J Neurosurg 83:438–444, 1995

Early detection of delayed traumatic intracranial hematomas using near-infrared spectroscopy

SHANKAR P. GOPINATH, M.D., CLAUDIA S. ROBERTSON, M.D., CHARLES F. CONTANT, PH.D., RAJ K. NARAYAN, M.D., ROBERT G. GROSSMAN, M.D., AND BRITTON CHANCE, PH.D.

Department of Neurosurgery, Baylor College of Medicine, Houston, Texas; and Department of Biochemistry and Biophysics, University of Pennsylvania, Philadelphia, Pennsylvania

Delayed intracranial hematomas are an important treatable cause of secondary brain injury in patients with head trauma. Early identification and treatment of these lesions, which appear or enlarge after the initial computerized tomography (CT) scan, may improve neurological outcome. Serial examinations using near-infrared spectroscopy (NIRS) to detect the development of delayed hematomas were performed in 167 patients. The difference in absorbance of light (ΔOD) at 760 nm between the normal and the hematoma side was measured serially during the first 3 days after injury. Twenty-seven (16%) of the patients developed a type of late hematoma: intracerebral hematoma in eight, extracerebral hematoma in six, and postoperative hematoma in 13 patients. Eighteen of the delayed hematomas caused significant mass effect and required surgical evacuation. The hematomas appeared between 2 and 72 hours after admission. In 24 of the 27 patients, a significant increase (> 0.3) in the ΔOD occurred prior to an increase in intracranial pressure, a change in the neurological examination, or a change on CT scan. A favorable outcome occurred in 67% of the patients with delayed hematomas, which suggests that early diagnosis using NIRS may allow early treatment and reduce secondary injury caused by delayed hematomas.

KEY WORDS • near-infrared spectroscopy • head injury • subdural hematoma • epidural hematoma • intracerebral hematoma • delayed traumatic hematoma

CLINICAL studies have documented the importance of secondary brain insults in determining neurological outcome after head injury. Delayed intracranial hematomas are one of the most easily remedied causes of secondary injury if identified early but can cause significant disability or death if not promptly recognized and treated. Computerized tomography (CT) scanning has revealed that delayed hematomas after head trauma are more common than had been previously suspected.[3,7,10,11,14,18,22,26,28,29] The incidence of delayed traumatic intracerebral hematoma varies from 2.3% to 8.4% of severely head injured patients.[7,8,14,17,18,24,26,27] The incidence of delayed epidural hematoma varies from 9% to 23%.[4,22,25] Postoperative intracranial hematomas can also occur, with an incidence ranging from 7.8% to 61%.[15,16,19,23] In a recent large series of 850 patients who underwent craniotomy for evacuation of a traumatic hematoma, 88 (10.4%) required a second operation for removal of a second intracranial lesion.[6] Mortality rate and the incidence of a poor neurological recovery are significantly increased in patients who develop delayed traumatic intracranial hematomas.[9,14,20,21,29]

Serial CT scans are the most reliable method for detecting a delayed hematoma. However, CT scans require that patients, many of whom are critically ill, be taken out of the intensive care unit, and the yield is relatively low if serial scans are obtained in all patients. A clinical monitoring technique for accurate selection of patients requiring follow-up CT would improve the yield.

Current clinical monitoring techniques, such as intracranial pressure (ICP) monitoring and serial neurological examinations, are not ideal for detecting delayed hematomas. Patients with delayed hematomas may appear to be relatively normal and later demonstrate sudden neurological deterioration,[29] or they may not exhibit a change in their neurological examination.[1,9,14,25] Intracranial pressure may be normal in up to 20% of patients harboring delayed hematomas that require surgery.[2,5,14]

The ideal clinical monitor would be capable of making online continuous measurements in the intensive care unit and would identify the development of a hematoma prior to the onset of clinical neurological deterioration. A CT scan could then be performed to obtain more information about the size and location of the hematoma. A previous study demonstrated that near-infrared spectroscopy (NIRS) performed in the emergency room reliably identified the presence of a traumatic intracranial hematoma in each of 40 patients in whom a CT scan also revealed the

presence of a hematoma.[13] The majority of the hematomas detected were subdural (22) or epidural (10). Only eight patients had intracerebral hematomas. The difference in absorbance of near-infrared light at 760 nm between the normal side and the side of the brain harboring a hematoma was directly related to the thickness of the hematoma and was specific for acute collections of blood. The purpose of this study was to determine whether NIRS would be useful in identifying the development of delayed intracranial hematomas in patients in the intensive care unit.

