Acute kidney failure

E. Fiaccadori (IT)
Nutrition support in renal disease

Acute Kidney Injury

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Presentation overview

- Epidemiological aspects and pathogenesis of protein-energy wasting (PEW) in AKI
- Nutrient needs in patients with AKI
- Integration between artificial nutrition and renal replacement therapy (RRT) in patients with AKI
## AKI: diagnosis and staging

<table>
<thead>
<tr>
<th>Stage</th>
<th>Serum creatinine</th>
<th>Urine output</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1.5–1.9 times baseline or ≥0.3 mg/dl (≥26.5 μmol/l) increase</td>
<td>&lt;0.5 ml/kg/h for 6–12 hours</td>
</tr>
<tr>
<td>2</td>
<td>2.0–2.9 times baseline</td>
<td>&lt;0.5 ml/kg/h for ≥12 hours</td>
</tr>
<tr>
<td>3</td>
<td>3.0 times baseline or Increase in serum creatinine to ≥4.0 mg/dl (≥353.6 μmol/l) or Initiation of renal replacement therapy or In patients &lt;18 years, decrease in eGFR to &lt;35 ml/min per 1.73 m²</td>
<td>&lt;0.3 ml/kg/h for ≥24 hours or Anuria for ≥12 hours</td>
</tr>
</tbody>
</table>

In stage 3 AKI, dialysis/hemofiltration need is likely, especially in ICU patients.
Epidemiology and prognosis of AKI: Three major problems

• Incidence is high
• Incidence is increasing
• Negative impact on short- and long-term outcome
# World Incidence of AKI: A Meta-Analysis

Pawee Na Suwitapong,† Dinna N. Cruz,§ Jorge Cerda,¶ Mahir Abafaraj,* Fahad Alqahtani,* Ioannis Koulouridis,* and Bertrand L. Jaber,*† for the Acute Kidney Injury Advisory Group of the American Society of Nephrology

## Table 2. Pooled incidence rate of AKI according to the KDIGO-equivalent definition

<table>
<thead>
<tr>
<th>Subgroup</th>
<th>Studies (n)</th>
<th>Patients (n)</th>
<th>Patients with AKI (n)</th>
<th>AKI Incidence Rate (%)</th>
<th>95% Confidence Interval</th>
<th>Test for Heterogeneity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>All</td>
<td>154</td>
<td>3,585,911</td>
<td>573,424</td>
<td>23.2</td>
<td>21.0 to 25.7</td>
<td>99.9</td>
</tr>
<tr>
<td>Age category</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adults</td>
<td>130</td>
<td>3,571,691</td>
<td>569,861</td>
<td>21.6</td>
<td>19.3 to 24.1</td>
<td>99.9</td>
</tr>
<tr>
<td>Children</td>
<td>24</td>
<td>14,220</td>
<td>3563</td>
<td>33.7</td>
<td>26.9 to 41.3</td>
<td>98.3</td>
</tr>
<tr>
<td>Clinical setting</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Community acquired</td>
<td>7</td>
<td>548,398</td>
<td>4897</td>
<td>8.2</td>
<td>1.6 to 33.0</td>
<td>99.9</td>
</tr>
<tr>
<td>Critical care</td>
<td>41</td>
<td>888,604</td>
<td>272,580</td>
<td>31.7</td>
<td>28.6 to 35.0</td>
<td>99.7</td>
</tr>
<tr>
<td>Cardiac surgery</td>
<td>42</td>
<td>164,333</td>
<td>33,157</td>
<td>24.8</td>
<td>20.4 to 28.8</td>
<td>98.7</td>
</tr>
<tr>
<td>Trauma</td>
<td>4</td>
<td>14,947</td>
<td>2557</td>
<td>19.9</td>
<td>13.6 to 28.2</td>
<td>—</td>
</tr>
<tr>
<td>Heart failure</td>
<td>1</td>
<td>682</td>
<td>221</td>
<td>32.4</td>
<td>29.0 to 36.0</td>
<td>—</td>
</tr>
<tr>
<td>Hematology/oncology</td>
<td>3</td>
<td>2401</td>
<td>453</td>
<td>21.3</td>
<td>7.5 to 47.6</td>
<td>99.9</td>
</tr>
<tr>
<td>Nephrotoxins</td>
<td>4</td>
<td>17,786</td>
<td>1681</td>
<td>12.2</td>
<td>6.2 to 22.7</td>
<td>98.7</td>
</tr>
<tr>
<td>Hospital acquired, unspecified</td>
<td>52</td>
<td>1,948,760</td>
<td>257,878</td>
<td>20.9</td>
<td>17.2 to 25.2</td>
<td>99.9</td>
</tr>
</tbody>
</table>

\[ n = 3.585.911 \]
Hospitalizations for acute kidney injury, with or without dialysis

