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Near-infrared spectroscopic localization of intracranial hematomas

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✓ Near-infrared spectroscopy (NIRS) of the cerebral hemispheres, applied transcranially through the intact scalp and skull, was evaluated for its ability to detect the presence of an intracranial hematoma in 46 head-injured patients. In 40 patients intracranial hematomas (22 subdural, 10 epidural, eight intracerebral) were identified on computerized tomography (CT); in all 40 cases, NIRS demonstrated greater absorption of light at 760 nm on the side of the hematoma. The mean difference in optical density (OD) between the hemisphere with the hematoma and the normal hemisphere was 0.99 ± 0.30 for epidural hematomas, 0.87 ± 0.31 for subdural hematomas, but only 0.41 ± 0.11 for intracerebral hematomas. In 36 patients, the asymmetry in OD resolved after surgical evacuation of the hematoma or with spontaneous resorption of the hematoma. Four patients who developed postoperative or delayed hematomas exhibited persistence of the asymmetry in OD. Six patients had only diffuse injuries and exhibited only minor differences in OD between the hemispheres, similar to 10 patients in the control group with no head injury. It appears that NIRS is useful in the initial examination of the head-injured patient, as an adjunct to CT, and in following patients postoperatively in the intensive care unit.

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

THE application of near-infrared spectroscopy (NIRS) to the study of human cerebral metabolism was described by Jöbsis.[12] Unlike visible light, near-infrared light can penetrate the scalp, skull, dura, and brain for several centimeters, and thus information about intracerebral attenuation of light can be obtained.[6,9] In the brain, there are three major light-absorbing molecules;[1,12,15] these chromophores include oxyhemoglobin, deoxyhemoglobin, and cytochrome aa_3. The absorption of oxyhemoglobin and reduced hemoglobin is detected at a variety of wavelengths near 760 nm. Increasing absorbance at 760 nm represents increases in deoxyhemoglobin levels.[14] Oxidized cytochrome aa_3 is detected near 830 nm,[5] the contributions from aa_3 being small.[1]

Transcranial NIRS has been used to detect cerebral hypoxia and changes in cerebral blood volume.[2–5,8,11,16] In addition, NIRS has been used to detect disturbances in autoregulation of cerebral blood flow (CBF) in newborns,[13] to identify the presence of hyperoxia after circulatory arrest and reperfusion,[14] to detect metabolic brain injury in infants,[7] and to measure CBF in preterm infants.[10] The potential of NIRS as a method of localizing intracranial hematomas has not been systematically studied. Extravascular blood should strongly absorb near-infrared light compared to intravascular blood because of the greater concentration of hemoglobin in the hematoma than in brain tissue. The present study was carried out to test the hypothesis that absorbance of near-infrared light would be greater on the side of the brain containing a hematoma than on the uninjured side.

Clinical Material and Methods

Forty-six patients who were admitted to Ben Taub General Hospital, in Houston, Texas, between August, 1991, and March, 1992, with a severe head injury were studied with NIRS and computerized tomography (CT). The patients ranged in age from 4 months to 74 years; 41 patients were male and five were female. Thirty-nine patients were studied in the emergency room prior to obtaining a CT scan, while the remaining seven patients were examined in the operating room prior to surgery or in the intensive care unit if surgery was not required. In all cases the examiner was blinded to the results of the CT scan.

TABLE 1

*Difference in optical density in patients with epidural hematomas**

Case No.	Age (yrs)	Side of Hematoma	Preop ΔOD	Postop ΔOD	Postop CT Scan
evacuation of hematoma					
1	23	lt	1.37	0.02	no hematoma
2	17	rt	1.30	0.03	no hematoma
3	7	lt	0.64	0.02	no hematoma
4	30	lt	0.90	0.02	no hematoma
5	11	rt	1.12	0.02	no hematoma
6	39	lt	1.30	0.02	no hematoma
7	29	rt	0.64	0.52	recurrent hematoma
8	20	lt	1.00	0.94	delayed epidural hematoma (opposite side)
nonsurgical treatment					
9	17	rt	0.52		
10	$\frac{1}{3}$	lt	1.08		
mean ± SD			0.99 ± 0.30	0.02 ± 0.01	no hematoma
				1.01 ± 0.27	postop hematoma

* ΔOD = difference in optical density between the hematoma site and the opposite side of the head; CT = computerized tomography; SD = standard deviation.