Clinical Material and Methods

Patient Characteristics and Management

One hundred sixty-seven patients who were admitted to Ben Taub General Hospital with a moderate (Glasgow Coma Scale (GCS) score 9–12) or severe (GCS score ≤ 8) head injury were studied with NIRS and CT. The age of the patients ranged from 4 months to 101 years. One hundred forty-one patients were male and 26 were female. The GCS score was 3 to 8 in 77 patients and 9 to 12 in 90 patients.

All patients were evaluated with an initial CT scan and were followed with serial neurological examinations. Patients with GCS scores of less than or equal to 8 also had ICP monitoring. A repeat CT scan was obtained at 24 to 48 hours postinjury. A CT scan was also obtained after the occurrence of neurological deterioration, increasing ICP, or suggestion of an intracranial hematoma by NIRS examination. Indications for surgery were a midline shift greater than 5 mm, intracranial hypertension, or neurological deterioration.

Method of NIRS Examination

An NIRS examination was performed on the patient in the emergency room at the time of the admission CT scan, and then serial measurements were obtained during the hospital course, along with follow-up CT scans. A dual wavelength NIRS unit (RunMan, NIM, Inc., Philadelphia, PA) was used to quantitate hemispheric differences in light absorbance. The NIRS unit is compact, battery operated, and easily transported to the emergency room or intensive care unit. The probe consists of two tungsten filament lamps on either side of a 760- and 850-nm light detector. The 4-cm separation of light source and detector allows measurement of near-infrared light absorbance in a volume of tissue approximately 2 cm wide by 2 to 3 cm deep. The detector measures the intensity of the unabsorbed or reflected light.

The probe of the NIRS unit was placed successively on the frontal, parietal, occipital, parasagittal, and suboccipital regions of the scalp on both sides of the head. The intensity of unabsorbed light at 760 nm was recorded in each of these regions. The difference in optical density (ΔOD) between the hemispheres in each of these regions was calculated by the formula: $\Delta OD = \log_{10} (I_N \div I_H)$, where I_N is the intensity of the reflected light on the normal side and I_H is the intensity of the reflected light on the hematoma side. The maximum ΔOD among the various regions examined was recorded for each patient.

Statistical Analysis

Data are expressed as the median and range. Differences in median values were compared by Kruskal–Wallis analysis of variance and Dunn's method when multiple comparisons were made. The ΔOD was compared to the thickness of the hematoma on CT scan by nonlinear regression analysis. A p value of less than 0.05 was considered significant.

Results

Initial Diagnosis: Relationship to ΔOD

The maximum ΔOD, measured in the emergency room, correlated with the findings on the initial CT scan. As in the previous study,[13] there were characteristic abnormalities of near-infrared light absorbance found in patients with intracranial hematomas.

Diffuse Brain Injury. The initial diagnosis was diffuse brain injury in 49 of the patients. The median ΔOD was 0.03 (range 0–0.05) in these patients. The distribution of ΔOD among these patients is shown in Fig. 1. These values were not significantly different from those observed in normal adults (0.02 ± 0.01) and previously reported.[13]

Intracranial Hematomas. The initial diagnosis included an intracranial hematoma in 118 of the patients. Figure 1 shows the frequency distribution of the ΔOD obtained in the different types of intracranial hematoma. The ΔOD in patients with an extracerebral hematoma (subdural or epidural hematoma) was significantly greater than in patients with intracerebral hematomas ($p < 0.001$). The ΔOD in the patients with all types of hematomas was significantly greater than in patients with diffuse brain injury ($p < 0.001$).

