tocol, we generated one based on our data (Fig. 1). We hope that this protocol, or classification of high-risk mild head injury, will be widely accepted, as it is practical, simple, and inexpensive. As it is not quite perfected, however, we would not yet call it “standard.”

We are glad that this topic can arouse so much interest, as shown by Dr. Stein’s constructive comments and the many requests for article reprints. Hopefully, this discussion will help to answer how can we safely discharge a patient with mild head injury from the Emergency Department without obtaining a head CT scan.

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References

Thrombin–Antithrombin Complex Levels in Subarachnoid Hemorrhage

TO THE EDITOR: Dr. Peltonen and his coworkers recently presented a study on hemostatic activation in acute subarachnoid hemorrhage (SAH) (Peltonen S, Juvela S, Kaste M, et al: Hemostasis and fibrinolysis activation after subarachnoid hemorrhage. J Neurosurg 87:207–214, August, 1997), which included incorrect assertions based on inadequate knowledge of changes in hemostatic parameters associated with neurosurgery.6 They wrote that the levels of thrombin–antithrombin complex (TAT) in our study4 were 10-fold higher than in their study because of a delay in contact between blood and the anticoagulant agent during blood collection in which a multiple-syringe technique was used. Actually, a time delay between blood drawing and contact between blood and the anticoagulant agent is less than 3 seconds. Is it long enough to activate hemostatic systems? During my tenure with Professor Eberhard F. Mammmen, editor-in-chief of Seminars in Thrombosis and Hemostasis, I performed a number of clinical procedures in which TAT2,3 was used. I conducted thousands of TAT assays including blood collection and enzyme immunoassay. Therefore I believe that my knowledge and technical ability in conducting practical TAT assays is accurate enough to perform reliable blood coagulation studies in which TAT assays are used. The multiple-syringe blood collection technique is routinely used in the field of blood coagulation research to avoid artificial activation of hemostatic systems. We must use the multiple-syringe technique for reliable blood coagulation studies.

The discrepancy in the TAT levels between our study and the Peltonen study seems to be attributable to a large difference in the patient population. The clinical status on admission in the patients in our study, graded according to the World Federation of Neurological Surgeons Grading Scale1 (Grade 3.3 ± 0.1, mean ± standard error of the mean), was considerably worse than that in the Peltonen study (Grade 2.35 ± 1.35). We reported that the levels of TAT at admission significantly increased with developing severity of the neurological grade at admission.4 There was also a wide difference between the two studies in the time from onset to collection of the first blood sample. In our study we reviewed the patients admitted within 24 hours of onset, and the time from onset to blood collection was 3.1 ± 0.2 hours. On the other hand, the patients in the Peltonen study included those admitted within 3 days after onset. The time from discovery of bleeding to blood collection was 1.5 ± 0.2 days. We recently reported that the levels of TAT in patients with subarachnoid hemorrhage (SAH) were the highest at admission and rapidly decreased after admission while the levels of D-dimer increased over the value at admission and peaked on Days 3 to 6 of onset.5 Additionally I performed a statistical analysis to assess a relationship between the levels of TAT and the time delay from onset to blood collection in the 235 SAH patients admitted within 72 hours of onset (Table 1). The majority of the patients were admitted within 1 hour of onset. The levels of TAT in the patients admitted within 2 hours of onset were extremely high. From a statistical point of view this analysis, although a univariate analysis, demonstrates that the levels of TAT significantly decrease with increasing time (measured in hours) from onset of SAH. Hence, I believe the authors missed blood collection from the SAH patients in an ultraacute stage (within several hours of onset). They should have mentioned this important point in their paper.

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References

TABLE 1
Relationship between time interval from onset and levels of thrombin–antithrombin complex in 235 patients admitted within 72 hours from onset of spontaneous subarachnoid hemorrhage (1990–1995)

