

**GUIDELINES FOR THE SURGICAL MANAGEMENT  
OF TRAUMATIC BRAIN INJURY  
AUTHOR GROUP**

**M. Ross Bullock**

Department of Neurological Surgery  
Virginia Commonwealth University Medical Center  
Richmond, Virginia

**David Gordon**

Department of Neurological Surgery  
Montefiore Medical Center  
Bronx, New York

**Franco Servadei**

Department of Neurological Surgery  
M. Bufalini Hospital  
Cesena, Italy

**Randall Chesnut**

Department of Neurological Surgery  
University of Washington, School of Medicine  
Harborview Medical Center  
Seattle, Washington

**Roger Hartl**

Department of Neurological Surgery  
Weill Cornell Medical College  
of Cornell University  
New York, New York

**Beverly C. Walters**

Department of Neurological Surgery  
New York University,  
School of Medicine  
New York, New York

**Jamshid Ghajar**

Department of Neurological Surgery  
Weill Cornell Medical College  
of Cornell University  
New York, New York

**David W. Newell**

Department of Neurological Surgery  
Swedish Medical Center  
Seattle, Washington

**Jack E. Wilberger**

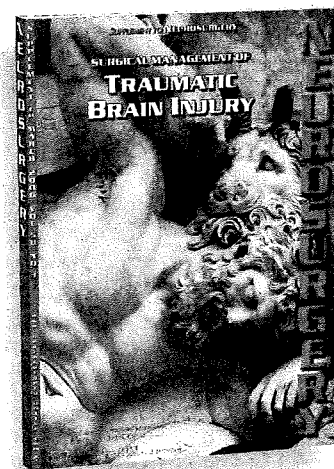
Department of Neurological Surgery  
Allegheny General Hospital  
Pittsburgh, Pennsylvania

A JOINT VENTURE OF:

**THE BRAIN TRAUMA FOUNDATION AND  
THE CONGRESS OF NEUROLOGICAL SURGEONS**

*The content of "Guidelines for the Surgical Management of  
Traumatic Brain Injury" has undergone the peer-review process  
by the Editorial Board of **NEUROSURGERY**.*

*Supported through a restricted educational grant from  
**Integra NeuroSciences**.*



**M. Ross Bullock, M.D., Ph.D.**

Department of Neurological Surgery,  
Virginia Commonwealth University  
Medical Center,  
Richmond, Virginia

**Randall Chesnut, M.D.**

Department of Neurological Surgery,  
University of Washington  
School of Medicine,  
Harborview Medical Center,  
Seattle, Washington

**Jamshid Ghajar, M.D., Ph.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David Gordon, M.D.**

Department of Neurological Surgery,  
Montefiore Medical Center,  
Bronx, New York

**Roger Hartl, M.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David W. Newell, M.D.**

Department of Neurological Surgery,  
Swedish Medical Center,  
Seattle, Washington

**Franco Servadei, M.D.**

Department of Neurological Surgery,  
M. Bufalini Hospital,  
Cesena, Italy

**Beverly C. Walters, M.D., M.Sc.**

Department of Neurological Surgery,  
New York University  
School of Medicine,  
New York, New York

**Jack E. Wilberger, M.D.**

Department of Neurological Surgery,  
Allegheny General Hospital,  
Pittsburgh, Pennsylvania

**Reprints requests:**

Jamshid Ghajar, M.D., Ph.D.,  
Brain Trauma Foundation,  
523 East 72nd Street,  
New York, NY 10021.  
Email: ghajar@braintrauma.org

**INTRODUCTION**

*Neurosurgery* 58:S2-1-S2-3, 2006

DOI: 10.1227/01.NEU.0000210361.83548.D0

www.neurosurgery-online.com

**T**raumatic brain injury (TBI) affects up to 2% of the population per year, and constitutes the major cause of death and severe disability among young people. By far, the most important complication of TBI is the development of an intracranial hematoma, which complicates 25 to 45% of severe TBI cases, 3 to 12% of moderate TBI cases, and approximately 1 in 500 patients with mild TBI (20). Without effective surgical management, an intracranial hematoma may transform an otherwise benign clinical course with the expectation of recovery to a situation in which death or permanent vegetative survival will occur. Moreover, prolonged delay in the diagnosis or evacuation of an intracranial hematoma may produce a similar result.

As many as 100,000 patients per year may require surgical management for a posttraumatic intracranial hematoma in the United States alone. For these reasons, the impact that neurosurgeons can have on the care of such patients is enormous, and perhaps, more than in any other area of emergency medicine, the aggressiveness and rapidity with which care is provided for an intracranial hematoma will determine the outcome (8). Picard et al. (13) have shown that craniotomy for evacuation of an acute epidural hematoma is one of the most cost-effective of all surgical procedures. For this particular subgroup, which may represent up to 5% of patients with severe and moderate TBI, the quality of outcome has been shown to vary dramatically among different hospitals with different levels of commitment to acute neurotrauma care (2, 6, 11). It is for this reason, more than any other, that neurosurgical consultation in the Emergency Room should be promptly available and is a mandated requirement for Level I certification of Trauma centers (1).

Although there is evidence that posttraumatic intracranial mass lesions have been removed surgically up to 4000 years ago by the Egyptians and Meso-Americans, it was not until a series of publications emerged in the late 1960s that it became generally accepted that excellent results could be achieved with craniotomy for removal

of extradural hematomas (9). For acute subdural hematomas and intraparenchymal lesions, such as contusions and traumatic intracerebral hematomas, the outcome has historically been much worse, because up to 60% of patients with acute subdural hematomas will die or remain severely disabled (10).

During the early 1970s, a series of publications from the Medical College of Virginia demonstrated that wide decompressive craniotomy with duraplasty was one of the most effective forms of therapy for raised intracranial pressure in patients with mass lesions (4). Subsequently, most neurosurgical centers with an interest in TBI have also applied the same craniotomy technique to patients with intraparenchymal contusions, with improvements in outcome. However, there is also widespread dissent and controversy regarding the surgical management of intraparenchymal lesions, with some neurosurgeons maintaining that aggressive surgical intervention, although able to preserve life, will result in a very poor quality of life for survivors (7, 15).

With our increasing understanding of the pathomechanisms in severe and moderate TBI have come changes in our approach to management of patients with posttraumatic intracranial mass lesions. For example, it is now well accepted that most intraparenchymal mass lesions (contusions and intracerebral hematomas) will enlarge with time, necessitating serial computed tomographic scanning, and usually intracranial pressure monitoring during the first few days (12, 17). Similarly, the propensity of patients with posttraumatic coagulation disorders to develop intraparenchymal bleeding that is more severe is now well accepted, and has led to management of coagulation disorders in the head injured population that is much more aggressive (18).

In turn, these practices led to an increase in the performance of craniotomy, both for evacuation of intraparenchymal mass lesions and as a decompressive measure. Recently, several publications have shown that, within the context of modern aggressive neuro-intensive

care therapy, decompressive craniotomy is an effective means of controlling raised intracranial pressure after severe TBI, especially in those patients with intraparenchymal lesions (14, 19). Many neurosurgeons, however, are reluctant to implement such aggressive surgical techniques in patients with raised intracranial pressure after severe TBI, claiming that improved quality of life has never been conclusively demonstrated.

It is, therefore, the overall aim of these *Guidelines* is to present rigorous literature-based recommendations for the surgical management of patients with posttraumatic intracranial mass lesions. We have chosen to focus on those acute mass lesions that develop within 10 days of injury and, thus, we have chosen not to cover chronic subdural hematoma, subdural hygroma, and posttraumatic hydrocephalus, which usually are delayed. Similarly, we have chosen to focus on closed TBI in general because a comprehensive set of management guidelines for patients with penetrating TBI has already been formulated (3).

Compared with the *Guidelines for The Management of Severe Traumatic Brain Injury* (5), the literature regarding surgical management after TBI suffers from extensive limitations, in both quality and scope. Most notably, although our group reviewed more than 700 manuscripts for the preparations of these *Guidelines*, there are no controlled clinical trials in the literature to support different forms of surgical management, or to support surgical versus conservative therapy. Consistent with these limitations, we have been unable to formulate recommendations at the standard level requiring Class I evidence.

As in all other areas of "evidence-based medicine," these *Guidelines* have been formulated strictly in accordance with externally imposed constraints. Only clinical human-based literature has been reviewed. Only literature from 1975 through 2001 has been reviewed. Mainly literature in English, with far fewer articles in other languages, was reviewed. For these reasons, the reader must clearly understand that the scope and level of magnitude of the recommendations made here are distilled from the available literature and interpreted according to the rules of "evidence-based medicine" (16).

An important aspect of this document is, therefore, to also formulate critical questions that need to be resolved by future clinical trials or prospective cohort studies, to determine the most effective forms of therapy for the future. As with the other guidelines in severe TBI, therefore, this is a document in evolution, and frequent revisions will be made to keep up with the evolving state of knowledge in this area.

These *Guidelines* have been organized on the basis of the traditional literature-based classification of posttraumatic mass lesions: namely, epidural hematoma, acute subdural hematoma, intraparenchymal lesions (contusion and intracerebral hematoma), acute posterior fossa mass lesions, and depressed fractures of the cranium. We recognize, however, that, for most patients with severe TBI, and for some patients with moderate TBI, more than one of these acute posttraumatic mass lesions may coexist at the same time. For example, the majority of patients with acute subdural hematomas will also demonstrate

concomitant intraparenchymal contusions on their computed tomographic scan. In some patients, there may be multiple sites in which intraparenchymal mass lesions occur—e.g., bifrontal contusions, bitemporal contusions, or temporal and frontal lesions. For high-volume lesions (>50 cm<sup>3</sup>), management decisions are easier, and generally are in favor of surgery. Low-volume lesions (<25 cm<sup>3</sup>) are usually not operated on, however, for lesions between high and low volumes, the decisions may be very difficult and associated factors, e.g., shift, cisterns, and Glasgow Coma Scale, become especially important.

Within the literature, the terms surgical decompression, decompressive craniectomy, evacuation, and internal decompression are often used interchangeably. This aspect is clarified as much as possible in the individual sections.

We describe methods for posttraumatic mass volume measurement in *Appendix 1* and simple methods and definitions of midline shift, subarachnoid hemorrhage, and status of basal cisterns in *Appendix 2*.

## REFERENCES

1. ACS-COT: American College of Surgeons-Committee on Trauma: *Resources for Optimal Care of the Injured Patient*. Chicago, American College of Surgeons, 1999.
2. Alberico A, Ward J, Choi S, Marmarou A, Young H: Outcome after severe head injury. Relationship to mass lesions, diffuse injury, and ICP course in pediatric and adult patients. *J Neurosurg* 67:648-656, 1987.
3. Anonymous: Management and prognosis of penetrating brain injury. *J Trauma* 51[Suppl]: S1-S49, 2001.
4. Becker D, Miller J, Ward J, Greenberg R, Young H, Sakalas R: The outcome from severe head injury with early diagnosis and intensive management. *J Neurosurg* 47:491-502, 1977.
5. Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care: Guidelines for the management of severe traumatic brain injury. *J Neurotrauma* 17:457-554, 2000.
6. Bricolo A, Pasut L: Extradural hematoma: Toward zero mortality. A prospective study. *Neurosurgery* 14:8-12, 1984.
7. Clark K, Nash TM, Hutchison GC: The failure of circumferential craniotomy in acute traumatic cerebral swelling. *J Neurosurg* 29:367-371, 1968.
8. Consensus conference: Rehabilitation of persons with traumatic brain injury. NIH Consensus Development Panel on Rehabilitation of Persons with Traumatic Brain Injury. *JAMA* 282:974-983, 1999.
9. Jamieson KG, Yelland JD: Extradural hematoma: Report of 167 cases. *J Neurosurg* 29:13-23, 1968.
10. Jamieson KG, Yelland JD: Surgically treated subdural hematomas. *J Neurosurg* 37:137-149, 1972.
11. Klauber MR, Marshall LF, Luerssen TG, Frankowski R, Tabaddor K, Eisenberg HM: Determinants of head injury mortality: Importance of the low risk patient. *Neurosurgery* 24:31-36, 1989.
12. Lobato RD, Gomez PA, Alday R, Rivas JJ, Dominguez J, Cabrera A, Turanzas FS, Benitez A, Rivero B: Sequential computerized tomography changes and related final outcome in severe head injury patients. *Acta Neurochir (Wien)* 139:385-391, 1997.
13. Picard J, Bailey S, Sanderson H, Reese M, Garfield JS: Steps towards cost benefit analysis of regional neurosurgical care. *BMJ* 301:629-635, 1990.
14. Polin R, Shaffrey M, Bogaev C, Tisdale N, Germanson T, Bocchicchio B, Jane J: Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. *Neurosurgery* 41:84-92, 1997.
15. Ransohoff J, Benjamin MV, Gage EL Jr, Epstein F: Hemispheric craniectomy in the management of acute subdural hematoma. *J Neurosurg* 34:70-76, 1971.
16. Sackett DL, Rosenberg WM, Gray JA, Haynes RB, Richardson WS: Evidence based medicine. What it is and what it isn't. *BMJ* 312:71-72, 1996.

17. Soloniuk D, Pitts LH, Lovely M, Bartkowski H: Traumatic intracerebral hematomas: Timing of appearance and indications for operative removal. *J Trauma* 26:787-794, 1986.
18. Stein SC, Young GS, Talucci RC, Greenbaum BH, Ross SE: Delayed brain injury after head trauma: Significance of coagulopathy. *Neurosurgery* 30: 160-165, 1992.
19. Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, Klug G, Wallace D, Henning R, Tibballs J: A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. *Childs Nerv Syst* 17:154-162, 2001.
20. Thurman D, Guerrero J: Trends in hospitalization associated with traumatic brain injury. *JAMA* 282:954-957, 1999.

### CONGRESS OF NEUROLOGICAL SURGEONS' MISSION STATEMENT

"The *Congress of Neurological Surgeons* exists for the purpose of promoting the public welfare through the advancement of neurosurgery, by a commitment to excellence in education, and by dedication to research and scientific knowledge. The *Congress of Neurological Surgeons* maintains the vitality of our learned profession through the altruistic volunteer efforts of our members and the development of leadership in service to the public, to our colleagues in other disciplines, and to the special needs of our fellow neurosurgeons throughout the world and at every stage of their professional lives."

### CONGRESS OF NEUROLOGICAL SURGEONS / AMERICAN ASSOCIATION OF NEUROLOGICAL SURGEONS JOINT SECTION CHAIRMEN

- Cerebrovascular Surgery:** Robert H. Rosenwasser, Philadelphia, Pennsylvania
- Disorders of the Spine and Peripheral Nerves:** Robert F. Heary, Newark, New Jersey
- History of Neurological Surgery:** Dennis E. McDonnell, LaCrosse, Wisconsin
- Neurotrauma and Critical Care:** P. David Adelson, Pittsburgh, Pennsylvania
- Pain:** Richard K. Osenbach, Durham, North Carolina
- Pediatric Neurological Surgery:** Rick Abbott, New York, New York
- Stereotactic and Functional Neurosurgery:** Andres Lozano, Toronto, Ontario
- Tumors:** Raymond Sawaya, Houston, Texas

### FUTURE MEETINGS—CONGRESS OF NEUROLOGICAL SURGEONS

The following are the planned sites and dates for future annual meetings of the Congress of Neurological Surgeons:

2006	Chicago, IL	October 7-12
2007	San Diego, CA	September 15-20
2008	Orlando, FL	September 20-25

### FUTURE MEETINGS—AMERICAN ASSOCIATION OF NEUROLOGICAL SURGEONS

The following are the planned sites and dates for future annual meetings of the American Association of Neurological Surgeons:

2006	San Francisco, CA	April 22-27
2007	Washington, DC	April 14-19
2008	Chicago, IL	March 29-April 3
2009	San Diego, CA	May 2-7

## METHODOLOGY

### M. Ross Bullock, M.D., Ph.D.

Department of Neurological Surgery,  
Virginia Commonwealth University  
Medical Center,  
Richmond, Virginia

### Randall Chesnut, M.D.

Department of Neurological Surgery,  
University of Washington  
School of Medicine,  
Harborview Medical Center,  
Seattle, Washington

### Jamshid Ghajar, M.D., Ph.D.

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

### David Gordon, M.D.

Department of Neurological Surgery,  
Montefiore Medical Center,  
Bronx, New York

### Roger Hartl, M.D.

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

### David W. Newell, M.D.

Department of Neurological Surgery,  
Swedish Medical Center,  
Seattle, Washington

### Franco Servadei, M.D.

Department of Neurological Surgery,  
M. Bufalini Hospital,  
Cesena, Italy

### Beverly C. Walters, M.D., M.Sc.

Department of Neurological Surgery,  
New York University  
School of Medicine,  
New York, New York

### Jack E. Wilberger, M.D.

Department of Neurological Surgery,  
Allegheny General Hospital,  
Pittsburgh, Pennsylvania

#### Reprints requests:

Jamshid Ghajar, M.D., Ph.D.,  
Brain Trauma Foundation,  
523 East 72nd Street,  
New York, NY 10021.  
Email: ghajar@braintrauma.org

*Neurosurgery* 58:52-4-52-6, 2006

DOI: 10.1227/01.NEU.0000210362.83548.0B

www.neurosurgery-online.com

The concept of formulating a set of recommendations for patient management has its basis in the advent of scientific investigation in clinical medicine. Along with this came the realization that deriving recommendations from our own experience as clinicians, although tempting, has limitations related to the fact that humans remember the best or the worst of their experiences, and cannot easily objectify their results. What clinical experience does do for us is to help with the formulation of hypotheses that can be tested through human clinical trials experimentation. Rather than seeing clinical experience and clinical trials as dichotomous, polar sources of information regarding outcomes of patient care, it is probably more useful to think of the gamut of clinical testing along a continuum. On one end, we have the more-subjective impressions of care delivery that are attractive because they are grounded in personal experience in the real world of caring for patients. This is expressed well by one of the fathers of clinical epidemiology, Alvan Feinstein. Dr. Feinstein, in his seminal work, *Clinical Judgment* (2), writes: "...any decent doctor reflects on alternatives, is aware of uncertainties, modifies judgments on the basis of accumulated evidence, balances risks of various kinds, considers the potential consequences of his or her diagnoses or treatments, and synthesizes all of this in making a reasoned decision that he or she deems right for the patient." He further goes on to underscore the value of clinical experience when he writes, "In caring for patients, clinicians constantly perform experiments. During a single week of active practice, a busy clinician conducts more experiments than most of his laboratory colleagues do in a year."

This philosophy underscores and supports the notion that we can make recommendations from the wealth of clinical experience provided by direct, day-to-day clinical care. However, a close look at the process of true experimentation yields a long list of potential errors that can be made in the gathering and

interpretation of data, even in highly controlled circumstances. Sources of error have been classified into two broad categories: systematic error (bias) and random error. In general, the former source of error is controlled by careful study design, and the latter is dealt with by randomization. Without these protectors against error, the chances of making mistakes in deriving recommendations from practice are great. For this reason, the notion of categorizing recommendations into groups associated with various strengths of clinical studies on the basis of scientific rigor was introduced in the last decade-and-a-half of the 20th century.

In 1990, the Institute of Medicine published a landmark work entitled *Clinical Practice Guidelines: Directions for a New Program* (1). The instigating factors for this publication were the observation of wide variability of practice throughout American healthcare and the development of mechanisms for establishing quality assurance and review within Medicare, along with the increasing use of technology and concomitant costs of care delivery. In this document, the authors made recommendations regarding attributes defining the validity of recommendations. These include the following: 1. The available scientific literature should be searched using appropriate and comprehensive search terminology. 2. A thorough review of the scientific literature should precede guideline development. 3. The evidence should be evaluated and weighted, reflecting the scientific validity of the methodology used to generate the evidence. 4. There should be a link between the available evidence and the recommendations, with the strength of the evidence being reflected in the strength of the recommendations, reflecting scientific certainty (or lack thereof). 5. Empirical evidence should take precedence over expert judgment in the development of guidelines. 6. Expert judgment should be used to evaluate the quality of the literature and to formulate guidelines when the evidence is weak or nonexistent. 7. Guideline development should be a multidis-

ciplinary process, involving key groups affected by the recommendations.

### SEARCH OF THE LITERATURE

The literature is generally searched using the National Library of Medicine computerized database, either through individual search engines in academic medical centers or through the internet. This, by definition, will limit the scope of the search to 1966 and forward. In addition, if modern neuroimaging techniques are pertinent, as in this document, the search should be limited to the years since the modality was available. Therefore, our searches were limited to the period of 1975 to the present. Because guidelines are applicable to patient care, the literature is limited to human studies in the area of traumatic brain injury and surgical management, along with the imaging characteristics in patients with surgically amenable lesions. In addition, further limitations in the search were imposed by the fact that most of the literature is in English, and limited ability existed for reading the useful articles that appear in different languages. Therefore, most searches were limited to the English language. Appropriate search terms were chosen, as demonstrated in each of the individual *Guideline* sections.

### EVALUATION AND WEIGHTING OF THE LITERATURE

The journal articles found have been carefully read and evaluated, including an assessment of the methodology used in the studies. This not only includes the establishment of the clinical question addressed (e.g., therapeutic effectiveness, diagnostic tests, prognostic studies, etc.) and type of study (randomized controlled trial, case-control study, case series, etc.), but also the quality of the study with respect to potential errors in design, execution, or conclusions reached. Therefore, studies that might, on the surface, represent evidence supporting one level of recommendation, may instead be flawed enough to be devalued to support a recommendation of lesser strength. The quality of the literature was evaluated in this way according to well-established criteria (3). All articles were cross-reviewed and disagreements were resolved by consensus.

### LINK BETWEEN EVIDENCE AND GUIDELINES

The general concept of relating strength of recommendations to strength of evidence reflecting varying degrees of clinical certainty was formalized into a scheme that has been followed by medical societies, including organized neurosurgery, from the inception of the *Guideline* development process. Despite problems with the strict application of this paradigm (some of which are displayed and discussed in this supplement), the scheme has the benefit of using scientific evidence rather than expert opinion for the substrate of the recommendations, although expert opinion is used to formulate the recommendations themselves, as well as to make judgments regarding the quality of the evidence. The evidence-based scheme used in these and all *Guidelines* regarding therapeutic effectiveness endorsed by the American Association of Neurological Surgeons and the Congress of Neurological Surgeons begins with classification of the literature into three categories of evidence, as outlined next and in *Table 1*.

The classification of evidence into these three categories leads to the formulation of recommendations called *Standards*, *Guidelines*, and *Options*. *Class I* evidence is used to support treatment recommendations of the strongest type, practice *Standards*, reflecting a *high degree of clinical certainty*. *Class II* evidence is used to support *Guidelines*, reflecting a *moderate degree of clinical certainty*. *Class III* evidence supports practice *Options* reflecting *unclear clinical certainty*. This terminology was developed to indicate, in normal vocabulary, the strength of the recommendations on the basis of strong to weak medical evidence. In neurosurgery, this scheme has been used to formulate *Guidelines*, rather than a scheme that uses letters or numbers that have no grounding in language and are, therefore, more easily misinterpreted. The link between scientific evidence and recommendations has been highlighted in these *Guidelines* by presenting those studies in the scientific foundation that support the stated recommendation in boldface type.