Thirty-five patients had an epidural hematoma on their initial CT scan. The median ΔOD was 1.12 (range 0.12–1.60). The thickness of the epidural hematoma on the initial CT scan and the ΔOD in the emergency room were significantly related (Fig. 2 *left*; $r^2 = 77\%$, $p < 0.01$). The ΔOD increased with the size of the hematoma up to a thickness of approximately 1.5 cm and then plateaued.

Sixty-two patients had a subdural hematoma on their initial CT scan. The median ΔOD was 0.82 (range 0.32–1.82). The thickness of the subdural hematoma on the initial CT scan and the ΔOD in the emergency room were significantly related (Fig. 2 *right*; $r^2 = 46\%$, $p < 0.01$).

Fifteen patients had an intracerebral hematoma and six patients had a contusion revealed on the initial CT scan. The median ΔOD was 0.38 (range 0.29–0.75) with an intracerebral hematoma and 0.18 (range 0.05–0.38) with a contusion. The ΔOD was not significantly related to the size of the intracerebral hematoma, perhaps because a significant volume of the hematoma was deeper than the 2-cm light penetration afforded by the design of the NIRS probe used in this study.

Delayed Lesions: Relationship to ΔOD

Late lesions included delayed traumatic intracerebral hematomas (new intracerebral hematoma or coalescence of a preexisting contusion into a hematoma), delayed extracerebral hematomas (new subdural or epidural hema-

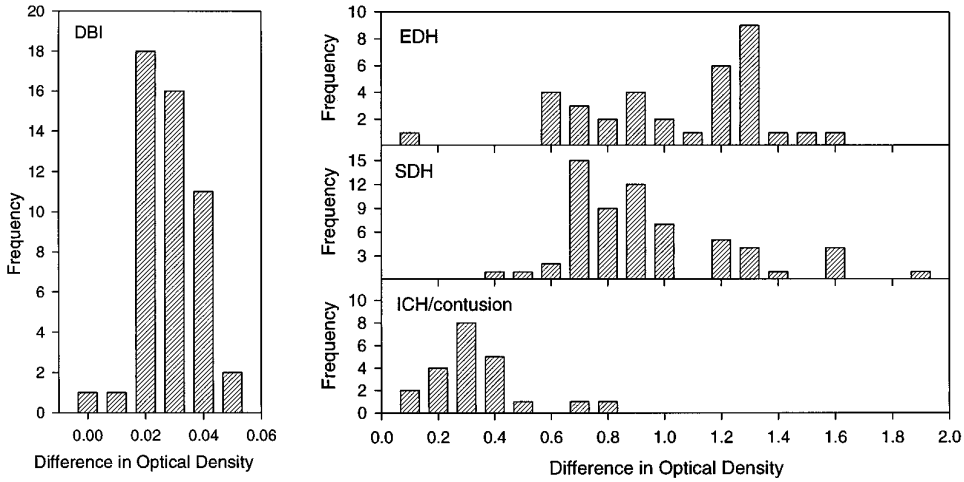

FIG. 1. Bar graphs showing frequency of distribution of initial measurements of optical density (ΔOD) in patients with diffuse brain injury (DBI) *(left)* and with an intracranial hematoma *(right)*. With DBI, the scale is 0 to 0.06 OD and in patients with intracranial hematomas, the scale is 0 to 2.0 OD. EDH = epidural hematoma; SDH = subdural hematoma; ICH = intracerebral hematoma.

toma or enlargement of a preexisting hematoma), and development of a hematoma at the operative site. Twenty-seven (16%) of the 167 patients developed some type of late intracranial hematoma, either an intracerebral (eight patients), extracerebral (six patients), or postoperative hematoma (13 patients). The types of late lesions are summarized in Table 1.

Diffuse Brain Injury. Three of the 49 patients with diffuse brain injury developed lesions that were not present on the initial CT, at times varying from 2 to 48 hours postinjury. One patient with multiple skull fractures developed an epidural hematoma 2 days postinjury. The ΔOD in this patient was initially 0, and it gradually increased over a period of 48 hours to 1.42, prompting a CT scan that revealed the epidural hematoma. A second patient developed a delayed traumatic intracerebral hematoma 2 hours after a normal CT scan. The ΔOD increased from 0.05 to 0.37 over the same 2-hour period. A third patient developed a subdural hematoma and frontal contusion 5 hours after a normal CT scan. The ΔOD increased from 0.03 to 0.82 over the 5 hours between CT scans. All three late lesions required surgical evacuation.