<table>
<thead>
<tr>
<th>Time After Onset (hrs)</th>
<th>No. of Patients</th>
<th>Thrombin–Antithrombin Complex* (ng/mL, mean ± SEM)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1–1</td>
<td>100</td>
<td>123.8 ± 14.0</td>
</tr>
<tr>
<td>1.1–2</td>
<td>36</td>
<td>121.4 ± 28.3</td>
</tr>
<tr>
<td>2.1–4</td>
<td>35</td>
<td>57.3 ± 14.6</td>
</tr>
<tr>
<td>4.1–12</td>
<td>39</td>
<td>46.3 ± 8.3</td>
</tr>
<tr>
<td>12.1–24</td>
<td>7</td>
<td>18.3 ± 5.9</td>
</tr>
<tr>
<td>24.1–48</td>
<td>11</td>
<td>7.1 ± 1.6</td>
</tr>
<tr>
<td>48.1–72</td>
<td>7</td>
<td>4.7 ± 1.4</td>
</tr>
<tr>
<td>total (0.1–72)</td>
<td>235</td>
<td>88.6 ± 8.3</td>
</tr>
</tbody>
</table>

*The level significantly decreased with increasing time (hours) elapsed after onset (analysis of variance in linear regression). SEM = standard error of the mean.
RESPONSE: Because our study made very similar observations to those in a study by Dr. Fujii and colleagues regarding activation of coagulation and fibrinolysis, we find it unreasonable that he should state that our knowledge of changes in hemostatic parameters would be inadequate. As evidence of our experience with this matter, we refer to various studies we have authored (impact factors ranging between 3 and 8). And by no means was our intention to question the reliability, knowledge, and technique of Dr. Fujii and his colleagues; in fact, we repeatedly agreed with their previous work throughout our Discussion section.

Unfortunately, Fujii, et al., did not provide us with the information regarding the “ultraacute” sampling (3.1 hours after onset), which explains the 10-fold difference in thrombin generation compared with our sampling taken 1.5 days later, while this paper was in progress. This conclusion is strongly supported by the Table 1 provided in Dr. Fujii’s letter. We did not have available the recent article by Fujii, et al., on the peaking of D-dimer at a later stage (3–6 days). These data also explain the discrepancy in thrombin–antithrombin/D-dimer ratio between our studies. Without this information, we were left with the possibility that the laboratory-related technical differences could explain the quantitative differences. Thus, our conclusions stated that the cut-off values need to be set separately for each laboratory. In conclusion, because our results accord very well with those of Fujii, et al., we very much regret that our discussion has offended Dr. Fujii. We sincerely hope that this reply will settle the issue.

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References

Zero Drift in Pressure Monitors

To the Editor: We took notice of the findings of Bavetta, et al., on the zero drift of fiberoptic intracranial pressure monitors (Bavetta S, Norris JS, Wyatt M, et al: Prospective study of zero drift in fiberoptic pressure monitors used in clinical practice. J Neurosurg 86:927–930, June, 1997). The authors have used Camino fiberoptic transducers, located either subdurally or intraventricularly. At the time of removal, the fiberoptic tip was exposed to room air and the zero drift was noted. The zero drift was not related to the time of measurement and varied from −12 to +13 with a clear negative bias. The authors conclude that undue reliance should not be placed on the results of this system alone. In the neuroscience intensive care unit in Rotterdam, it is common practice to monitor, among vital parameters, the intracranial pressure by using the Camino fiberoptic intracranial pressure transducer in which the pressure-sensitive tip is located directly in the brain parenchyma. During the last 2 years we have recorded the zero and sensitivity drifts (at 15 and 30 mm Hg) of the used catheters directly after removal. Seventy-five catheters were calibrated in this way. Four were clearly damaged by patient movements and could not be considered functioning for the calibration. Among the remaining 71 catheters, the adverse zero drift was −2.5 ± 4.0 mm Hg. In 10% of the cases the drift was larger than 8 mm Hg. As also observed in the study of Bavetta, we could not find a relation between the time of use and the zero drift. The histogram (Fig. 1) shows the distribution of zero drift values. One catheter had even drifted −7 mm Hg within 16 hours. Because several monitors were in use, we looked for but did not find a relationship with the system used. No sensitivity drift was observed after lowering the probes in a water column at depths corresponding with pressures of 15 and 30 mm Hg.

The first 37 of the used catheters were tested in a lab bench test. This bench test consisted of zero drift assessments over a 5-day period with the probes in 22°C saline. The temperature coefficient was measured by exposing the probes to 22°C saline and 37°C saline. Static sensitivity was tested with a manometer over a range of 100 to 0

![Zero Drift Camino's after use in ICU (mmHg)](image-url)