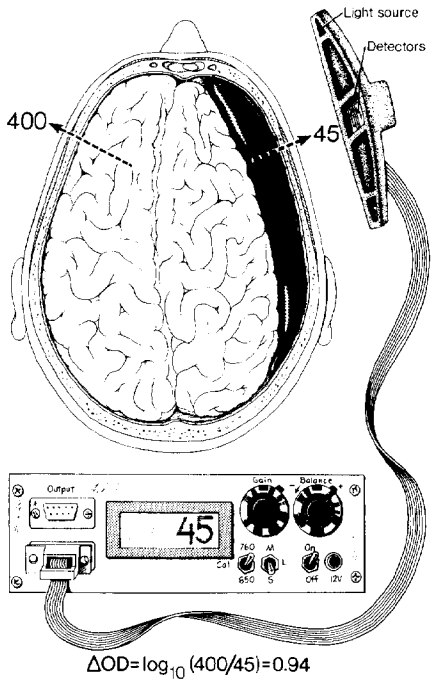

FIG. 1. Diagram of the procedure used to measure the difference in optical density (ΔOD) between the hemispheres. The intensity of the unabsorbed or reflected light was measured by placing the near-infrared spectroscopy probe on each side of the head. The right and left values were similar in the control patients and the patients with diffuse injuries. In patients with intracranial hematomas, there was a marked reduction in the amount of unabsorbed light on the side of the hematoma.

TABLE 2

*Difference in optical density in patients with subdural hematomas**

Case No.	Age (yrs)	Side of Hematoma	Preop ΔOD	Postop ΔOD	Postop CT Scan
evacuation of hematoma					
1	30	rt	0.83	0.02	no hematoma
2	2	lt	0.69	0.01	no hematoma
3	34	rt	0.43	0.02	no hematoma
4	16	lt	1.30	0.03	no hematoma
5	17	lt	0.78	0.03	no hematoma
6	74	rt	1.37	0.03	no hematoma
7	25	rt	0.62	0.03	no hematoma
8	41	lt	0.82	0.05	no hematoma
9	43	rt	0.69	0.02	no hematoma
10	20	rt	0.78	0.03	no hematoma
11	17	lt	0.82	0.02	no hematoma
12	39	lt	0.86	0.02	no hematoma
13	28	lt	0.76	0.03	no hematoma
14	40	rt	0.94	0.03	no hematoma
15	39	lt	1.00	0.02	no hematoma
16	20	rt	1.12	0.03	no hematoma
17	30	rt	0.86	0.02	no hematoma
18	53	lt	1.82	1.20	epidural hematoma
19	64	rt	0.52	0.82	epidural hematoma
nonsurgical treatment					
20	45	lt	0.77		
21	3	lt	0.61		
22	11	rt	0.82		
mean ± SD			0.87 ± 0.31	0.03 ± 0.01	no hematoma
				1.01 ± 0.27	postop hematoma

* ΔOD = difference in optical density between the hematoma site and the opposite side of the head; CT = computerized tomography; SD = standard deviation.

A dual wavelength NIRS unit* 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 placed 3.5 cm on either side of a 760- and 850-nm light detector. The 3.5-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. Optical density (OD) can be calculated from the formula:

$$OD = \log_{10} \frac{I_O}{I_A},$$

where I_O is the intensity of the illuminating beam and I_A is the intensity of the 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 OD (ΔOD) between the hemispheres in each of these regions

* RunMan dual wavelength near-infrared spectroscopy unit manufactured by NIM, Inc., Philadelphia, Pennsylvania.

TABLE 3

*Difference in optical density in patients with intracerebral hematomas**

Case No.	Age (yrs)	Site of Hematoma	Preop ΔOD	Postop ΔOD
evacuation of hematoma				
1	29	lt frontal	0.60	0.04
2	28	lt temporal	0.30	0.05
3	72	rt parietal	0.39	0.03
4	35	lt temporal	0.56	0.02
5	33	rt temporal	0.41	0.02
6	65	rt parietal	0.30	0.03
nonsurgical treatment				
7	28	lt temporal	0.43	
8	45	rt cerebellar	0.32	
mean ± SD			0.41 ± 0.11	0.03 ± 0.01

* ΔOD = difference in optical density between the hematoma site and the opposite side of the head; SD = standard deviation.