Epidural Hematomas. Twenty-three of the 35 patients with an initial diagnosis of epidural hematoma had surgical evacuation of the hematoma on admission to the hospital; 12 patients were treated without surgical evacuation because the epidural hematoma was small and localized. Late lesions developed in three of the patients initially treated surgically and in two of the patients initially treated medically.

Figure 3 *upper* shows the average ΔOD on the admission NIRS examination in all 23 patients treated surgically, postoperatively in the 20 patients with uncomplicated postoperative courses, and in the three patients who developed some type of intracranial hematoma postoperatively. These postoperative complications included: recurrence

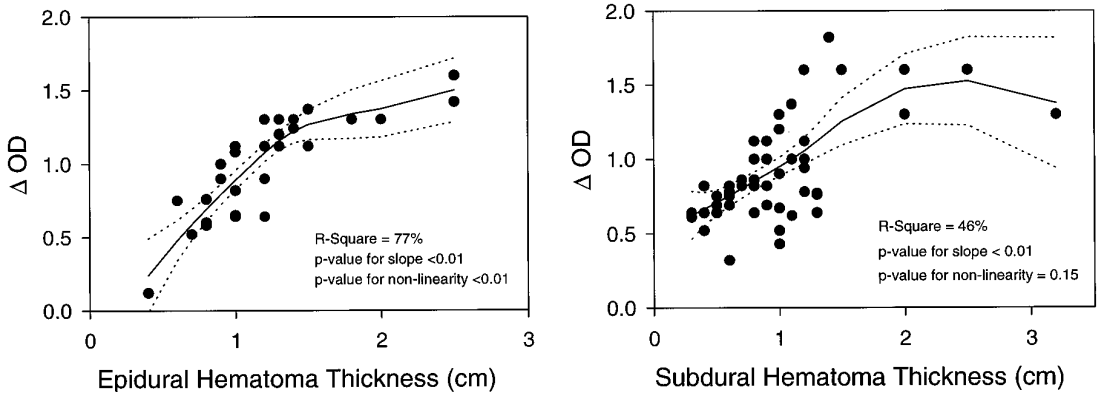

FIG. 2. Scatterplots showing that the initial optical density (OD) was significantly related to the thickness of the hematoma on the admission computerized tomography scan in patients with epidural hematoma *(left)* and subdural hematoma *(right)*. The solid lines are the estimated regression line, and the dotted lines are the 95% confidence intervals.

TABLE 1

*Types of delayed lesions observed in 167 head-injured patients**

Initial Lesion	Late Lesion: Recurrence of Initial Lesion	Late Lesion: Postop EDH	Late Lesion: New Lesion, No.	Late Lesion: Increase in Size of Preexisting Lesion, No.	Incidence
DBI	0	0	EDH, 1 DTICH, 1 SDH + CONT, 1	0	3/49 (6)
EDH	1	0	EDH, 2	EDH, 2	5/35 (14)
SDH	6	3	DTICH, 1	0	10/62 (16)
ICH/CONT	2	1	0	DTICH, 6	9/21 (43)
total	9	4	6	8	27/167 (16)

* EDH = epidural hematoma; DBI = diffuse brain injury; DTICH = delayed traumatic intracerebral hematoma; SDH = subdural hematoma; CONT = contusion; ICH = intracerebral hematoma. Numbers in parentheses are percentages.

of the epidural hematoma; and delayed development of epidural hematoma on the side opposite the original lesion. Two of these late lesions were surgically evacuated.

In addition, two of the 12 patients who were initially managed medically had significant increases in the size of the epidural hematoma during the early hospitalization, with one patient ultimately requiring surgery. Figure 3 *upper* shows the average ΔOD on the admission NIRS examination in the 12 patients who were treated nonsurgically, and the average ΔOD in the two patients who had enlargement of the epidural hematoma during the hospital course.

Of the five late lesions identified after an initial diagnosis of epidural hematoma, three required surgical evacuation. Two of the late lesions were small and resolved spontaneously.

