Refer to: Hoff JT, Spetzler R, Winestock D: Head injury and early signs of tentorial herniation—A management dilemma. West J Med 128:112-116, Feb 1978

Head Injury and Early Signs of Tentorial Herniation

A Management Dilemma

JULIAN T. HOFF, MD; ROBERT SPETZLER, MD, *and* DAVID WINESTOCK, MD
San Francisco

In 100 patients intracranial exploration was done soon after severe head injury when signs of transtentorial herniation were present. A third of patients had extracerebral hematomas shown on initial burr hole examination. An additional 18 percent had parenchymal clots of clinical significance disclosed only by angiography after burr hole exploration or at autopsy. Half the patients in whom exploration was done did not have intracranial hematomas of sufficient size to warrant an operative approach. Detailed neurological examinations soon after injury and the availability of more rapid diagnostic tools may improve the accuracy of early diagnosis and allow more specific prognostication in patients with severe head injury.

WHEN PHYSICAL SIGNS of brain stem compromise are present in patients soon after severe head trauma, the injury may be primarily to the brain stem or secondarily from compression of the midbrain by supratentorial structures herniating through the tentorial notch.[1] In either circumstance, similar physical signs may be present—coma, pupillary abnormalities, and extensor rigidity or flaccidity of the extremities.[2-4] The correct diagnosis, from initial physical signs alone, may then be difficult.[5]

Patients with transtentorial herniation may have one or all of a variety of supratentorial lesions—epidural or subdural hematomas (or both), intracerebral clots and diffuse cerebral contusions with brain swelling. Most intracranial hematomas, when present, are extracerebral,[1,6-8] lending themselves to discovery by trephination, by angiography, by radionuclide scanning and by computed axial tomography (CT scanning).

From the Departments of Neurological Surgery and Radiology, University of California, School of Medicine, San Francisco.

Submitted June 8, 1977.

This study was supported in part by the Trauma Center Program Project GM 18470 and NS 11539, Trauma Center Fellowship Grant NIGMO 7032 (Dr. Spetzler), grants of the National Institutes of Health, USPHS.

Dr. Hoff is recipient of Teacher-Investigator Award NS 11051 of the NIH, USPHS.

Presented in part at the Annual Meeting of American Association for Surgery of Trauma, Scottsdale, Arizona, September, 1975.

Reprint requests to: Julian T. Hoff, MD, Editorial Office, 350 Parnassus Avenue, Suite 807, University of California, San Francisco, CA 94143.

Surgeons agree that optimal care of patients with head injuries is best provided when a specific diagnosis is available before operative treatment. However, to provide an accurate diagnosis by current radiographic techniques requires time. For example, cerebral angiography rarely can be accomplished in less than two hours;[9] CT scanning requires a comparable amount of time, although more rapid diagnosis by this technique may be possible in the future.

A physician may elect to observe and support the patient for an indefinite period, or use diagnostic tools available to him (such as angiography, CT scanning) before beginning treatment of a specific lesion. Alternatively, he may treat the patient immediately by all medical and surgical means including exploration, on the premise that time lost testing is better spent treating. In general, a poor prognosis for patients with brain stem signs soon after injury has provided an adequate rationale for each of these management approaches.[8,10,11]

On the assumption that tentorial herniation is an emergency which becomes more profound with time, we elected to explore patients with head injury who had signs of herniation upon admission or soon after. This approach, while not new, was designed to focus on obvious neurological signs, rather than upon a specific disease entity. Information gathered from this approach could be helpful in a physician's first moments of contact with an injured patient. To date similar data have not appeared in the literature. We sought to (1) establish the incidence of extracerebral hematomas in trauma victims who have early brain stem signs, (2) determine what percentage of patients might benefit from early operation and (3) based on these data, assess the effectiveness of traditional burr holes as a diagnostic and therapeutic technique.

Patients and Methods

From 1972 through 1974 we carried out exploration by multiple burr holes in 100 patients with severe head injury who had signs of brain stem compression immediately upon or soon after admission. Of the patients, 72 were male and 28 were female. The average age was 38 years (range, 2 to 88 years). Most injuries were the result of traffic accidents, falls or assault. The time from injury to hospital admission was usually less than a hour because of the rapid delivery and triage of patients seen on an emergency basis in the San Francisco area.