Recommendations are not necessarily weak in cases in which the evidence is weak. Examples of this appear in many of the *Guidelines* developed in neurosurgery to date, and authors and readers alike feel frustrated at the impotency of the recommendations when the evidence is weak, especially when the logic of the recommendation and all of the evidence supporting it, however weak, support the recommendation. One example of this, among many that can be found in this sup-

**TABLE 1. Classification of Evidence on Therapeutic Effectiveness**

Class I	Evidence from one or more well-designed, randomized, controlled clinical trials, including overviews of such trials
Class II	Evidence from one or more well-designed comparative clinical studies, such as nonrandomized cohort studies, case-control studies, and other comparable studies
Class III	Evidence from case series, comparative studies with historical controls, case reports, and expert opinion

plement, is in the recommendation regarding timing of evacuation of epidural hematomas. According to the paradigm embraced and used in this set of *Guidelines*, case series indicating that patients who have a Glasgow Coma Scale score of 8 or below with evidence of a "blown pupil" and who are operated on early achieve better outcomes can only support a practice "Option." However, no competent neurosurgeon would allow a patient in this clinical scenario to be neglected when the need for surgical relief of brain compression is so clear. It is fairly certain to say that there will never be a randomized controlled trial for this circumstance, and, thus, never a practice "Standard." However, a patient database could be used to generate a case-control study, thus, yielding a higher recommendation. However, there is no evidence that waiting to operate on such a patient is beneficial, and, therefore, an "Option" to delay surgical evacuation will also probably never be promulgated. If, indeed, such a recommendation were put forward, it would never be accepted by the profession, and rightfully so.

### EXPERT JUDGMENT AND EMPIRICAL EVIDENCE

There are two ways in which expert judgment comes into *Guideline* development. The most common use of expert opinion is in developing recommendations for practice. This has been a usual method in the past (as well as the present, in the form of textbook chapters), but has more recently given way to more formalized approaches embraced by evidence-based medicine methodology, such as that used in this supplement. However, even in evidence-based methodology, expert opinion is used to evaluate the literature as well as to frame the concepts and wording of the recommendations. In addition, if the evidence is weak and conflicting, expert opinion is used to derive recommendations. This use is unavoidable, but the expert opinion is guided by the evidence published in the literature, rather than from personal experience alone.

### OPTION LEVEL RECOMMENDATION

All of the recommendations in the *Surgical Management of Traumatic Brain Injury* are at the option level, supported only

by Class III scientific evidence. Unfortunately, option can mean choice and, as discussed above, it would be unethical not to operate on surgical mass lesions in salvageable patients. Currently, all neurological guidelines approved by the American Association of Neurological Surgeons and the Congress of Neurological Surgeons use option recommendations for Class III evidence. In the future, this may change because these surgical guidelines put maximized strain on using such terminology.

Given that the recommendations in these surgical guidelines are at the option level (Class III evidence) we state the recommendation at the beginning of the chapter on each topic, and refer the reader to this methodology section for an explanation of the level of evidence.

### MULTIDISCIPLINARY PROCESS

In all *Guidelines* published under the auspices of the Brain Trauma Foundation and the American Association of Neurological Surgeons, other professional organizations were involved in either developing the *Guidelines* or reviewed and approved them. In these *Surgical Management of Traumatic Brain Injury Guidelines*, however, only neurosurgeons were involved. These neurosurgeons represent a wide range of organizations. There were representatives from the American Association of Neurological Surgeons, the Congress of Neurological Surgeons, the European Brain Injury Consortium, the American College of Surgeons (Committee of Trauma) and the World Federation of Neurological Surgeons (Neurotrauma section) involved in the development of these *Surgical Management of Traumatic Brain Injury Guidelines*.

### REFERENCES

1. Committee to Advise the Public Health Service on Clinical Practice Guidelines (Institute of Medicine): *Clinical Practice Guidelines: Directions for a New Program*. Washington, D.C., National Academy Press, 1990.
2. Feinstein A: *Clinical Judgement*. Baltimore, Williams & Wilkins, 1967.
3. Walters B: Clinical Practice Parameter Development, in Bean J (ed): *Neurosurgery in Transition*. Baltimore, Williams & Wilkins, 1998, pp 99-111.

---

### SUBMISSIONS, PEER-REVIEW, AND DISCLOSURE

All original material presented in **NEUROSURGERY**, Operative **NEUROSURGERY**, and **NEUROSURGERY-Online** undergoes rigorous multi-factorial peer-review by carefully selected panels of knowledgeable and dedicated individuals who are highly versed in the academic process and the given topic.

For some time the burden of full disclosure of financial or other personal interests that may bias presentation has been placed on submitting authors. Neurosurgery will now extend this strict requirement of disclosure to those engaged in the review process in an effort to reduce bias and potential conflict in analysis and decision-making.

---

**M. Ross Bullock, M.D., Ph.D.**

Department of Neurological Surgery,  
Virginia Commonwealth University  
Medical Center,  
Richmond, Virginia

**Randall Chesnut, M.D.**

Department of Neurological Surgery,  
University of Washington  
School of Medicine,  
Harborview Medical Center,  
Seattle, Washington

**Jamshid Ghajar, M.D., Ph.D.**

Department of Neurological Surgery,  
Weill Cornell Medical College of  
Cornell University,  
New York, New York

**David Gordon, M.D.**

Department of Neurological Surgery,  
Montefiore Medical Center,  
Bronx, New York

**Roger Hartl, M.D.**

Department of Neurological Surgery,  
Weill Cornell Medical College of  
Cornell University,  
New York, New York

**David W. Newell, M.D.**

Department of Neurological Surgery,  
Swedish Medical Center,  
Seattle, Washington

**Franco Servadei, M.D.**

Department of Neurological Surgery,  
M. Bufalini Hospital,  
Cesena, Italy

**Beverly C. Walters, M.D., M.Sc.**

Department of Neurological Surgery,  
New York University  
School of Medicine,  
New York, New York

**Jack E. Wilberger, M.D.**

Department of Neurological Surgery,  
Allegheny General Hospital,  
Pittsburgh, Pennsylvania

**Reprints requests:**

Jamshid Ghajar, M.D., Ph.D.,  
Brain Trauma Foundation,  
523 East 72nd Street,  
New York, NY 10021.  
Email: ghajar@braintrauma.org

## SURGICAL MANAGEMENT OF ACUTE EPIDURAL HEMATOMAS

### RECOMMENDATIONS

(see *Methodology*)

#### Indications for Surgery

- An epidural hematoma (EDH) greater than 30 cm<sup>3</sup> should be surgically evacuated regardless of the patient's Glasgow Coma Scale (GCS) score.
- An EDH less than 30 cm<sup>3</sup> and with less than a 15-mm thickness and with less than a 5-mm midline shift (MLS) in patients with a GCS score greater than 8 without focal deficit can be managed nonoperatively with serial computed tomographic (CT) scanning and close neurological observation in a neurosurgical center.

#### Timing

- It is strongly recommended that patients with an acute EDH in coma (GCS score < 9) with anisocoria undergo surgical evacuation as soon as possible.

#### Methods

- There are insufficient data to support one surgical treatment method. However, craniotomy provides a more complete evacuation of the hematoma.

**KEY WORDS:** Coma, Computed tomographic parameters, Craniotomy, Epidural, Head injury, Hematoma, Surgical technique, Timing of surgery, Traumatic brain injury

*Neurosurgery* 58:S2-7-S2-15, 2006

DOI: 10.1227/01.NEU.0000210363.91172.A8

www.neurosurgery-online.com

## OVERVIEW

### Incidence

Since the introduction of CT scanning as the imaging study of choice to detect intracranial lesions after trauma, the incidence of surgical and nonsurgical EDH among traumatic brain injury (TBI) patients has been reported to be in the range of 2.7 to 4% (8, 11, 25, 41). Among patients in coma, up to 9% harbored an EDH requiring craniotomy (10, 35). The peak incidence of EDH is in the second decade, and the mean age of patients with EDH is between 20 and 30 years of age (3, 8, 9, 13, 16–18, 20, 22, 26, 29, 32, 37, 39). EDH are a rare entity in patients older than 50 to 60 years of age. In pediatric patients, the mean age of patients harboring EDH is between 6 and 10 years (21, 34), and EDH is less frequent in very young children and neonates (27, 30).

### Pathogenesis

Traffic-related accidents, falls, and assaults account for 53% (range, 30–73%), 30% (range,

7–52%), and 8% (range, 1–19%), respectively, of all EDH (3, 8, 20, 22, 26, 27, 36, 40). In pediatric patients, falls are the leading cause of EDH in 49% of cases (range, 25–59%) and traffic-related accidents are responsible for 34% (range, 25–41%) of all EDH (21, 25–27, 30, 34). EDH can result from injury to the middle meningeal artery, the middle meningeal vein, the diploic veins, or the venous sinuses. Historically, bleeding from the middle meningeal artery has been considered the main source for EDH. In a recent report on EDH in 102 pediatric patients and 387 adults, arterial bleeding was identified as the source of the EDH in 36% of the adults and only in 18% of the children (27). In 31% of the pediatric patients, a bleeding source could not be identified and venous bleeding accounted for approximately 32% of EDH in both age groups.

### Location

In surgical series, EDH are more frequently located in the temporoparietal and temporal regions as compared with other locations (3, 6, 25,



27, 29, 32). In 2 to 5% of patients, bilateral EDH are found (11, 18, 40), and there seems to be a slight predominance of right-sided EDH over left-sided lesions (6, 40).

### Clinical Presentation

In patients with EDH, 22 to 56% are comatose on admission or immediately before surgery (3, 17, 20, 22, 25, 32). The classically described "lucid interval," i.e., a patient who is initially unconscious, then wakes up and secondarily deteriorates, was observed in a total of 456 of 963 patients (47%) undergoing surgery for EDH in seven studies (3, 8, 18, 22, 28, 31, 39). Between 12 and 42% of patients remained conscious throughout the time between trauma and surgery (3, 8, 17, 22). Pupillary abnormalities are observed in between 18 and 44% of patients, and up to 27% (3–27%) of patients are neurologically intact. Other presenting symptoms include focal deficits, such as hemiparesis, decerebration, and seizures. Early seizures are noted in 8% of pediatric patients presenting with EDH (21).

### Mortality

The mortality in patients in all age groups and GCS scores undergoing surgery for evacuation of EDH is approximately 10% (range, 7–12.5%) (7, 8, 14, 17, 18, 20, 22, 28, 31, 32). Mortality in comparable pediatric case series is approximately 5% (25, 30).

### *Determinants of Outcome in Patients Undergoing Surgical Evacuation of an EDH*

GCS, age, pupillary abnormalities, associated intracranial lesions, time between neurological deterioration and surgery, and intracranial pressure (ICP) have been identified as important factors determining outcome from EDH.

### *Age and GCS*

The influence of age on outcome in the subgroup of patients with EDH is not as pronounced as it is in TBI patients overall. Three studies using multiple regression analysis found that GCS was a better predictor of outcome than age in patients undergoing surgery for EDH (20, 22, 38). In a retrospective analysis of 98 patients of all age groups with EDH undergoing craniotomy, van den Brink et al. (39) investigated determinants of outcome at 6 months. They identified GCS, age, and the CT diagnosis of subarachnoid hemorrhage as significant factors correlated with outcome, using multivariate analysis. Admission GCS or GCS before surgery is the single most important predictor of outcome in patients with EDH undergoing surgery (3, 10, 20, 22, 24, 38, 39). In three studies using multivariate analysis in a total of 284 patients, the admission GCS score was identified as the most significant factor determining outcome at 6 months (20, 38, 39). In one study with 200 patients undergoing craniotomy, admission and preoperative GCS both correlated with functional outcome at 1 year (22). Gennarelli et al. (10) analyzed the relationship between type of lesion, GCS score on admission, and 3-months outcome in

1107 comatose patients with TBI. The highest mortality was found in patients with a subdural hemorrhage and a GCS between 3 and 5 (74%). Patients with an EDH and a GCS of 3 to 5 had a mortality of 36%, and patients with an EDH and a GCS of 6 to 8 had a mortality of only 9%.

### *Pupils*

Pupillary abnormalities, such as pupillary asymmetry or fixed and dilated pupils occur in approximately 20 to 30% of patients with EDH undergoing surgery (3, 16, 20, 40) and in 62% of patients who are comatose on admission (32). One study showed that ipsilateral mydriasis was not associated with adverse outcome and was reversible when operated on within 70 minutes after pupillary dilation (6). Bilateral mydriasis, however, is associated with a high mortality (3, 6, 8, 24, 32, 39). Mydriasis contralateral to the hematoma is also associated with high mortality (24, 32). Van den Brink et al. (39), in a multivariate model evaluating the relative prognostic value of predictive parameters, found that, in patients in all age groups and GCS scores, pupillary abnormalities were significantly related to unfavorable outcome. Adverse outcome was observed in 30% of normal pupillary responses, in 35% of unilateral fixed pupils, and in 50% of bilateral fixed pupils. Bricolo and Pasut (3) achieved a good outcome in 100% of patients who presented with anisocoria and in 90% of patients who presented with anisocoria and hemiparesis. The only patient with bilateral mydriasis in their case series died.

### *Associated Lesions*

Associated intracranial lesions are found in between 30 and 50% of adult patients with surgically evacuated EDH (3, 8, 13, 16, 20, 22, 23, 27, 29, 31, 32, 35). These are predominantly contusions and intracerebral hemorrhage followed by subdural hematoma (SDH) and diffuse brain swelling (8, 16, 29, 32, 35). The incidence of associated lesions is less in the pediatric age group (25, 27, 30). SDH and/or parenchymal lesions in association with EDH lower the chance of a good outcome. In two studies with a total of 315 patients operated on for evacuation of an EDH, the frequency of associated intracranial lesions was 33% (20, 22). In both studies, a significant relationship was found between the presence of other lesions in addition to the EDH and an adverse outcome. Lee et al. (22) identified associated brain lesions as one of four independent predictors of unfavorable outcome after surgery for EDH and this has been confirmed by several others (8, 13, 16, 23, 32). Cranial fractures are present in between 70 and 95% of cases (15, 17, 20, 25, 30, 37). The impact of fractures on outcome is controversial. Kuday et al. (20) observed a significant relationship between cranial fractures and adverse outcome in 115 patients undergoing surgery for EDH. Lee et al. (22) did not see this relationship in a series of 200 patients managed similarly, and Rivas et al. (32) actually reported a significantly lower mortality rate in patients with cranial fractures. Significant extracranial injury is present in 7 to 23% of patients operated on for an EDH (8, 13, 17, 24, 27). Lobato et al. (24)

found extracranial injury in 20% of their patients, and the mortality rate in this subgroup was lower than the overall mortality (7.6% versus 28%). No data were found on the association of hypotension and outcome in patients with an acute EDH.

### ICP

There is only one study available in which postoperative ICP and its relationship to outcome 6 months after trauma was studied. Lobato et al. (24) monitored ICP in 54 (83%) of 64 comatose patients after removal of an EDH. Elevated ICP (>15 mm Hg) was found in 67% of cases, and ICP greater than 35 mm Hg was significantly associated with a higher mortality.

## PROCESS

A MEDLINE computer search using the following keywords for the years 1975 to 2001 was performed: "traumatic brain injury" or "head injury" and "epidural" or "extradural" and "hematoma" or "hematoma" or "hemorrhage." The search was narrowed by including the keywords "surgical treatment" or "surgery" or "operation" or "craniotomy" or "craniectomy" or "craniostomy" or "burr holes" and excluding "spinal." These searches combined yielded 168 articles. The reference lists of these publications were reviewed and an additional 22 articles were selected for analysis. Case reports, publications in books, and publications regarding penetrating brain injuries on spinal EDH and on exploratory burr holes without a preoperative CT scan were not included. Articles were excluded if the diagnosis of EDH was not based on CT scanning, or if subgroups of patients who did not undergo CT scanning were not clearly identified. Publications with fewer than 10 patients or publications that did not include information on outcome were excluded. Of these 190 articles, 18 were selected for analysis.

## SCIENTIFIC FOUNDATION

### Indication for Surgery

The decision to operate on an acute EDH is based on the patient's GCS score, pupillary exam, comorbidities, CT findings, age, and, in delayed decisions, the patient's ICP. Neurological deterioration over time is also an important factor influencing the decision to operate. Trauma patients presenting to the emergency room with altered mental status, pupillary asymmetry, and abnormal flexion or extension are at high risk for either an SDH and/or EDH compressing the brain and brainstem.

### CT Characteristics and Outcome

CT is the imaging study of choice for the diagnosis of an EDH. CT scanning is recommended in patients at risk for harboring an acute EDH. It allows not only diagnosis of the primary lesion but also identification of additional features that affect outcome, such as MLS, traumatic subarachnoid

hemorrhage, obliteration of the basal cisterns, thickness of the blood clot, and hematoma volume.

In a series of 200 patients who were treated surgically for EDH, Lee et al. (22) found that a hematoma volume greater than 50 cm<sup>3</sup> was significantly related to higher mortality and unfavorable functional outcome. Unfavorable functional recovery was observed in 6.2% of patients with a hematoma volume less than 50 cm<sup>3</sup>, and in 24% of patients with a hematoma volume greater than 50 cm<sup>3</sup>. Mixed density of the blood clot, indicating acute bleeding, was observed in 32% of their patients and correlated with unfavorable outcome but not with mortality. Patients with an MLS greater than 10 mm showed a higher mortality and more unfavorable outcome when compared with those with less displacement. Partial or total obliteration of the basal cisterns was observed in 59% of their patients and correlated with both mortality and functional outcome. Multivariate analysis identified only hematoma volume as an independent predictor of unfavorable outcome.

In contrast, in 98 patients with EDH who underwent surgery, van den Brink et al. (39) found that the status of the basal cisterns, MLS, and hematoma volume were not related to outcome. The authors only identified the presence of traumatic subarachnoid hemorrhage to be significantly associated with unfavorable outcome. Patients with favorable outcome had a hematoma volume of 56 ± 30 cm<sup>3</sup> and, with unfavorable outcome, the hematoma volume was 77 ± 63 cm<sup>3</sup>, but this difference was not significant.

Rivas et al. (32) found that hematoma volume and severity of MLS were related to preoperative coma in patients with EDH. In comatose patients, a hematoma volume greater than 150 cm<sup>3</sup> and an MLS greater than 12 mm were associated with increased mortality. Mixed-density blood clots were observed in 62% of their patients and were related to poor outcome. Location of the lesion did not influence outcome. Seelig et al. (35) did not find a relationship between location of blood clot, MLS, and outcome in 51 comatose patients undergoing surgery for EDH.

In summary, most authors could not detect a relationship between blood clot location and outcome. However, it is likely that hematoma volume, MLS, mixed density of the blood clot, and traumatic subarachnoid hemorrhage are related to outcome, but more studies are needed to clarify this issue.

### Surgery and Nonoperative Treatment

Prospective, randomized trials comparing surgical treatment with nonoperative management are not available. Some studies compared patients who were treated either surgically or nonoperatively, and used logistic regression analysis and multivariate analysis models to determine factors that were associated with either treatment (36, 37). Some investigators looked at patient series that were initially all treated nonoperatively and analyzed the factors associated with subsequent, delayed surgery (2, 7, 19, 37). There are no studies on nonoperative treatment of comatose patients with EDH.

What are the factors leading to surgery? The following studies compared characteristics between patients who were treated with either surgery or were managed nonoperatively. The value of such analyses is doubtful because it merely documents the criteria used to select patients for surgery.

Servadei et al. (36) conducted a prospective study including 158 consecutive patients with GCS 14 and 15 with EDH who were admitted to three neurosurgical units. A treatment protocol was not defined for these hospitals. One hundred-sixteen patients underwent surgery and 42 patients were managed nonoperatively. Ninety-three percent of patients with an MLS greater than 5 mm, and 91% of patients with a hematoma thickness greater than 15 mm underwent surgery. A logistic regression analysis identified hematoma thickness and MLS as the factors associated with surgery. Location and the presence of associated lesions did not reach significance. Outcome was good in all patients. Similar results were obtained in 33 pediatric patients, 20 of whom were treated surgically (1). Both groups did not differ in terms of age, GCS, and outcome. Multivariate logistic regression analysis revealed that MLS, hematoma thickness, and volume, as well as temporal location of the blood clot were related to surgery. Hematoma volume and MLS were 41 cm<sup>3</sup> and 8 cm<sup>3</sup>, and 4 mm and 0.5 mm, for the surgical and nonsurgical groups, respectively.

A review of 30 patients who were treated with craniotomy and 18 patients treated nonoperatively revealed that patients managed with surgery had lower GCS scores, were more likely to present with pupillary abnormalities and hemiparesis, and had larger blood clots and more MLS (12). Temporal location of the hematoma and the presence and location of a fracture were not related to surgery.

### Factors Determining Delayed Surgery

Bezircioglu et al. (2) conducted a prospective study on the nonoperative management of 80 patients with EDH and GCS scores between 9 and 15. Patients with a GCS score greater than 8, an EDH volume less than 30 ml, a hematoma thickness less than 2 cm, and without neurological deficit were treated nonoperatively. Five patients deteriorated and underwent craniotomy. One of these patients died, the others had good outcomes. The only factor significantly associated with delayed surgery was a temporal location of the hematoma, which was observed in all five surgical patients but only in 24% of the 75 patients treated without operation.

In a study of 74 patients with initially asymptomatic EDH managed nonoperatively, 14 required delayed surgery because of neurological deterioration or increase in the size of the hematoma (5). The authors found that a hematoma volume greater than 30 cm<sup>3</sup>, a hematoma thickness greater than 15 mm, and an MLS greater than 5 mm were significantly more frequent in patients requiring surgery. A hematoma volume greater than 30 cm<sup>3</sup>, an EDH thickness greater than 15 mm, and an MLS greater than 5 mm were observed in 5%, 27%, and 28% of patients managed without surgery, and in 57%, 71%, and 79% of patients who had surgery, respectively. The au-

thors used the "ellipsoid" or "ABC/2" method to estimate the volume of EDHs (see *Appendix 1*). Hematoma location, bone fractures, and time-to-initial CT scan were not related to outcome. In a small study on 22 patients, 7 of whom required later surgery, a time interval of less than 6 hours after injury to the first CT scan and a cranial fracture that crossed a major vessel were significantly related to surgery (19).

### Studies Describing Successful Nonsurgical Management

Bullock et al. (4) treated 12 of 123 patients presenting with EDH nonsurgically. All patients were conscious (GCS 12–15) with a hematoma volume between 12 and 38 cm<sup>3</sup> (mean, 26.8 cm<sup>3</sup>) and an MLS less than 10 mm on the initial CT scan. None of the hematomas were in the temporal region. All patients made a good outcome. Cucciniello reported on 57 patients with EDH who were treated nonoperatively (9). Initial GCS was between 10 and 15. Five hematomas were in the temporal region. The maximum hematoma thickness ranged between 6 and 12 mm. Only one patient had an MLS. All patients made a good recovery.

