td>
<td>17.1%</td>
<td>8.8%</td>
</tr>
</tbody>
</table>

Karnik JA et al. KI 2001; 60: 350

<table>
<thead>
<tr>
<th>SCD</th>
<th>Controls</th>
</tr>
</thead>
<tbody>
<tr>
<td>Serum $K^+$</td>
<td>4.78±0.9</td>
</tr>
</tbody>
</table>

Pun PH et al. KI 2011; 79: 218
Interaction between serum $K^+$ and dialysate $K^+$ conc.

Pun PH et al. KI 2011; 79: 218
<table>
<thead>
<tr>
<th>Pre dialysis serum K⁺</th>
<th>Suggested dialysate K⁺</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;4.0 mmol/l</td>
<td>4.0 mmol/l</td>
</tr>
<tr>
<td>4.0-4.5 mmol/l</td>
<td>3.5 mmol/l</td>
</tr>
<tr>
<td>4.5-5.5 mmol/l</td>
<td>2.0 to 3.0 mmol/l</td>
</tr>
<tr>
<td>5.5-8.0 mmol/l</td>
<td>2.0 mmol/l (use 2.5-3.0 mmol/l if digoxin)</td>
</tr>
<tr>
<td>&gt;8.0 mmol/l</td>
<td>1.0 mmol/l</td>
</tr>
</tbody>
</table>
Potassium profiling

Santoro A et al. NDT 2008;23:1415
Dialysate calcium

- Low calcium dialysate associated with SCD (<1.25mmol/l)
  Karnik JA et al. KI 2001; 60: 350

- High calcium dialysate improves BP (1.75mmol/l)
  Kriazis et al. KI 2002; 61(1): 276

- Risk of hypercalcaemia (17%)

IDH 6.9% versus 18-20% with lower D.Ca^{2+}
Calcium and mortality

• Epidemiological studies: hypocalcaemia assoc. with worse outcomes

• Administering Ca$^{2+}$ to rats with septic shock worsened mortality

Malcolm DS Crit Care Med 1989; 17: 900
Magnesium and haemodialysis: potential benefits

- Serum magnesium predicts mortality in HD patients

- Potential mechanisms:
  - Direct
    - Low serum magnesium (<0.8mmol/L) associated with prolonged QT interval and increased arrhythmias
    - Coronary vasomotor tone
    - Myocardial ischaemia
  - Indirect
    - Reduction in intradialytic haemodynamic instability, with secondary reduction in HD-induced acute cardiac injury

Ishimura et al, 2007; Wu et al, 1991; Kyriasis et al, 2004
**0.5 versus 1.0mmol/l dialysate Mg²⁺**

<table>
<thead>
<tr>
<th>Dialysate Mg</th>
<th>0.5mmol/l</th>
<th>1.0mmol/l</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre dialysis Mg (mmol/L)</td>
<td>1.05 0.13</td>
<td>1.42 0.20</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Delta Mg (mmol/L)</td>
<td>-0.19 0.09</td>
<td>+0.02 0.13</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Fall in BP (mmHg)</td>
<td>-46 27</td>
<td>-32 30</td>
<td>0.52</td>
</tr>
<tr>
<td>No. of dysfunctional LV regions</td>
<td>3.4 1.9</td>
<td>3.7 1.9</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Jefferies HJ and McIntyre CW
BRS conference 2010
Temperature

- Key role in dialysis stability

- Effective in dialysis and CVVH

Rokyta R. NDT 2004

Selby NM & McIntyre CW. NDT 2006;21:1883
Reduction in RWMA number and severity with cooled dialysate

Selby NM, Burton JO, Chesterton LJ, McIntyre CW. Clin J Am Soc Nephrol
Buffer mass balance in bicarbonate dialysis

<table>
<thead>
<tr>
<th></th>
<th>Blood</th>
<th>Dialysate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetate</td>
<td>0</td>
<td>4 (3-9)</td>
</tr>
<tr>
<td>$\text{HCO}_3^-$</td>
<td>20</td>
<td>30 (28-38)</td>
</tr>
</tbody>
</table>

$\Delta = 4$
$\Delta = 10$
Total $\Delta = 14$
Original Articles

Effects of Acetate-Free Double-Chamber Hemodiafiltration and Standard Dialysis on Systemic Hemodynamics and Troponin T Levels

Nicholas M. Selby, Richard J. Fluck, Maarten W. Taal, and Christopher W. McIntyre

Journal of Artificial Organs
Volume 14, Number 2, 112-119, DOI: 10.1007/s10047-010-0551-7

Acetate-free blood purification can impact improved nutritional status in hemodialysis patients

Kazuhiro Matsuyama, Tadashi Tomo and Jun-ichi Kadota
Summary

• Flexibility in dialysate prescription for IHD

• Individualise to specific clinical situation