Subdural Hematomas. Fifty-three of the 62 patients with an initial diagnosis of subdural hematoma had surgical evacuation of the hematoma on admission to the hospital; nine patients were not treated surgically because the subdural hematoma was small and not associated with mass effect. Late lesions developed in 10 of the patients who were treated surgically.

Figure 3 *center* shows the average ΔOD on the initial scan in all 53 patients who were treated surgically, as well as the average postoperative ΔOD in the 43 patients with uncomplicated courses, and in the 10 patients who developed some type of postoperative intracranial hematoma that was significantly different ($p < 0.05$). These postoperative complications included: recurrence of the subdural hematoma (six patients), epidural hematoma at the operative site (three patients), and delayed traumatic intracerebral hematoma (one patient). Seven of these 10 late lesions were lesions that had to be treated surgically.

The nine patients who were treated medically all had uneventful hospital courses. The NIRS examination revealed a gradual return to normal values for ΔOD (Fig. 3 *center*).

Intracerebral Hematomas or Contusions. Thirteen of the 15 patients in whom an initial diagnosis of intracerebral hematoma had been made received surgical evacuation of the hematoma on admission to the hospital, and two were treated medically. All six patients in whom a contusion was revealed on the initial CT scan were treated medically. Figure 3 *lower* shows the average ΔOD on the initial scan in the 13 patients who were treated surgically, as well as the average postoperative ΔOD in the 10 patients who had uncomplicated courses, and in the three patients who developed some type of postoperative intracerebral hematoma that was significantly different ($p < 0.05$). These postoperative complications included recurrence of the intracerebral hematoma (two patients), and epidural hematoma at the operative site (one patient). Two of the three late lesions required surgical evacuation.

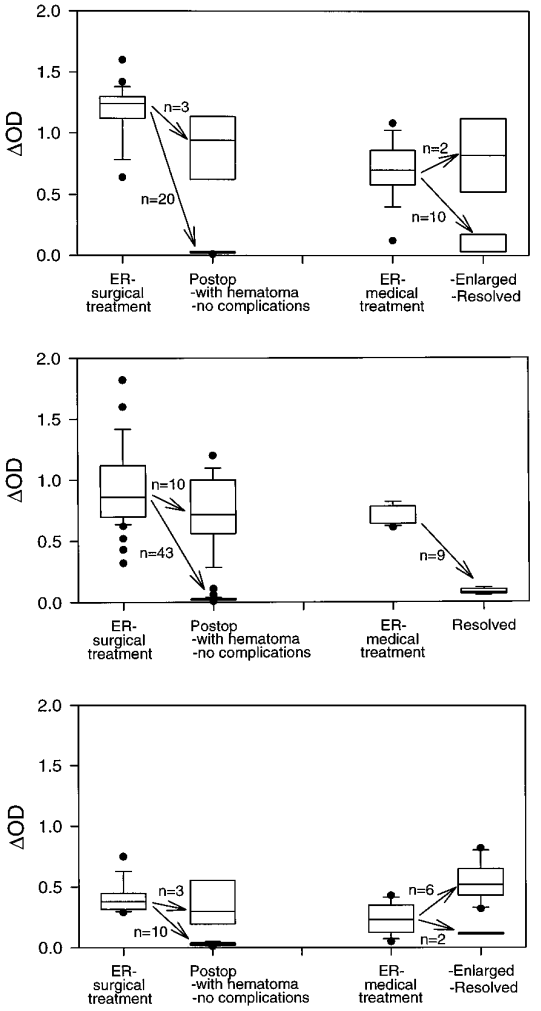

FIG. 3. Graphs illustrating the distribution of the optical density (ΔOD) in patients with and without late hematomas, after an injury resulting in an initial diagnosis of epidural hematoma *(upper)*, subdural hematoma *(center)*, and intracerebral hematoma or contusion *(lower)*. In each circumstance, the patients who developed late hematomas had significantly higher values for ΔOD than did patients with uncomplicated courses. In each graph, the box plots show the median of the distribution as a horizontal line across the box. The vertical edges of the box mark the 25th and 75th percentiles and the error bars mark the 10th and 90th percentiles. The closed circles mark any data points outside the 10th and 90th percentiles. ER = emergency room.