### Timing of Surgery

#### *Time between Injury and Surgery*

The effect of surgical timing on outcome from EDH is relevant for a subgroup of patients in whom the EDH causes compression of brain structures that, with time, could cause poor outcome. This subgroup is usually categorized as having pupillary abnormalities and/or a GCS score less than 9 (coma). Generally, studies of EDH reveal that only 21 to 34% of patients present to the hospital with a GCS score less than 8 or 9 (3, 20, 22, 25). Studies do not find a relationship between surgical timing and outcome if patients of all GCS scores are included. In 200 patients with EDH that were surgically evacuated, Lee et al. (22) failed to demonstrate a significant relationship between surgery within 4 hours of trauma or surgery within 2 hours of admission and outcome, using multivariate analysis. However, a significant correlation was observed between the duration of brain herniation, as evidenced by anisocoria, and the outcome. The time lapse between the onset of pupillary abnormalities and surgery is related to outcome. Cohen et al. (6) studied 21 patients with EDH and GCS score less than 9 who underwent surgery. Ten of these patients developed anisocoria after admission. All 5 patients with anisocoria for longer than 70 minutes before surgical evacuation of the EDH died. Patients with anisocoria for shorter than 70 minutes achieved a good outcome. Haselsberger et al. (13) studied 60 patients with EDH, and 34 patients developed coma before surgery. They found that patients treated within 2 hours after loss of consciousness exhibited a mortality rate of 17% and good recovery in 67%, compared with a mortality rate of 56% and good results in 13% in patients operated on later. Sakas et al. (33) found that all patients with either SDH or EDH with fixed and dilated pupils for longer than 6 hour died.

### Patient Transfer and Timing of Surgery

The question of whether a patient with acute EDH should be treated at the nearest hospital or transferred to a specialized trauma center has been debated but poorly documented in studies. This is an important timing issue and is significant in the group of patients who are deteriorating. Another issue is the surgical evacuation of EDH by nonneurosurgeons with subsequent transfer to a neurosurgical center. Obviously, these studies are uncontrolled with regard to the efficacy of surgery and the type of patients included in both arms. In the above timing of surgery, the group of patients in a coma and with pupillary abnormalities can be expected to do worse the longer the interval to evacuation of the EDH. Thus, because of the delay, transferred patients would have longer interval times to surgery.

Wester (40) studied 83 patients with acute EDH that underwent craniotomy. Twenty-eight patients were transferred from other hospitals and 11 of these underwent emergency surgery at those outside institutions. Patients who underwent surgery outside the parent institution by nonneurosurgeons had a significantly worse outcome at 3 months as compared with patients who were directly admitted to the study hospital. This was mainly attributed to the technical inadequacy of the primary operation at the outside institution. The authors interpreted this as support for the strategy of directly transferring patients to an adequate trauma center, but they did not control for other confounding variables, such as admission GCS and pupillary exam.

Another study analyzed 107 patients operated on for EDH (3). The majority (67%) of these patients were transferred from outlying hospitals. The authors noted that only 6% of the direct admissions experienced a poor outcome, as compared with 18% of patients who were transferred after undergoing CT scanning at an outside institution. This difference failed to reach statistical significance. Poon and Li (31) studied 71 patients with EDH managed surgically primarily at a neurosurgical hospital and 33 patients transferred from an outside institution. Time delay from neurological deterioration to surgery was  $0.7 \pm 1$  hour and  $3.2 \pm 0.5$  hours for the direct versus indirect transferred group, respectively. Six-months outcome was significantly better in patients who were directly admitted with a minimal delay from deterioration in neurological exam to surgery.

### SUMMARY

In patients with an acute EDH, clot thickness, hematoma volume, and MLS on the preoperative CT scan are related to outcome. In studies analyzing CT parameters that may be predictive for delayed surgery in patients undergoing initial nonoperative management, a hematoma volume greater than  $30 \text{ cm}^3$ , an MLS greater than 5 mm, and a clot thickness greater than 15 mm on the initial CT scan emerged as significant. Therefore, patients who were not comatose, without focal neurological deficits, and with an acute EDH with a thickness

of less than 15 mm, an MLS less than 5 mm, and a hematoma volume less than  $30 \text{ cm}^3$  may be managed nonoperatively with serial CT scanning and close neurological evaluation in a neurosurgical center (see *Appendix II* for measurement techniques). The first follow-up CT scan in nonoperative patients should be obtained within 6 to 8 hours after TBI. Temporal location of an EDH is associated with failure of nonoperative management and should lower the threshold for surgery. No studies are available comparing operative and nonoperative management in comatose patients. The literature supports the theory that patients with a GCS less than 9 and an EDH greater than  $30 \text{ cm}^3$  should undergo surgical evacuation of the lesion. Combined with the above recommendation, it follows that all patients, regardless of GCS, should undergo surgery if the volume of their EDH exceeds  $30 \text{ cm}^3$ . Patients with an EDH less than  $30 \text{ cm}^3$  should be considered for surgery but may be managed successfully without surgery in selected cases.

Time from neurological deterioration, as defined by onset of coma, pupillary abnormalities, or neurological deterioration to surgery, is more important than time between trauma and surgery. In these patients, surgical evacuation should be performed as soon as possible because every hour delay in surgery is associated with progressively worse outcome.

### KEY ISSUES FOR FUTURE INVESTIGATION

- Effect of transfer versus direct admission to a trauma center on timing of surgery and outcome from EDH.
- Identification of subgroups that do not benefit from surgery: old patients with low GCS scores, pupillary abnormalities, and associated intracerebral lesions.
- Surgical technique.

### REFERENCES

1. Bejjani G, Donahue D, Rusin J, Broemeling L: Radiological and clinical criteria for the management of epidural hematomas in children. *Pediatr Neurosurg* 25:302-308, 1996.
2. Bezircioglu H, Ersahin Y, Demircivi F, Yurt I, Donertas K, Tektas S: Nonoperative treatment of acute extradural hematomas: Analysis of 80 cases. *J Trauma* 41:696-698, 1996.
3. Bricolo A, Pasut L: Extradural hematoma: toward zero mortality. A prospective study. *Neurosurgery* 14:8-12, 1984.
4. Bullock R, Smith R, van Dellen JR: Nonoperative management of extradural hematoma. *Neurosurgery* 16:602-606, 1985.
5. Chen T, Wong C, Chang C, Lui T, Cheng W, Tsai M, Lin T: The expectant treatment of "asymptomatic" supratentorial epidural hematomas. *Neurosurgery* 32: 176-179, 1993.
6. Cohen J, Montero A, Israel Z: Prognosis and clinical relevance of anisocoria-craniotomy latency for epidural hematoma in comatose patients. *J Trauma* 41:120-122, 1996.
7. Cook R, Dorsch N, Fearnside M, Chaseling R: Outcome prediction in extradural haematomas. *Acta Neurochir (Wien)* 95:90-94, 1988.
8. Cordobes F, Lobato R, Rivas J, Munoz M, Chillón D, Portillo J, Lamas E: Observations on 82 patients with extradural hematoma. Comparison of results before and after the advent of computerized tomography. *J Neurosurg* 54:179-186, 1981.
9. Cucciniello B, Martellotta N, Nigro D, Citro E: Conservative management of extradural haematomas. *Acta Neurochir (Wien)* 120:47-52, 1993.

10. Gennarelli T, Spielman G, Langfitt T, Gildenberg P, Harrington T, Jane J, Marshall L, Miller J, Pitts L: Influence of the type of intracranial lesion on outcome from severe head injury. *J Neurosurg* 56:26-32, 1982.
11. Gupta S, Tandon S, Mohanty S, Asthana S, Sharma S: Bilateral traumatic extradural haematomas: Report of 12 cases with a review of the literature. *Clin Neurol Neurosurg* 94:127-131, 1992.
12. Hamilton M, Wallace C: Nonoperative management of acute epidural hematoma diagnosed by CT: The neuroradiologist's role. *AJNR Am J Neuroradiol* 13:853-859, 1992.
13. Haselsberger K, Pucher R, Auer L: Prognosis after acute subdural or epidural haemorrhage. *Acta Neurochir (Wien)* 90:111-116, 1988.
14. Heinzelmann M, Platz A, Imhof H: Outcome after acute extradural haematoma, influence of additional injuries and neurological complications in the ICU. *Injury* 27:345-349, 1996.
15. Hunt J, Hill D, Besser M, West R, Roncal S: Outcome of patients with neurotrauma: The effect of a regionalized trauma system. *Aust N Z J Surg* 65:83-86, 1995.
16. Jamjoom A: The influence of concomitant intradural pathology on the presentation and outcome of patients with acute traumatic extradural haematoma. *Acta Neurochir (Wien)* 115:86-89, 1992.
17. Jamjoom A: The difference in the outcome of surgery for traumatic extradural hematoma between patients who are admitted directly to the neurosurgical unit and those referred from another hospital. *Neurosurg Rev* 20:227-230, 1997.
18. Jones N, Molloy C, Kloeden C, North J, Simpson D: Extradural haematoma: Trends in outcome over 35 years. *Br J Neurosurg* 7:465-471, 1993.
19. Knuckey N, Gelbard S, Epstein M: The management of "asymptomatic" epidural hematomas. A prospective study. *J Neurosurg* 70:392-396, 1989.
20. Kудay C, Uzan M, Hanci M: Statistical analysis of the factors affecting the outcome of extradural haematomas: 115 cases. *Acta Neurochir (Wien)* 131:203-206, 1994.
21. Lahat E, Sheinman G, Feldman Z, Barzilay A, Harel R, Barzilay Z, Paret G: Metabolic and clinical markers of prognosis in the era of CT imaging in children with acute epidural hematomas. *Pediatr Neurosurg* 33:70-75, 2000.
22. Lee E, Hung Y, Wang L, Chung K, Chen H: Factors influencing the functional outcome of patients with acute epidural hematomas: Analysis of 200 patients undergoing surgery. *J Trauma* 45:946-952, 1998.
23. Lobato R, Cordobes F, Rivas J, de la Fuente M, Montero, A, Barcena A, Perez C, Cabrera A, Lamas E: Outcome from severe head injury related to the type of intracranial lesion. A computerized tomography study. *J Neurosurg* 59:762-774, 1983.
24. Lobato R, Rivas J, Cordobes F, Alted E, Perez C, Sarabia R, Cabrera A, Diez I, Gomez P, Lamas E: Acute epidural hematoma: An analysis of factors influencing the outcome of patients undergoing surgery in coma. *J Neurosurg* 68:48-57, 1988.
25. Maggi G, Aliberti F, Petrone G, Ruggiero C: Extradural hematomas in children. *J Neurosurg* 42:95-99, 1998.
26. Meier U, Heinitz A, Kintzel D: Surgical outcome after severe craniocerebral trauma in childhood and adulthood. A comparative study [in German]. *Unfallchirurg* 97:406-409, 1994.
27. Mohanty A, Kolluri V, Subbakrishna D, Satish S, Mouli B, Das B: Prognosis of extradural haematomas in children. *Pediatr Neurosurg* 23:57-63, 1995.
28. Otsuka S, Nakatsu S, Matsumoto S, Sato S, Motozaki T, Ban S, Yamamoto T: Study on cases with posterior fossa epidural hematoma—Clinical features and indications for operation. *Neurol Med Chir (Tokyo)* 30:24-28, 1990.
29. Paterniti S, Fiore P, Macri E, Marra G, Cambria M, Falcone F, Cambria S: Extradural haematoma. Report of 37 consecutive cases with survival. *Acta Neurochir (Wien)* 131:207-210, 1994.
30. Pillay R, Peter J: Extradural haematomas in children. *S Afr Med J* 85:672-674, 1995.
31. Poon W, Li A: Comparison of management outcome of primary and secondary referred patients with traumatic extradural haematoma in a neurosurgical unit. *Injury* 22:323-325, 1991.
32. Rivas J, Lobato R, Sarabia R, Cordobes F, Cabrera A, Gomez P: Extradural hematoma: Analysis of factors influencing the courses of 161 patients. *Neurosurgery* 23:44-51, 1988.
33. Sakas D, Bullock M, Teasdale G: One-year outcome following craniotomy for traumatic hematoma in patients with fixed dilated pupils. *J Neurosurg* 82:961-965, 1995.
34. Schutzman S, Barnes P, Mantello M, Scott R: Epidural hematomas in children. *Ann Emerg Med* 22:535-541, 1993.
35. Seelig J, Marshall L, Toutant S, Toole B, Klauber M, Bowers S, Varnell J: Traumatic acute epidural hematoma: Unrecognized high lethality in comatose patients. *Neurosurgery* 15:617-620, 1984.
36. Servadei F, Faccani G, Roccella P, Seracchioli A, Godano U, Ghadirpour R, Naddeo M, Piazza G, Carrieri P, Taggi F, Pagni CA: Asymptomatic extradural haematomas. Results of a multicenter study of 158 cases in minor head injury. *Acta Neurochir (Wien)* 96:39-45, 1989.
37. Sullivan T, Jarvik J, Cohen W: Follow-up of conservatively managed epidural hematomas: Implications for timing of repeat CT. *AJNR Am J Neuroradiol* 20:107-113, 1999.
38. Uzan M, Yentur E, Hanci M, Kaynar MY, Kafadar A, Sarioglu AC, Bahar M, Kудay C: Is it possible to recover from uncal herniation? Analysis of 71 head injured cases. *J Neurosurg Sci* 42:89-94, 1998.
39. van den Brink WA, Zwienenberg M, Zandee SM, van der Meer L, Maas AI, Avezaat CJ: The prognostic importance of the volume of traumatic epidural and subdural haematomas revisited. *Acta Neurochir (Wien)* 141:509-514, 1999.
40. Wester K: Decompressive surgery for "pure" epidural hematomas: Does neurosurgical expertise improve the outcome? *Neurosurgery* 44:495-500, 1999.
41. Wu J, Hsu C, Liao S, Wong Y: Surgical outcome of traumatic intracranial hematoma at a regional hospital in Taiwan. *J Trauma* 47:39-43, 1999.

---

**CONTACT THE EDITORIAL OFFICE**

To reach the Editorial Office, please use the following information.

**NEUROSURGERY**

Michael L.J. Apuzzo, Editor  
 1420 San Pablo Street, PMB A-106  
 Los Angeles, CA 90033  
 Phone: 323/442-3001  
 Fax: 323/442-3002  
 Email: neurosurgery-journal@hsc.usc.edu  
 Website: www.neurosurgery-online.com

---

EVIDENTIARY TABLE

TABLE 1. Surgical management of acute epidural hematomas<sup>a</sup>

Authors (ref. no.)	No. of patients	Class	Inclusion GCS	Treatment	Outcome	Description	Conclusion																																
Bejjani et al. (1)	33	III	All GCS	Surgery and nonsurgical	Discharge	Retrospective analysis of factors affecting the decision to operate in 33 pediatric patients with EDH. 13 patients underwent operative treatment.	Mass effect, temporal location of blood clot, MLS, thickness of clot, and volume were independently related to surgery. Other clinical factors, such as age, GCS, and associated fractures were not. Outcome was good in >90% of patients.																																
Bezircioglu et al. (2)	80	III	GCS > 8	Surgery and nonsurgical	GOS at 2 mo	Prospective study on nonoperative management of patients with EDH. Patients with a EDH volume <30 ml, thickness <2 cm, GCS > 8, no neurological deficit, and admitted within 24 h of TBI were treated nonoperatively.	<p>EDH</p> <table border="1"> <tr> <th>Operative treatment</th> <th>Nonoperative treatment</th> </tr> <tr> <td>Thickness (mm)</td> <td>20</td> </tr> <tr> <td>Volume (cm<sup>3</sup>)</td> <td>41</td> </tr> <tr> <td>MLS (mm)</td> <td>4</td> </tr> </table> <p>Of 80 patients who were treated nonoperatively, 5 deteriorated and needed surgery. One died, and 4 had a good outcome. Temporal lobe EDH was related to delayed surgery.</p>	Operative treatment	Nonoperative treatment	Thickness (mm)	20	Volume (cm <sup>3</sup> )	41	MLS (mm)	4																								
Operative treatment	Nonoperative treatment																																						
Thickness (mm)	20																																						
Volume (cm <sup>3</sup> )	41																																						
MLS (mm)	4																																						
Bricolo and Pasut (3)	107	III	All GCS	All surgery	GOS at 6 mo		<p>Neurological signs on admission</p> <table border="1"> <tr> <th>No. of patients</th> <th>Good outcome (%)</th> </tr> <tr> <td>None</td> <td>17</td> </tr> <tr> <td>One dilated pupil</td> <td>8</td> </tr> <tr> <td>Hemiparesis only</td> <td>23</td> </tr> <tr> <td>Hemiparesis and one dilated pupil</td> <td>10</td> </tr> <tr> <td>Decortication</td> <td>9</td> </tr> <tr> <td>Decerebration</td> <td>4</td> </tr> <tr> <td>Both pupils fixed</td> <td>1</td> </tr> </table> <p>GCS on admission</p> <table border="1"> <tr> <th>No. of patients</th> <th>GR/MD (%)</th> <th>SD/VS (%)</th> <th>Dead (%)</th> </tr> <tr> <td>3-4</td> <td>4</td> <td>25</td> <td>50</td> </tr> <tr> <td>5-7</td> <td>32</td> <td>78</td> <td>13</td> </tr> <tr> <td>8-15</td> <td>71</td> <td>97</td> <td>3</td> </tr> </table> <p>All patients made a good recovery. No temporal EDH patients were included, and basal cisterns were open or only partially effaced in all patients. MLS was &lt;10 mm in all patients. Mean volume of the EDH was 26.8 cm<sup>3</sup>.</p> <p>All patients achieved a good GOS. Patients requiring delayed surgery presented more frequently with an EDH volume &gt;30 cm<sup>3</sup>, clot thickness &gt;15 mm, or MLS &gt; 5 mm on the initial CT scan.</p>	No. of patients	Good outcome (%)	None	17	One dilated pupil	8	Hemiparesis only	23	Hemiparesis and one dilated pupil	10	Decortication	9	Decerebration	4	Both pupils fixed	1	No. of patients	GR/MD (%)	SD/VS (%)	Dead (%)	3-4	4	25	50	5-7	32	78	13	8-15	71	97	3
No. of patients	Good outcome (%)																																						
None	17																																						
One dilated pupil	8																																						
Hemiparesis only	23																																						
Hemiparesis and one dilated pupil	10																																						
Decortication	9																																						
Decerebration	4																																						
Both pupils fixed	1																																						
No. of patients	GR/MD (%)	SD/VS (%)	Dead (%)																																				
3-4	4	25	50																																				
5-7	32	78	13																																				
8-15	71	97	3																																				
Bullock et al. (4)	12	III	GCS 12-15	Nonsurgical	Discharge	Retrospective analysis of the nonoperative treatment of 12 patients with EDH.																																	
Chen et al. (5)	74	III	GCS > 12	Surgery and nonsurgical	GOS at 1 mo	Retrospective analysis of 111 of 465 patients with EDH who underwent initial nonoperative treatment. All nonoperative cases had a GCS > 12, and 14 patients underwent delayed surgery because of neurodeterioration or increase in the size of the EDH. 37 patients were excluded because of incomplete CT data.	<table border="1"> <tr> <th>Operative treatment</th> <th>Nonoperative treatment</th> <th>P value</th> </tr> <tr> <td>No. of patients</td> <td>97</td> <td></td> </tr> <tr> <td>Temporal/frontotemporal location of EDH</td> <td>36.6%</td> <td>n.s.</td> </tr> <tr> <td>EDH &gt; 30 cm<sup>3</sup></td> <td>3</td> <td>P &lt; 0.001</td> </tr> <tr> <td>MLS &gt; 5 mm</td> <td>17</td> <td>P &lt; 0.001</td> </tr> <tr> <td>EDH &gt; 15 mm thickness</td> <td>16</td> <td>P &lt; 0.01</td> </tr> </table>	Operative treatment	Nonoperative treatment	P value	No. of patients	97		Temporal/frontotemporal location of EDH	36.6%	n.s.	EDH > 30 cm <sup>3</sup>	3	P < 0.001	MLS > 5 mm	17	P < 0.001	EDH > 15 mm thickness	16	P < 0.01														
Operative treatment	Nonoperative treatment	P value																																					
No. of patients	97																																						
Temporal/frontotemporal location of EDH	36.6%	n.s.																																					
EDH > 30 cm <sup>3</sup>	3	P < 0.001																																					
MLS > 5 mm	17	P < 0.001																																					
EDH > 15 mm thickness	16	P < 0.01																																					

(Table continues)

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Class	Inclusion GCS	Treatment	Outcome	Description	Conclusion
Poon and Li (31)	104	III	All GCS	All surgery	GOS at 6 mo	A prospective study of 104 patients of all age groups with isolated EDH. One-third of the patients were transferred from an outside hospital.	A significant increase in mortality and morbidity was related to increased time from neurological deterioration to surgery.
Sakas et al. (33)	11	III	"Comatose"	All surgery	GOS at 1 yr	Analysis of 40 severe TBI patients who underwent craniotomy after developing bilateral fixed and dilated pupils.	<p>No. of patients</p> <p>Time between deterioration of conscious level and surgery (h)</p> <p>D (%)</p> <p>VS/SD/MD (%)</p> <p>GR (%)</p> <p>71</p> <p>0.7 ± 1</p> <p>4</p> <p>10</p> <p>86</p> <p>33</p> <p>3.2 ± 0.5</p> <p>24</p> <p>27</p> <p>49</p> <p>Patients with SDH had a significant increase in mortality (64%) compared with those with EDH (18%). Patients who underwent delayed (&gt;3 h) surgery had a worse outcome.</p>
Seelig et al. (35)	51	III	"Comatose"	All surgery	GOS at 3 mo-2 yr	Prospective multicenter database analyzed retrospectively, looking at the factors influencing outcome after surgery for EDH.	<p>Time from pupillary nonreactivity to surgery</p> <p>No. of patients</p> <p>GR/MD (%)</p> <p>SD (%)</p> <p>VS/D (%)</p> <p>&lt;3 h</p> <p>20</p> <p>30</p> <p>30</p> <p>40</p> <p>&gt;3 h</p> <p>16</p> <p>25</p> <p>12</p> <p>63</p> <p>The most powerful indicator of outcome was motor score before surgery.</p>
Servadei et al. (36)	158	III	GCS 14/15	Surgery and nonsurgical	GOS at 6 mo	Prospective study of 158 consecutive patients with minor TBI (GCS 14/15) admitted to 3 neurosurgical units. No treatment protocol was specified and a logistic regression analysis was conducted to identify the factors leading to surgery.	<p>Motor score</p> <p>No. of patients</p> <p>GR/MD/SD (%)</p> <p>VS/D (%)</p> <p>1-3</p> <p>19</p> <p>32</p> <p>68</p> <p>4-6</p> <p>32</p> <p>69</p> <p>31</p> <p>116 patients underwent surgery and 42 patients were managed nonoperatively. Of all factors analyzed, only thickness of hematoma and MLS were related to the decision to operate. Outcome was the same in both groups.</p>
van den Brink et al. (39)	98	III	All GCS	All surgery	GOS at 6 mo	Retrospective analysis of CT parameters and outcome in 98 patients of all age groups with acute EDH.	<p>MLS (mm)</p> <p>No. of patients</p> <p>Craniotomy (%)</p> <p>Nonoperative management (%)</p> <p>0-5</p> <p>130</p> <p>70</p> <p>30</p> <p>6-10</p> <p>19</p> <p>89.5</p> <p>10.5</p> <p>&gt;10</p> <p>8</p> <p>100</p> <p>0</p> <p>Volume of the EDH did not correlate with outcome. Subarachnoid blood and pupillary dysfunction, age &gt;20 yr, and GCS on admission were parameters correlating with outcome.</p>
Wester (40)	83	III	All GCS	All surgery	GOS at 3 mo	Retrospective study of the management of pure EDH in patients of all age groups in Norway. Information on their initial neurological exam is limited.	<p>Age (yr)</p> <p>SD/VS/D (%)</p> <p>&lt;20</p> <p>6</p> <p>21-40</p> <p>17</p> <p>41-60</p> <p>31</p> <p>&gt;60</p> <p>50</p> <p>Motor score</p> <p>SD/VS/D (%)</p> <p>1-3</p> <p>50</p> <p>4-6</p> <p>7</p> <p>Pupils</p> <p>SD/VS/D (%)</p> <p>Normal</p> <p>3</p> <p>Unilateral fixed and dilated</p> <p>35</p> <p>Bilateral fixed and dilated</p> <p>50</p> <p>The outcome of 11 patients treated at the primary hospital was worse than the outcome of patients that were admitted directly. The authors conclude that patients should be transferred directly for neurosurgical management.</p>

<sup>a</sup> GCS, Glasgow Coma Scale; EDH, epidural hematoma; MLS, midline shift; GOS, Glasgow outcome score; TBI, traumatic brain injury; GR, good recovery; MD, moderate disability; SD, severe disability; VS, vegetative state; D, death; CT, computed tomographic scan; n.s., not significant.