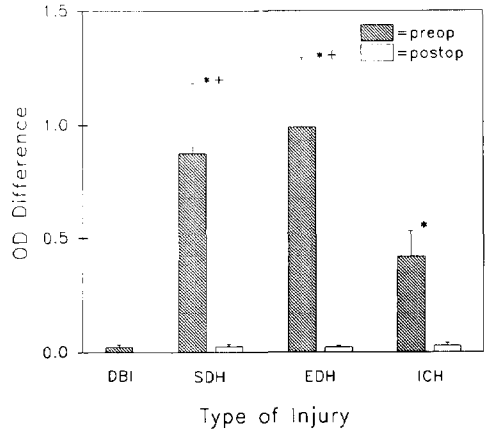

FIG. 2. Bar graph showing the mean difference in optical density (OD) in the four types of injuries examined. An *asterisk* indicates a significant difference ($p < 0.05$) in OD from the patients with diffuse brain injury (DBI). A *cross* indicates a significant difference ($p < 0.05$) in OD from the patients with intracerebral hematoma (ICH). SDH = subdural hematoma; EDH = epidural hematoma.

was calculated by the formula:

$$\Delta OD = \log_{10} \frac{I_O}{I_N} - \log_{10} \frac{I_O}{I_H} = \log_{10} \frac{I_N}{I_H},$$

where I_O is the intensity of the illuminating beam, 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. These calculations are illustrated in Fig. 1. The maximum ΔOD among the various regions examined was recorded for each patient. A control group of 10 patients without cranial injuries were examined in a similar manner, and the maximum ΔOD between the right and the left hemispheres was recorded for each.

The diagnosis based on the admission CT scan of the head-injured patients included: acute subdural hematoma (22 patients), epidural hematoma (10 patients), intracerebral hematoma (eight patients), and diffuse brain injury (six patients). In 34 patients, the hematoma was evacuated soon after admission, CT was repeated 24 to 48 hours postoperatively, and the NIRS was repeated during the first 3 days following surgery. The remaining 12 patients were treated conservatively; all underwent follow-up CT scanning and NIRS at 24 to 48 hours after treatment and at weekly intervals for up to 3 weeks until the hematoma resolved.

Data are expressed as the mean ± standard deviation. Differences in mean values were compared by analysis of variance and Fisher's least-significant difference test when multiple comparisons were made. The ΔOD was compared to the thickness of the hematoma on CT scan by linear regression analysis. A p value < 0.05 was considered significant.

Results

In the 10 control patients without head injury, the maximum ΔOD at 760 nm between the right and left sides averaged 0.02 ± 0.01. The six head-injured patients with diffuse brain injury had only minor differences in OD between the right and left sides, averaging 0.02 ± 0.01, which was not significantly different from that observed in the control patients.

In all 40 patients with intracranial hematomas, a significant difference was detected in OD between the hematoma side and the normal side. The preoperative ΔOD varied from 0.30 to 1.82 (Tables 1 to 3); it was highest in the patients with extracerebral hematomas, averaging 0.99 in the 10 patients with epidural hematomas and 0.87 in the 22 patients with subdural hematomas, compared to only 0.41 in the eight patients with intracerebral hematomas ($p < 0.01$) (Fig. 2). In general, acute epidural and subdural hematomas had a ΔOD of greater than 0.60 and intracerebral hematomas had a ΔOD of less than 0.60. There was sufficient overlap, however, that an extracerebral hematoma could not be distinguished from an intracerebral hematoma with absolute certainty.

In the 32 patients with extracerebral hematomas (Fig. 3), there were 33 occasions where simultaneous measurement of ΔOD was significantly related to the thickness of the hematoma ($r = 0.45$, $p = 0.007$). In the eight patients with intracerebral hematomas, this relationship was not so clear, perhaps because the bulk of the hematoma was beyond the penetration of near-infrared light or because there was a variable amount of normal and/or edematous brain between the hematoma and the near-infrared light source and detector.

Of the 22 patients with a subdural hematoma, 19 underwent rapid evacuation of the hematoma. Postoperatively, the ΔOD returned to near normal, or an average of 0.03 ± 0.01, in the 17 patients who had successful evacuation of the hematoma. In the other two patients who developed postoperative epidural hematomas the ΔOD was still 1.20 and 0.82, respectively, after the initial surgery; in one of these, NIRS suggested the presence of a recurrent hematoma before the intracranial pressure increased to abnormally high levels.

Of the 10 patients with an epidural hematoma, eight underwent rapid evacuation of the hematoma. Post-

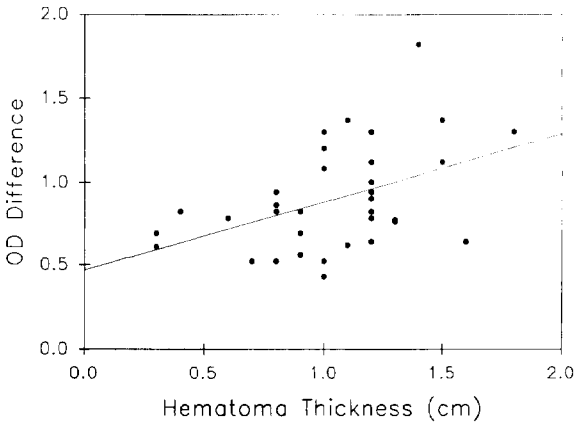

FIG. 3. Scattergram showing the relationship between the difference in optical density (OD) and the thickness of the hematoma measured on computerized tomography scans in 32 patients with extracerebral hematomas ($r = 0.45$, $p < 0.007$).