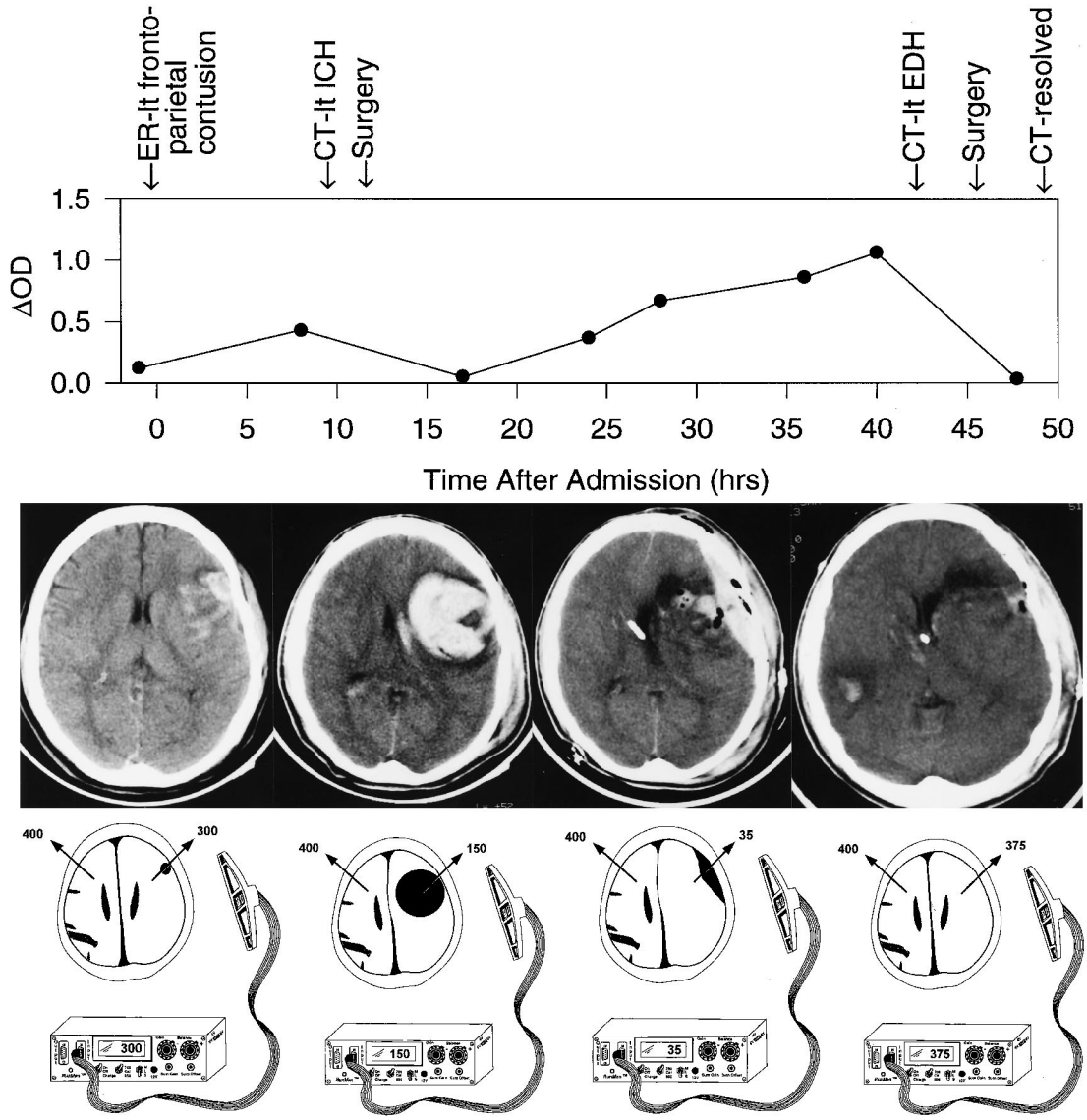

FIG. 4. Illustrative case of a patient who developed both intracerebral and extracerebral hematomas. *Upper:* Graph illustrating the entire sequence of events. *Center:* Serial computerized tomography (CT) scans. *Lower:* Drawings showing the near-infrared spectroscopy (NIRS) examination at the time of the CT scan. This patient was admitted to the hospital with a Glasgow Coma Scale score of 6 after an auto–pedestrian accident. A CT scan obtained at the time of admission revealed a left frontoparietal contusion. He was monitored, and 8 hours later the NIRS suggested the development of an intracranial hematoma. A CT scan was obtained and demonstrated a large left frontal intracerebral hematoma. He was taken to surgery and the hematoma was evacuated. Postoperatively, the optical density returned to normal values, but then gradually increased over several hours. A repeat CT scan on the 2nd postoperative day showed an epidural hematoma, which was surgically evacuated.

In addition, the six patients who initially had a contusion and were treated medically had coalescence of the contusion into an intracerebral hematoma. Three of the six patients required surgical evacuation of the hematoma. Figure 3 *lower* shows the average ΔOD on the admission NIRS examination in the six patients who were treated nonsurgically and the average ΔOD in these patients, who had coalescence of the contusion to an intracerebral hematoma during the hospital course.