Percent of patients

- **AKI**
- **AKI & dialysis**

<table>
<thead>
<tr>
<th>Year</th>
<th>AKI</th>
<th>AKI &amp; Dialysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>1995</td>
<td>0.2</td>
<td>0.2</td>
</tr>
<tr>
<td>1998</td>
<td>0.4</td>
<td>0.4</td>
</tr>
<tr>
<td>2001</td>
<td>0.6</td>
<td>0.6</td>
</tr>
<tr>
<td>2004</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>2007</td>
<td>1.5</td>
<td>1.5</td>
</tr>
</tbody>
</table>

**AKI: an epidemic?**
AKI has negative effects on outcome

Epidemiology and Outcomes of Acute Renal Failure in Hospitalized Patients: A National Survey

- Review of discharge data on a projected total of 29,039,599 hospitalizations
- 558,032 cases of ARF identified (192/100,000 hospitalizations)

ARF diagnosis on the basis of a ICD-9-CM code in discharge records

Artificial nutrition in AKI: A difficult task

- Dysmetabolism of critical illness worsened by the acute loss of kidney homeostatic function

- Nutritional approach made difficult by the complexity of the syndrome itself and by the frequent need of renal replacement therapy (RRT)

- No data from RCTs

- Clinical practice mostly based on expert’s opinions
ESPEN Guidelines on Enteral Nutrition: Adult Renal Failure

N. Cano\textsuperscript{a,b,\*}, E. Fiaccadori\textsuperscript{b}, P. Tesinsky\textsuperscript{c}, G. Toigo\textsuperscript{d}, W. Druml\textsuperscript{e}, DGEM: \textsuperscript{\*\*} M. Kuhlmann, H. Mann, W.H. Hörl

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\textsuperscript{b}Department of Clinical Medicine and Nephrology, Università degli Studi di Parma, Parma, Italy
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Received 20 January 2006; accepted 20 January 2006

ESPEN Guidelines on Parenteral Nutrition: Adult Renal Failure

N.J.M. Cano\textsuperscript{a,b,c}, M. Aparicio\textsuperscript{d}, G. Brunori\textsuperscript{e}, J.J. Carrero\textsuperscript{f}, B. Cianciaruso\textsuperscript{g}, E. Fiaccadori\textsuperscript{h}, B. Lindholm\textsuperscript{f}, V. Teplan\textsuperscript{i}, D. Fouque\textsuperscript{j}, G. Guarnieri\textsuperscript{k}
No major differences as compared to the 2006-2009 ESPEN guidelines
A proposed nomenclature and diagnostic criteria for protein–energy wasting in acute and chronic kidney disease

D Fouque1,17, K Kalantar-Zadeh2,17, J Kople1, N Cano3, P Chauveau4, L Cuppari5, H Franch6, G Guarnieri7, TA Ikizler8, G Kaysen9,10, B Lindholm11, Z Massy12,13, W Mitch14, E Pineda15, P Stenvinkel11, A Treviso-Becerra15 and C Wanner16

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Diagnosis of PEW when at least one parameter is found below recommendation in three of the four nutritional variable groups.
A major open problem in AKI is the lack of adequate tools for nutritional status evaluation at the individual level, and for monitoring of the effects of nutritional support.

