Head-injured patients who talk and deteriorate into coma

Analysis of 211 cases studied with computerized tomography

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Of 838 patients with severe head injuries admitted since the introduction of computerized tomography, 211 (25.1%) talked at some time between trauma and subsequent deterioration into coma. Of these 211 patients, 89 (42.2%) had brain contusion/hematoma, 46 (21.8%) an epidural hematoma, 35 (16.6%) a subdural hematoma, and 41 (19.4%) did not show focal mass lesions. Thus, four of every five patients who deteriorated into coma after suffering an apparently nonsevere head injury had a mass lesion potentially requiring surgery; the mass was intracerebral in 52.3% of the cases and extracerebral in 47.6%. Patients aged 20 years or less had a 39% chance of having a nonfocal mass lesion (diffuse brain damage), a 29% chance of having an epidural hematoma, and a 32% chance of having an intradural mass lesion; patients over 40 years had only a 3% chance of having a nonfocal mass lesion, an 18% chance of having an epidural hematoma, and a 79% chance of having an intradural mass lesion.

Sixty-eight (32.2%) patients died and 143 (67.8%) survived. The following were independent outcome predictors (in order of significance): Glasgow Coma Scale score following deterioration into coma, the highest intracranial pressure during the patient's course, the degree of midline shift, the type of intracranial lesion, and the age of the patient. In contrast, the mechanism of injury, the verbal Glasgow Coma Scale score during the lucid interval, and the length of time until deterioration or until operative intervention did not influence the final result.

Key Words • head injury • epidural hematoma • subdural hematoma • brain contusion • lucid interval • coma

Among patients rendered comatose by a head injury, 12% to 32% are capable of speaking at some time between trauma and subsequent deterioration.13,17 Delayed neurological deterioration, which is most commonly caused by an expanding focal mass lesion, is followed by a fatal result or severe disability in 30% to 40% of cases. Many studies have been addressed to identify patients with minor head injuries at risk for secondary neurological deterioration,2,3,7,8,18-21 but few have specifically analyzed the mechanisms of deterioration and the avoidable factors responsible for the unfavorable outcome in this potentially salvageable group of patients.10,13,31,24

In this study we analyze the clinical profile and pathological findings in 211 patients who talked after sustaining an apparently nonsevere head injury and then deteriorated into coma. We also compare some clinical features in this group and in another group of 627 patients treated during the same period who did not experience a lucid interval.

Clinical Material and Methods

The 211 patients included in the study represent 25.1% of a series of 838 severely head-injured patients treated consecutively and examined with computerized tomography (CT) at our unit from November, 1977, to July, 1989. Nearly two-thirds of these patients were admitted either directly from the scene of the accident or from local hospitals located in the Madrid conurbation. The remaining patients were referred from regional hospitals located more than 50 km away. Patient transportation to the hospital was by road in the majority of cases and by helicopter in a few. The CT scan was usually obtained at our hospital, but some patients were scanned at peripheral hospitals.

All 211 patients in this series were known to have talked at some time before going into coma. Seventy-five (35.5%) patients were fully oriented during their lucid interval (verbal score of 5 in the Glasgow Coma Scale (GCS)23), 65 (30.8%) were confused (verbal GCS
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score of 4), and 71 (33.6%) uttered at least some comprehensible speech prior to going into coma (verbal GCS score of 3). Deterioration occurred within 24 hours after injury in 71% of cases and most of the remaining patients became comatose during the 2nd day posttrauma. Once in coma, patients were managed according to a standardized protocol reported elsewhere. Intracranial pressure (ICP) was measured in 179 patients by means of an intraventricular catheter or less commonly by an epidural fiberoptic sensor.

For the purpose of this study and based on the CT and the operative findings, the cases were separated into four major pathological categories: epidural hematoma, subdural hematoma, brain contusion/hematoma, and diffuse brain damage (absence of focal lesions). Nearly two-thirds of the patients in the last category showed more or less marked effacement of the ventricles and cisterns and 30% showed either subarachnoid, intraventricular, or small parenchymal hemorrhages, or a combination of these. A few patients with large extraxial hematomas had small associated brain contusions, and some in the brain contusion/hematoma category had associated laminar extracerebral hematomas. Three patients with posttraumatic meningitis, who died after arriving to our unit critically ill, were excluded from the study.