TABLE I.—*Physical Signs Before Operation in 100 Patients*

Signs	*Percent*
Stupor	4
Coma	96
Flaccid extremities	14
Extensor rigidity	51
Fixed pupil dilatation	84
Unilateral	47
Bilateral	37
Other major injury	30

During the same period (1972 through 1974) operations were carried out in 147 other patients with less severe head injuries, after angiography had identified specific intracranial hematomas. Clinical signs of herniation were not obvious in those 147 patients before the radiographic study; therefore they were excluded from this review.

Diagnosis

All 100 patients had severe depression of consciousness on admission to hospital. Physical signs during the interval from admission to operation (less than two hours) are listed in Table 1. The most common neurologic sign leading to urgent operation was profound disturbance of consciousness that failed to improve promptly. In all, 96 patients were unresponsive to all stimulation except pain before operation. Most (65 percent) showed either extensor rigidity or flaccidity of the extremities; the other 35 percent retained variable degrees of purposive, spontaneous movement of the arms and legs. In 84 patients there were pupillary signs of transtentorial herniation before operation. A unilateral dilated pupil which failed to respond to light was common (47 percent); 37 patients (37 percent) had bilateral fixed, dilated pupils. Therefore, the classical triad of physical signs indicating transtentorial herniation[2,4]—coma, extensor rigidity or flaccidity of the extremities, and fixed dilatation of one or both pupils—was common soon after injury.

In 57 patients skull radiographs were obtained before operation; in 37, a fracture was shown. Plain skull films showed no abnormalities in 20 patients; a pineal shift was present in three patients. In 43, skull films were not made before operation because neurological deterioration was precipitous during the initial evaluation, or physical signs of herniation were present on arrival at

TABLE 2.—*Autopsy Findings in 48 Patients*

Findings	*Percent*
Cortical hemorrhages	98
Cortical necrosis, lacerations	52
Intracranial hematoma	21
Extracerebral	10
Intracerebral	9
Cerebellar	2
Brain stem hemorrhages	62

the hospital. When skull films were obtained, cervical spine films were taken also; one patient had a cervical fracture (2 percent). Thirty patients had multiple injuries. In 19 an emergency operative procedure was required for abdominal, thoracic or extremity trauma.

Treatment

When initial resuscitation was complete all patients received dexamethasone intravenously (10 mg). Mannitol (500 ml, 20 percent) was begun intravenously in 91 patients before operation. Ventilation was assisted, if necessary, to maintain arterial carbon dioxide pressure between 25 and 35 mm of mercury.

Burr holes were made bilaterally at three sites: frontal, occipitoparietal and temporal. A clinically significant hematoma was encountered epidurally in four patients. In 30 patients acute subdural hematoma was noted. Swollen, contused cortex was evident, without an extracerebral mass, in the remaining 66 patients. If an extracerebral clot was evident through a burr hole, it was removed immediately by craniotomy. If no extracerebral hematoma was present, needle aspiration of the brain through the burr holes was not carried out.

When no hematoma could be found by burr holes, cerebral angiography was then done in 32 patients. The circulation of the carotid and one vertebral artery was studied by the transfemoral route. An intracerebral mass effect was apparent in 18 of the patients (56.2 percent) who were analyzed in this manner. Seventeen had a supratentorial parenchymal mass; one had a cerebellar hematoma. Of these deep masses not shown by burr holes, but identified by angiography, nine were judged sufficiently large and localized to require operative removal by craniotomy.

Postoperative support included dexamethasone administration, intermittent mannitol infusion, temperature control and ventilatory support.

TABLE 3.—*Physical Signs Before Operation in Survivors and Nonsurvivors, 100 patients*

Signs	*Survivors (47) Percent**	*Nonsurvivors (53) Percent**
Stupor	6	2
Coma	94	98
Flaccid extremities	9	19
Extensor rigidity	49	53
Fixed pupil dilatation	70	96
Unilateral	57	38
Bilateral	13	59
Other major injury	28	32

*Percent of patients in each group.

Results

Survival

Of the 100 patients, 53 died. The autopsy findings in 48 are shown in Table 2. Primary parenchymal injury was severe, occurring in both the cerebral hemispheres and the brain stem. There were five cases of significant (greater than 50 cc) extracerebral hematomas, four of intracerebral clots and one of cerebellar hematoma. Nine of the ten patients with intracranial hematomas shown at autopsy *had not had angiography carried out* at any time during their hospital course.

Of the 47 patients who survived, a significant intracranial hematoma was removed after injury in 22. Included in these 22 survivors are four (of a total of nine) who survived removal of an intracranial hematoma shown by angiography after burr hole exploration had proved to be nondiagnostic. In the remaining 25 survivors operation for removal of an intracranial clot was not required.