<table>
<thead>
<tr>
<th>Nutritional parameters</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin, prealbumin, cholesterol</td>
<td>They can be reduced even independently from PEW (negative markers of inflammation)</td>
</tr>
<tr>
<td>Lymphocyte count</td>
<td>Low specificity</td>
</tr>
<tr>
<td>Body weight (BW) changes</td>
<td>Total body water increased in AKI</td>
</tr>
<tr>
<td>Fluid overload can mask lean body mass changes</td>
<td></td>
</tr>
<tr>
<td>Anthropometry (triceps skinfold, midarm circumference, etc.)</td>
<td>Interference by arm edema</td>
</tr>
<tr>
<td>Protein catabolic rate (PCR) or protein equivalent of nitrogen appearance (PNA)</td>
<td>Measurement requires calculations based on urea kinetic during RRT + dialysis fluid collection/proportional sampling</td>
</tr>
<tr>
<td>Energy expenditure (EE)</td>
<td>Formulas for EE prediction not always reliable in critically ill patients (often based on body weight)</td>
</tr>
</tbody>
</table>

CT and/or MRI: No data in AKI

PEW, protein-energy wasting; RRT, renal replacement therapy; SGA, Subjective Global Assessment.
PEW is associated with high mortality in AKI

In-hospital mortality according to nutritional status in 309 AKI pts

Nutritional status by SGA (Subjective Global Assessment of nutritional status, Baker JP et al., NEJM 1982; 306:969-72)

Fiaccadori E et al., JASN 1999; 10:581-93
Catabolism

Nutrient losses on RRT

Inadequate intakes

Why PEW in AKI?

PEW in AKI patients is the result of the coexistence of two kinds of pathogenetic factors: those common to the other critically ill patients and those specific of renal patients
Pathogenesis of PEW in AKI is complex

Associated conditions (prolonged critical illness, hospital-associated PEW, chronic comorbidities)

Immobilization

Acute critical illness (metabolic stress)

- Anorexia and starvation
- Delayed starting of nutritional support
- Insufficient EN without PN supplementation
- Low protein intake to delay RRT start
- Problems in the calculation of nutrient needs

Acute Kidney Injury

Metabolic Acidosis

Catabolism, lean body mass wasting

Inadequate delivery of proteins and calories

Protein-energy debt

- Increased mortality
- Increased morbidity
- Increased LOS
- Increased health resource utilization

PEW

Fiaccadori E et al., Curr Opin Clin Nutr Metab Care 2013; 16:217-224
Amino acids have small molecular weight (average 140 Daltons, range 75-215)

RRT can be associated with the loss of up to 10–20 g amino acids in each session, depending on RRT modality and filter type

With CRRT 10 to 15% of infused amino acids are lost every day (up to 15-20% in the case of glutamine, 0.5-6.8 g/day when supplementation is 0.32 g/Kg/day)

Kihara M et al., Int Care Med 1997; 23:110-13
Kuhlmann MK et al., Anaesthetist 2000; 49:353-8
Berg A et al., Int Care Med 2007; 33:660-6
Btaiche EF et al., Pharmacotherapy 2008; 28:600-613
• Protein needs
• Energy needs
Protein catabolic rate in critically ill patients with AKI on RRT

Macias WL et al, J Parent Ent Nutr 1996; 20:56
Fiaccadori E et al., Nephrol Dial Transpl 2005; 20:1976
Measured REE in AKI not different from that of the other ICU pts

REE in AKI patients

1835 Kcal/69 Kg

= 27 Kcal/Kg/day

Faisy C et al., Am J Clin Nutr 2003; 78:241-9
Energy and protein intake interactions on N balance

Best compromise for N balance at 25-30 Kcal/Kg and 1.5 g/Kg/proteins → calorie/nitrogen ratio about 100

Macias WL et al., JPEN 1996; 20:56-62
Modulation of nitrogen balance in patients with AKI

- Any advantage in increasing Kcal intake?
- Any advantage in increase N intake?
No advantages on N balance in increasing calories in AKI