A multivariate regression analysis of the variables that might influence the final outcome was carried out to assess their relative importance.

Results

Comparison Between Patients With and Without a Lucid Interval

The mortality rate in patients with and those without a lucid interval was not significantly different (Table 1). Although the mechanisms of injury were similar in both groups, patients with a lucid period had a lower incidence of generalized injury and peritraumatic hypotension or hypoxia, indicating that they suffered less severe injuries and presumably less frequent hypoxic brain damage. However, they were comparatively older and had a higher incidence of focal mass lesions, most of which required surgical intervention.

Clinical and CT Findings in Patients With a Lucid Interval

Table 2 shows several clinical and CT variables and the final outcome in patients with a lucid interval, in both the 68 (32.2%) who died and the 143 (67.7%) who survived. Five percent of the survivors developed a severe disability. Multivariate analysis indicates that the GCS score following deterioration and the highest mean ICP during the course were the most powerful prognostic predictors (p < 0.001), followed by the degree of midline shift (p < 0.01), the type of intracranial lesion (p < 0.01), and the age of the patient (p < 0.05). In contrast, the mechanism of injury, the verbal score during the lucid interval, and the interval between

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**TABLE 1**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Lucid Interval</th>
<th>No Lucid Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of cases</td>
<td>211</td>
<td>627</td>
</tr>
<tr>
<td>Percent</td>
<td>25.1</td>
<td>74.8</td>
</tr>
<tr>
<td>Fatal result</td>
<td>68</td>
<td>233</td>
</tr>
<tr>
<td>Percent</td>
<td>32.2</td>
<td>37.1</td>
</tr>
<tr>
<td>Polytrauma</td>
<td>58</td>
<td>260</td>
</tr>
<tr>
<td>Percent</td>
<td>27.4</td>
<td>41.4*</td>
</tr>
<tr>
<td>Hypotension-hypoxia</td>
<td>14</td>
<td>129</td>
</tr>
<tr>
<td>Percent</td>
<td>6.6</td>
<td>20.5*</td>
</tr>
<tr>
<td>Age &gt; 40 yrs</td>
<td>90</td>
<td>131</td>
</tr>
<tr>
<td>Percent</td>
<td>42.6</td>
<td>20.8*</td>
</tr>
<tr>
<td>Focal mass lesion</td>
<td>170</td>
<td>317</td>
</tr>
<tr>
<td>Percent</td>
<td>80.5</td>
<td>50.5*</td>
</tr>
</tbody>
</table>

*Statistical significance of difference between the two groups: p < 0.001.