Comparison of Survivors and Nonsurvivors

Physical signs before operation did not differ substantially between patients who survived and those who died (see Table 3). Flaccid extremities were more common in those who died (19 percent) than in those who lived (9 percent). Fixed pupillary dilatation, particularly when bilateral, suggested a poor outcome. Not surprisingly, patients who died were usually older than those who survived. But none of the physical signs we elicited consistently predicted the ultimate result.

When the three signs of transtentorial herniation were present before operation, a good result was unlikely. Of the 53 patients who died, 34 (64 percent) had classical herniation before operation, while such signs were apparent in only 15 of the 47 who survived (32 percent). Of in-

TABLE 4.—*Physical Signs Before Operation and Quality of Survival, 100 Patients*

Signs	Functional Recovery: Good (22) Percent*	Functional Recovery: Poor (25) Percent*	Died (53) Percent*
Stupor	9	4	2
Coma	91	96	98
Flaccid extremities	5	12	19
Extensor rigidity	36	60	53
Fixed pupil dilatation	64	76	97
Unilateral	55	60	38
Bilateral	9	16	59
Other major injury	36	20	32
Average age	28	29	38

*Percent of patients in each group.

terest was the finding that four patients who survived and then recovered fully had a complete triad of herniation signs before operation.[12] Two who became functional survivors had bilateral pupillary dilatation before burr hole exploration.

Quality of Survival

The quality of survival six months after injury ranged from complete functional recovery to persistent vegetative state.[13] A total of 22 patients were either independent or minimally dependent (good), while 25 were dependent or vegetative (poor) six months or longer after injury (Table 4). The actual presence or absence of a traumatic intracranial hematoma had little effect on the quality of life of patients who survived in our series.

Discussion

Patients included in this review had in common severe, traumatic brain injury and evidence of early brain stem compromise. Some had epidural hematomas, some subdural hematomas, some intracerebral clots and others diffuse injury without localized masses. The prognosis for each injury might have been different had the pathological entity (for example, epidural hematoma) itself been considered and the clinical state excluded.[7,9,14,15] But considering *clinical signs* as determinants of outcome, all patients were neurologically moribund at the time of exploration, suggesting that the prognosis would be poor regardless of the specific intracranial injury.

We proceeded to exploration early, assuming that both the correct diagnosis could be established and treatment rendered if an extracerebral mass causing transtentorial herniation was present. We further assumed that exploration, if findings were negative, would do no harm, provided standard supportive treatment was given simultaneously. When exploratory burr holes failed to reveal an extracerebral clot, many patients then underwent cerebral angiography to identify "missed" intracranial hematomas requiring removal. When a missed hematoma of clinical significance was found by angiography (nine patients), it was removed during a second operation.

In 34 patients there were extracerebral hematomas of clinical significance diagnosed by burr holes. All were evacuated by craniotomy. In the remaining 66 patients findings on exploratory burr holes were negative. Therefore, only in a third of our patients, most of whom had signs of transtentorial herniation within two hours of injury, were there extracerebral clots that were removed as early as possible.

Burr holes had given negative findings in 18 patients (18 percent) in whom an intracerebral mass was identified angiographically. The mass was sufficiently large in nine of them to warrant removal by craniotomy. Angiography showed one extracerebral mass, a parietal epidural hematoma, which had been missed by previous exploratory burr holes.

Of the 18 patients with negative burr hole findings in whom postoperative angiography was not done, 16 died from the brain injury; of these, a clinically significant intracerebral hematoma was found at autopsy in nine. Therefore, a total of 18 of the 100 patients reviewed had significant deep clots that were missed on burr hole exploration, and were or would have been recognized by angiography.

Nearly half (48 percent) of our patients in whom exploration was done did not have a mass sufficiently large to benefit from its removal. Thus nonoperative, supportive care was initially and subsequently the only therapeutic alternative.

It has been reported that patients who experience transtentorial herniation after head injury rarely survive, and that a good functional recovery is even less likely.[8,10,11] The results of our study confirm that prediction. Only four patients (8 percent) of the 49 with classical herniation before exploration recovered fully. Of the patients who died, 64 percent had a complete triad of herniation signs before operation, while 32 percent of those who survived had classical transtentorial herniation preoperatively. Therefore, while herniation suggested a poor outcome despite aggressive management, it did not necessarily foretell it. Previous reports have verified that complete recovery may occur with proper therapy even though herniation may already have occurred.[9,12,16]

Survival from severe head injury is not only of medical interest, but also of socioeconomic importance. Recent reports have focused on this problem,[10,13] stressing the need for early clinical criteria that may predict outcome. Our study suggests that prognosis from very early signs may not be sufficiently accurate, using the simple bedside examinations reviewed in this study. For example, four of our 49 patients with classical signs of herniation from brain injury not only survived, but eventually recovered completely. The coma scale recently advocated by Teasdale and Jennett[17] and the more detailed clinical assessment used by Overgaard and co-workers[10] may afford more accurate prognostication at the crucial early stage of management.

