Outcome Index for Head-Injured Patients

To The Editor: Most neurosurgeons interested in measuring outcome from head injuries would agree with Dr. Langfitt (Langfitt TW: Measuring the outcome from head injuries. J Neurosurg 48:673–678, May, 1978) about the importance of using the coma scale and measurements of outcome as defined by the Glasgow group.

Nevertheless, the Glasgow criteria for the evaluation of the head-injured patient cannot always be applied retrospectively to large series of head injuries from the literature in an attempt to compare current results. Moreover, these criteria have not been universally adopted by all investigators. Indeed, Becker, et al.,1 in presenting their superb results in the management of over 160 head injuries did not use the Glasgow Coma Scale to evaluate individual patients, although outcome was defined in terms of the Glasgow criteria.

Thus, when we wanted to compare the results of intensive management of our own head-injured patients with other series, we were somewhat stymied. Because most investigators report the number or percentage of decerebrate or flaccid patients admitted to their series, we decided to employ the following outcome index: % deaths, persistent vegetative, or severely disabled patients/% patients admitted decerebrate or flaccid = outcome index. The calculated outcome index for each of the series shown in Table 1 is computed from Table 3 of Langfitt’s article. The last column represents our own results. It is obvious that the higher the ratio of bad outcome to patients admitted decerebrate or flaccid, the worse the results, and the higher the outcome index. For example, the Los Angeles series had 3.6 deaths for each patient admitted decerebrate or flaccid, and Richmond had 1.19 deaths for each patient admitted decerebrate or flaccid.

While we were initially discouraged by what we felt were poor results (56% dead, vegetative, or severely disabled) in our own patients (average age 24 years) in spite of intensive therapy as described by Becker, et al.,1 we also realized that we had an extraordinarily high percentage of patients admitted decerebrate or flaccid (52%). Our outcome index of 1.08 in the 60 patients we have thus far studied, however, compares very favorably with the other adult series in the literature.

This outcome index should in no way be seen as supplanting the Glasgow evaluation or outcome criteria. Indeed, we continue to use them. Rather, it is a useful tool for evaluating and comparing results between series where these criteria have not always been employed for one reason or another.

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Kemp Clark, M.D.
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<table>
<thead>
<tr>
<th>Institution</th>
<th>% Deaths, Vegetative, or Severely Disabled</th>
<th>% Patients Admitted Decerebrate or Flaccid</th>
<th>Outcome Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Los Angeles</td>
<td>72</td>
<td>20</td>
<td>3.60</td>
</tr>
<tr>
<td>Netherlands</td>
<td>58</td>
<td>22</td>
<td>2.64</td>
</tr>
<tr>
<td>Glasgow</td>
<td>62</td>
<td>21</td>
<td>2.95</td>
</tr>
<tr>
<td>Richmond</td>
<td>43</td>
<td>36</td>
<td>1.19</td>
</tr>
<tr>
<td>UTSMS*</td>
<td>56</td>
<td>52</td>
<td>1.08</td>
</tr>
</tbody>
</table>

*UTSMS = University of Texas Southwestern Medical School.

Reference


Response: The outcome index described by Drs. Cooper and Clark is a useful contribution. It emphasizes the need to define the population of head-injured patients admitted to a neurosurgical unit before comparing results with other units. In order to make cross-comparisons of outcome, and thereby
attempt to establish the relative effectiveness of various forms of management in head-injured patients, one must either demonstrate that the two patient populations are equivalent in such variables as age and the severity of the injury, or, if the populations are different, quantify the difference. The latter requirement is impossible to fulfill at this time, because we are unable to quantify the importance of age, time from injury to admission to definitive therapy, the presence of intracranial hematoma, and a host of other variables.

Although it is not possible to precisely scale the relationship of the neurological status of the patient on admission to the outcome, I believe there is agreement that of all the variables that influence outcome, the neurological status of the patient during the first few hours following injury is the most important one. This relationship is addressed in the outcome index used by Drs. Cooper and Clark. Decerebration or flaccidity are signs of very severe brain injury, and therefore, the incidence of decerebration and flaccidity in a patient population is a reasonable index of the nature of the patients included in the series. Using this criterion of the severity of injury and comparing the outcome index in Dallas with the index calculated from other published series, Drs. Cooper and Clark demonstrate convincingly that their results are better than the outcome alone would suggest, because they had a much larger incidence of patients admitted decerebrate or flaccid. These results point up again the pitfalls in interpreting outcome and the dangers of attributing success to a particular therapeutic approach without a detailed comparison of the two populations.

There is strong evidence that age greatly influences the outcome from severe head injuries. It would be interesting to see the results produced by substituting the percentage of patients under the age of 21 years in the denominator of each of the reported series.

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Kaolin-Induced Hydrocephalus and Central Cord Canal

TO THE EDITOR: In a recent article Dr. James and his co-workers reported their observations on the central canal of the spinal cord in an experimental model of communicating hydrocephalus (James AE, Flor WJ, Novak GR, et al: Evaluation of the central canal of the spinal cord in experimentally induced hydrocephalus. J Neurosurg 48: 970–974, June, 1978). Their method of producing hydrocephalus does not cause a significant inflammatory response or obstruction of the fourth ventricular outlet foramina and in this experimental model the central canal is not affected. In other experiments in which hydrocephalus was caused by cisternal injection of kaolin, the central canal dilated and functioned as a cerebrospinal fluid (CSF) pathway. Noting this difference they attribute special significance to their finding and in this paper, and in an earlier paper reporting similar methods and results, they question the importance of the central canal in the pathology of hydrocephalus.

The finding that the central canal of the spinal cord is undisturbed in their model of hydrocephalus should, however, be of no surprise. In contrast, when hydrocephalus is induced by injection of kaolin into the cisterna magna, obstruction of the fourth ventricular outlet foramina occurs and fluid escapes the ventricular system by way of a dilated central canal entering the spinal subarachnoid space at the filum terminale; absorption takes place from that space. One would not expect the central canal to be disturbed in a model which did not obstruct the outlets of the fourth ventricle and in which fluid from the ventricular system had normal access to the spinal subarachnoid space.

Furthermore, they state that they are unaware of the central canal being commonly affected in human forms of hydrocephalus. It has, however, been clearly shown that there is a relationship between hydrocephalus, hydromyelia, and syringomyelia, particularly when the outlets for the fourth ventricle are obstructed by the Arnold-Chiari malformation. Perhaps less widely recognized is the association of myelomeningocele and hydro-myelia. In these cases the hydromyelia that occurs when the hydrocephalus is not treated was shown to be of clinical significance, resulting in specific physical deficits and in some cases, as in the kaolin hydrocephalus, acting as a compensatory CSF pathway.

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