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	GCS	Treatment	Outcome	Description	Conclusion
Cohen et al. (6)	21	III	GCS < 8	All surgery	Not documented	Prospective data collection on 21 adult patients admitted during 3 yr with acute EDH and GCS < 8, who underwent surgery. 10 patients developed new anisocoria after admission. Only 14 patients had CT scans. 3 patients had emergency craniotomy without radiographic studies.	Anisocoria for more than 70 min was associated with 100% mortality, and for less than 70 min with GOS 4 and 5.
Cucciniello et al. (9)	57	III	GCS 10-15	Nonsurgical	Not documented	Retrospective analysis of a case series of 57 patients out of 144 with EDH who were managed nonoperatively.	<b>Time from anisocoria to craniotomy</b> <71 min 5 >89 min 5 All patients had good outcome. Only one patient demonstrated MLS and maximum hematoma thickness was between 6 and 12 mm.
Hamilton and Wallace (12)	48	III	All GCS	Surgery and nonsurgical	Discharge	Retrospective analysis of patients who underwent surgical and nonoperative treatment for EDH.	Patients who underwent surgical treatment had lower GCS scores, more pupillary abnormalities, larger EDH, greater MLS, and were more likely to show uncal herniation on CT.
Haselsberger et al. (13)	60	III	All GCS	All surgery	Not documented	A review of 171 patients who presented with either subdural (111 patients) or epidural (60 patients) hematoma. The influence of surgical timing on mortality and functional recovery was analyzed.	Patients with an acute subdural or epidural hematoma had a lower mortality and improved functional recovery when operated on <2 h after onset of coma.
Knuckey et al. (19)	22	III	All GCS	Surgery and nonsurgical	Not documented	Retrospective analysis of 22 patients of all age groups who were initially treated nonoperatively. 7 patients needed subsequent craniotomy.	<b>Time from coma onset to surgery</b> <2 h 18 >2 h 16 GR/MD (%) 67 SD/VS (%) 16 D (%) 17
Kuday et al. (20)	115	III	All GCS	All surgery	GOS at 6 mo	Retrospective analysis of prospectively collected data on 115 patients of all age groups undergoing surgery for "signs of herniation."	Early CT scan <6 h after trauma and cranial fractures crossing big vessels or the middle meningeal artery were associated with deterioration. Initial GCS, age, and size of the EDH were not.
Lee et al. (22)	200	III	All GCS	All surgery	GOS at 1 yr	A retrospective study of 200 patients with epidural hematomas requiring operation. Analysis of factors leading to a poor outcome.	GCS is the parameter correlating most closely with outcome. The presence of a cranial fracture and "long" interval between onset of symptoms and intervention also affect outcome.
						<b>Interval between anisocoria and decompression (h)</b> No 126 <1.5 18 1.5-2.5 27 2.5-3.5 13 3.5-4.5 13 >4.5 3 SD/VS/D (%) 7.9 22.2 25.9 30.8 53.8 66.7	
						<b>Hematoma volume (ml)</b> <50 81 51-100 81 101-150 31 >150 7 MLS (mm) <5 85 6-10 69 11-15 35 >15 11 SD/VS/D (%) 6.2 14.8 38.7 71.4	
						<b>No. of patients</b> 126 18 27 13 13 3	
						<b>No. of patients</b> 81 81 31 7 85 69 35 11	
						<b>SD/VS/D (%)</b> 6.2 14.8 38.7 71.4 9.4 8.7 37.1 63.6	(Table continues)



**M. Ross Bullock, M.D., Ph.D.**

Department of Neurological Surgery,  
Virginia Commonwealth University  
Medical Center,  
Richmond, Virginia

**Randall Chesnut, M.D.**

Department of Neurological Surgery,  
University of Washington  
School of Medicine,  
Harborview Medical Center,  
Seattle, Washington

**Jamshid Ghajar, M.D., Ph.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David Gordon, M.D.**

Department of Neurological Surgery,  
Montefiore Medical Center,  
Bronx, New York

**Roger Hartl, M.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David W. Newell, M.D.**

Department of Neurological Surgery,  
Swedish Medical Center,  
Seattle, Washington

**Franco Servadei, M.D.**

Department of Neurological Surgery,  
M. Bufalini Hospital,  
Cesena, Italy

**Beverly C. Walters, M.D., M.Sc.**

Department of Neurological Surgery,  
New York University  
School of Medicine,  
New York, New York

**Jack E. Wilberger, M.D.**

Department of Neurological Surgery,  
Allegheny General Hospital,  
Pittsburgh, Pennsylvania

**Reprints requests:**

Jamshid Ghajar, M.D., Ph.D.,  
Brain Trauma Foundation,  
523 East 72nd Street,  
New York, NY 10021.  
Email: ghajar@braintrauma.org

## SURGICAL MANAGEMENT OF ACUTE SUBDURAL HEMATOMAS

### RECOMMENDATIONS

(see *Methodology*)

#### Indications for Surgery

- An acute subdural hematoma (SDH) with a thickness greater than 10 mm or a midline shift greater than 5 mm on computed tomographic (CT) scan should be surgically evacuated, regardless of the patient's Glasgow Coma Scale (GCS) score.
- All patients with acute SDH in coma (GCS score less than 9) should undergo intracranial pressure (ICP) monitoring.
- A comatose patient (GCS score less than 9) with an SDH less than 10-mm thick and a midline shift less than 5 mm should undergo surgical evacuation of the lesion if the GCS score decreased between the time of injury and hospital admission by 2 or more points on the GCS and/or the patient presents with asymmetric or fixed and dilated pupils and/or the ICP exceeds 20 mm Hg.

#### Timing

- In patients with acute SDH and indications for surgery, surgical evacuation should be performed as soon as possible.

#### Methods

- If surgical evacuation of an acute SDH in a comatose patient (GCS < 9) is indicated, it should be performed using a craniotomy with or without bone flap removal and duraplasty.

**KEY WORDS:** Coma, Computed tomographic parameters, Craniotomy, Decompressive craniectomy, Head injury, Hematoma, Intracranial pressure monitoring, Salvageability, Subdural, Surgical technique, Timing of surgery, Traumatic brain injury

*Neurosurgery* 58:S2-16-S2-24, 2006

DOI: 10.1227/01.NEU.0000210364.29290.C9

www.neurosurgery-online.com

## OVERVIEW

SDH represents one type of intracranial mass lesion, and surgical management attempts to define the subset of patients who would benefit from surgical evacuation of an acute SDH. SDH are diagnosed on a CT scan as extracranial, hyperdense, crescentic collections between the dura and the brain parenchyma. They can be divided into acute and chronic lesions. Herein, "acute SDH" is defined as an SDH diagnosed within 14 days of traumatic brain injury (TBI).

### Incidence

Studies conducted after the introduction of CT scanning report an incidence of acute SDH between 12 and 29% in patients admitted with

severe TBI. Combining several publications, acute SDH was diagnosed in 21% of 2870 patients (8, 26, 27, 36). When including mild, moderate, and severe head injuries, 11% (360 of 3397 patients) present with SDH (21, 28). The mean age for this combined group is between 31 and 47 years, with the vast majority of patients being men (6, 11, 17, 21).

### Mechanism

The mechanism of injury responsible for the development of an SDH differs between age groups. Most SDH are caused by motor vehicle-related accidents (MVA), falls, and assaults. In one study, 56% of SDH in the younger group (18–40 yr) were caused by MVA and only 12% were caused by falls, whereas, in the older groups (>65 yr), these

mechanisms were responsible for 22% and 56% of SDH, respectively (15). Falls have been identified as the main cause of traumatic SDH in two studies looking specifically at patients older than 75 and 80 years (3, 16). Studies with comatose patients describe MVA as the mechanism of injury in 53 to 75% of SDH. This indicates that MVA causes more severe injury, possibly because of high-velocity accidents and diffuse axonal injury (18, 26, 36).

### Clinical Presentation

Between 37 and 80% of patients with acute SDH present with initial GCS scores of 8 or less (4, 6, 21, 28, 33). A lucid interval has been described in 12 to 38% of patients before admission but there is no conclusive evidence that this correlates with outcome (1, 9, 31, 34, 36). The definition of lucid interval is vague. Authors interpret the lucid interval differently and analysis of its frequency requires documentation during the prehospital phase. Pupillary abnormalities are observed in 30 to 50% of patients on admission or before surgery (6, 10, 28, 33).

### Mortality

Studies looking at patients from all age groups with GCS scores between 3 and 15 with SDH requiring surgery quote mortality rates between 40 and 60% (10, 11, 13, 14, 17, 27, 39). Mortality among patients presenting to the hospital in coma with subsequent surgical evacuation is between 57 and 68% (7, 12, 18, 20, 26, 36).

### Associated Injuries

Only 30 to 40% of SDH requiring surgery are isolated lesions (21, 28). In the majority of cases, the SDH is associated with other intracranial and extracranial injuries. Contusions and intracerebral hematomas are the most frequently associated intracranial abnormalities. Associated intracranial and extracranial lesions have been reported in larger series to occur in 47 to 57% of patients presenting with GCS scores between 3 and 15 (4, 11, 15, 28) and in 65 to 82% of patients with GCS scores less than 10 (18, 26). In patients with SDH, contusions and fractures are frequent associated injuries (16, 19, 25, 31, 36, 37). An associated subarachnoid hemorrhage has been observed in 14 to 25% of patients with SDH (4, 28) and epidural hematomas are observed in 6 to 14% of patients (4, 28). Significant extracranial injuries are observed in 18 to 51% of patients and the majority of these cases include facial fractures, extremity fractures, and thoracic and abdominal trauma (5, 6, 11, 28, 31). Because of the frequent association of SDH with parenchymal injury, surgical management decisions should take into consideration the recommendations for both lesion types.

## PROCESS

A MEDLINE computer search using the following keywords for the years 1975 to 2001 was performed: "traumatic brain injury" or "head injury" and "subdural" or "intradural"

and "hematoma" or "hemorrhage." The search was narrowed by including the keywords "surgical treatment" or "surgery" or "operation" or "craniotomy" or "craniectomy" or "craniostomy" or "burr holes" and excluding "chronic" and "spinal." These searches combined yielded 161 articles. The reference lists of these publications were reviewed and an additional 18 articles were selected for analysis. Case reports, publications in books, and publications regarding penetrating brain injuries, or spinal or chronic SDH were not included. Chronic SDH was defined as an SDH occurring or diagnosed more than 14 days after trauma. Articles were excluded if the diagnosis of SDH was not based on CT scanning, or if subgroups of patients who did not undergo CT scanning were not clearly identified. Publications with fewer than 10 patients or publications that did not include information on outcome were excluded. Of these 179 articles, 21 were selected for analysis.

## SCIENTIFIC FOUNDATION

### Indications for Surgery

The decision to operate on an SDH is based on the patient's GCS score, pupillary exam, comorbidities, CT findings, age, and, in delayed decisions, ICP. Neurological deterioration over time is also an important factor influencing the decision to operate. Trauma patients presenting to the emergency room with altered mental status, pupillary asymmetry, and abnormal flexion or extension are at high risk for either an SDH and/or an epidural hematoma compressing the brain and brainstem.

### CT Parameters

Many investigators have tried to define a relationship between CT parameters, such as hematoma volume, clot thickness, midline shift (MLS), and patency of the basal cisterns, and outcome. Two studies using multivariate analysis to identify factors affecting outcome from SDH found contradictory results. Howard et al. (15) reported on 67 patients, with GCS scores between 3 and 15, who were undergoing surgery, and found a significant correlation between poor outcome and the volume of the SDH and the MLS. The volume of the SDH, the MLS, and mortality were significantly greater in older patients. van den Brink et al. (33) found no difference in hematoma volumes, MLS or status of the basal cisterns when comparing surgical patients, who had a GCS of 3 to 15, and favorable versus unfavorable outcome. Zumkeller et al. (39) investigated CT scan parameters in 174 patients with SDH and a GCS between 3 and 15 undergoing surgery. The findings revealed a 10% mortality rate in patients with a clot thickness of less than 10 mm, and a 90% mortality for patients with clots thicker than 30 mm. For an MLS greater than 20 mm, there was a steep increase in mortality. Both parameters correlated well with the Glasgow outcome score (GOS). In a mixed group of patients treated with or without surgery, Servadei et al. (28) also found a correlation between outcome and clot thickness, MLS, and status of the basilar cisterns. Kotwica and Brzezinski (18) found a significant relationship between MLS and out-

come in 200 patients with GCS scores lower than 10, who were undergoing surgery for SDH. In summary, there seems to be a relationship between CT parameters and outcome, but it is difficult to determine specific threshold values.

### **Surgical Versus Nonoperative Treatment of SDH**

The decision for nonoperative versus surgical management of SDH is influenced by the GCS score; CT parameters, such as MLS, SDH clot thickness and volume, and patency of the basal cisterns; and the salvageability of the patient (i.e., whether the primary injury is so extensive that evacuation of the SDH will not make a difference in outcome). On the basis of the reviewed literature, a clot thickness greater than 10 mm or a MLS greater than 5 mm are suggested as critical parameters for surgical evacuation of an acute SDH, regardless of the GCS.

Wong (37) tried to identify parameters that would predict the failure of initial nonoperative management. No treatment protocol was defined. Six of 31 patients with GCS scores between 6 and 15 who were initially treated without surgery required a later craniotomy because of neurological deterioration (performed within 3 d). The authors found that an MLS greater than 5 mm in patients with a GCS score of lower than 15 on the initial CT scan was significantly related to the failure of nonoperative treatment. Hematoma volume and thickness of the hematoma were not predictive. Good outcome was achieved in all patients.

Matthew et al. (22) reviewed the data on 23 patients with GCS scores between 13 and 15 who were initially treated nonoperatively. No criteria were defined for nonoperative management. All patients had an isolated SDH and all were observed in the neurosurgical intensive care unit. Six patients required delayed (mean, 14 d) evacuation of their SDH. Significant differences in clot thickness and hematoma volume were found between the operative and the nonoperative groups. In addition, all patients with an initial hematoma thickness greater than 10 mm required surgery. Finally, Servadei et al. (27) developed a protocol to select comatose patients with SDH for nonoperative management. The criteria used to select comatose patients for nonoperative treatment were clinical stability or improvement during the time from injury to evaluation at the hospital, hematoma thickness less than 10 mm and MLS less than 5 mm on the initial CT scan, and ICP monitoring in the neurosurgical intensive care unit. Surgery was performed if the ICP exceeded 20 mm Hg. Fifteen of 65 comatose patients with SDH were treated nonoperatively. Of these, two patients were identified that required delayed surgery based on increasing ICP and the development of intracerebral hematomas. Good outcome was achieved in 23% of the patients in the surgery group and 67% of the patients in the nonoperative group. The authors concluded that nonoperative treatment can be safely used for a defined group of comatose patients with SDH.

### **Age and Salvageability**

Increasing age is a strong independent factor in prognosis from severe TBI, with a significant increase in poor outcome in patients older than 60 years of age (2). Among patients with acute SDH, there is also a tendency for older patients to have a poorer outcome, especially those patients presenting with low GCS scores (3, 16, 18, 19, 36). In comatose patients with GCS scores less than 9 who underwent craniotomy for SDH, Wilberger et al. (36) found that age older than 65 years was statistically correlated with poorer outcome. In patients with GCS scores less than 10 undergoing surgery for SDH, Kotwica and Brzezinski (18) found that there was a statistically significant difference in 3-months outcome between younger patients (18–30 yr of age, 25% mortality) and older patients (>50 yr, 75% mortality). Three smaller studies looked specifically at patients between 70 and 100 years of age with an admission GCS (one study) or preoperative GCS (two studies) equal to or less than 9. The 49 patients from these three studies all underwent surgery. Forty-eight patients died and one had a poor outcome (severely disabled or vegetative) (3, 16, 19). No patient older than the age of 75 years who preoperatively was extensor posturing, flaccid to pain, or had unilateral or bilateral fixed and dilated pupils made a good recovery (GOS, 3–5) (16). In 23 comatose patients aged 66 years and older who presented with an acute SDH, Howard et al. (15) found that 17 died and the others survived in a vegetative state or with severe disabilities.

Functional outcomes in older patients with low GCS scores have also been reported. However, these articles did not document whether patients showed signs of cerebral herniation. Hatashita et al. (14) reported 9 deaths in 12 patients older than 65 years who presented with GCS scores between 4 and 6 and underwent surgery for SDH, as compared with 34% for those aged 19 to 40 years. Two older patients survived with a GOS of 4 or 5. In another publication, 1 of 28 comatose patients older than 65 years made a functional recovery after craniotomy for SDH (36). Although some studies that included patients with all GCS scores undergoing surgery for SDH found a relationship between age and outcome (15, 21, 28), other authors failed to describe such a relationship (13, 17, 26, 33, 39). Three studies using multivariate analysis in patients operated on for SDH did not identify age as an independent predictor of outcome (15, 26, 33). In summary, there is a relationship between poor outcome and age, low GCS, and signs of herniation, but it is not possible to predict death on the basis of old age and poor GCS with certainty.

### **Timing of Surgery**

The time from injury to entering the operating room is one of the few factors that can be affected by intervention. Unfortunately, the relationship between time from injury to operation and outcome is difficult to study because patients who are operated on soon after TBI tend to have more severe injuries than those who undergo delayed surgery. Therefore, outcome in patients operated on a short time after injury is frequently

worse when compared with patients undergoing delayed surgery. Furthermore, time from TBI to surgery may not be as important as time from clinical deterioration or onset of cerebral herniation to surgery. The literature supports the statement that the length of time from clinical deterioration to operative treatment of an SDH is significantly related to outcome. Haselsberger et al. (13) studied the time interval from onset of coma to surgery in 111 patients with SDH. Thirty-four patients were operated on within 2 hours after onset of coma. Of those patients, 47% died and 32% recovered with good outcome or moderate disability. However, 54 patients who underwent surgery longer than 2 hours after the onset of coma had a mortality of 80% and only 4% had a favorable outcome. These differences were statistically significant.

Seelig et al. (26) studied the delay to surgery in 82 patients with SDH who were all comatose on admission. They found a 30% mortality rate in patients operated on within 4 hours after injury and a 90% mortality in patients who had surgery more than 4 hours after injury. The mean time for evacuation was  $390 \pm 39$  minutes in patients who died and  $170 \pm 18$  minutes in patients who made a functional recovery. Multivariate analysis identified time to surgery as one of the factors determining outcome from SDH. The weaknesses of this study are that a proportion of patients did not undergo CT scanning and that SDH was diagnosed using air ventriculography. In comatose patients undergoing surgery for SDH, Wilberger et al. (36) found that the time interval from TBI to surgery was  $374 \pm 31$  minutes for patients who died and  $280 \pm 26$  minutes for patients who made a functional recovery. Mortality in patients undergoing surgery within 4 hours of injury was 59% versus 69% in patients operated on after 4 hours. A statistically significant difference could only be found in patients who underwent surgery after 12 hours, in which case, mortality rose to 82%.

Sakas et al. (24) looked at outcome from surgery for intracerebral hematoma, epidural hematoma, and SDH in 40 patients who developed bilateral pupillary abnormalities during their hospital course. The authors found a significant relationship between the time from onset of bilateral pupillary abnormalities and 6-months outcome. Patients who had surgery more than 3 hours after herniation had a higher morbidity and mortality than those undergoing surgery earlier (mortality, 63% versus 30%).

Most studies focusing on the time between injury and surgery did not find a correlation with outcome (15, 17, 18, 21, 28, 32). Some investigators even reported that early surgery was associated with worse results than delayed surgery (6, 14, 29). As mentioned, this may be related to the fact that most investigators do not control for other variables affecting outcome, such as prehospital hypotension, hypoxia, GCS score, and associated intracranial lesions. In 82 patients undergoing surgery for SDH, Dent et al. (6) found that time to surgery of less than 4 hours was associated with a significantly lower rate of functional outcome when compared with surgery delayed for longer than 4 hours (24% versus 51%). Mortality was approximately 30% in both groups. The authors also found that

patients who underwent surgery within 4 hours were more likely to have obliterated basal cisterns and showed a tendency for lower GCS scores and more associated intracranial injuries, suggesting a more severe TBI.

The only large study with patients with low GCS scores (GCS < 10) that did not find a relationship between early surgery and better outcome was the study by Kotwica and Brzezinski (18). In that study, mortality was approximately 60% in all patients, regardless of whether they had surgery within 4 hours or between 4 and 16 hours after TBI. A detailed analysis reveals that although GCS scores were the same, almost 90% of patients undergoing early surgery had associated intracranial lesions. Associated lesions were found in 78% and 64% of patients surviving the first 5 and 12 hours, respectively. This indicates that patients with early surgery had more severe injuries. In summary, there is evidence that patients who undergo surgery within 2 to 4 hours after clinical deterioration have a better outcome than those who undergo delayed surgery.

### Surgical Technique

Different surgical techniques have been advocated for the evacuation of an SDH. The most commonly used techniques are:

- Twist drill trephination/craniostomy procedures.
- Burr hole trephination.
- Craniotomy with or without dural grafting.
- Subtemporal decompressive craniectomy.
- Large decompressive hemicraniectomy, with or without dural grafting.

Most investigators do not specify the type of surgical treatment used for evacuation of the SDH and, if they do, they usually do not address the effectiveness of the procedure. Except for two studies (14, 30) no papers were found looking at the impact of procedure type on outcome. The choice of operative technique is influenced by the surgeon's expertise, training, and evaluation of the particular situation. Some centers treat all SDH with decompressive craniectomies (18, 23), whereas other centers used solely osteoplastic craniotomies (36). Most studies report a mixture of procedures depending on the clinical and radiographic evaluation (13–15, 17, 38), or combined approaches in the same patient, i.e., subtemporal decompression plus subsequent craniotomy (26) or craniotomies with contralateral decompressive craniectomies in some children (31). One study evaluated decompressive hemicraniectomies for the treatment of selected patients with SDH (30).