Problems with high calorie regimens in AKI patients

- Increased fluid administration
- Increased insulin needs
- High glucose levels

**Higher AA/Protein intake in AKI?**

<table>
<thead>
<tr>
<th>Patient Population</th>
<th>CRRT Modality and Effluent Rate</th>
<th>Amino Acid and Caloric Intake</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults (surgical or medical): cohort 1 (n=24), cohort 2 (n=16)</td>
<td>CVVHD, CAVHD; 1 or 2 L/hr</td>
<td>Parenteral nutrition: Cohort 1: amino acids 1.2 (range 0.41–2.4) g/kg/day, Cohort 2: amino acids 2.5 g/kg/day; nonprotein calories 30–35 kcal/kg/day</td>
<td>High amino acid intake resulted in less negative nitrogen balance (-1.92 g/day) vs low amino acid intake (-5.5 g/day, p=0.176) Effluent daily amino acid losses similar between groups Survival similar between groups High amino acid intake caused higher plasma urea concentrations, requiring more aggressive dialysis</td>
</tr>
<tr>
<td>Adults with MODS (n=7)</td>
<td>CVVHDF; 1 or 2 L/hr</td>
<td>Parenteral nutrition: amino acids 2.5 g/kg/day, nonprotein calories 35 kcal/kg/day</td>
<td>Median amino acid losses of 12 g/day (5–21% of daily amino acid intake) Median nitrogen balance -1.8 g/day (range -21 to +17.9 g/day) Positive nitrogen balance 35% of time No effect on improvement in patient outcome</td>
</tr>
</tbody>
</table>

No clear advantages in increasing AA/protein intake to 2.5 g/Kg/day or more

Bellomo R et al., Ren Fail 1997; 19:111-20
Bellomo R et al., Int J Artif Org 2002; 25:261-8
**Energy metabolism in acute and chronic renal failure**


Resting energy expenditure and energy expenditure of major fuels as determined by indirect calorimetry*

<table>
<thead>
<tr>
<th></th>
<th>Control subjects (n = 24)</th>
<th>Acute renal failure with septicemia (n = 18)</th>
<th>Acute renal failure without septicemia (n = 11)</th>
<th>Chronic renal failure, conservative treatment (n = 17)</th>
<th>Severe untreated azotemia (n = 15)</th>
<th>Chronic renal failure, hemodialysis (n = 25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temperature (°C)</td>
<td>36.3 ± 0.06</td>
<td>37.7 ± 0.21†</td>
<td>36.7 ± 0.12</td>
<td>36.5 ± 0.17</td>
<td>36.3 ± 0.13</td>
<td>36.6 ± 0.09</td>
</tr>
<tr>
<td>( \dot{V}O_2 ) (mL·min(^{-1})·1.73 m(^{-2}))</td>
<td>201.2 ± 4.2</td>
<td>270.8 ± 9.4†</td>
<td>222.1 ± 15.8</td>
<td>190.8 ± 5.8</td>
<td>211.9 ± 7.3</td>
<td>216.6 ± 8.9</td>
</tr>
<tr>
<td>( \dot{V}CO_2 ) (mL·min(^{-1})·1.73 m(^{-2}))</td>
<td>168.7 ± 4.6</td>
<td>213.0 ± 7.5†</td>
<td>174.5 ± 10.9</td>
<td>156.9 ± 5.2</td>
<td>168.5 ± 6.1</td>
<td>172.1 ± 6.3</td>
</tr>
<tr>
<td>RQ (mg·min(^{-1})·1.73 m(^{-2}))</td>
<td>0.84 ± 0.01</td>
<td>0.79 ± 0.01†</td>
<td>0.79 ± 0.02</td>
<td>0.82 ± 0.02</td>
<td>0.80 ± 0.01†</td>
<td>0.80 ± 0.01†</td>
</tr>
<tr>
<td>UN appearance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(mg·min(^{-1})·1.73 m(^{-2}))</td>
<td>7.25 ± 0.37</td>
<td>8.72 ± 0.79</td>
<td>7.88 ± 0.69</td>
<td>5.00 ± 0.34†</td>
<td>4.06 ± 0.48†</td>
<td>5.05 ± 0.31†</td>
</tr>
<tr>
<td>npRQ (mg·min(^{-1})·1.73 m(^{-2}))</td>
<td>0.84 ± 0.01</td>
<td>0.79 ± 0.02†</td>
<td>0.79 ± 0.03</td>
<td>0.83 ± 0.02</td>
<td>0.79 ± 0.01†</td>
<td>0.80 ± 0.01†</td>
</tr>
<tr>
<td>BEE (kcal·min(^{-1})·1.73 m(^{-2}))</td>
<td>1.04 ± 0.01</td>
<td>1.00 ± 0.02†</td>
<td>0.99 ± 0.03</td>
<td>0.96 ± 0.02†</td>
<td>0.98 ± 0.03†</td>
<td>0.97 ± 0.02†</td>
</tr>
<tr>
<td>REE (kcal·min(^{-1})·1.73 m(^{-2}))</td>
<td>0.96 ± 0.02</td>
<td>1.28 ± 0.04†</td>
<td>1.05 ± 0.07</td>
<td>0.91 ± 0.03</td>
<td>1.01 ± 0.04</td>
<td>1.03 ± 0.04</td>
</tr>
<tr>
<td>Fat oxidation rate (% of REE)</td>
<td>41.0 ± 2.7</td>
<td>59.2 ± 5.1†</td>
<td>56.3 ± 8.4</td>
<td>49.1 ± 6.8</td>
<td>61.2 ± 3.2†</td>
<td>58.3 ± 4.5†</td>
</tr>
<tr>
<td>CHO oxidation rate (% of REE)</td>
<td>38.5 ± 3.0</td>
<td>22.6 ± 5.1†</td>
<td>23.0 ± 8.2</td>
<td>35.8 ± 6.4</td>
<td>27.5 ± 3.4†</td>
<td>27.9 ± 4.3</td>
</tr>
<tr>
<td>Pro oxidation rate (% of REE)</td>
<td>20.5 ± 1.1</td>
<td>18.2 ± 1.3†</td>
<td>20.7 ± 1.7</td>
<td>15.1 ± 1.2†</td>
<td>11.3 ± 1.6†</td>
<td>13.8 ± 1.1†</td>
</tr>
</tbody>
</table>