Comparison of Changes in ΔOD to Clinical Monitors. Clearly abnormal changes in ΔOD (> 0.3) preceded the detection of the hematoma from clinical signs (change in neurological examination or increase in ICP) or from a routine follow-up CT scan in 24 of the 27 patients who developed a late lesion. On two occasions in patients who developed a recurrent intracerebral hematoma, the ΔOD increased but not to more than 0.3, and in one patient who had bilateral lesions, the ΔOD did not change as both lesions increased in size.

An example of the serial changes in ΔOD that occurred as an intracerebral hematoma, then a postoperative epidural hematoma developed, is shown in Fig. 4. Summary

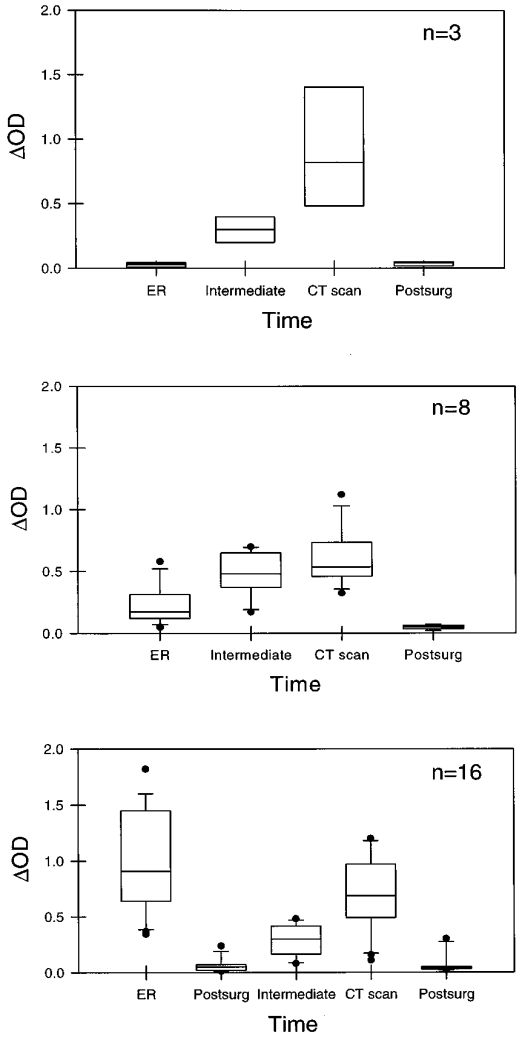

FIG. 5. Box plots illustrating the three patterns of serial changes in optical density (ΔOD) observed in patients who developed late hematomas. Patients who developed a hematoma after having a normal computerized tomography scan initially *(upper)*, had normal ΔOD on the emergency room examination but developed increasing ΔOD during the hospitalization. Patients who had enlargement of a preexisting lesion *(center)* had elevated ΔOD in the emergency room, followed by increasing values during the hospitalization. Patients who developed a new hematoma after craniotomy for a traumatic hematoma *(lower)* had initially high ΔOD values on the emergency room examination, normal ΔOD immediately postoperatively, then gradually increasing ΔOD during the hospitalization. In each graph, the box plots show the median of the distribution as a horizontal line across the box. The vertical edges of the box mark the 25th and 75th percentiles and the error bars mark the 10th and 90th percentiles. The closed circles mark any data points outside the 10th and 90th percentiles. ER = emergency room.

graphs demonstrating these changes in all 27 patients are shown in Fig. 5.