Whether or not surgeons should still resort to exploratory burr holes remains an unanswered question. Occasional extracerebral clots and a substantial number (18 percent) of deep clots will be overlooked if burr holes are the first and last diagnostic procedure used. Fundamental to the problem is our inability to diagnose with certainty the most important of a host of intracranial lesions in trauma victims. Similarly, we do not know when signs of mid-brain compression are reversible and when they are not. And finally, we cannot yet accurately diagnose within the first few hours after injury those patients whose immediate, primary and permanent injury is to the brain stem itself.[18] Further development of visual, auditory and somatosensory evoked potentials may improve early accuracy.[19]

While our approach to the problem of severe head injury failed to differentiate with certainty patients who would recover from those who would not, it did yield practical information useful to surgeons who must consider early management alternatives in moribund trauma victims. When the physician is confronted with a patient who shows signs of tentorial herniation soon after head injury, immediate exploration via burr holes can probably help in about a third of the cases. Preoperative angiography or computerized scanning can identify a mass of clinical significance in half of the patients if the diagnostic delay imposed by the procedure can be tolerated, and almost all of the remaining patients will benefit only by aggressive, nonoperative, supportive care.

REFERENCES

1. Cohen SI, Aronson SM: Secondary brain stem hemorrhages. Arch Neurol 19:257-263, Sept 1968

2. Finney LA, Walker AE: Transtentorial Herniation. Springfield, Ill, Charles C Thomas, 1962, pp 162

3. Jefferson G: The tentorial pressure cone. Arch Neurol Psych 40:857-876, Nov 1938

4. Johnson RT, Yates PO: Clinico-pathological aspects of pressure changes at the tentorium. Acta Radiol 46:242-249, Jul-Dec 1956

5. Plum F, Posner J (Eds.): Stupor and Coma, Philadelphia, FA Davis Co., 1972, pp 286

6. Jamieson KG: A First Notebook on Head Injury, 2nd Ed. London, Butterworth, 1971, pp 148

7. Jamieson KG: Extradural and subdural hematomas. J Neurosurg 33:632-635, Dec 1970

8. Richards T, Hoff J: Factors affecting survival from acute subdural hematoma. Surgery 75:253-258, Feb 1974

9. Cooper PR, Rovit RL, Ransohoff J: Hemicraniectomy in the treatment of acute subdural hematoma: A re-appraisal. Surg Neurol 5:25-28, Jan 1976

10. Overgaard J, Huid-Hansen O, Land AM, et al: Prognosis after head injury based on early clinical examination. Lancet 2:631-635, Sep 1973

11. Vapalahti M, Troupp H: Prognosis for patients with severe brain injuries. Br Med J 3:404-407, Aug 1971

12. Brendler SJ, Selverstone B: Recovery from decerebration. Brain 93:381-392, 1970

13. Jennett B, Plum F: Persistent vegetative state after brain damage. Lancet 1:734-737, Apr 1972

14. McLaurin RL, Tutor FT: Acute subdural hematoma. J Neurosurg 18:61-67, Jan 1961

15. Talalla A, Morin MA: Acute traumatic subdural hematoma: A review of one hundred consecutive cases. J Trauma 11:771-777, Jul-Dec 1971

16. Hoff J, Grollmus J, Barnes B, et al: Clinical, arteriographic, and cisternographic observations after removal of acute subdural hematoma. J Neurosurg 43:27-31, Jul 1975

17. Teasdale G, Jennett B: Assessment of coma and impaired consciousness. Lancet 2:81-84, July 1974

18. Mitchell DE, Adams JH: Primary focal impact damage to the brain stem in blunt head injuries: Does it exist? Lancet 2:215-218, Aug 1973

19. Greenberg RP, Mayer DJ, Becker DP: The prognostic value of evoked potentials in human mechanical head injury, *In* McLaurin RL (Ed): Head Injuries. New York, Grune and Stratton, 1976, pp 81-88