*\( \bar{x} \pm \text{SEM} \), \( \dot{V}O_2 \), oxygen consumption; \( \dot{V}CO_2 \), carbon dioxide production; RQ, respiratory quotient; UN appearance, urinary nitrogen appearance rate; npRQ, nonprotein respiratory quotient; BEE, basal energy expenditure; REE, resting energy expenditure; Fat oxidation rate, percentage of total REE derived from fat; CHO oxidation rate, percentage of total REE derived from carbohydrate; and Pro oxidation rate, percentage of total REE derived from protein.

†Significantly different from control subjects, \( P < 0.05 \).

Glucose oxidation rate is reduced and fat oxidation rate is increased in AKI

Quantitative and qualitative aspects of nutrient intake in AKI patients on RRT

- **Proteins**: at least 1.5 g/Kg/day (+ 0.2 g/kg/day, taking into account also that about 10–15% of infused amino acids in PN during RRT are lost in the dialysate/ultrafiltrate)
- **Aminoacids**: EAA+NEAA
- **Energy**: not more than 25 Kcal/Kg/day (non protein), 2/3 of nonprotein calories as glucose (not > 5 g/Kg/day) and 1/3 as lipids (1-1.5 g/Kg/day, 18-24 hour infusion)

Total body water is increased in AKI → actual BW is not the right reference for nutrient calculations

Urea space and total body water measurements by stable isotopes in patients with acute renal failure

T. Alp Ikizler, M. Tugrul Sezer, Paul J. Flakoll, Sree Hariachar, N. Suren Kanagasundaram, Nancy Gritter, Stephanie Knights, Yu Shyr, Emil Paganini, Raymond M. Hakim, and Jonathan Himmelfarb for the PICARD Study Group

Vanderbilt University Medical Center, Division of Nephrology, Nashville, Tennessee; Vanderbilt University Medical Center, Department of Surgery, Nashville, Tennessee; Cleveland Clinic Foundation, Division of Nephrology, Cleveland, Ohio; Maine Medical Center, Division of Nephrology, Portland, Maine; and Vanderbilt University Medical Center, Division of Biostatistics, Nashville, Tennessee