Neurological Outcome

Outcome was graded according to the Glasgow Outcome Scale and was assessed at 3 months or more postinjury. Eighteen (67%) of the 27 patients had a favorable outcome (good recovery and moderate disability), whereas nine patients (33%) had a poor outcome (severe disability, vegetative state, and death). Of the 18 patients with a surgical lesion identified by the NIRS, 12 (67%) had a favorable outcome and six (33%) had a poor outcome.

Discussion

The 16% incidence of delayed traumatic hematomas in the present study was comparable to that reported in the literature. The primary difference in the present study was the identification of the delayed hematomas at a relatively early time, prior to any neurological consequences in the majority of patients. The hematomas were identified as early as 2 hours, and as late as 72 hours after admission. Even if serial CT scans had been performed on every patient within the first 24 hours postinjury, no more information would have been obtained than with the serial NIRS examinations. The NIRS examination was successful in detecting intracerebral as well as subdural and epidural hematomas.

Earlier identification and treatment of intracranial hematomas should prevent one cause of secondary injury, resulting in a better neurological outcome. This study was not a randomized trial, and caution should be taken in comparing studies with possibly differing entry criteria and severities of injury. Nevertheless, the 67% incidence of favorable outcomes in the current series was better than might have been expected based on previous reports. Mortality rates of 36.5% to 50% have been reported for delayed traumatic intracerebral hematoma.[9,14,20,21,29] Postcraniotomy hematomas have been reported to result in a poor outcome in nearly two-thirds of patients.[6]

One possible explanation for the better outcome in the present study could be that hematomas that would not have resulted in subsequent neurological deterioration were identified. This is not likely because the incidence of hematomas is similar to previous studies, and because the outcome was better even if patients with only surgical lesions were examined.

There are several potential limitations in the identification of intracranial hematoma using NIRS in its current form. The location of the hematoma cannot be precisely determined. Intracerebral hematomas tend to absorb light less intensely than extracerebral hematomas, but there is some overlap. A CT scan must be obtained to gather information necessary to make surgical decisions.

Because the NIRS examination relies on comparison of light absorption of normal brain to the hematoma, bilateral hematomas might not be detected. One patient in the present series had enlarging bilateral temporal lesions that did not change the ΔOD. The NIRS delineates the edges of the hematoma; therefore an adjacent unaffected area could be used as a reference site.

Because the NIRS examination relies on light absorption by hemoglobin, initially in the oxy form and subsequently in the met form, the eventual metabolic products of hemoglobin that may occur in chronic subdural hematoma would not be detected. In a previous report, the light absorbance was found to be increased, decreased, or not changed on the side of a chronic subdural hematoma.[12]

With the probe configuration that was used in these studies, the mean depth of brain examined was not more than 2 to 2.5 cm; thus, the relationship between ΔOD and the thickness of the extracerebral hematomas plateaued at a thickness of approximately 1.5 cm. In addition, an intracerebral hematoma more than 2 cm below the surface of the brain would not be detected with this probe configuration. The separation of the optical detectors and the light source could be readily widened to increase the depth of the brain that could be examined.

Near-infrared spectrosocpy has promise as a technology that will allow early identification and treatment of intracranial hematomas. Development of the technology may improve the resolution and depth of the NIRS examination.

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Manuscript received August 25, 1994.
Accepted in final form November 7, 1994.
This work was supported by National Institutes of Health Grants PO1-NS27616 and PO1-NS27346 and by a research grant from the International Trauma Anesthesia and Critical Care Society.

Address reprint requests to: Shankar P. Gopinath, M.D., Department of Neurosurgery, Baylor College of Medicine, One Baylor Plaza, Houston, Texas 77030.