**Prognosis**

**Patients who survived the first 30 days after a first episode of status epilepticus had increased mortality at 10 years**


**Questions**
What is the long-term mortality after a first episode of afebrile status epilepticus (SE)? Do seizure type, cause, and duration of SE affect long-term mortality?

**Design**
Inception cohort followed until death or 10 years after initial SE.

**Setting**
Rochester, Minnesota, USA.

**Patients**
145 patients (50% women) who had a first episode of afebrile SE (a seizure lasting ≥ 30 min or repeated seizures over a period ≥ 30 min without recovery between episodes) and survived the first 30 days. Exclusion criteria were seizure lasting < 30 minutes and stopped by antiseizure medication; electroencephalographic SE in the absence of clinical features; or a flurry of seizures, each < 30 minutes, with intervening periods of consciousness. All patients were included in the analysis.

**Assessment of prognostic factors**
Seizure type (generalized, secondary generalized, partial, myoclonic, and absence), cause (acute symptomatic, progressive symptomatic, remote symptomatic, or idiopathic cryptogenic), duration (30 min to < 2 h, 2 to 24 h, or > 24 h), and age (< 1 y, 1 to 19 y, 20 to 64 y, ≥ 65 y).

**Main outcome measure**
Mortality at 10 years after initial SE.

**Main results**
62 deaths occurred during the 10-year follow-up period. This mortality rate of 43% was 2.8 times higher than that expected in the Minnesota population, which was standardized by age, sex, and time period (standardized mortality ratio 2.8, CI 2.1 to 3.5). Multivariate analysis using the Cox proportional hazards model showed that long-term mortality was increased in patients who had acute symptomatic SE, myoclonic SE, or SE lasting > 24 hours and in those who were 20 to 64 years of age or ≥ 65 years of age (Table).

**Conclusions**
10-year mortality rates were increased after a first episode of afebrile status epilepticus (SE), especially in patients with acute symptomatic SE, myoclonic SE, or SE lasting > 24 hours and in those who were 20 years of age or older.

Source of funding: National Institute of Neurological Disorders and Stroke.

For correspondence: Dr. W.A. Hauser, G.H. Sergievsky Center, New York, NY, USA. E-mail wahl@columbia.edu.

<table>
<thead>
<tr>
<th>Prognostic factor</th>
<th>Reference (RR = 1.0)</th>
<th>RR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoclonic type</td>
<td>Generalized type</td>
<td>4.0 (1.3 to 13)</td>
</tr>
<tr>
<td>Acute symptomatic cause</td>
<td>Idiopathic or cryptogenic</td>
<td>2.2 (1.0 to 5.1)</td>
</tr>
<tr>
<td>Duration &gt; 24 h</td>
<td>&lt; 2 h</td>
<td>2.3 (1.1 to 5.1)</td>
</tr>
<tr>
<td>Age 20 to 64 y</td>
<td>Age 1 to 19 y</td>
<td>13.3 (1.7 to 103)</td>
</tr>
<tr>
<td>Age ≥ 65 y</td>
<td>Age 1 to 19 y</td>
<td>67 (8.9 to 503)</td>
</tr>
</tbody>
</table>

*RR = relative risk. Other abbreviations defined in Glossary.

**Commentary**
Cause is the key consideration in making a prognosis for survival after SE, and not all SE episodes should be regarded as equal. For example, survival is dramatically reduced in patients with SE (usually myoclonic) resulting from hypoxic–ischemic brain injury after cardiac arrest (1, 2). Acute brain insults producing SE explain the substantial mortality seen in studies looking at short-term outcomes. The analysis of long-term survival after SE by Logroscino and colleagues confirms this notion and emphasizes other important aspects. First, patients with idiopathic or cryptogenic SE alone did not have a higher risk for death than did the general population. Second, the substantial increase in mortality in patients with SE associated with progressive or underlying neurologic or systemic disorders was probably no different than that seen in patients with those disorders who did not have SE. Third, although persons ≥ 65 years of age with SE had a higher mortality rate, this difference was largely explained by age and not by SE. Finally, seizure type had no effect on mortality with the exception of myoclonic seizures, in which the higher mortality rate was directly related to its dire cause (e.g., cardiac arrest). Because the cause of SE greatly affects survival, prognostic statements should follow a careful search for that cause.

Important aspects that deserve further exploration include determining the effects of modern neurointensive care and of newer drugs for SE, describing cognitive and functional status outcomes, and assessing the long-term prognosis of subclinical (electroencephalographic) SE.

Samuel Wiebe, MD, MSc
University of Western Ontario
London, Ontario, Canada

**References**