• Trace elements
• Vitamins
• Electrolytes
<table>
<thead>
<tr>
<th>Micronutrient</th>
<th>ARF</th>
<th>CRRT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trace elements</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Zinc</td>
<td>↓</td>
<td>0 à ↑</td>
</tr>
<tr>
<td>Selenium</td>
<td>↓</td>
<td>↓↓</td>
</tr>
<tr>
<td>Copper</td>
<td>—</td>
<td>↓↓</td>
</tr>
<tr>
<td>Chromium</td>
<td></td>
<td>↓↓</td>
</tr>
<tr>
<td>Manganese</td>
<td>↑ (controversial)</td>
<td>↓↓</td>
</tr>
<tr>
<td>Iron</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Nickel</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Vitamins</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Folic acid (B&lt;sub&gt;9&lt;/sub&gt;)</td>
<td>—</td>
<td>↓</td>
</tr>
<tr>
<td>Pyridoxal phosphate (B&lt;sub&gt;6&lt;/sub&gt;)</td>
<td>↓</td>
<td>↓</td>
</tr>
<tr>
<td>Thiamine (B&lt;sub&gt;1&lt;/sub&gt;)</td>
<td>↓</td>
<td>↓↓</td>
</tr>
<tr>
<td>Vitamin C</td>
<td>↓ (controversial)</td>
<td>↓</td>
</tr>
<tr>
<td>Vitamin A</td>
<td>↑</td>
<td>—</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>↓</td>
<td>—</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>↓</td>
<td>↓</td>
</tr>
</tbody>
</table>

↓, decrease; ↑, increase.

• In general, daily supplementation with standard doses of parenteral multitrace element preparations results in enough trace elements to overcome the amount lost by CRRT

• No data on whether multitrace element preparations give the patients on CRRT the optimal dose of trace elements
In ICU patients with AKI, plasma levels of water-soluble vitamins, such as vitamin C, thiamine and folic acid, may be lower than normal [51, 56], due mainly to the losses occurring through the extracorporeal circuit: in CVVH vitamin C losses can reach up to 600 μmol/day, i.e. 100 mg/day, and folate losses up to 600 nmol/day [51–56]; in CVVHDF thiamine losses may amount more than 1.5 times the daily provision of the vitamin from standard total parenteral nutrition solutions.

Recommended vitamin C administration in patients with AKI is 50–100 mg/day; higher intakes (up to 150–200 mg) may be needed when continuous modalities of RRT are used. No supplementation of fat-soluble vitamins is usually necessary in AKI.

Fiaccadori E et al., NDT Plus 2010, 3:1-7
Problems with highly efficient RRT modalities (CRRT and SLED)

• Many important substances are removed by the treatment
• → Risk for hypophosphatemia and hypomagnesiemia
• Low risk for hypokalemia (SLED dialysis fluid or CRRT fluids contain potassium at 4 mmoles/L)
Artificial nutrition and outcome in AKI patients
Available studies on artificial nutrition and outcome in AKI: many methodological problems!

- Few patients
- Suboptimal selection of patients
- Population and syndrome heterogeneity
- No stratification for severity of illness
- No stratification for nutritional status
- Use of historical controls or no controls at all
- In most cases retrospective studies
- Quantitative inadequacy of Kcal and/or N intake
- Qualititative inadequacy of Kcal and/or N intake
- Inadequate duration of nutritional support
Artificial nutrition likely to be beneficial in AKI

Effect of acute renal failure requiring renal replacement therapy on outcome in critically ill patients*

Philipp G. H. Metnitz, MD, PhD, DEAA; Claus G. Krenn, MD; Heinz Steltzer, MD; Thomas Lang, PhD; Jürgen Polder, MS; Kurt Lenz, MD; Jean-Roger Le Gall, MD; Wilfred Druml, MD

Table 5. Multivariate predictors: Results of stepwise logistic regression analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>Estimate</th>
<th>p Value</th>
<th>Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>−2.58460</td>
<td>&lt;.001</td>
<td></td>
</tr>
<tr>
<td>SAPS II score</td>
<td>0.00798</td>
<td>.166</td>
<td>1.08</td>
</tr>
<tr>
<td>Cardiopulmonary resuscitation</td>
<td>0.06160</td>
<td>.004</td>
<td>1.86</td>
</tr>
<tr>
<td>Multiple vasoactive medication</td>
<td>0.02930</td>
<td>&lt;.001</td>
<td>1.34</td>
</tr>
<tr>
<td>Mechanical ventilation</td>
<td>0.02930</td>
<td>&lt;.001</td>
<td>1.34</td>
</tr>
<tr>
<td>Single vasoactive medication</td>
<td>0.01160</td>
<td>.012</td>
<td>1.13</td>
</tr>
<tr>
<td>Treatment of complicated metabolic acidosis/alkalosis</td>
<td>0.00768</td>
<td>.084</td>
<td>1.08</td>
</tr>
<tr>
<td>Total enteral nutrition</td>
<td>−0.00883</td>
<td>.002</td>
<td>0.91</td>
</tr>
<tr>
<td>Enteral nutrition</td>
<td>−0.01480</td>
<td>&lt;.001</td>
<td>0.86</td>
</tr>
</tbody>
</table>