Only two investigators addressed the effect of the operative technique on outcome from SDH. Hatashita et al. (14) looked at 3-months GOS in 60 patients with GCS scores between 3 and 15 admitted for SDH evacuation. All patients underwent surgery. The authors performed 24 burr holes, 25 craniotomies, 8 craniotomies with dural grafting, and 3 decompressive craniectomies. In patients with GCS scores between 4 and 6, the authors found a statistically significant increased mortality

and reduced functional recovery rate in patients undergoing burr hole trephination versus craniotomy. Koc et al. (17) compared craniotomy, craniotomy with dural grafting, and decompressive craniectomy in 113 patients with GCS scores between 3 and 15 undergoing SDH evacuation. Seventeen patients underwent decompressive craniectomy and all died. No other significant differences were found between treatment groups. The results of all of these studies have to be viewed with caution because groups undergoing different types of surgical treatment were not comparable.

**SUMMARY**

In patients with an acute SDH, clot thickness or volume and the MLS on the preoperative CT correlate with outcome. In studies analyzing CT parameters that may be predictive for delayed surgery in patients undergoing initial nonoperative management, an MLS greater than 5 mm or a clot thickness greater than 10 mm on the initial CT scan emerged as significant prognostic factors (see *Appendices* for measurement techniques). Therefore, patients with SDH presenting with a clot thickness greater than 10 mm or an MLS greater than 5 mm should undergo surgical evacuation, regardless of their GCS. Patients who present in a coma (GCS < 9) but with an SDH with a thickness less than 10 mm and an MLS less than 5 mm can be treated nonoperatively, providing that they undergo ICP monitoring, they are neurologically stable since the injury, they have no pupillary abnormalities, and they have no intracranial hypertension (ICP > 20 mm Hg). Because of the frequent association of SDH with parenchymal injury, surgical management decisions should take into consideration the recommendations for both lesion types.

**KEY ISSUES FOR FUTURE INVESTIGATION**

- Craniotomy versus decompressive craniectomy and dural grafting for the initial evacuation of SDH. Effect of different prehospital ambulance systems on timing of surgery and outcome from SDH.
- Incidence and impact of prehospital hypotension and hypoxia on outcome from SDH.
- Identification of subgroups that do not benefit from surgery: older patients with low GCS scores, pupillary abnormalities, and associated intracerebral lesions.
- Prospective evaluation of the treatment option for comatose patients (GCS < 9) presented above: does operating on all comatose patients, regardless of their hematoma thickness and MLS lead to a better outcome than following the treatment option presented above.

**REFERENCES**

1. Bowers S, Marshall L: Outcome in 200 consecutive cases of severe head injury treated in San Diego County: A prospective analysis. *Neurosurgery* 6:237-242, 1980.
2. Brain Trauma Foundation: Early indicators of prognosis in severe traumatic brain injury. *J Neurotrauma* 17:535-627, 2000.

3. Cagetti B, Cossu M, Pau A, Rivano C, Viale G: The outcome from acute subdural and epidural intracranial haematomas in very elderly patients. *Br J Neurosurg* 6:227-231, 1992.
4. Cordobes F, Lobato R, Rivas J, Munoz M, Chillon D, Portillo J, Lamas E: Observations on 82 patients with extradural hematoma. Comparison of results before and after the advent of computerized tomography. *J Neurosurg* 54:179-186, 1981.
5. Croce M, Dent D, Menke P, Robertson J, Hinson M, Young B, Donovan T, Pritchard F, Minard G, Kudsk K, Fabian TC: Acute subdural hematoma: Nonsurgical management of selected patients. *J Trauma* 36:820-826, 1994.
6. Dent D, Croce M, Menke P, Young B, Hinson M, Kudsk K, Minard G, Pritchard F, Robertson J, Fabian T: Prognostic factors after acute subdural hematoma. *J Trauma* 39:36-42, 1995.
7. Domenicucci M, Strzelecki J, Delfini R: Acute posttraumatic subdural hematomas: "Intradural" computed tomographic appearance as a favorable prognostic factor. *Neurosurgery* 42:51-55, 1998.
8. Ersahin Y, Mutluer S: Posterior fossa extradural hematomas in children. *Pediatr Neurosurg* 19:31-33, 1993.
9. Espersen J, Petersen O: Computerized tomography (CT) in patients with head injuries. Relation between CT scans and clinical findings in 96 patients. *Acta Neurochir (Wien)* 56:201-217, 1981.
10. Fell D, Fitzgerald S, Moiel R, Caram P: Acute subdural hematomas. Review of 144 cases. *J Neurosurg* 42:37-42, 1975.
11. Gabl M, Mohsenipour I, Benedetto K: Acute posttraumatic subdural hematoma in advanced age [in German]. *Unfallchirurgie* 15:273-278, 1989.
12. Gennarelli T, Spielman G, Langfitt T, Gildenberg P, Harrington T, Jane J, Marshall L, Miller J, Pitts L: Influence of the type of intracranial lesion on outcome from severe head injury. *J Neurosurg* 56:26-32, 1982.
13. Haselsberger K, Pucher R, Auer L: Prognosis after acute subdural or epidural haemorrhage. *Acta Neurochir (Wien)* 90:111-116, 1988.
14. Hatashita S, Koga N, Hosaka Y, Takagi S: Acute subdural hematoma: Severity of injury, surgical intervention, and mortality. *Neurol Med Chir (Tokyo)* 33:13-18, 1993.
15. Howard MA 3rd, Gross AS, Dacey RJ Jr, Winn HR: Acute subdural hematomas: An age-dependent clinical entity. *J Neurosurg* 71:858-863, 1989.
16. Jamjoom A: Justification for evacuating acute subdural haematomas in patients above the age of 75 years. *Injury* 23:518-520, 1992.
17. Koc R, Akdemir H, Oktem I, Meral M, Menku A: Acute subdural hematoma: Outcome and outcome prediction. *Neurosurg Rev* 20:239-244, 1997.
18. Kotwica Z, Brzezinski J: Acute subdural haematoma in adults: An analysis of outcome in comatose patients. *Acta Neurochir (Wien)* 121:95-99, 1993.
19. Kotwica Z, Jakubowski J: Acute head injuries in the elderly. An analysis of 136 consecutive patients. *Acta Neurochir (Wien)* 118:98-102, 1992.
20. Lobato R, Cordobes F, Rivas J, de la Fuente M, Montero A, Barcana A, Perez C, Cabrera A, Lamas E: Outcome from severe head injury related to the type of intracranial lesion. A computerized tomography study. *J Neurosurg* 59:762-774, 1983.
21. Massaro F, Lanotte M, Faccani G, Triolo C: One hundred and twenty-seven cases of acute subdural haematoma operated on. Correlation between CT scan findings and outcome. *Acta Neurochir (Wien)* 138:185-191, 1996.
22. Mathew P, Oluoch-Olunya D, Condon B, Bullock R: Acute subdural haematoma in the conscious patient: Outcome with initial nonoperative management. *Acta Neurochir (Wien)* 121:100-108, 1993.
23. Paterniti S, Fiore P, Macri E, Marra G, Cambria M, Falcone F, Cambria S: Extradural haematoma. Report of 37 consecutive cases with survival. *Acta Neurochir (Wien)* 131:207-210, 1994.
24. Sakas D, Bullock M, Teasdale G: One-year outcome following craniotomy for traumatic hematoma in patients with fixed dilated pupils. *J Neurosurg* 82:961-965, 1995.
25. Schechter W, Peper E, Tuatoo V: Can general surgery improve the outcome of the head-injury victim in rural America? A review of the experience in American Samoa. *Arch Surg* 120:1163-1166, 1985.
26. Seelig J, Becker D, Miller J, Greenberg R, Ward J, Choi S: Traumatic acute subdural hematoma: Major mortality reduction in comatose patients treated within four hours. *N Engl J Med* 304:1511-1518, 1981.
27. Servadei F, Nasi M, Cremonini A, Giuliani G, Cenni P, Nanni A: Importance of a reliable admission Glasgow Coma Scale score for determining the need for evacuation of posttraumatic subdural hematomas: A prospective study of 65 patients. *J Trauma* 44:868-873, 1998.

28. Servadei F, Nasi M, Giuliani G, Cremonini A, Cenni P, Zappi D, Taylor G: CT prognostic factors in acute subdural haematomas: The value of the 'worst' CT scan. *Br J Neurosurg* 14:110-116, 2000.
29. Shenkin H: Acute subdural hematoma. Review of 39 consecutive cases with high incidence of cortical artery rupture. *J Neurosurg* 57:254-257, 1982.
30. Shigemori M, Syojima K, Nakayama K, Kojima T, Ogata T, Watanabe M, Kuramoto S: The outcome from acute subdural haematoma following decompressive hemicraniectomy. *Acta Neurochir (Wien)* 54:61-69, 1980.
31. Spanu G, Pezzotta S, Silvani V, Leone V: Outcome following acute supratentorial subdural hematoma in pediatric age. *J Neurosurg Sci* 29:31-35, 1985.
32. Uzan M, Yentur E, Hanci M, Kaynar MY, Kafadar A, Sarioglu AC, Bahar M, Kunday C: Is it possible to recover from uncal herniation? Analysis of 71 head injured cases. *J Neurosurg Sci* 42:89-94, 1998.
33. van den Brink WA, Zwienerberg M, Zandee SM, van der Meer L, Maas AI, Avezaat CJ: The prognostic importance of the volume of traumatic epidural and subdural haematomas revisited. *Acta Neurochir (Wien)* 141:509-514, 1999.
34. van den Heever CM, van der Merwe DJ: Management of depressed skull fractures. Selective conservative management of nonmissile injuries. *J Neurosurg* 71:186-190, 1989.
35. Wilberger JJ, Harris M, Diamond D: Acute subdural hematoma: Morbidity and mortality related to timing of operative intervention. *J Trauma* 30:733-736, 1990.
36. Wilberger JJ, Harris M, Diamond D: Acute subdural hematoma: Morbidity, mortality, and operative timing. *J Neurosurg* 74:212-218, 1991.
37. Wong CW: Criteria for conservative treatment of supratentorial acute subdural haematomas. *Acta Neurochir (Wien)* 135:38-43, 1995.
38. Yanaka K, Kamezaki T, Yamada T, Takano S, Meguro K, Nose T: Acute subdural hematoma—Prediction of outcome with a linear discriminant function. *Neurol Med Chir (Tokyo)* 33:552-558, 1993.
39. Zumkeller M, Behrmann R, Heissler H, Dietz H: Computed tomographic criteria and survival rate for patients with acute subdural hematoma. *Neurosurgery* 39:708-712, 1996.



### CALL FOR SUBMISSIONS

NEUROSURGERY is now accepting electronic submissions through PEGASUS, our online submission system, at: <http://www.editorialmanager.com/neu>

While we prefer to receive manuscripts through the PEGASUS system, we will continue to accept hard copy submissions via postal mail. Authors should consult the "Author Tutorial" located on the web site listed above for information including but not limited to online registration, and submission instructions. Official *Instructions for Authors*, including updated manuscript submission requirements, are available online through PEGASUS (web site listed above) and the NEUROSURGERY web site (<http://www.neurosurgery-online.com>). Complete versions of the *Instructions for Authors* also appear in the January and July issues of NEUROSURGERY, with abbreviated versions appearing in subsequent issues.

### CALL FOR CLINICAL TRIALS CONTRIBUTIONS

Clinical trials are an increasingly important part of daily neurosurgical practice that can change management paradigms and influence decision-making. Accordingly, NEUROSURGERY is pleased to announce a new *Clinical Trials* section effective January 2006. This section will focus on clinical trial design and comprehensive reviews of trials treating neurosurgically relevant disease processes. Submissions describing results of single or multi-center clinical trials are also encouraged. A future online offering to this section will allow neurosurgeons to list their own clinical trials as part of NEUROSURGERY-Online. We look forward to better informing our readers about clinical trials so that the most recent, validated results can be integrated into daily practice.

Please contact Andrew T. Parsa M.D., Ph.D. directly regarding any questions attendant to this expanding area of NEUROSURGERY'S content and focus at: [parsaa@neurosurg.ucsf.edu](mailto:parsaa@neurosurg.ucsf.edu)

TABLE 1. Surgical management of acute subdural hematomas<sup>a</sup>

Authors (ref. no.)	No. of patients	Inclusion Class	GCS	Treatment	Outcome	Description	Conclusion																																																
Cagetti et al. (3)	26	III	All GCS	All surgery	Early mortality	Retrospective study of 28 patients between 80 and 100 yr of age. 2 patients had an EDH and 26 patients had a SDH.	<table border="0"> <tr> <td colspan="2">Patients older than 80 yr had a 88% mortality. All 19 patients with a GCS between 3 and 9 died.</td> </tr> <tr> <td>GCS</td> <td>No. of patients</td> </tr> <tr> <td>13-15</td> <td>4</td> </tr> <tr> <td>10-12</td> <td>5</td> </tr> <tr> <td>3-9</td> <td>19</td> </tr> <tr> <td colspan="2">Admission GCS, injury severity score, and pupillary reactivity were independent predictors of outcome in the entire patient population.</td> </tr> <tr> <td colspan="2"> <table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>&gt;4 h (%)</td> <td>P value</td> </tr> <tr> <td>24</td> <td>51</td> <td>0.02</td> </tr> <tr> <td>GR/MD</td> <td>19</td> <td></td> </tr> <tr> <td>SD/MS</td> <td>48</td> <td></td> </tr> <tr> <td>D</td> <td>28</td> <td>n.s.</td> </tr> <tr> <td>Open cisterns</td> <td>5</td> <td>0.0004</td> </tr> <tr> <td>Effaced cisterns</td> <td>76</td> <td>0.0002</td> </tr> <tr> <td>GCS</td> <td>7.0 points</td> <td>n.s.</td> </tr> </table> </td> </tr> </table>	Patients older than 80 yr had a 88% mortality. All 19 patients with a GCS between 3 and 9 died.		GCS	No. of patients	13-15	4	10-12	5	3-9	19	Admission GCS, injury severity score, and pupillary reactivity were independent predictors of outcome in the entire patient population.		<table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>&gt;4 h (%)</td> <td>P value</td> </tr> <tr> <td>24</td> <td>51</td> <td>0.02</td> </tr> <tr> <td>GR/MD</td> <td>19</td> <td></td> </tr> <tr> <td>SD/MS</td> <td>48</td> <td></td> </tr> <tr> <td>D</td> <td>28</td> <td>n.s.</td> </tr> <tr> <td>Open cisterns</td> <td>5</td> <td>0.0004</td> </tr> <tr> <td>Effaced cisterns</td> <td>76</td> <td>0.0002</td> </tr> <tr> <td>GCS</td> <td>7.0 points</td> <td>n.s.</td> </tr> </table>		<4 h from TBI to surgery (%)	>4 h (%)	P value	24	51	0.02	GR/MD	19		SD/MS	48		D	28	n.s.	Open cisterns	5	0.0004	Effaced cisterns	76	0.0002	GCS	7.0 points	n.s.										
Patients older than 80 yr had a 88% mortality. All 19 patients with a GCS between 3 and 9 died.																																																							
GCS	No. of patients																																																						
13-15	4																																																						
10-12	5																																																						
3-9	19																																																						
Admission GCS, injury severity score, and pupillary reactivity were independent predictors of outcome in the entire patient population.																																																							
<table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>&gt;4 h (%)</td> <td>P value</td> </tr> <tr> <td>24</td> <td>51</td> <td>0.02</td> </tr> <tr> <td>GR/MD</td> <td>19</td> <td></td> </tr> <tr> <td>SD/MS</td> <td>48</td> <td></td> </tr> <tr> <td>D</td> <td>28</td> <td>n.s.</td> </tr> <tr> <td>Open cisterns</td> <td>5</td> <td>0.0004</td> </tr> <tr> <td>Effaced cisterns</td> <td>76</td> <td>0.0002</td> </tr> <tr> <td>GCS</td> <td>7.0 points</td> <td>n.s.</td> </tr> </table>		<4 h from TBI to surgery (%)	>4 h (%)	P value	24	51	0.02	GR/MD	19		SD/MS	48		D	28	n.s.	Open cisterns	5	0.0004	Effaced cisterns	76	0.0002	GCS	7.0 points	n.s.																														
<4 h from TBI to surgery (%)	>4 h (%)	P value																																																					
24	51	0.02																																																					
GR/MD	19																																																						
SD/MS	48																																																						
D	28	n.s.																																																					
Open cisterns	5	0.0004																																																					
Effaced cisterns	76	0.0002																																																					
GCS	7.0 points	n.s.																																																					
Dent et al. (6)	211	III	All GCS	Surgery and nonsurgical	GOS at a mean of 253 d	Retrospective analysis of factors affecting outcome in 211 patients with SDH. 83 patients underwent surgery and 128 did not. A treatment protocol was not specified.																																																	
Haselberger et al. (13)	111	III	All GCS	All surgery	GOS at 9 mo	A retrospective review of 171 patients who presented with either a subdural (111 patients) or an epidural (60 patients) hematoma. The influence of surgical timing was analyzed in relation to mortality and functional recovery.	<table border="0"> <tr> <td colspan="2">Patients with an acute subdural or epidural hematoma had a lower mortality and improved functional recovery when operated on &lt;2 h after onset of coma.</td> </tr> <tr> <td colspan="2"> <table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>&gt;4 h (%)</td> <td>P value</td> </tr> <tr> <td>24</td> <td>51</td> <td>0.02</td> </tr> <tr> <td>GR/MD</td> <td>19</td> <td></td> </tr> <tr> <td>SD/MS</td> <td>48</td> <td></td> </tr> <tr> <td>D</td> <td>28</td> <td>n.s.</td> </tr> <tr> <td>Open cisterns</td> <td>5</td> <td>0.0004</td> </tr> <tr> <td>Effaced cisterns</td> <td>76</td> <td>0.0002</td> </tr> <tr> <td>GCS</td> <td>7.0 points</td> <td>n.s.</td> </tr> </table> </td> </tr> </table>	Patients with an acute subdural or epidural hematoma had a lower mortality and improved functional recovery when operated on <2 h after onset of coma.		<table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>&gt;4 h (%)</td> <td>P value</td> </tr> <tr> <td>24</td> <td>51</td> <td>0.02</td> </tr> <tr> <td>GR/MD</td> <td>19</td> <td></td> </tr> <tr> <td>SD/MS</td> <td>48</td> <td></td> </tr> <tr> <td>D</td> <td>28</td> <td>n.s.</td> </tr> <tr> <td>Open cisterns</td> <td>5</td> <td>0.0004</td> </tr> <tr> <td>Effaced cisterns</td> <td>76</td> <td>0.0002</td> </tr> <tr> <td>GCS</td> <td>7.0 points</td> <td>n.s.</td> </tr> </table>		<4 h from TBI to surgery (%)	>4 h (%)	P value	24	51	0.02	GR/MD	19		SD/MS	48		D	28	n.s.	Open cisterns	5	0.0004	Effaced cisterns	76	0.0002	GCS	7.0 points	n.s.																				
Patients with an acute subdural or epidural hematoma had a lower mortality and improved functional recovery when operated on <2 h after onset of coma.																																																							
<table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>&gt;4 h (%)</td> <td>P value</td> </tr> <tr> <td>24</td> <td>51</td> <td>0.02</td> </tr> <tr> <td>GR/MD</td> <td>19</td> <td></td> </tr> <tr> <td>SD/MS</td> <td>48</td> <td></td> </tr> <tr> <td>D</td> <td>28</td> <td>n.s.</td> </tr> <tr> <td>Open cisterns</td> <td>5</td> <td>0.0004</td> </tr> <tr> <td>Effaced cisterns</td> <td>76</td> <td>0.0002</td> </tr> <tr> <td>GCS</td> <td>7.0 points</td> <td>n.s.</td> </tr> </table>		<4 h from TBI to surgery (%)	>4 h (%)	P value	24	51	0.02	GR/MD	19		SD/MS	48		D	28	n.s.	Open cisterns	5	0.0004	Effaced cisterns	76	0.0002	GCS	7.0 points	n.s.																														
<4 h from TBI to surgery (%)	>4 h (%)	P value																																																					
24	51	0.02																																																					
GR/MD	19																																																						
SD/MS	48																																																						
D	28	n.s.																																																					
Open cisterns	5	0.0004																																																					
Effaced cisterns	76	0.0002																																																					
GCS	7.0 points	n.s.																																																					
Hatashita et al. (14)	60	III	All GCS	All surgery	GOS at 3 mo	Retrospective analysis of 60 patients and the influence of surgical timing on the outcome from SDH.	<table border="0"> <tr> <td colspan="2">Patients operated on within 4 h of injury had a higher mortality compared with those operated on after 4 h. Patients with a GCS between 4 and 6, who underwent a craniotomy had a significantly better outcome compared with those who were treated with burr holes.</td> </tr> <tr> <td colspan="2"> <table border="0"> <tr> <td>Time from coma onset to surgery</td> <td>No. of patients</td> <td>GR/MD (%)</td> <td>SD/MS (%)</td> <td>D (%)</td> </tr> <tr> <td>&lt;2 h</td> <td>34</td> <td>32</td> <td>21</td> <td>47</td> </tr> <tr> <td>&gt;2 h</td> <td>55</td> <td>4</td> <td>16</td> <td>80</td> </tr> </table> </td> </tr> </table>	Patients operated on within 4 h of injury had a higher mortality compared with those operated on after 4 h. Patients with a GCS between 4 and 6, who underwent a craniotomy had a significantly better outcome compared with those who were treated with burr holes.		<table border="0"> <tr> <td>Time from coma onset to surgery</td> <td>No. of patients</td> <td>GR/MD (%)</td> <td>SD/MS (%)</td> <td>D (%)</td> </tr> <tr> <td>&lt;2 h</td> <td>34</td> <td>32</td> <td>21</td> <td>47</td> </tr> <tr> <td>&gt;2 h</td> <td>55</td> <td>4</td> <td>16</td> <td>80</td> </tr> </table>		Time from coma onset to surgery	No. of patients	GR/MD (%)	SD/MS (%)	D (%)	<2 h	34	32	21	47	>2 h	55	4	16	80																													
Patients operated on within 4 h of injury had a higher mortality compared with those operated on after 4 h. Patients with a GCS between 4 and 6, who underwent a craniotomy had a significantly better outcome compared with those who were treated with burr holes.																																																							
<table border="0"> <tr> <td>Time from coma onset to surgery</td> <td>No. of patients</td> <td>GR/MD (%)</td> <td>SD/MS (%)</td> <td>D (%)</td> </tr> <tr> <td>&lt;2 h</td> <td>34</td> <td>32</td> <td>21</td> <td>47</td> </tr> <tr> <td>&gt;2 h</td> <td>55</td> <td>4</td> <td>16</td> <td>80</td> </tr> </table>		Time from coma onset to surgery	No. of patients	GR/MD (%)	SD/MS (%)	D (%)	<2 h	34	32	21	47	>2 h	55	4	16	80																																							
Time from coma onset to surgery	No. of patients	GR/MD (%)	SD/MS (%)	D (%)																																																			
<2 h	34	32	21	47																																																			
>2 h	55	4	16	80																																																			
Howard et al. (15)	67	III	All GCS	All surgery	GOS at 2 mo	Retrospective analysis of 2 age groups (18-40 yr and >65 yr) of patients with acute SDH.	<table border="0"> <tr> <td colspan="2">Age &gt;65 yr, size of the hematoma, and MLS were related to outcome, but not independently. Size of the hematoma and MLS were greater in the older patient group. Mortality in the older patient group was 74%, in the younger group, it was 18% (P &lt; 0.001).</td> </tr> <tr> <td colspan="2"> <table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>4-10 h, n = 17 (%)</td> </tr> <tr> <td>26</td> <td>41</td> </tr> <tr> <td>GR/MD</td> <td>26</td> </tr> <tr> <td>D</td> <td>63</td> </tr> </table> </td> </tr> </table>	Age >65 yr, size of the hematoma, and MLS were related to outcome, but not independently. Size of the hematoma and MLS were greater in the older patient group. Mortality in the older patient group was 74%, in the younger group, it was 18% (P < 0.001).		<table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>4-10 h, n = 17 (%)</td> </tr> <tr> <td>26</td> <td>41</td> </tr> <tr> <td>GR/MD</td> <td>26</td> </tr> <tr> <td>D</td> <td>63</td> </tr> </table>		<4 h from TBI to surgery (%)	4-10 h, n = 17 (%)	26	41	GR/MD	26	D	63																																				
Age >65 yr, size of the hematoma, and MLS were related to outcome, but not independently. Size of the hematoma and MLS were greater in the older patient group. Mortality in the older patient group was 74%, in the younger group, it was 18% (P < 0.001).																																																							
<table border="0"> <tr> <td>&lt;4 h from TBI to surgery (%)</td> <td>4-10 h, n = 17 (%)</td> </tr> <tr> <td>26</td> <td>41</td> </tr> <tr> <td>GR/MD</td> <td>26</td> </tr> <tr> <td>D</td> <td>63</td> </tr> </table>		<4 h from TBI to surgery (%)	4-10 h, n = 17 (%)	26	41	GR/MD	26	D	63																																														
<4 h from TBI to surgery (%)	4-10 h, n = 17 (%)																																																						
26	41																																																						
GR/MD	26																																																						
D	63																																																						
Jamjoom (16)	27	III	All GCS	All surgery	GOS at 6 mo	A review of 27 patients aged 75 yr or older who required operation for an acute SDH.	<table border="0"> <tr> <td colspan="2">No patient older than 75 yr who preoperatively was extensor posturing, flaccid to pain, or had unilateral or bilateral fixed and dilated pupils made a good recovery (GOS 3-5). Evacuation of acute subdural hematoma is not recommended in this subgroup of patients.</td> </tr> <tr> <td colspan="2"> <table border="0"> <tr> <td>No. of patients</td> <td>GR/MD (%)</td> <td>SD/MS (%)</td> <td>D (%)</td> </tr> <tr> <td>13</td> <td>8</td> <td>92</td> <td></td> </tr> <tr> <td colspan="4">Preoperative deterioration to GCS &lt; 8</td> </tr> <tr> <td colspan="4">Pupils reactive</td> </tr> <tr> <td>15</td> <td>27</td> <td>27</td> <td>46</td> </tr> <tr> <td colspan="4">Uni/bilateral unreactive pupils</td> </tr> <tr> <td>12</td> <td></td> <td></td> <td>100</td> </tr> <tr> <td colspan="4">Admission GCS &gt; 4</td> </tr> <tr> <td>19</td> <td>21</td> <td>16</td> <td>63</td> </tr> <tr> <td colspan="4">Admission GCS &lt; 5</td> </tr> <tr> <td>8</td> <td></td> <td>13</td> <td>87</td> </tr> </table> </td> </tr> </table>	No patient older than 75 yr who preoperatively was extensor posturing, flaccid to pain, or had unilateral or bilateral fixed and dilated pupils made a good recovery (GOS 3-5). Evacuation of acute subdural hematoma is not recommended in this subgroup of patients.		<table border="0"> <tr> <td>No. of patients</td> <td>GR/MD (%)</td> <td>SD/MS (%)</td> <td>D (%)</td> </tr> <tr> <td>13</td> <td>8</td> <td>92</td> <td></td> </tr> <tr> <td colspan="4">Preoperative deterioration to GCS &lt; 8</td> </tr> <tr> <td colspan="4">Pupils reactive</td> </tr> <tr> <td>15</td> <td>27</td> <td>27</td> <td>46</td> </tr> <tr> <td colspan="4">Uni/bilateral unreactive pupils</td> </tr> <tr> <td>12</td> <td></td> <td></td> <td>100</td> </tr> <tr> <td colspan="4">Admission GCS &gt; 4</td> </tr> <tr> <td>19</td> <td>21</td> <td>16</td> <td>63</td> </tr> <tr> <td colspan="4">Admission GCS &lt; 5</td> </tr> <tr> <td>8</td> <td></td> <td>13</td> <td>87</td> </tr> </table>		No. of patients	GR/MD (%)	SD/MS (%)	D (%)	13	8	92		Preoperative deterioration to GCS < 8				Pupils reactive				15	27	27	46	Uni/bilateral unreactive pupils				12			100	Admission GCS > 4				19	21	16	63	Admission GCS < 5				8		13	87
No patient older than 75 yr who preoperatively was extensor posturing, flaccid to pain, or had unilateral or bilateral fixed and dilated pupils made a good recovery (GOS 3-5). Evacuation of acute subdural hematoma is not recommended in this subgroup of patients.																																																							
<table border="0"> <tr> <td>No. of patients</td> <td>GR/MD (%)</td> <td>SD/MS (%)</td> <td>D (%)</td> </tr> <tr> <td>13</td> <td>8</td> <td>92</td> <td></td> </tr> <tr> <td colspan="4">Preoperative deterioration to GCS &lt; 8</td> </tr> <tr> <td colspan="4">Pupils reactive</td> </tr> <tr> <td>15</td> <td>27</td> <td>27</td> <td>46</td> </tr> <tr> <td colspan="4">Uni/bilateral unreactive pupils</td> </tr> <tr> <td>12</td> <td></td> <td></td> <td>100</td> </tr> <tr> <td colspan="4">Admission GCS &gt; 4</td> </tr> <tr> <td>19</td> <td>21</td> <td>16</td> <td>63</td> </tr> <tr> <td colspan="4">Admission GCS &lt; 5</td> </tr> <tr> <td>8</td> <td></td> <td>13</td> <td>87</td> </tr> </table>		No. of patients	GR/MD (%)	SD/MS (%)	D (%)	13	8	92		Preoperative deterioration to GCS < 8				Pupils reactive				15	27	27	46	Uni/bilateral unreactive pupils				12			100	Admission GCS > 4				19	21	16	63	Admission GCS < 5				8		13	87										
No. of patients	GR/MD (%)	SD/MS (%)	D (%)																																																				
13	8	92																																																					
Preoperative deterioration to GCS < 8																																																							
Pupils reactive																																																							
15	27	27	46																																																				
Uni/bilateral unreactive pupils																																																							
12			100																																																				
Admission GCS > 4																																																							
19	21	16	63																																																				
Admission GCS < 5																																																							
8		13	87																																																				

