To the Editor:—The mortality rate of septic patients increases progressively with increasing severity of encephalopathy caused by sepsis. Several mechanisms have been suggested as the cause of septic encephalopathy, including direct central nervous system infection, endotoxin and cytokine effects on the brain, inadequate cerebral perfusion, metabolic derangements, or complications of medical therapy. Neuroradiologic examinations of 12 septic patients who died during protracted, severe septic encephalopathy showed microabscesses in eight cases and vascular lesions in approximately half.

We assessed the ability of the neuron-specific enolase (NSE), the neuronal isomer of the glycolytic enzyme 2-phospho-D-glycerate hydratase, and the central nervous system–specific isoforms of S100 to predict mortality in patients with severe sepsis or septic shock. Both NSE and S100 have been shown to be specific parameters for the assessment of
cerebral damage caused by hypoxia–ischemia. In addition, NSE has been found to be increased in nonsurvivors in a baboon model of sepsis.2

Twenty-nine consecutive patients of the surgical intensive care unit were enrolled in the study within the first 24 h after onset of severe sepsis or septic shock according to the criteria of the American College of Chest Physicians/Society of Critical Care consensus conference.3 At enrollment, we measured NSE and S100 in the serum of these patients using immunoluminometric assays (Byk-Sangtec, Dietzenbach, Germany). In addition, we determined interleukin 6 values by enzyme-linked immunosorbent assay (Bender MedSystems, Vienna, Austria). All patients were mechanically ventilated and were cared for by the intensive care unit staff. The severity of the patients’ illness was estimated using the APACHE II score. Patients with chronic altered mental status, acute primary central nervous system disorders (e.g., meningitis or cerebrovascular accident), previous cardiac arrest, acute metabolic disorders, and acute primary liver disease were excluded from the study.

In the present study, APACHE II score, mean arterial pressure, heart rate, arterial oxygen partial pressure/fraction of inspired oxygen ratio, and serum interleukin-6 concentration did not distinguish between survivors and nonsurvivors (table 1). Although S100 was increased in septic patients compared with the normal range (< 0.12 μg/l), no significant difference was observed between survivors and nonsurvivors. In contrast, serum concentrations of NSE were significantly higher in nonsurvivors than in survivors. The receiver operating characteristic curve in prediction of mortality for NSE is shown in figure 1. Based on 14 μg/l as the best cutoff point, NSE predicted mortality in patients with severe sepsis and septic shock with a sensitivity and specificity of 85% and 94%, respectively.

Increased serum NSE may serve as a useful prognostic marker of outcome in severe sepsis and septic shock. However, it must be kept in mind that NSE can also be elevated in the case of small-cell lung cancer or benign pulmonary disease.4 Because pulmonary dysfunction as estimated by the arterial oxygen partial pressure/fraction of inspired oxygen ratio was not significantly worse in nonsurvivors and did not correlate with NSE values, it is unlikely that the difference in NSE in the present study reflects pulmonary dysfunction in septic patients. False-positive increases in NSE can also be caused by hemolysis, because erythrocytes contain NSE.5 The diverging result of NSE and S100 could possibly be explained by different release kinetics of these two proteins6 or the finding that NSE more sensitively reflects small cerebral infarcts or transient ischemic attacks.5 Furthermore, NSE has a long half-life compared with S100. In contrast to S100, which is present in high concentrations in glial cells and Schwann cells,11 NSE originates predominantly from neurons and neuroendocrine cells.12 Therefore, in sepsis, it is possible that S100 just reflects a glial inflammatory reaction, whereas NSE may serve as a marker of neuronal damage. Further studies involving a greater number of patients are necessary to evaluate NSE as a parameter of outcome in sepsis.

Table 1. APACHE II Score, Mean Arterial Pressure, Heart Rate, PaO2/FI02 Ratio, Interleukin-6, S100, and Neuron-Specific Enolase in 29 Patients with Severe Sepsis or Septic Shock

<table>
<thead>
<tr>
<th></th>
<th>Survivor (n = 16)</th>
<th>Nonsurvivor (n = 13)</th>
</tr>
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<tbody>
<tr>
<td>APACHE II</td>
<td>25.5 ± 5.8</td>
<td>26.9 ± 5.4</td>
</tr>
<tr>
<td>MAP (mmHg)</td>
<td>68 ± 16</td>
<td>71 ± 13</td>
</tr>
<tr>
<td>HR (beats/min)</td>
<td>121 ± 20</td>
<td>115 ± 16</td>
</tr>
<tr>
<td>PaO2/FI02</td>
<td>139 ± 61</td>
<td>116 ± 52</td>
</tr>
<tr>
<td>Interleukin-6 (μg/ml)</td>
<td>251 ± 142</td>
<td>266 ± 160</td>
</tr>
<tr>
<td>S100 (μg/l)</td>
<td>0.35 ± 0.39</td>
<td>0.24 ± 0.24</td>
</tr>
<tr>
<td>NSE (μg/l)</td>
<td>11.1 ± 3.2</td>
<td>19.2 ± 6.4*</td>
</tr>
</tbody>
</table>

Values are mean ± SD.

MAP = mean arterial pressure; HR = heart rate; S100 = normal range < 0.12 μg/l; NSE = neuron-specific enolase (normal range, < 12.5 μg/l).

*P < 0.05 for survivors versus nonsurvivors, unpaired Student t test.

References


To the Editor:—We wish to report the uncoiling and separation of the internal wire of an epidural catheter (Arrow International, Reading, PA) on removal. We placed a Flextip catheter into the L3–L4 interspace of a 14-yr-old gravida 1, 80-kg, 160-cm tall patient for labor analgesia, using a 17-gauge Tuohy needle and the technique of loss of resistance to air. We attempted to introduce the epidural catheter, but it would not pass beyond the end of the needle. We then advanced the epidural needle approximately 1 mm in accordance with the directions supplied by the manufacturer and were then able to introduce the epidural catheter into the epidural space. The catheter was advanced 4 cm into the epidural space, and the needle was withdrawn. The epidural catheter functioned normally for labor analgesia. Subsequently, the local anesthetic infusion was discontinued, and the conduction block was allowed to recede. On removal of the catheter, there was considerable resistance to withdrawal. We asked the patient to flex her back to facilitate the removal of catheter. With increased flexion, we were able to withdraw the catheter. However, on continued attempts to withdraw, we realized that the plastic outer portion of the catheter had been removed from the patient’s back, but the wire was uncoiling and still within the patient. At this time, because the patient had good sensation and movement in all dermatomes where epidural analgesia had been present, we believed that it was acceptable to continue with gentle traction as long as there were no paresthesias during this procedure. The wire gradually was removed from the patient’s back with no symptoms or sequelae.

We believe this is the first report of the internal wire of an epidural catheter uncoiling within the patient’s back. This may pose risk to the patient because the sharp wire may increase the potential for internal lacerations. We examined the catheter and wire using a microscope and found that the plastic catheter appeared intact, but a sharp hook was present at the end of the wire (fig. 1). Figure 2 shows the epidural catheter with 19 cm of the extended uncoiled wire.

The possibility exists that structures in the epidural space may be lacerated by the sharp wire during removal from the patient. This may increase the possibility of epidural hematoma. We removed the catheter after the block had resolved, and no symptoms were present at that time. In a recent case report, 1 an epidural catheter fragment was retained after coiling around a nerve root. One can speculate that if an epidural catheter has coiled around a nerve root and also separates, as it did in our patient, injury to the nerve root may be more likely.

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(Accepted for publication October 22, 1999.)