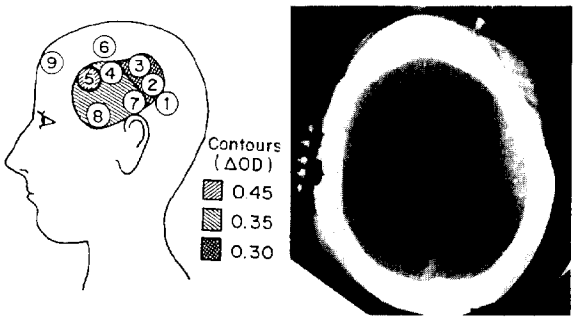

FIG. 4. Studies in a 20-year-old man admitted with an epidural hematoma in the right temporal region, which was surgically evacuated. Forty-eight hours later, the difference in optical density (ΔOD) was 0.94, highest in the left temporoparietal region on the side opposite the original hematoma. A contour map of the ΔOD was drawn *(left)*, outlining the localized epidural hematoma which was found on the computerized tomography scan *(right)*. Numbers denote brain areas studied.

operatively, the ΔOD returned to an average of 0.02 ± 0.1 in the six patients who had undergone successful evacuation of the hematoma. Two patients developed epidural hematomas postoperatively, one at the operative site and one on the opposite side of the head. In the patient with a recurrent hematoma, the ΔOD remained 0.52 postoperatively. In the other patient, who developed a hematoma on the side opposite the initial surgery, the ΔOD was 0.94, with the absorbance now greater on the side with the new hematoma. Two of the patients had small localized epidural hematomas that were allowed to resolve spontaneously. It was possible in these cases to actually outline the blood collection (Fig. 4). The ΔOD between the hematoma and the normal side decreased as the hematoma resolved.

In the eight patients with intracerebral hematomas, seven lesions were located in the cerebral hemispheres and one was in the cerebellum. Six of these patients had surgical evacuation of the hematoma, and the ΔOD averaged 0.03 ± 0.01 postoperatively. Two other patients were managed conservatively and the ΔOD returned to near-normal values over the 3-week period.

Discussion

Our study demonstrates the usefulness of NIRS in localizing intracranial hematomas in head-injured patients. We found NIRS sufficiently sensitive to identify all extracerebral and intracerebral collections of blood. An NIRS examination can be performed at the bedside and can be repeated frequently with a minimum of expense.

There are several potential limitations of NIRS. The location of the hematoma could not be precisely determined in all cases. Intracerebral hematomas tended to absorb light less intensely than extracerebral hematomas, but there was some overlap. Because the method relies on light absorption of normal brain compared with the hematoma, bilateral hematomas might not be detected, and because it relies on light absorption by hemoglobin, only acute collections can be reliably identified. Chronic subdural hematomas, where hemoglobin has been metabolized, may not have the same light-absorbing properties. Nevertheless, NIRS can be useful in the emergency room, as an adjunct to CT or when CT is not immediately available, and in the intensive care unit as a method for following patients postoperatively for recurrence of hematomas or for the development of delayed hematomas.

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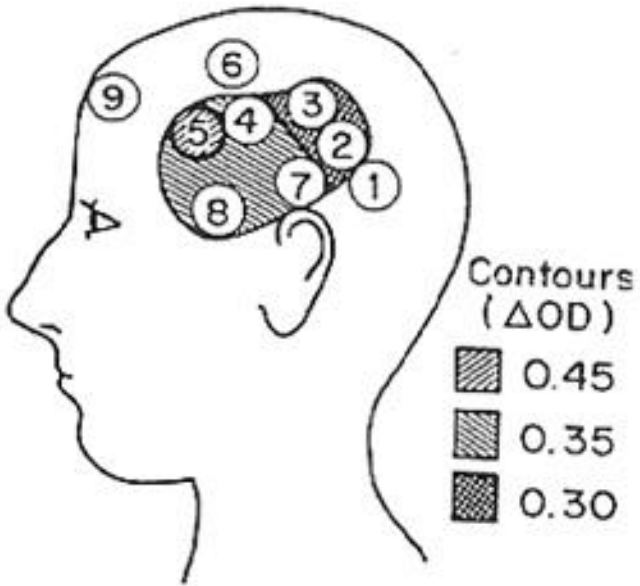
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