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Should a Renal Dose of Norepinephrine Stimulate Hyperfiltration in Head Trauma Patients?

To the Editor:

In the August 2004 issue of CHEST, Albane and colleagues evaluated the effect of norepinephrine (NE) in 26 patients, especially 12 patients with head trauma without infection. The glomerular filtration rate (GFR) was assessed using the formula (urinary creatinine × urinary flow rate)/serum creatinine in the head trauma group, and the Cockroft and Gault formula, published in 1976, in the septic shock group. We were surprised, despite missing data, that mean the GFR was above the normal value in the head trauma patients, approximately 165 mL/min/1.73 m². This remains significant after NE infusion, with a mean value of approximately 130 mL/min/1.73 m². A similar observation was previously published by Bennmalek et al in 1999. Using the same formula to evaluate GFR in 20 young head trauma patients, also treated with NE, they found values of 132 ± 22 mL/min/1.73 m². No more explanation was provided. Renal failure is a common complication observed after acute brain injury. Indeed, in our knowledge, no other data concerning the increase of GFR in head trauma patients have been published. To confirm this fact, we retrospectively reviewed the charts of brain-dead patients undergoing renal sharing for transplantation. Data were provided by the Etablissement Français des Greffes when one of our patients was selected to undergo transplantation. During the last 6 months, 58 renal grafts were proposed, obtained in 58 heart-beating donors. In 20 cases, data concerning the donor were insufficient to evaluate GFR before brain death. We also excluded four patients dead after cardiocirculatory arrest and two who died from meningitis. We studied the 32 remaining patients classified as head trauma or stroke (ischemic or hemorrhagic). With one exception, they were treated with NE to obtain a mean arterial pressure > 75 mm Hg. Data were obtained before brain death. The GFR was calculated using the Cockroft and Gault formula, or the recently proposed Modified Diet in Renal Disease formula. We confirmed the existence of hyperfiltration in patients with head trauma (Table 1). No known factors may explain this fact. There was no difference in age, incidence of diabetes mellitus (one in both groups), body mass index, or use of medications as cimetidine or trimethoprim available to diminish the tubular secretion of creatinine. No woman was pregnant. Two questions arise from this. First is the physiologic comprehension: difference in sympathetic tone, neurotransmitters, use of renal functional reserve, difference in nutritional support? Second is the value to study the "renal" effect of NE in patients with such "supranormal" renal function. Other studies are needed to get answers.

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Table 1—Renal Variables in Organ Donors Before Brain Death*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Male/Female</th>
<th>Gender, No.</th>
<th>Body Mass Index</th>
<th>Creatinine, µmol/L</th>
<th>Creatinine Clearance, MDRD, mL/min/1.73 m²</th>
<th>Creatinine Clearance, Cockroft and Gault², mL/min/1.73 m²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke 11/17</td>
<td>50.2 ± 14.51</td>
<td>27.8 ± 7.41</td>
<td>88.31 ± 37.22†</td>
<td>63.58 ± 16.95‡</td>
<td>70.69 ± 33.51§</td>
<td></td>
</tr>
<tr>
<td>Head trauma 14/1</td>
<td>57.2 ± 15.71</td>
<td>23.3 ± 4.51</td>
<td>64.5 ± 13.85†</td>
<td>129.07 ± 43.19‡</td>
<td>117.37 ± 39.49§</td>
<td></td>
</tr>
</tbody>
</table>

*Data are presented as mean ± SD. MDRD = Modified Diet in Renal Disease.
†p not significant, paired Student t test.
‡p < 0.002, paired Student t test.
§p < 0.001, paired Student t test.
Relationship of Baseline Glucose and Mortality During Medical Critical Illness?

To the Editor:

We read with interest the study by Cely et al (September 2004) on the relationship of baseline glucose homeostasis to hyperglycemia in critically ill patients. The authors show that hyperglycemia generally is present in critical illness, and that patients with low “baseline” hemoglobin A1c levels are less inclined to hyperglycemia than patients with higher hemoglobin A1c levels. Furthermore, they mention that “patients with normal and abnormal baseline glucose control had similar survival rates.” We don’t think that this statement has statistical validity, considering the limited number of evaluated patients (75 patients; ICU mortality rate, 29%).

To determine the relation between blood glucose regulation and mortality, we conducted an analysis among all patients admitted to our medical ICU over a 2-year period. A total of 1,209 consecutive patients were included. ICU mortality was 19.4%. A total of 10,954 glucose measurements were obtained. Mortality was significantly lower in the group with a mean glucose level of 4.0 to 6.0 mmol/L vs the group with a mean glucose level of 6.0 to 10.0 mmol/L: 12% vs 17.6% (p = 0.03). Patients who died in the ICU (n = 235) also had significantly higher baseline glucose values than patients who left the ICU alive (n = 974): 9.0 ± 5.3 mmol/L vs 7.6 ± 4.3 mmol/L (mean ± SD). We also calculated mortality rate after classifying patients into groups based on average glucose levels during ICU admission. The mortality rate in the group with an average glucose level of 4.4 to 6.1 mmol/L (n = 300) was 12%, while mortality with an average glucose level of >11.1 mmol/L (n = 96) was 37.5%. Maximal glucose values during hospital admission appeared to be lower in survivors than in nonsurvivors: 9.6 ± 5.2 mmol/L vs 11.7 ± 6.0 mmol/L, respectively.

Acute hyperglycemia is frequently found in stress situations. It is common in critically ill patients. Furthermore, it has been shown that strict regulation of glucose has beneficial effects on morbidity and even mortality.

Based on our own findings and those of others, we assume that hyperglycemia is correlated with mortality in critically ill patients. The next important step is to design and start up feasible glucose regulation protocols and study the effect of strict glucose control also in medical intensive care patients.

REFERENCES


Cardiovascular Risks Associated With β-Agonist Therapy

To the Editor:

The recent metaanalysis that was published in CHEST (June 2004) examining the cardiovascular risks associated with β-agonist therapy for obstructive lung diseases has significant implications for the millions of individuals who are currently using this therapy. The potential therapeutic impact and concerns that were raised for the management of these patients from the conclusions of this metaanalysis is demonstrated by the widespread reporting of this extremely crucial information on numerous news services. However, I disagree strongly that “physicians who recommend regular use of β-agonists [such as myself] may actually be putting their patients at risk.” I also strongly disagree with the conclusion drawn by the authors of this metaanalysis that short-term or long-term β-agonist use increases the risk for untoward cardiovascular events to the point of suggesting that the recommendations of these widely accepted guidelines be revised. The fact that the information from this metaanalysis was rapidly incorporated into personal injury legal web sites even further speaks to the magnitude of this recommendation and the impact that this may have on the patient use of, and even the physician prescribing of, these agents.

Despite the apparent concerns raised by the authors from the interpretation of their results, I was reassured that there was no evidence of a statistically significant increase in major cardiovascular effects from β-agonist use in their analysis.

REFERENCES


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