**TABLE 2**

<table>
<thead>
<tr>
<th>Factor</th>
<th>Functional Survival</th>
<th>Fatal Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>No. of cases</td>
<td>143</td>
<td>68</td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 40 yrs</td>
<td>83</td>
<td>28</td>
</tr>
<tr>
<td>41–60 yrs</td>
<td>28</td>
<td>19</td>
</tr>
<tr>
<td>&gt; 60 yrs</td>
<td>22</td>
<td>21</td>
</tr>
<tr>
<td>Interval to detection of deterioration</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 6 hrs</td>
<td>48</td>
<td>30</td>
</tr>
<tr>
<td>6–24 hrs</td>
<td>43</td>
<td>19</td>
</tr>
<tr>
<td>&gt; 24 hrs</td>
<td>40</td>
<td>17</td>
</tr>
<tr>
<td>Verbal GCS score (first determination at lucid interval)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>43</td>
<td>32</td>
</tr>
<tr>
<td>4</td>
<td>42</td>
<td>23</td>
</tr>
<tr>
<td>3</td>
<td>58</td>
<td>13</td>
</tr>
<tr>
<td>GCS score (following deterioration)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7 or 8</td>
<td>55</td>
<td>7</td>
</tr>
<tr>
<td>5 or 6</td>
<td>58</td>
<td>20</td>
</tr>
<tr>
<td>3 or 4</td>
<td>30</td>
<td>41</td>
</tr>
<tr>
<td>Mechanism of injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fall</td>
<td>44</td>
<td>35</td>
</tr>
<tr>
<td>Vehicle accident</td>
<td>69</td>
<td>20</td>
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<tr>
<td>Pedestrian accident</td>
<td>13</td>
<td>9</td>
</tr>
<tr>
<td>Other or unknown</td>
<td>16</td>
<td>6</td>
</tr>
<tr>
<td>Type of lesion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>All focal lesions</td>
<td>108</td>
<td>62</td>
</tr>
<tr>
<td>Epidural hematoma</td>
<td>37</td>
<td>9</td>
</tr>
<tr>
<td>Subdural hematoma</td>
<td>16</td>
<td>19</td>
</tr>
<tr>
<td>Brain contusion/hematoma</td>
<td>55</td>
<td>34</td>
</tr>
<tr>
<td>Diffuse brain damage</td>
<td>35</td>
<td>6</td>
</tr>
<tr>
<td>Midline shift</td>
<td></td>
<td></td>
</tr>
<tr>
<td>≤ 5 mm</td>
<td>62</td>
<td>14</td>
</tr>
<tr>
<td>6–15 mm</td>
<td>66</td>
<td>23</td>
</tr>
<tr>
<td>&gt; 15 mm</td>
<td>15</td>
<td>31</td>
</tr>
<tr>
<td>Highest mean ICP</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt; 15 mm Hg</td>
<td>26</td>
<td>3</td>
</tr>
<tr>
<td>15–35 mm Hg</td>
<td>74</td>
<td>12</td>
</tr>
<tr>
<td>&gt; 35 mm Hg</td>
<td>14</td>
<td>50</td>
</tr>
</tbody>
</table>

*CT = computerized tomography; GCS = Glasgow Coma Scale; ICP = intracranial pressure.
injury and deterioration or surgical intervention were not significant predictors of outcome (p > 0.05).

Of the 211 patients in the series, 170 (80.5%) had a focal mass lesion: this was a brain contusion/hematoma in 89 cases (42.2% of the total 211 patients in the series), an epidural hematoma in 46 cases (21.8%), and a subdural hematoma in 35 cases (16.6%). Forty-one patients (19.4%) did not exhibit focal mass lesions. Thus, four of every five patients who became comatose after suffering a seemingly minor or moderate head injury had a mass lesion potentially requiring surgery; the mass was intracerebral in 52.3% of the cases and extracerebral in the remaining 47.6%. The mortality rate was higher in patients with focal pathology, most of whom required craniotomy, than in those without focal mass lesions (36.4% vs. 14.5%). Of all the mass lesions, subdural hematoma carried the highest mortality rate (54.3%), followed by brain contusion/hematoma (38.2%) and epidural hematoma (19.6%). More than two-thirds of the patients under 20 years of age had no mass lesions (38.9% of cases) or an epidural hematoma (28.8%). In contrast, more severe lesions (brain contusion/hematoma and subdural hematoma) occurred with greater frequency in patients over 40 years of age (56.6% and 22.6% of cases, respectively).

Deterioration into coma occurred sooner on average in patients with extracerebral hematoma than in patients without focal mass lesions or in those with brain contusion/hematoma. The percentages of patients with subdural hematoma, epidural hematoma, diffuse brain damage, and brain contusion/hematoma who deteriorated within 6 hours after injury were 60%, 54.3%, 34.1%, and 20.2%, respectively, and the percentages of those who deteriorated later than 24 hours posttrauma were 11.4%, 15.2%, 29.2%, and 38.2%, respectively. Patients with epidural or brain hematoma who deteriorated within 6 hours after injury had a higher mortality rate than those who showed a more protracted course of deterioration, but the difference was not significant. Patients with subdural hematoma and those without focal lesions had the highest and lowest mortality rate, respectively, regardless of the time lapse between trauma and deterioration.

Avoidable Factors Contributing to Delayed Neurological Deterioration

Delayed diagnosis at, or transfer from, other hospitals occurred in 16% of the cases with extracerebral hematoma and in 4% of those with brain contusion/hematoma. Delayed diagnosis of intracranial hematoma at our unit occurred in at least 6% of patients. Among these were five patients who were not subjected to CT on admission although they had severe headache or minimal focal signs; in two cases this was due to lack of cooperation. Five more patients deteriorated while undergoing alignment of cervical spine fractures (two cases) or general anesthesia for emergency extracranial surgery (three cases); two of the latter three patients and two more with GCS scores of 14 or 15 for more than 6 hours after trauma suffered delayed extracerebral hematoma formation (a new hematoma after a normal admission CT scan). In three more cases, CT failure accounted for delay in diagnosis.

