Traumatology Without Knowledge of the Trauma: Diagnosis, Scaling, and/or Scoring?

To the Editor: The increasingly lively discussion during the past 30 years concerning improvements in the examination and care of head injuries has followed various patterns.

Early efforts were concentrated on diagnostic terminology, applicable comparatively late in the post-traumatic course and related to the nature of the lesion and to its prognostic importance. It was quite clear that the old concept of terms such as “brain contusion” had often been incorrectly applied, in spite of the inherent pathomorphological meaning of the word “contusion.” Terminology for the primary injury, such as that describing various degrees of concussion, was thought to be more reliable with regard to coma duration and prognosis, and some used even more realistic terms within a few hours after trauma, such as “posttraumatic brief coma” or “prolonged coma.” Thus, further impairment in the patient’s condition could be defined, when indicated, by adding to the primary diagnosis well known pathomorphological terms, such as “focal brain contusion,” “subdural hematoma,” and “extradural hematoma.” Such descriptive terminology may immediately suggest the nature of the lesion and the prognosis, as well as indications for treatment. Other neurosurgeons and neurophysiologists tried to define the primary injury, with its symptoms and signs, by the part of the brain in which it probably occurred by using distinctly anatomical terms.

Some of these investigators included in their diagnostic terms (International Classification of Diseases — ICD) severity-related symptoms and signs well known for their prognostic importance. Thus, a diagnosis such as prolonged coma could already be applied 1 or 2 hours after the injury if there were no verbal response or extension movement on pain, fixed or dilated pupils, absent oculovestibular response, or other signs known to indicate delayed recovery.

A clear need arose for a more differentiated grouping of such signs according to the degree of influence on the outcome. This was the so-called “scaling” of injuries. Among the injury scales, the Abbreviated Injury Scale (AIS) of 1968 became widely used. However, because of its emphasis on morphological aspects, attempts were later made to partly substitute these with clinical observations in the early stages. This system facilitated the study of groups of patients with similarly serious injuries, or at least with injuries with symptoms and signs of comparable severity.

The AIS defines severity of injury as follows:
1. Minor
2. Moderate
3. Severe (not life-threatening)
4. Serious (life-threatening)
5. Critical (survival uncertain)
6. Maximum (currently untreatable).

The possibilities of relating this kind of grouping to an already existing or extended diagnostic terminology are apparent.

A great number of different symptoms and signs have been tested by computer analysis to establish which information is related to prognosis. To put too much weight on one or two of the graded symptoms or signs has been shown to give false scoring numbers with regard to outcome. This type of scoring does not consider directly the cause of the symptoms and can hardly result in diagnostic-prognostic differentiation, or guide the treatment by itself. A more simplified evaluation of the injured patient can easily be performed at the bedside by nurses, and is a very reliable, practical, and continuous monitor of changes in the patient’s condition. Irrespective of some drawbacks, careful observation of the patient has been of utmost importance, and should be extended into the early posttraumatic period.

During recent years, surprisingly little has been done to include existing work on classification of diagnoses and scaling of injuries in forming a useful basis for overall comparison during the whole course, from trauma to the final result of care. However, with improved scoring including all the other well known signs of prognostic importance, such as pupillary disturbances, change in vital signs, and variations in age, we are again approaching the above mentioned scaling groups. I think that better scoring and an appropriate grouping of scoring numbers could result from the use of the already well known severity categories of the AIS scale, later related to a more clearly defined ICD classification.

A simple example of our present state is as follows: Everyone knows that a “brain concussion” or “brief posttraumatic coma” (AIS No. 2: Moderate primary brain injury), complicated with subsequent symptoms and signs of epidural hematoma (AIS No. 4 to 5), should have a good prognosis and outcome if predicted, diagnosed, and treated early enough. It should be possible to make this clear in diagnostic as well as scaling terminology from the information received on admittance, examination, and observation, scoring already done during the development of the condition. However, the earliest available information of the seriousness of the head injury has so far been neglected: that is, the knowledge of the severity and of the site of the trauma. In spite of new methods facilitating the observation of the patient, such as...
computerized tomography scanning, continuous intracranial pressure measurements, and more or less continuous electroencephalographic monitoring, the importance of the findings varies depending on the time of observation after the head injury. Even in this respect we have seen that knowledge of the severity of the site of the trauma on the head has greatly facilitated both early prediction of the prognosis of the primary injury and prediction of subsequent intracranial complications, thereby improving the planning of the care of the injured patient.