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Class	Inclusion GCS	Treatment	Outcome	Description	Conclusion																																												
Koc et al. (17)	113	III	All GCS	All surgery	GOS at 3 mo	Review of 113 consecutive patients of all age groups operated on for acute SDH.	Preoperative GCS and pupillary exam correlated with functional outcome. Associated intracerebral hematomas, cerebral contusions, and SAH were also related to poorer outcome. Time from injury to surgery did not affect mortality.																																												
						<table border="1"> <tr> <th>No. of patients</th> <th>GR (no.)</th> <th>MD (no.)</th> <th>VS (no.)</th> <th>D (no.)</th> </tr> <tr> <td>37</td> <td>2</td> <td></td> <td></td> <td>35<sup>b</sup></td> </tr> <tr> <td>28</td> <td>4</td> <td>2</td> <td></td> <td>22<sup>b</sup></td> </tr> <tr> <td>25</td> <td>2</td> <td>1</td> <td>11</td> <td>25</td> </tr> <tr> <td>11</td> <td>10</td> <td>1</td> <td></td> <td></td> </tr> <tr> <td>12</td> <td>11</td> <td></td> <td>1</td> <td></td> </tr> <tr> <td>63</td> <td>37</td> <td>3</td> <td>2</td> <td>21</td> </tr> <tr> <td>11</td> <td>1</td> <td>1</td> <td></td> <td>9<sup>c</sup></td> </tr> <tr> <td>39</td> <td></td> <td>1</td> <td></td> <td>38<sup>c</sup></td> </tr> </table>	No. of patients	GR (no.)	MD (no.)	VS (no.)	D (no.)	37	2			35 <sup>b</sup>	28	4	2		22 <sup>b</sup>	25	2	1	11	25	11	10	1			12	11		1		63	37	3	2	21	11	1	1		9 <sup>c</sup>	39		1		38 <sup>c</sup>
No. of patients	GR (no.)	MD (no.)	VS (no.)	D (no.)																																															
37	2			35 <sup>b</sup>																																															
28	4	2		22 <sup>b</sup>																																															
25	2	1	11	25																																															
11	10	1																																																	
12	11		1																																																
63	37	3	2	21																																															
11	1	1		9 <sup>c</sup>																																															
39		1		38 <sup>c</sup>																																															
						<i>b</i> P < 0.05, <i>c</i> P < 0.01																																													
Kowicz and Brzezinski (18)	200	III	GCS < 10	All surgery	GOS at 3 mo	Retrospective analysis of 200 adult patients who underwent surgical evacuation of an acute SDH.	A GCS between 3 and 6, age >50 yr, and MLS > 1.5 cm were associated with poor outcome. There was no difference in outcome between early (less than 4 h) and late surgical evacuation.																																												
						<table border="1"> <tr> <th>No. of patients</th> <th>GR (no.)</th> <th>MD (no.)</th> <th>VS (no.)</th> <th>D (no.)</th> </tr> <tr> <td>166</td> <td>6</td> <td>25</td> <td>7</td> <td>99</td> </tr> <tr> <td>34</td> <td>6</td> <td>2</td> <td>6</td> <td>15</td> </tr> <tr> <td>41</td> <td>5</td> <td>2</td> <td>2</td> <td>16</td> </tr> <tr> <td>96</td> <td>18</td> <td>6</td> <td>10</td> <td>50</td> </tr> <tr> <td>63</td> <td>1</td> <td>4</td> <td>9</td> <td>4</td> </tr> <tr> <td>70</td> <td>16</td> <td>5</td> <td>12</td> <td>29</td> </tr> <tr> <td></td> <td>3</td> <td>4</td> <td>7</td> <td>53</td> </tr> </table>	No. of patients	GR (no.)	MD (no.)	VS (no.)	D (no.)	166	6	25	7	99	34	6	2	6	15	41	5	2	2	16	96	18	6	10	50	63	1	4	9	4	70	16	5	12	29		3	4	7	53					
No. of patients	GR (no.)	MD (no.)	VS (no.)	D (no.)																																															
166	6	25	7	99																																															
34	6	2	6	15																																															
41	5	2	2	16																																															
96	18	6	10	50																																															
63	1	4	9	4																																															
70	16	5	12	29																																															
	3	4	7	53																																															
						Old age is associated with a high mortality from SDH. All 17 patients with a GCS < 9 died.																																													
Kowicz and Jakubowski (19)	27	III	All GCS	All surgery	Not documented	Retrospective analysis of 27 patients >70 yr with SDH undergoing surgery.	Old age is associated with a high mortality from SDH. All 17 patients with a GCS < 9 died.																																												
Massaro et al. (21)	127	III	All GCS	All surgery	GOS at 18 mo	Retrospective analysis of 127 patients undergoing surgery for SDH.	GCS is the most important predictor of outcome.																																												
Mathew et al. (22)	23	III	CCS 13-15	Surgery and nonsurgical	GOS at 3-8 mo	Retrospective analysis of 23 patients with SDH who were initially managed nonoperatively.	6 patients underwent delayed surgery. All patients had a good outcome at 3-8 mo. All patients with a clot >10 mm thick on initial CT scan required surgery.																																												
Sakas et al. (24)	22	III	Comatose	All surgery	GOS at 1 yr	Analysis of 40 severe TBI patients who underwent craniotomy after developing bilateral fixed and dilated pupils.	Patients with SDH had a significant increase in mortality (64%) compared with those with EDH (18%). Patients who underwent delayed (>3 h) surgery had a worse outcome.																																												
						<table border="1"> <tr> <th>Time from pupillary nonreactivity to surgery</th> <th>No. of patients</th> <th>GR/MD (%)</th> <th>SD (%)</th> <th>VS/D (%)</th> </tr> <tr> <td>&lt;3 h</td> <td>20</td> <td>30</td> <td>30</td> <td>40</td> </tr> <tr> <td>&gt;3 h</td> <td>16</td> <td>25</td> <td>12</td> <td>63</td> </tr> </table>	Time from pupillary nonreactivity to surgery	No. of patients	GR/MD (%)	SD (%)	VS/D (%)	<3 h	20	30	30	40	>3 h	16	25	12	63																														
Time from pupillary nonreactivity to surgery	No. of patients	GR/MD (%)	SD (%)	VS/D (%)																																															
<3 h	20	30	30	40																																															
>3 h	16	25	12	63																																															
Servadei et al. (27)	65	III	GCS < 9	Surgery and nonsurgical	GOS at 6 mo	Retrospective analysis of comatose patients with SDH who were treated nonoperatively according to predefined criteria.	15 of 65 comatose patients with SDH were treated nonoperatively and 2 patients required delayed surgery because of increasing ICP and intracerebral hematomas. The authors concluded that nonoperative treatment can be safely used for selected comatose patients with SDH.																																												
Servadei et al. (28)	206	III	All GCS	Surgery and nonsurgical	GOS at 6 mo	Prospective analysis of 206 patients of all age groups presenting with an acute SDH of at least 5 mm thickness. 148 patients underwent operative treatment.	The initial CT scan identifies patients at risk for unfavorable outcome. Hematoma thickness, MLS, status of basal cisterns, and presence of SAH in all patients (surgical and nonsurgical) are related to outcome.																																												
Shigemori et al. (30)	15	III	GCS < 9	All surgery	GOS at 6 mo	Retrospective analysis of 15 patients with SDH undergoing decompressive hemicraniotomy because of rapid neurological deterioration.	10 patients died and 2 patients made a good recovery. Good recovery was only seen in patients with low postoperative ICP. The patients who made a good recovery were both 9 yr old.																																												



TABLE 1. Continued

Authors (ref. no.)	No. of patients	Class	Inclusion GCS	Treatment	Outcome	Description	Conclusion
Uzan et al. (32)	18	III	Comatose	All surgery	GOS at 6 mo	Prospective study of 71 patients with EDH, SDH, and intracerebral hemorrhage, of all age groups operated on for signs of uncal herniation.	Timing of surgery did not affect outcome. GCS correlated with GOS.
van den Brink et al. (33)	91	III	All GCS	All surgery	GOS at 6 mo	Retrospective analysis of the CT scan parameters in 91 patients with acute SDH.	Volume of the subdural blood did not correlate with outcome. Subarachnoid blood and pupillary dysfunction were the only significant parameters correlating with a poor outcome.  SD/VS/D (%) Bilateral abnormal pupils GCS motor score < 4 GCS motor score > 3
Wilberger et al. (35)	101	III	GCS < 9	All surgery	GCS after 18 mo	Retrospective analysis of 101 comatose patients of all age groups with acute SDH.	A significant increase in mortality was associated with GCS 3 or 4, age greater than 65 yr, ICP greater than 45 mm Hg and evacuation of hematoma >12 h after injury.  No. of patients 101 Time from injury to operation Age (yr) <35 35-50 51-65 65 No. of patients 35 21 17 28 GR/MD (%) 280 ± 26 min 374 ± 31 min D n.s. P value 54.3 61.9 70.6 82.1 % GR/MD 5 10 18 44 % D 90 76 62 51 P value P < 0.05 P < 0.05 n.s. n.s.
Wong (37)	31	III	All GCS	Surgery and nonsurgical	GCS at 2 wk to 5 yr	Retrospective analysis of 31 adult patients (1 child) of 300 patients with SDH who were initially treated nonoperatively. Patients required delayed surgery for neurologic deterioration. No difference was found in outcome.	MLs > 5 mm (only in patients with GCS < 15) and thickness of the hematoma > 10 mm on the initial CT scan are significantly related to the failure of nonoperative treatment. Hematoma volume was not predictive.
Yanaka et al. (38)	170	III	All GCS	Surgery and nonsurgical	GOS at 3 mo	A retrospective study on 170 patients of all age groups with SDH admitted during 7 yr, treated either surgically or nonsurgically. A strict protocol was followed and patients with MLs > 5 mm underwent surgery.	77 patients underwent surgery. Prognostic indicators of outcome for the whole group of patients were: GCS, pupils, age, hematoma size, MLS, clot thickness, associated contusions, SAH, status of basal cisterns, and ICP.
Zumkeller et al. (39)	174	III	All GCS	All surgery	Postoperative mortality	Retrospective analysis of 174 patients operated on for acute SDH.	SDH thickness of less than 1 cm or midline shift less than 12 mm are well tolerated, with high survival rates. Mortality climbs steeply when MLS surpasses clot thickness.

<sup>a</sup> GCS, Glasgow Coma Scale; EDH, epidural hematoma; SDH, subdural hematoma; GOS, Glasgow outcome score; TBI, traumatic brain injury; GR, good recovery; MD, moderate disability; SD, severe disability; VS, vegetative state; D, death; n.s., not significant; MLS, midline shift; SAH, subarachnoid hemorrhage; ICP, intracranial pressure; CT, computed tomographic.

**M. Ross Bullock, M.D., Ph.D.**

Department of Neurological Surgery,  
Virginia Commonwealth University  
Medical Center,  
Richmond, Virginia

**Randall Chesnut, M.D.**

Department of Neurological Surgery,  
University of Washington  
School of Medicine,  
Harborview Medical Center,  
Seattle, Washington

**Jamshid Ghajar, M.D., Ph.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David Gordon, M.D.**

Department of Neurological Surgery,  
Montefiore Medical Center,  
Bronx, New York

**Roger Hartl, M.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David W. Newell, M.D.**

Department of Neurological Surgery,  
Swedish Medical Center,  
Seattle, Washington

**Franco Servadei, M.D.**

Department of Neurological Surgery,  
M. Bufalini Hospital,  
Cesena, Italy

**Beverly C. Walters, M.D., M.Sc.**

Department of Neurological Surgery,  
New York University  
School of Medicine,  
New York, New York

**Jack Wilberger, M.D.**

Department of Neurological Surgery,  
Allegheny General Hospital,  
Pittsburgh, Pennsylvania

**Reprints requests:**

Jamshid Ghajar, M.D., Ph.D.,  
Brain Trauma Foundation,  
523 East 72nd Street,  
New York, NY 10021.  
Email: ghajar@braintrauma.org

## SURGICAL MANAGEMENT OF TRAUMATIC PARENCHYMAL LESIONS

### RECOMMENDATIONS

(see *Methodology*)

#### Indications

- Patients with parenchymal mass lesions and signs of progressive neurological deterioration referable to the lesion, medically refractory intracranial hypertension, or signs of mass effect on computed tomographic (CT) scan should be treated operatively.
- Patients with Glasgow Coma Scale (GCS) scores of 6 to 8 with frontal or temporal contusions greater than 20 cm<sup>3</sup> in volume with midline shift of at least 5 mm and/or cisternal compression on CT scan, and patients with any lesion greater than 50 cm<sup>3</sup> in volume should be treated operatively.
- Patients with parenchymal mass lesions who do not show evidence for neurological compromise, have controlled intracranial pressure (ICP), and no significant signs of mass effect on CT scan may be managed nonoperatively with intensive monitoring and serial imaging.

#### Timing and Methods

- Craniotomy with evacuation of mass lesion is recommended for those patients with focal lesions and the surgical indications listed above, under Indications.
- Bifrontal decompressive craniectomy within 48 hours of injury is a treatment option for patients with diffuse, medically refractory posttraumatic cerebral edema and resultant intracranial hypertension.
- Decompressive procedures, including subtemporal decompression, temporal lobectomy, and hemispheric decompressive craniectomy, are treatment options for patients with refractory intracranial hypertension and diffuse parenchymal injury with clinical and radiographic evidence for impending transtentorial herniation.

**KEY WORDS:** Coma, Computed tomographic parameters, Craniotomy, Decompressive craniectomy, Head injury, Herniation, Intracranial pressure monitoring, Parenchymal mass lesion, Surgical technique, Timing of surgery, Traumatic brain injury

*Neurosurgery* 58:S2-25-S2-46, 2006

DOI: 10.1227/01.NEU.0000210365.36914.E3

www.neurosurgery-online.com

### OVERVIEW

Traumatic parenchymal mass lesions are common sequelae of traumatic brain injury (TBI), occurring in up to 8.2% of all TBI (37) and 13 to 35% of severe TBI, (6, 35, 44, 47, 57, 58) and comprising as much as 20% of operative intracranial lesions in representative series (44, 68). Most small parenchymal lesions do not require surgical evacuation (18, 19, 57). However, the development of mass effect from larger lesions may result in secondary brain injury, placing the patient at risk of further neu-

rological deterioration, herniation, and death (6). Because parenchymal lesions tend to evolve, (36, 54, 55, 58) and because timing of surgery with respect to the occurrence of neurological deterioration clearly affects outcome (41), much effort has been directed at defining patients at risk of progressive neurological compromise as a result of their traumatic injuries (6, 50). Becker et al. (3) advocated early evacuation of traumatic intracranial hematomas and contusions for the purpose of avoiding secondary complications. A selective approach was envisioned by Galbraith and Teasdale (18), who stated, "if those

who are going to deteriorate could be identified soon after the hematoma has been detected they could be operated on immediately without incurring the risks of delay, and the remainder would be spared unnecessary operation." The literature addressing these issues in the CT era is reviewed here in an attempt to define appropriate surgical indications, methods, and timing for the patient with traumatic parenchymal injuries.

Although the majority of relevant data retrospectively addresses prognostic variables, differential classification, and clinical course of parenchymal lesions, several studies attempt to assess the efficacy of surgical intervention in a retrospective, historically controlled fashion. Only one randomized, controlled trial has been published (61). In addition, the topics of delayed traumatic intracerebral hematoma (DTICH) and decompressive operations for diffuse parenchymal injury and persistent intracranial hypertension deserve specific consideration, and are also addressed here.