SAPS, Simplified Acute Physiology Score.

Variables express the proportion of days on which this activity was performed. The odds ratios reflect the change in the risk of dying during the intensive care unit stay if the proportion of days with intervention increases by 10%.
Decision tree for nutritional support in AKI patients with PEW or at risk of PEW

Normal GI tract function?

- **YES**
  - Enteral feeding
  - Are nutritional goals achieved?
    - **NO**
      - Integration with parenteral feeding
    - **YES**

- **NO**
  - Parenteral feeding
    - Peripheral (short-term, with or without fluid restriction)
    - Central (long-term, fluid restriction, catabolism)
Enteral nutrition as the preferred modality in AKI nutritional support

Enteral nutrition in patients with acute renal failure

Enrico Fiaccadori, Umberto Maggiore, Roberto Giacosa, Carlo Rotelli, Edoardo Picetti, Sibilla Sagripanti, Luigi Melfa, Tiziana Meschi, Loris Borghi, and Aderville Cabassi

Dipartimento di Clinica Medica, Nefrologia & Scienze della Prevenzione, Dipartimento di Scienze Cliniche Università degli Studi di Parma, Italy; and Servizio Anestesia & Rianimazione Azienda Ospedaliera, Parma, Italy

Enteral nutrition safe in ARF patients: no clinically relevant increase in complications (increased gastric residuals common)

A combination of enteral and parenteral support often needed to reach the targeted intake of proteins

The key role of the integration between artificial nutrition and renal replacement therapy (RRT) in AKI

daily RRT as hemodialysis (HD), sustained low efficiency dialysis (SLED) or continuous renal replacement therapy (CRRT) allows better nutritional support!
Prescribed vs administered nutrients in AKI pts on daily SLED during TPN

- 35 consecutive ICU patients with oliguric AKI
- APACHE II score at SLED start 27.4 (20 – 43)
- 8-10 hours a day (mean 9 hours)

Fiaccadori E et al., JASN 2002; 13:604A
Summary

a) Protein-Energy Wasting is frequent in AKI and represents an independent predictor of mortality and morbidity

b) At the present time no definitive demonstration from RCTs is available concerning the positive effects of artificial nutrition on prognosis in AKI patients; however, artificial nutrition should be considered a key component of therapeutic strategy of the syndrome

c) Energy at not more than 25-30 Kcal/Kg/day and proteins at 1.5-2 g/Kg/day should be provided in critically ill patients with AKI on RRT

d) Enteral nutrition should be the initial modality for artificial nutrition in AKI; in most cases it should be integrated with PN

e) More adequate nutritional support with daily RRT
Backup slides
Nutritional support for acute kidney injury

Yi Li², Xi Tang², Juqian Zhang², Taixiang Wu¹

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Cochrane Database of Systematic Reviews 2010,

No enough (and good) data for a systematic review!

8 studies
257 patients
6 studies 1978-1983
1 study 2005
1 study 2007 (no RRT)

Implications for practice

There is no strong evidence to conclude that ELAA, high calorie-TPN, high-dose amino acids or nitrogen and fat improves the survival and recovery from AKI in critically ill patients.
New RRT modalities: Protein and AA losses are increased with the use of high cut-off membranes for sepsis treatments

World Incidence of AKI: A Meta-Analysis

Pawee Boonpitapong,*,†‡ Dinna N. Cruz,§ Jorge Cerda,‖ Maher Abulfaraj,* Fahad Alqhtani,* Ioannis Koulouridis,*† and Bertrand L. Jaber,*† for the Acute Kidney Injury Advisory Group of the American Society of Nephrology

n = 3.585.911