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Mannitol and Head Injury

TO THE EDITOR: The well designed study by Frederick D. Brown, et al. (Detailed monitoring of the effects of mannitol following experimental head injury, J Neurosurg 50:423-432, April, 1979), documented many of the hemodynamic changes occurring after mannitol administration. Our understanding of fluid administration in head injury would be increased by knowing the volumes of saline required to maintain normal pulmonary artery wedge pressures and the technique used to measure serum viscosity.

The effects of mannitol on cardiac output and cerebral blood flow were intriguing. Mannitol was noted to strikingly increase cardiac output, but the increase was apparently not of statistical significance. The effect on cardiac output was not felt to be attributable to an increased systemic blood volume, although volume was measured only once during the test period and not at the time (30 minutes after administration was begun) when cardiac output was increased to the greatest extent. The improvement in cerebral blood flow was related to lowered blood viscosity, but flow did not increase as the viscosity declined.

The mannitol-induced improvements in cerebral blood flow and brain metabolism led the authors to suggest the use of mannitol in the early stages after head injury before a rise in intracranial pressure occurs. The administration of mannitol for durations longer than the 6 hours used in the authors' study may well be associated with alterations in systemic blood volume, viscosity, and osmolarity. The use of mannitol in humans before intracranial pressure only once during the test period not at the time, cardi output was increased to the greatest extent. The improvement in cerebral blood flow was related to lowered blood viscosity, but flow did not increase as the viscosity declined.

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RESPONSE: The authors wish to thank Dr. Albright for his thoughtful letter. The quantities of saline had to be titrated for each monkey to maintain normal wedge pressures, but on the average an animal required 4 cc/kg/hr. Blood viscosity was measured with a LVT-SCP Wells-Brookfield Micro Viscometer (Brookfield Engineering Laboratory, Inc., Stoughton, Massachusetts).

The increase in cardiac output was very near but not quite at a p value of 0.05, owing to the fact that a minority of the untreated animals underwent cardiac output measurements. We decided to measure systemic blood volume only once during the test period to avoid excessive blood loss from sampling. The 2-hour interval was chosen because at this point the animal had received almost all of the mannitol doses; thus, if a blood volume increase were to be seen, 2 hours would seem a reasonable time to look for it; only one or in some cases two boluses of mannitol had been given by 30 minutes. Figures 4 and 9 point out that blood viscosity is low from 30 minutes to 6 hours and that cerebral blood flow is significantly increased throughout this time.

We agree that mannitol administration for longer than 6 hours may be associated with alterations in systemic blood volume, viscosity, and osmolarity. One of the most frequent complications of mannitol administration is hypovolemia, which can result in renal failure, from inadequate replacement of urinary fluid losses. Certainly mannitol treatment should include precise monitoring of fluid intake and output, serum electrolytes, and osmolality, and a central venous pressure line if not a Swann Ganz catheter for pulmonary artery wedge pressure determinations. Through careful monitoring of the above, we can maintain a monovolemic state. Viscosity and osmolality changes will depend greatly on the doses of mannitol employed.

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Compensatory Sweating after Upper Dorsal Sympathectomy

TO THE EDITOR: Dts. Shih and Lin recently reported abnormalities in quantitative distribution of thermoregulatory sweating in patients after upper dorsal sympathectomy (UDS) for palmar hyperhidrosis (Shih C-J, Lin M-T: Thermoregulatory sweating in palmar hyperhidrosis before and after upper thoracic sympathectomy, J Neurosurg 50:88-94, January, 1979). This conclusion was based on changes in local sweating rate (LSR) showing a significant increase in sweating of the ventral thigh and lumbar regions (areas that were not denervated) at 41°C in patients after UDS.

In our initial series of UDS for palmar hyperhidrosis,1 now extended to over 250 patients, we noticed that almost all patients complained of increased sweating in the non-denervated areas. Shoenfeld, et al.,2 studied 60 UDS patients, tested by 1 hour of exercise at 30°C and 50% relative humidity, and reported that the total amount of sweating from the whole body was unchanged after bilateral UDS. They reported preoperative values of 5.7 ± 4.2 and