Twelve patients with discrete extra- and intra-axial focal mass lesions were initially managed without operation; however, they had deteriorated to a GCS score of less than 9 by the time the mass was eventually evacuated. Finally, seven children showing diffuse brain swelling in their admission CT scans suffered abrupt neurological deterioration 3 to 16 hours after injury, at a time when gasometric deterioration was documented; discrete hyperventilation resulting in a clear neurological improvement in four of them, suggesting that controlled ventilation might have prevented increasing brain swelling and further deterioration.

Discussion

In our series, 25.1% of severely head-injured patients talked prior to deteriorating into coma; this figure is in the lower range of that found in an international study in which 25% to 32% of the patients had a lucid interval,13 and twice that recorded in the plot phase of the National Traumatic Coma Data Bank (12%).15 Differences in percentages may relate to the number of primary versus secondary admissions to each neurological center and to the quality of prehospital and in-hospital care delivered by different systems in different epochs. It is interesting to note that the percentage of patients who “talked and died” in our series has decreased since CT scanners became available in some regional hospitals. Early CT scanning and an increased level of observation in the hospital setting are decisive for reducing the excessive death rate in head-injured patients who were initially predicted to be at low risk.14 28

Deterioration from Contusion/Hematoma

The major finding in this study is that 80.5% of the patients who deteriorated into coma following a seemingly minor or moderate head injury had a focal mass lesion, which required neurosurgical intervention in the majority of the cases. Brain contusion/hematoma was more commonly the cause of delayed deterioration (42.2% of cases) than extracerebral hematoma (38.4% of cases). Patients aged 20 years or less had a 39% chance of having a nonfocal mass lesion, a 29% chance of having an epidural hematoma, and a 32% chance of having an intradural mass lesion. In patients over 40 years of age, there was only a 3% chance of having a nonfocal mass lesion, an 18% chance of having an epidural hematoma, and a 79% chance of having an intradural mass lesion. These figures may be of significance when planning direct surgery on a head-injured patient presenting with rapid neurological deterioration after showing a lucid period.

In addition to age, the duration of the lucid interval may suggest the pathological diagnosis. The shorter the interval, the higher the probability that an extracerebral
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Fig. 1. Computerized tomography scans in a 15-year-old girl who was admitted to our unit after she had been struck by a car. Her Glasgow Coma Scale score was 13 and a skull x-ray film was normal. Ten hours after admission she deteriorated into coma. Left: Scan performed following endotracheal intubation showing diffuse brain swelling but the posterior fossa cisterns were still visible. Intracranial pressure (ICP) was over 35 mm Hg and could not be controlled even with high-dose thiopental. Right: Control scan performed 24 hours after deterioration showing complete obliteration of the cerebrospinal fluid spaces. Perhaps a more aggressive initial management including controlled ventilation and ICP monitoring might have prevented increased brain swelling and death in this patient.

Fig. 2. Computerized tomography scans in a 16-year-old boy who was admitted 1 hour after he had been struck by a motorcycle. His Glasgow Coma Scale (GCS) score was 14 and there was a skull fracture. Twenty hours after admission he suddenly deteriorated to a GCS score of 8. The scans showed generalized brain swelling and a small right frontal epidural hematoma with a midline shift. Following hematoma removal, the intracranial pressure ranged between 20 and 30 mm Hg and was easily controlled. The patient recovered consciousness 3 days after the operation. The epidural clot did not appear big enough to cause coma in the absence of associated diffuse brain swelling.

hematoma is present. Nearly 60% of the patients with extracerebral hematoma deteriorated with 6 hours of injury, whereas 77.5% of the patients with brain contusion/hematoma became comatose more than 6 hours after trauma. However, a short lucid interval may also occur in the absence of focal pathology; 

![Image](https://via.placeholder.com/150)