### PROCESS

A MEDLINE computer search using the following key words: "intracerebral" or "intraparenchymal" or "ICH" or "IPH" and "hematoma" or "hematoma" or "hemorrhage" and "surgery" or "craniotomy" or "craniectomy" or "burr hole" or "craniostomy" and "trauma" or "traumatic" or "TBI" or "CHI" between 1975 and 2001 and limited to humans was performed. A total of 330 documents were found. An additional search using the following key words: "brain" or "cortex" and "laceration" between 1975 and 2001 was performed, yielding 101 additional articles. This search was narrowed to include the following key words: "surgery" or "operative" or "craniotomy" or "craniectomy" or "decompressive craniectomy" or "repair" and "outcome," yielding 49 articles. A third search using the following key words: "contusion" or "hemorrhagic contusion" and "brain" and "surgery" or "craniotomy" or "craniectomy" or "burr hole" or "craniostomy" was performed, yielding 174 articles. A fourth search, using the key words "DTICH" or "DTIPH," yielded 11 articles. A fifth search, using the key word "TICH," yielded eight articles. All five searches were combined. Duplicates between searches were discarded. A total of 495 references resulted. In addition, the reference lists of selected articles were reviewed, and a total of 51 articles were selected for critical analysis. The results of this analysis were incorporated into the review presented here. Papers primarily addressing the following topics were not included: nontraumatic lesions (e.g., spontaneous intraparenchymal hemorrhage or infarction), patients with other associated nontraumatic lesions (e.g., tumors or arteriovenous malformations), posttraumatic aneurysms, chronic lesions, penetrating trauma, carotid-cavernous fistulae, patients undergoing anticoagulation therapy, patients with associated illnesses (e.g., acquired immunodeficiency syndrome; concomitant infection; hemophilia; thrombotic thrombocytopenic purpura; or hemolysis, elevated liver enzymes, and low platelet count), pregnant patients, birth trauma, traumatic intraventricular hemorrhage, traumatic hy-

drocephalus, external ventricular drainage, sagittal sinus injury, pre-CT era reports, and book chapters. Papers with the following characteristics were also excluded: case series with fewer than 10 patients evaluated by CT scan and with incomplete outcome data (mortality or Glasgow outcome score [GOS]), case reports, operative series with operations occurring greater than 14 days from injury. Posterior fossa parenchymal injuries are addressed in *Surgical Management of Posterior Fossa Mass Lesions*. Selected articles were evaluated for design, prognostic significance, therapeutic efficacy, and overall outcome. In addition, several articles were reviewed for the purposes of historical perspective.

### SCIENTIFIC FOUNDATION

#### Traumatic Parenchymal Lesions

Traumatic parenchymal lesions are a heterogeneous group, and are traditionally divided into focal and nonfocal lesions. Focal lesions include intracerebral hematomas (ICH), DTICH, contusions, and infarctions. Nonfocal lesions include cerebral edema, hemispheric swelling, and diffuse injury. Although these lesions often do not occur in isolation, their presence has been shown to adversely affect prognosis (16, 44). Indeed, Fearnside et al. (16) prospectively collected data on 315 severely head-injured patients and found that "the model which provided the most accurate prediction of poor outcome included age, hypotension and three different CT characteristics: subarachnoid blood, ICH or intracerebral contusion (accuracy 72.5%)." Parenchymal lesions have been further subclassified by multiple authors, and outcome has clearly been shown to differ among lesion types (19, 35, 39–41, 44, 65). Marshall (39) demonstrated that CT-defined injury type was a highly significant independent predictor of mortality, even when age and GCS motor score were included in the predictive model. Given the heterogeneity of the pathophysiology and prognostic significance of "parenchymal" lesions, the task of defining clear surgical indications and methods becomes difficult.

#### Prognostic Factors

Despite proven differences among lesion types, outcome within the broad category of "parenchymal" lesions correlates with known prognostic variables of TBI in general (5). These include age (26, 44, 45, 56, 58, 66, 70), admission or postresuscitation GCS (6, 19, 23, 40, 44, 45, 49, 51, 58), presence of cranial fracture (56), presence of pupillary response/brainstem reflexes (7, 44), respiratory insufficiency (8), ICP (6, 7, 23, 39, 44, 50, 51), and the status of the basal cisterns (6, 41, 62) or third ventricle (6, 41, 62) on CT scan. Moreover, other variables significantly correlate with outcome. These include location of the lesion (2, 32, 50, 58), ICH volume (8, 63), GCS at time of follow-up CT (63), lowest recorded GCS (7), severity of surrounding edema (6), timing of surgery (41, 51, 53), occurrence of preoperative neurological deterioration (41), and presence of acute hemispheric swelling or concomitant subdural hematoma (7, 35). Although their study included nontraumatic lesions, Andrews et al.

(2) showed that patients with a temporal or temporoparietal ICH of 30 cm<sup>3</sup> or greater, as defined by product of anteroposterior, mediolateral, and superoinferior diameters on CT scan, were significantly more likely to develop signs of brainstem compression or tentorial herniation, implying that these patients should undergo early evacuation of the offending mass lesion. However, these prognostic variables alone do not define the patient who should undergo operative intervention.

### Operative Indications

As stated above, in Overview, several studies have focused on defining the patient at risk for subsequent neurological deterioration, making the assumption that operative intervention for this patient will improve the likelihood of a more favorable outcome. Predictors of failure of nonoperative management (defined by subsequent neurological deterioration and need for craniotomy) include lesion location (50), intracranial hypertension (6, 18, 50), presence of subarachnoid hemorrhage (41), cisternal effacement (41), lesion volume (41), and hypoxic events (41).

Bullock et al. (6) prospectively studied 85 patients with ICH whose initial need for craniotomy was uncertain. These patients underwent ICP monitoring in an attempt to better define the need for surgical intervention. The authors then retrospectively reviewed the CT scans of those patients for whom ICP monitoring failed to predict late deterioration and, thus, the need for ICH evacuation. With multiple linear regression analysis, they found the peak ICP to be the strongest predictor of outcome. However, ICP monitoring failed to predict late deterioration or death secondary to high ICP in 5 of 30 patients who did not undergo initial surgery. After critical analysis of CT factors, they concluded that the decision to operate "should be based on a spectrum of clinical, CT scanning and ICP findings". CT and clinical predictors included cisternal status, edema severity, and admission GCS. Interestingly, the authors found that the weight of each predictor depended on the location of the ICH. For temporoparietal lesions, hematoma size, degree of edema, GCS, basal cistern status, and ICP data correlated with outcome. However, for frontal lesions, peak ICP alone was predictive of outcome. These findings expand on those reported by Gallbraith and Teasdale (18), who found that all patients with "intradural" lesions (including subdural hematoma, ICH, and "burst lobes") and sustained ICP greater than 30 mm Hg, versus only one patient with an ICP less than 20 mm Hg, required operative intervention, as defined by clinical deterioration or failure to improve in the setting of increased ICP.

Mathiesen et al. (41) reviewed data collected prospectively for the Head Injury Trial-2 nimodipine trial on 218 TBI patients not obeying commands within 24 hours of injury. These authors found that the initial CT characteristics of presence of subarachnoid hemorrhage, presence of focal lesion with volume greater than 40 cm<sup>3</sup>, and compressed or absent cisterns were associated with neurological deterioration, defined as a fall in GCS by 2 points or from 4 to 3, or as the development of pupillary dilation.

They also found that the incidence of secondary (greater than 5 d after injury) deterioration was associated with the occurrence of hypoxic events. The occurrence of neurological deterioration, in turn, was found to adversely affect outcome from craniotomy, suggesting that patients with factors strongly associated with neurological deterioration should be considered for early surgery (i.e., before the onset of neurological deterioration). The authors directly demonstrated that a subgroup of patients with admission GCS of at least 6 and focal lesion volume of at least 20 cm<sup>3</sup> who underwent surgery without previous neurological deterioration had significantly better outcomes compared with those who either did not undergo surgery or who underwent surgery after deterioration. Furthermore, if a radiological sign of mass effect (i.e., compression or obliteration of the cisterns and/or a midline shift  $\geq$  5 mm) was present, craniotomy significantly improved the outcome in this group. Craniotomy was also directly shown to improve outcome in a small subgroup of patients with admission GCS of at least 10, temporal contusions, and a radiological sign of mass effect (i.e., a midline shift and/or compression or obliteration of the basal cisterns). Additionally, patients admitted with a GCS of at least 6 and a lesion volume of at least 50 cm<sup>3</sup> had better outcome with surgery before or immediately after deterioration than without surgery or with delayed operation.

Although the goal of identifying those patients who are likely to deteriorate neurologically is paramount, the question of whether surgery itself is beneficial remains unanswered. Few studies compare surgical outcome with matched, nonsurgically managed controls. In a retrospective study of 21 patients with frontal lobe contusions and medically intractable intracranial hypertension (>40 mm Hg), mortality was significantly decreased in the surgical group compared with a nonsurgical historical group (22% versus 88%, respectively) (28). These patients were matched for age, sex, GCS, and ICP levels, although statistics for these variables are not provided by the authors. Choksey et al. (8) retrospectively reviewed 202 patients with traumatic ICH and showed, with logistic regression analysis, that craniotomy significantly improved the probability of good outcome. Factors taken into consideration for this analysis included low GCS and hematoma volume greater than 16 cm<sup>3</sup>, each of which independently predicted poor outcome in these patients. Several other studies examine outcome relative to specific decompressive procedures, and are discussed in the surgical treatment section.

For the purposes of CT classification, Marshall (39) defined a "mass lesion" as a lesion of volume greater than 25 cm<sup>3</sup>. They showed differential outcome between patients with evacuated and nonevacuated mass lesions (23% versus 11% favorable outcome, respectively) in a series of 746 severe TBI patients (i.e., after resuscitation, GCS  $\leq$  8). In contrast, a recent paper from the European Brain Injury Consortium (54) evaluating a series of 724 TBI patients with a GCS of 3 to 12 showed a 45% rate of favorable results in evacuated mass lesions versus 42% in nonevacuated mass lesions using the same classification system. Sample size between these two studies was noticeably different: the former series included 276 patients with evacu-

ated mass lesions and 36 with nonevacuated mass lesions, whereas the latter included 195 and 148 patients, respectively. These studies reviewed illustrate that a classification system based solely on lesion volume is unable to consistently show the relationship between surgery and outcome. Surgical indications are, in fact, related to many factors, including CT parameters (i.e., volume, midline shift, and basal cistern compression), clinical status, and the occurrence of clinical deterioration, among others.

One fundamental problem in using initial CT parameters as independent indications for surgery is that CT pathology has clearly been shown to be a dynamic process. Using the Traumatic Coma Data Bank classification system (39), Lobato et al. (36) showed that 51.2% of 587 severely injured patients (GCS  $\leq$  8) developed significant changes between initial and "control" CT scans, the latter of which more accurately predicted outcome. Similarly, Servadei et al. (54) showed that 16% of moderately-to-severely injured patients (GCS of 3–12) with diffuse injury showed radiological progression across Traumatic Coma Data Bank classes. Yamaki et al. (69) showed that only 56% of ICH greater than 3 cm in diameter developed within 6 hours of injury, and that only 84% of ICH reached maximal size within 12 hours. These studies highlight the dynamic nature of parenchymal injuries and the dangers inherent in placing too much emphasis on a single, static CT scan for management decisions.

The data reviewed shows that there are subpopulations of patients with traumatic intraparenchymal lesions that will benefit from surgical intervention. However, the precise characteristics of these subpopulations are not, as yet, clearly defined. The literature supports taking into account an amalgam of clinical and radiographic criteria, including GCS, location, volume, CT appearance, ICP, and the presence of neurological deterioration, to make an informed decision to subject a patient with a parenchymal lesion to a craniotomy. It seems that all factors must be taken into consideration to best define the patient population that will benefit from surgery.

### DTICH

ICH have been shown to evolve over time (55, 58, 69). The entity of DTICH was initially described by Bollinger in 1891 (4), and is now defined by most authors as occurring in areas of radiographically normal brain in patients with otherwise abnormal initial CT scans (20, 22, 59, 63). It is defined by Gentleman et al. (20) as a "lesion of increased attenuation developing after admission to hospital, in a part of the brain which the admission CT scan had suggested was normal". Other authors, however, have noted DTICH to occur in areas of contusion on initial, high-resolution CT scan (72). The incidence of DTICH ranges from 3.3 to 7.4% in patients with moderate-to-severe TBI (15, 20, 22, 29, 59, 63). Evacuated DTICH represent approximately 1.6% of all evacuated traumatic ICH (20), and mortality ranges from 16 to 72% (15, 20, 22, 59, 63). Therefore, the importance of careful monitoring and of serial CT scanning cannot be overemphasized (55, 63, 72).

In a retrospective review of 32 patients with DTICH, Tseng (63) found that greater lesion volume, cisternal compression, earlier timing of appearance, occurrence of clinical deterioration, and lower GCS at time of a second, follow-up CT adversely affected outcome. Mortality occurred only with clinical presentation of DTICH within 48 hours of injury. In this small series, no patient with DTICH requiring craniotomy presented after 72 hours of injury. Sprick et al. (59) similarly found that approximately 70% of clinically significant DTICH presented within 48 hours of injury. Additionally, DTICH has been shown to be significantly associated with an increased incidence of secondary, systemic insults (22), an increased incidence after decompressive surgery for other mass lesions (22), and an increased incidence of abnormal clotting parameters (29), suggesting a complex etiology for this lesion beyond the mechanical disruption of the parenchyma (22, 27).

Although DTICH is most likely a distinct pathophysiological entity, it is still a parenchymal lesion from which many patients either fail to recover or clinically deteriorate. The findings of Mathiesen et al. (41) indicate that patients with intracerebral lesions undergoing surgery before neurological deterioration have improved outcomes. It follows logically that a subset of DTICH patients would benefit from rapid surgical intervention after discovery of the lesion. However no CT-era studies have critically examined surgical outcome. Because the majority of studies show that all patients who develop clinically relevant DTICH have abnormal initial CT scans (20, 63, 72), it is essential that patients with initially abnormal scans undergo intensive monitoring and serial imaging to ensure rapid intervention, if necessary.

### Surgical Methods

The standard surgical treatment of focal lesions, such as intracerebral hemorrhages or contusions, is craniotomy with evacuation of the lesion. Location of the lesion and proximity to critical structures are considerations when contemplating the choice of surgical options. Evacuation of traumatic mass lesions is often effective in amelioration of brain shift and reduction of ICP, and can decrease the requirement for intensive medical treatment. Other methods, such as stereotactic evacuation of focal mass lesions, have also been used, although much less commonly (12). These procedures, however, become less effective when the patient's intracranial pathology is diffuse and involves intracranial hypertension as a result of posttraumatic edema or hemispheric swelling—factors known to be associated with poor outcome (6, 19, 35, 38, 44, 51). Even with focal ICH, a significant proportion of patients have medically intractable intracranial hypertension after standard craniotomy, and these patients fare the worst (44).

### Surgical Treatment of Intracranial Hypertension

#### Rationale

A variety of operations have been developed for, or applied to, decompression of the brain at risk for the sequelae of traumati-

cally elevated ICP. These include Cushing's subtemporal decompression (13), temporal lobectomy (34, 48), manual reduction of the temporal lobe (64), circumferential craniotomy (9), and the more widely used hemispheric decompressive craniectomy (52) and bifrontal decompressive craniectomy (30). The rationale for such an approach is supported from a physiological perspective by both human and animal studies. Particular attention has been directed at the study of changes in ICP and CT scan features, such as cisternal effacement and midline shift, after decompression, with the goal of correlating significant postoperative reduction of these parameters with improved outcome.

Hatashita and Hoff (25) showed that decompressive frontoparietal craniectomy in cats led to significant reduction in ICP, reduction in cortical gray and white matter tissue pressure, increased pressure-volume index, and increased tissue compliance. The authors found that craniectomy significantly increased the volumetric compensatory capacity of the intracranial cavity, a finding consistent with those of Hase et al. (24), in which ventricular catheter ICP measurements in 47 severe TBI patients, 33 of whom underwent external decompression, documented a dramatic increase in intracranial compliance after decompression. Yoo et al. (71) studied intraoperative ventricular pressures in a cohort of 20 patients with refractory intracranial hypertension after both traumatic and nontraumatic insults who underwent bilateral frontotemporoparietal decompressive craniectomy with dural expansion and grafting. These authors found a  $50.2 \pm 16.6\%$  reduction of initial ICP after craniectomy, and a further reduction to  $15.7 \pm 10.7\%$  of initial ICP after dural opening.

Polin et al. (51) showed a significant decrease in ICP after bifrontal decompressive craniectomy, as well as a significant difference in postoperative ICP when compared with ICP measured 48 to 72 hours after injury in a cohort of historically matched controls. Gower et al. (21) found that 7 of 10 patients who underwent subtemporal decompression for medically refractory intracranial hypertension had an average decrease in ICP of 34%. Kunze et al. (33) found a reduction in mean ICP from 41.7 to 20.6 mm Hg in 28 patients after unilateral or bilateral decompressive craniectomy for posttraumatic edema refractory to maximal medical therapy. And Whitfield et al. (67) demonstrated a significant reduction of ICP from 37.5 to 18.1 mm Hg ( $P = 0.003$ ) in 26 patients who underwent bifrontal decompressive craniectomy for refractory intracranial hypertension managed using a standardized ICP/cerebral perfusion pressure (CPP) treatment algorithm. The amplitude of the ICP waveform and of slow waves were significantly reduced, and compensatory reserve was significantly increased postoperatively in a subgroup of eight patients in this study. Munch et al. (45), however, failed to show a significant postoperative reduction in ICP or increase in CPP after unilateral hemispheric decompression. From a radiological perspective, however, this group found a significant improvement in the visibility of mesencephalic cisterns and a significant decrease in midline shift after decompressive craniectomy—both known to correlate with improved outcome. Furthermore, the change in cistern visibility correlated with the distance between the lower craniectomy border and cranial base. Additionally, Alexander et al. (1) found an average increase in

intracranial volume of 26 cm<sup>3</sup> in an analysis of CT scans in patients with traumatic and nontraumatic lesions undergoing subtemporal decompression. In contrast, experimental evidence from Cooper et al. (10) supports the notion that decompressive procedures may aggravate cerebral edema formation, thus, resulting in increased secondary injury. Such studies may help explain why, despite laboratory success, actual improvement in patient outcome has not been consistently demonstrated.

### *Procedures and Outcome*

There are several studies that suggest an important role for decompressive procedures in the management of parenchymal injury. In a retrospective review of 28 patients undergoing unilateral or bilateral decompressive craniectomy for posttraumatic edema and intracranial hypertension refractory to head elevation, moderate hyperventilation, osmодиuretics, barbiturates, tromethamine, and cerebrospinal fluid drainage, 57% of patients had good outcome or moderate disability at 1 year. However, the authors excluded patients with "vast" primary lesions, hypoxia, ischemic infarction, brainstem injury, "central" herniation, and primary anisocoria, thus, biasing results toward favorable recovery (33). Nussbaum et al. (48) showed that complete temporal lobectomy performed within 2 hours of the development of clinical signs of transtentorial herniation in 10 patients with unilateral hemispheric swelling and a GCS of less than 7 resulted in 40% functional independence. All patients demonstrated displacement of the brainstem, compression of the contralateral peduncle, and progressive obliteration of the parasellar and interpeduncular cisterns on CT scan, along with fixed pupillary dilation, and therefore, represent a particularly compromised patient population.

Guerra et al. (23) prospectively performed decompressive craniectomy following a standardized treatment protocol with standardized surgical technique for posttraumatic diffuse brain swelling in 57 severe TBI patients (GCS, 4–6). Thirty-nine of these patients underwent primary decompression, and 18 others underwent secondary decompression because of persistent intracranial hypertension after evacuation of a surgical mass lesion. Intracranial hypertension in these patients was refractory to a standardized medical protocol that included hemodynamic stabilization, head elevation, sedation with or without muscle relaxation, controlled hyperventilation to an arterial carbon dioxide pressure of 28 to 32 mm Hg, mannitol, tromethamine for acute rises in ICP, and electroencephalogram burst suppression with barbiturates. Fifty-eight percent of the first group and 65% of the second group experienced good outcome or moderate disability at 1 year. These results compare favorably with published outcomes of alternative second-tier therapy. However, a direct comparison with matched nonsurgical controls was not performed. Whitfield et al. (67) demonstrated a favorable outcome (GOS, 4–5) in 61% of a severe TBI population that underwent bifrontal decompressive craniectomy for ICP greater than 30 mm Hg with CPP less than 70 mm Hg, or for ICP greater than 35 mm Hg, irrespective of CPP, despite a medical management pro-

protocol that involved head elevation, propofol sedation, mannitol and/or hypertonic saline, normocarbia, mild hypothermia to 33°C to 35°C, and electroencephalogram burst suppression. Only 55% of eligible patients, however, underwent craniectomy, and the reasons for nonsurgical management despite eligibility were not documented or discussed.

Munch et al. (45) were able to show a significant difference in outcome between patients undergoing rapid decompressive craniectomy and those undergoing delayed decompressive craniectomy. However, the nature of the lesions differed between the two groups, and, thus, the outcomes cannot be meaningfully compared. Tseng (64) noted that the addition of gentle elevation of the temporal lobe until the tentorium was visualized and CSF egress was noted decreased mortality and improved the incidence of good outcome when compared with a standard craniotomy with hematoma evacuation and contusion resection in a series of 32 severe TBI patients with anisocoria, hemiparesis, and CT evidence for uncal herniation. However, no statistical analysis was performed, and the author notes that preoperative selection was biased and that operative timing and intraoperative judgements may have influenced the choice of surgical procedure. Gower et al. (21) retrospectively studied 115 patients with admission GCS of 8 or less who had adequate ICP monitoring data and no operative mass lesion on admission. These patients were managed under a standard treatment protocol. Outcome was compared between 10 patients who underwent subtemporal decompression and 17 patients managed by induction of pentobarbital coma. They found that subtemporal decompression afforded significantly lower mortality than pentobarbital coma, despite the fact that the surgical group had a lower (but not statistically significant) average admission GCS. However, 10 of the patients treated medically had been determined not to be operative candidates and subsequently died, greatly biasing results in favor of the operative group. In a retrospective review of 29 patients undergoing operation for a combination of acute subdural hematoma and severe contusion and swelling of the temporal lobe with uncal herniation, Lee et al. (34) documented a significant improvement in outcome with the addition of temporal lobectomy to subtemporal decompression and debridement of contused brain. Mortality decreased from 56% to 8%, with a concomitant increase in average GOS from 2.2 to 4.0. These two surgical groups did not differ with respect to preoperative GCS, age, or sex. However, patients with intraoperative "overswelling," defined as herniation of brain more than 2 cm above the craniectomy window, were excluded and underwent decompressive craniectomy with dural expansion, thus, potentially biasing these results.

Coplin et al. (11) retrospectively reviewed 29 consecutive patients with GCS of at most 9 and CT scans with a midline shift greater than the volume of a surgically amenable lesion to evaluate the safety and feasibility of decompressive craniectomy with duraplasty versus traditional craniotomy as the initial surgical procedure. No significant differences in age, gender, admission GCS, time to surgery, or serum ethanol concentration existed between the two groups. Despite a sig-

nificantly lower GCS at time of surgery and significantly greater percentage of Diffuse Injury III and IV injuries (39), there was no significant difference in mortality, GOS, acute hospital length of stay, functional independence measure score on admission to a rehabilitation unit, change in functional independence measure score, or length of rehabilitation stay between craniectomy and craniotomy groups. Although this study is subject to the biases inherent in a retrospective, uncontrolled design, it strongly supports the safety of decompressive craniectomy as a first-line surgical intervention as opposed to its traditional role as a salvage procedure.