Deterioration Without a Focal Mass

The mechanism for delayed deterioration into coma in the absence of focal lesions remains unclear. A lucid interval does not occur with severe brain damage due to impact injury, and brain hyperemia, brain edema, or an electrophysiological disturbance has been considered responsible for delayed unconsciousness in these cases. Only 12 (29.3%) of the 41 patients without focal mass lesions in this study showed an apparently normal CT scan through the course. The remaining patients, most of whom were children, showed variable degrees of basal cistern collapse indicative of generalized brain swelling in their admission or follow-up CT scans, and some also had small parenchymal or subarachnoid hemorrhages. In the majority of patients for whom we could compare the pre- and post-deterioration scans, an increased degree of obliteration of the cisterns and ventricles was documented in the post-deterioration studies (Fig. 1). Our findings in this subgroup of patients support the conclusion propounded by Bruce, et al., that diffuse brain swelling is the most frequent cause of secondary deterioration in children showing a lucid period after a head injury. Whatever the mechanism of delayed deterioration, these 41 patients had higher GCS scores during coma, were unconscious for shorter periods of time, and had a significantly better functional result than those with the same pathological category of diffuse brain damage who were comatose from the moment of the impact (unpublished observations). Although the mechanism for delayed neurological deterioration in the presence of a focal mass seems obvious, some patients had relatively small lesions at the time they went into coma, suggesting that they also suffered from a concomitant pathological mechanism of the type operating in patients with diffuse brain damage (Fig. 2).

Management Strategies

The high mortality rate of head-injured patients who become comatose after showing a lucid period, reported in this and other series, indicates that intracranial complications are often not recognized or treated as soon as they should be and suggests that current strategies for precautionary admission and CT examination in patients believed to have suffered a minor or moderate head injury need to be modified. Although routine CT scan usage in the presence of minimal posttraumatic deficits or skull fracture may detect early intracranial hematoma formation in some cases, there are still other patients who deteriorate during transportation to neurosurgical units or while undergoing radiological examination or treatment for associated extracranial injuries. Furthermore, some patients with hyperactive intracranial hematoma or diffuse brain swelling deteriorate while under observation and die despite rapid neurosurgical action.
During the period of the study we discharged home two patients without skull fracture or any deficit who were later readmitted showing advanced neurological deterioration caused by an intracranial hematoma. These two cases would likely have escaped the management strategy of minor head injuries recommended by Feuerman, et al., which is currently used at our unit; these authors perform emergency CT in all patients with a GCS score of 13 or 14 and in patients with a score of 15 if either an abnormal mental condition or a focal hemispheric deficit is present. Such a CT scanning regime, which renders skull radiographs superfluous, will likely reveal an intracranial hematoma in some patients who have not yet developed clinical evidence of brain compression. However, it should be noted that a normal CT scan obtained early after trauma does not exclude the occurrence of delayed brain swelling or hematoma formation, which may result in a fatal outcome.

In two of our five patients who developed intracranial hematoma after showing a normal admission CT scan, the complication occurred while anesthesia was being given for extracranial surgery. Some other patients who either showed no pathology at all or had punctate lobar hemorrhage on the emergency CT scan developed focal or multifocal brain contusions, which in some cases were not evident until advanced deterioration had occurred (Figs. 3 and 4). Patients with minimal or no changes on the initial CT scan but who show persistent neurological impairment may benefit from sequential CT scanning or magnetic resonance imaging, which is more sensitive for detecting brain contusion during the acute posttraumatic period.12,27

Although many patients with brain contusion/he- matoma are successfully managed without surgery,
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early removal of focal brain lesions causing marked midline shift is preferable even if the patient’s neurological status is relatively good and the ICP is normal. Postponement of operative intervention on the basis of a normal initial ICP resulted in severe secondary brain damage in some of our cases. The absence of midline displacement in patients with gross bilateral brain contusions may also give false security, as the majority eventually develop raised ICP which cannot be controlled with medical therapy.

Because of the risk of delayed brain swelling we recommend that children with an abnormal neurological examination (including GCS and mental status) be placed under close neurological observation and have blood gases, electrolytes, and fluid balance measured throughout the first 48 hours after injury. Those showing basal cistern effacement in the admission CT scan should be considered for management with epidural ICP monitoring and assisted ventilation.

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References


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