The studies briefly outlined here support the potential usefulness of decompressive procedures, but are clearly hampered by inherent biases.

The study by Polin et al. (51) deserves particular mention, because it offers more concrete evidence that a decompressive procedure may result in improved patient outcome. These authors report a retrospective evaluation of outcome in 35 patients undergoing bifrontal decompressive craniectomy for refractory posttraumatic cerebral edema matched for age, admission GCS, sex, and maximal ICP with historical controls selected from the Traumatic Coma Data Bank. Only patients with Diffuse Injury III (39) were eligible as controls. Highest postoperative ICP less than 24 hours after surgery in cases was matched with highest ICP 48 to 72 hours after injury in controls. Preoperative ICP in cases was matched with highest ICP less than 48 hours after injury in controls. Several findings are particularly relevant. In the operative group, surgery performed less than 48 hours after injury was significantly associated with favorable outcome when compared with surgery performed longer than 48 hours after injury (46% versus 0%, respectively). Medical management alone carried a 3.8 times relative risk of unfavorable outcome compared with decompressive craniectomy. Maximum benefit was achieved in patients undergoing decompression within 48 hours of injury and whose ICP elevations had not yet been sustained above 40 mm Hg (60% favorable outcome versus 18% in matched controls). This study argues strongly in favor of bifrontal decompressive craniectomy for patients with medically refractory posttraumatic cerebral edema and resultant intracranial hypertension not yet sustained above 40 mm Hg within 48 hours of injury, but does not have contemporaneous controls (51).

Overall, the literature suggests, but does not prove, that decompressive procedures may be the intervention of choice given the appropriate clinical context. A recent study by Taylor et al. (61) examined the use of early (median 19.2 h after injury) bitemporal craniectomy in addition to intensive medical management versus intensive medical management alone in 27 children with sustained intracranial hypertension in a prospective, randomized, controlled fashion. Their results showed a trend towards greater improvement in ICP, less time required in the intensive care unit, and improved outcome with surgical decompression. These trends, although promising, did not reach statistical significance. Additional prospective, controlled studies are, thus, needed to strengthen the argument for the use of surgical de-

compression in the management of intracranial hypertension and refractory cerebral edema.

SUMMARY

The majority of studies regarding surgical treatment of parenchymal lesions are case series. Only one prospective clinical trial of treatment using surgical versus nonsurgical management has been published (61). The majority of evidence indicates that the development of parenchymal mass lesions, which are associated with progressive neurological dysfunction, medically refractory intracranial hypertension, or radiological signs of mass effect, are associated with a poor outcome if treated nonsurgically. Specific surgical criteria, however, have not been firmly established.

Evidence also suggests that decompressive craniectomy may be the procedure of choice in patients with posttraumatic edema, hemispheric swelling, or diffuse injury, given the appropriate clinical context. This context has yet to be defined.

KEY ISSUES FOR FUTURE INVESTIGATION

The majority of studies on traumatic parenchymal lesions is observational, and the studies offer no means to meaningfully compare outcome between surgical and nonsurgical groups. Those studies that attempt this comparison fail to adequately control for known prognostic variables between surgically and nonsurgically managed groups in a prospective fashion. Prospective, controlled trials, such as that of Taylor et al. (61) need to be pursued and supported to define appropriate clinical criteria and surgical methods.

Additionally, the data of Coplin et al. (11) and Taylor et al. (61) strongly support the feasibility of performing trials of decompressive operations in the first instance, as opposed to a second- or third-tier therapy for posttraumatic intracranial hypertension.

REFERENCES

1. Alexander E, Ball M, Laster D: Subtemporal decompression: Radiological observations and current surgical experience. *Br J Neurosurg* 1:427-433, 1987.
2. Andrews BT, Chiles BW, Olsen WL, Pitts LH: The effect of intracerebral hematoma location on the risk of brain-stem compression and on clinical outcome. *J Neurosurg* 69:518-522, 1988.
3. Becker D, Miller J, Ward J, Greenberg R, Young H, Sakalas R: The outcome from severe head injury with early diagnosis and intensive management. *J Neurosurg* 47:491-502, 1977.
4. Bollinger O: Uber traumatische Spat-Apoplexie: Ein Beitrag zur Lehre der Hirnerschutterung, in *Internationale Beitrage zur Wissenschaftlichen Medizin*. Berlin: Festschrift, Rudolf Virchow, A Hirschwald, 1891, vol 2, pp 457-470.
5. Brain Trauma Foundation: Early indicators of prognosis in severe traumatic brain injury. *J Neurotrauma* 17:535-627, 2000.
6. Bullock R, Golek J, Blake G: Traumatic intracerebral hematoma—Which patients should undergo surgical evacuation? CT scan features and ICP monitoring as a basis for decision making. *Surg Neurol* 32:181-187, 1989.

7. Caroli M, Locatelli M, Campanella R, Balbi S, Martinelli F, Arienti C: Multiple intracranial lesions in head injury: Clinical considerations, prognostic factors, management, and results in 95 patients. *Surg Neurol* 56:82-88, 2001.
8. Choksey M, Crockard HA, Sandilands M: Acute traumatic intracerebral haematomas: Determinants of outcome in a retrospective series of 202 cases. *Br J Neurosurg* 7:611-622, 1993.
9. Clark K, Nash TM, Hutchison GC: The failure of circumferential craniotomy in acute traumatic cerebral swelling. *J Neurosurg* 29:367-371, 1968.
10. Cooper PR, Hagler H, Clark WK, Barnett P: Enhancement of experimental cerebral edema after decompressive craniectomy: Implications for the management of severe head injuries. *Neurosurgery* 4:296-300, 1979.
11. Coplin WM, Cullen NK, Policherla PN, Vinas FC, Wilseck JM, Zafonte RD, Rengachary SS: Safety and feasibility of craniectomy with duraplasty as the initial surgical intervention for severe traumatic brain injury. *J Trauma* 50:1050-1059, 2001.
12. Coraddu M, Floris F, Nurchi G, Meleddu V, Lobina G, Marcucci M: Evacuation of traumatic intracerebral haematomas using a simplified stereotactic procedure. *Acta Neurochir (Wien)* 129:6-10, 1994.
13. Cushing H: Subtemporal decompressive operations for the intracranial complications associated with bursting fractures of the skull. *Ann Surg* 47:641-644, 1908.
14. De Luca GP, Volpin L, Fornezza U, Cervellini P, Zanusso M, Casentini L, Curri D, Piacentino M, Bozzato G, Colombo F: The role of decompressive craniectomy in the treatment of uncontrollable posttraumatic intracranial hypertension. *Acta Neurochir* 76 [Suppl]:401-404, 2000.
15. Diaz FG, Yock DH, Larson D, Rockswold GL: Early diagnosis of delayed posttraumatic intracerebral hematomas. *J Neurosurg* 50:217-223, 1979.
16. Fearnside MR, Cook RJ, McDougall P, McNeil RJ: The Westmead Head Injury Project outcome in severe head injury. A comparative analysis of pre-hospital, clinical and CT variables. *Br J Neurosurg* 7:267-279, 1993.
17. Gaab M, Rittierodt M, Lorenz M, Heissler H: Traumatic brain swelling and operative decompression: A prospective investigation. *Acta Neurochir (Wien)* 51 [Suppl]:326-328, 1990.
18. Gallbraith S, Teasdale G: Predicting the need for operation in the patient with an occult traumatic intracranial hematoma. *J Neurosurg* 55:75-81, 1981.
19. Gennarelli T, Spielman G, Langfitt T, Gildenberg P, Harrington T, Jane J, Marshall L, Miller J, Pitts L: Influence of the type of intracranial lesion on outcome from severe head injury. *J Neurosurg* 56:26-32, 1982.
20. Gentleman D, Nath F, Macpherson P: Diagnosis and management of delayed traumatic intracerebral haematomas. *Br J Neurosurg* 3:367-372, 1989.
21. Gower D, Lee K, McWhorter J: Role of subtemporal decompression in severe closed head injury. *Neurosurgery* 23:417-422, 1988.
22. Gudeman S, Kishore P, Miller J, Girevendulis A, Lipper M, Becker D: The genesis and significance of delayed traumatic intracerebral hematoma. *Neurosurgery* 5:309-313, 1979.
23. Guerra W, Gaab M, Dietz H, Mueller J, Piek J, Fritsch M: Surgical decompression for traumatic brain swelling: Indications and results. *J Neurosurg* 90:187-196, 1999.
24. Hase U, Reulen HJ, Meinig G, Schurmann K: The influence of the decompressive operation on the intracranial pressure and the pressure-volume relation in patients with severe head injuries. *Acta Neurochir (Wien)* 45:1-13, 1978.
25. Hatashita S, Hoff JT: The effect of craniectomy on the biomechanics of normal brain. *J Neurosurg* 67:573-578, 1987.
26. Jamjoom A: The influence of concomitant intradural pathology on the presentation and outcome of patients with acute traumatic extradural hematoma. *Acta Neurochir (Wien)* 115:86-89, 1992.
27. Katayama Y, Tsubokawa T, Miyazaki S: Two types of delayed traumatic intracerebral hematoma: Differential forms of treatment. *Neurosurg Rev* 12:231-236, 1989.
28. Katayama Y, Tsubokawa T, Miyazaki S, Kawamata T, Yoshino A: Oedema fluid formation within contused brain tissue as a cause of medically uncontrollable elevation of intracranial pressure: The role of surgical therapy. *Acta Neurochir (Wien)* 51 [Suppl]:308-310, 1990.



29. Kaufman HH, Moake JL, Olson JD, Miner ME, duCret, RP, Pruessner JL, Gildenberg PL: Delayed and recurrent intracranial hematomas related to disseminated intravascular clotting and fibrinolysis in head injury. *Neurosurgery* 7:445-449, 1980.
30. Kjellberg RN, Prieto A: Bifrontal decompressive craniotomy for massive cerebral edema. *J Neurosurg* 34:488-493, 1971.
31. Kotwica Z, Jakubowski J: Acute head injuries in the elderly. An analysis of 136 consecutive patients. *Acta Neurochir (Wien)* 118:98-102, 1992.
32. Kumchev Y, Dimitrov Z, Kalnev B, Argirov S: Traumatic intracerebral hematomas—Diagnostic and therapeutic problem. *Folia Med (Plovdiv)* 40: 52-57, 1998.
33. Kunze E, Meixensberger J, Janka M, Sorensen N, Roosen K: Decompressive craniectomy in patients with uncontrollable intracranial hypertension. *Acta Neurochir (Wien)* 71 [Suppl]:16-18, 1998.
34. Lee EJ, Chio CC, Chen HH: Aggressive temporal lobectomy for uncocal herniation in traumatic subdural hematoma. *J Formos Med Assoc* 94:341-345, 1995.
35. Lobato R, Cordobes F, Rivas J, de la Fuente M, Montero, A, Barcena A, Perez C, Cabrera A, Lamas E: Outcome from severe head injury related to the type of intracranial lesion. A computerized tomography study. *J Neurosurg* 59:762-774, 1983.
36. Lobato RD, Gomez PA, Alday R, Rivas JJ, Dominguez J, Cabrera A, Turanzas FS, Benitez A, Rivero B: Sequential computerized tomography changes and related final outcome in severe head injury patients. *Acta Neurochir (Wien)* 139:385-391, 1997.
37. Manderam M, Zralek C, Krawczyk I, Zycinski A, Wencel T, Bazowski P: Surgery or conservative treatment in children with traumatic intracerebral haematoma. *Childs Nerv Syst* 15:267-269, 1999.
38. Marmarou A, Anderson RL, Ward JD, Choi SC, Young HF, Eisenberg HM, Foulkes MA, Marshall LF, Jane JA: Impact of ICP instability and hypotension on outcome in patients with severe head trauma. *J Neurosurg* 75 [Suppl]:S59-S66, 1991.
39. Marshall L: A new classification of head injury based on computerized tomography. *J Neurosurg* 75 [Suppl]:S14-S20, 1991.
40. Marshall L, Gaultille T, Klauber M, Eisenberg H, Jane J, Luerssen T, Marmarou A, Foulkes M: The outcome of severe closed head injury. *J Neurosurg* 75 [Suppl]:S28-S36, 1991.
41. Mathiesen T, Kakarieka A, Edner G: Traumatic intracerebral lesions without extracerebral haematoma in 218 patients. *Acta Neurochir (Wien)* 137:155-163, 1995.
42. Meier U, Heinitz A, Kintzel D: Surgical outcome after severe craniocerebral trauma in childhood and adulthood. A comparative study [in German]. *Unfallchirurg* 97:406-409, 1994.
43. Meier U, Zeilinger FS, Henzka O: The use of decompressive craniectomy for the management of severe head injuries. *Acta Neurochir* 76 [Suppl]:475-478, 2000.
44. Miller JD, Butterworth JF, Gudeman SK, Faulkner JE, Choi SC, Selhorst JB, Harbison JW, Lutz HA, Young HF, Becker DP: Further experience in the management of severe head injury. *J Neurosurg* 54:289-299, 1981.
45. Munch E, Horn P, Schurer L, Piepgras A, Paul T, Schmiedek P: Management of severe traumatic brain injury by decompressive craniectomy. *Neurosurgery* 47:315-322, 2000.
46. Nagabhand A, Sangcham K: The study of traumatic intracerebral hematoma at Buri Ram Hospital. *J Med Assoc Thai* 76:399-404, 1993.
47. Nordstrom C, Messeter K, Sundborg G, Wahlander S: Severe traumatic brain lesions in Sweden. Part I: Aspects of management in non-neurosurgical clinics. *Brain Inj* 3:247-265, 1989.
48. Nussbaum E, Wolf A, Sebring L, Mirvis S: Complete temporal lobectomy for surgical resuscitation of patients with transtentorial herniation secondary to unilateral hemispheric swelling. *Neurosurgery* 29:62-66, 1991.
49. Papo I, Caruselli G, Luongo A, Scarpelli M, Pasquini U: Traumatic cerebral mass lesions: Correlations between clinical, intracranial pressure, and computed tomographic data. *Neurosurgery* 7:337-346, 1980.
50. Patel N, Hoyt D, Nakaji P, Marshall L, Holbrook T, Coimbra R, Winchell R, Mikulaschek A: Traumatic brain injury: Patterns of failure of nonoperative management. *J Trauma* 48:367-374, 2000.
51. Polin R, Shaffrey M, Bogaev C, Tisdale N, Germanson T, Bocchicchio B, Jane J: Decompressive bifrontal craniectomy in the treatment of severe refractory posttraumatic cerebral edema. *Neurosurgery* 41:84-92, 1997.
52. Ransohoff J, Benjamin MV, Gage EL Jr, Epstein F: Hemispheric craniectomy in the management of acute subdural hematoma. *J Neurosurg* 34:70-76, 1971.
53. Sakas D, Bullock M, Teasdale G: One-year outcome following craniotomy for traumatic hematoma in patients with fixed dilated pupils. *J Neurosurg* 82:961-965, 1995.
54. Servadei F, Murray GD, Penny K, Teasdale GM, Dearden M, Iannotti F, Lapierre F, Maas AJ, Karimi A, Ohman J, Persson L, Stocchetti N, Trojanowski T, Unterberg A: The value of the "worst" computed tomographic scan in clinical studies of moderate and severe head injury. European Brain Injury Consortium. *Neurosurgery* 46:70-75, 2000.
55. Servadei F, Nanni A, Nasi M, Zappi D, Vergoni G, Giuliani G, Arista A: Evolving brain lesions in the first 12 hours after head injury: Analysis of 37 comatose patients. *Neurosurgery* 37:899-906, 1995.
56. Servadei F, Piazza GC, Padovani R, Fagioli L, Gaist G: "Pure" traumatic cerebral lacerations. A review of 129 cases with long-term follow-up. *Neurochirurgia (Stuttg)* 28:170-173, 1985.
57. Singounas E: Severe head injury in a paediatric population. *J Neurosurg Sci* 36:201-206, 1992.
58. Soloniuk D, Pitts LH, Lovely M, Bartkowski H: Traumatic intracerebral hematomas: Timing of appearance and indications for operative removal. *J Trauma* 26:787-794, 1986.
59. Sprick C, Bettag M, Bock W: Delayed traumatic intracranial hematomas—Clinical study of seven years. *Neurosurg Rev* 12 [Suppl 1]:228-230, 1989.
60. Statham PF, Johnston RA, Macpherson P: Delayed deterioration in patients with traumatic frontal contusions. *J Neurol Neurosurg Psychiatry* 52:351-354, 1989.
61. Taylor A, Butt W, Rosenfeld J, Shann F, Ditchfield M, Lewis E, Klug G, Wallace D, Herning R, Tibballs J: A randomized trial of very early decompressive craniectomy in children with traumatic brain injury and sustained intracranial hypertension. *Childs Nerv Syst* 17:154-162, 2001.
62. Teasdale E, Cardoso E, Galbraith S, Teasdale G: CT scan in severe diffuse head injury: Physiological and clinical correlations. *J Neurol Neurosurg Psychiatry* 47:600-603, 1984.
63. Tseng SH: Delayed traumatic intracerebral hemorrhage: A study of prognostic factors. *J Formos Med Assoc* 91:585-589, 1992.
64. Tseng SH: Reduction of herniated temporal lobe in patients with severe head injury and uncocal herniation. *J Formos Med Assoc* 91:24-28, 1992.
65. Uzzell BP, Dolinskas CA, Wisner RF, Langfitt TW: Influence of lesions detected by computed tomography on outcome and neuropsychological recovery after severe head injury. *Neurosurgery* 20:396-402, 1987.
66. Vollmer D, Torner J, Jane J, Sadovnic B, Charlebois D, Eisenberg H, Foulkes M, Marmarou A, Marshall L: Age and outcome following traumatic coma: Why do older patients fare worse? *J Neurosurg* 75:537-49, 1991.
67. Whitfield PC, Patel H, Hutchinson PJ, Czosnyka M, Parry D, Menon D, Pickard JD, Kirkpatrick PJ: Bifrontal decompressive craniectomy in the management of posttraumatic intracranial hypertension. *Br J Neurosurg* 15:500-507, 2001.
68. Wu J, Hsu C, Liao S, Wong Y: Surgical outcome of traumatic intracranial hematoma at a regional hospital in Taiwan. *J Trauma* 47:39-43, 1999.
69. Yamaki T, Hirakawa K, Ueguchi T, Tenjin H, Kuboyama T, Nakagawa Y: Chronological evaluation of acute traumatic intracerebral haematoma. *Acta Neurochir (Wien)* 103:112-115, 1990.
70. Yamaura A, Uemura K, Makino H: Large decompressive craniectomy in management of severe cerebral contusion. A review of 207 cases. *Neurol Med Chir (Tokyo)* 19:717-728, 1979.
71. Yoo DS, Kim DS, Cho KS, Huh PW, Park CK, Kang JK: Ventricular pressure monitoring during bilateral decompression with dural expansion. *J Neurosurg* 91:953-959, 1999.
72. Young HA, Gleave JR, Schmiedek HH, Gregory S: Delayed traumatic intracerebral hematoma: Report of 15 cases operatively treated. *Neurosurgery* 14:22-25, 1984.
73. Zumkeller M, Hollerhage HG, Proschl M, Dietz H: The results of surgery for intracerebral hematomas. *Neurosurg Rev* 15:33-36, 1992.

TABLE 1. Surgical Management of Traumatic Parenchymal Lesions\*

Authors (ref. no.)	No. of patients	Inclusion Class	GCS	Treatment	Outcome	Description	Conclusion
Bullock et al. (6)	85	III	Mean GCS 9	Nonsurg/cal	GOS 3 mo	Prospective study of 85 patients with ICH with uncertainty regarding need for immediate craniotomy. These patients were selected for ICP monitoring in an attempt to better define need for craniotomy. A retrospective analysis of CT scan characteristics was then performed for those patients for whom ICP monitoring did not predict late deterioration to create a better predictive model of those in need of ICH evacuation.	<p>● Management "should be based on a spectrum of clinical, CT scanning, and ICP findings."</p> <p>● Peak ICP was the strongest predictor of outcome, but failed to predict late deterioration or death in 16.7%.</p> <p>● For temporal/parietal lesions, hematoma size, degree of edema, GCS, basal cistern status, and peak ICP correlated with outcome; for frontal lesions, peak ICP alone predicted outcome.</p> <p>● Premenopausal cistern status falsely predicted low ICP in only 2.3% of patients with elevated ICP (false negative).</p> <p>● Authors conclude:</p> <ol style="list-style-type: none"> <li>1) Basal cistern effacement on initial CT scan is indication for surgery, regardless of GCS.</li> <li>2) Extensive surrounding edema should prompt ICP monitoring or serial CT scanning, if cisterns are effaced, evacuation should be considered.</li> <li>3) ICP monitoring should be carried out in patients in coma.</li> </ol>
Caroli et al. (7)	36	III	Average GCS 9.3	Surgery and nonsurgical	GOS 6 mo	Retrospective review of 95 patients with multiple traumatic intracranial lesions to identify clinical factors and radiologic predictors of prognosis to establish management criteria. Patients were divided into 3 groups based on predominant lesion type; 36 patients with pure ICH as the predominant lesion were included in this analysis. Patients with parenchymal lesions <2 cm were excluded by the authors.	<p>● Cisterns (<math>P &lt; 0.01</math>), shift (<math>P &lt; 0.05</math>), prolonged increased ICP (<math>P &lt; 0.0001</math>) differed significantly between surgical and nonsurgical groups.</p> <p>● 80.6% neurologically worsened within 1.2–2.4 h of initial assessment.</p> <p>● 44.4% incidence of DTICH.</p> <p>● Type of lesion (SDH + ICH), lowest recorded GCS, presence of prolonged increased ICP, absence of pupillary reflexes were significant predictors of bad outcome (VS/D) based on multiple regression analysis.</p> <p>● Decompressive craniectomy was a "useful means of controlling intracranial pressure in 19/22 patients undergoing procedure."</p>
Choksey et al. (8)	202	III	All GCS	Surgery and nonsurgical	GOS 6 mo	Retrospective study of 202 patients with traumatic ICH to ascertain factors influencing outcome.	<p>● Hematoma volume &gt;16 cm<sup>3</sup> increased probability of clinical deterioration.</p> <p>● Respiratory insufficiency, low GCS and hematoma volume &gt;16 cm<sup>3</sup> were significantly associated with a poor outcome.</p> <p>● With these factors taken into consideration, craniotomy significantly improved probability of good outcome.</p> <p>● Location of peripheral hematomas did not predict outcome.</p>

	No. of patients	GR/minimal disability (%)	MD (%)	VS (%)	D (%)
Surgery	55	47	20	33	30
No initial surgery	30	47	23	30	30

	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)
ICH	36	33.3	38.9	16.7	0	11.1
Operated					13.3	
Nonoperated	21					9.5

	No. of patients	GR/MD (%)	SD/VS/D (%)
1 Hematoma	169	58	42
2 Hematomas	19	21	79
≥ 3 Hematomas	14	0	100
Hypoxia, respiratory insufficiency	35	20	80
No respiratory insufficiency	167	57	43
Evacuation	84	62	38
Nonevacuation	118	42	58
Age 0–20 yr	56	29	71
Age 21–40 yr	54	67	33
Age 41–64 yr	44	68	32
Age ≥65 yr	48	42	58
GCS 3–7	112	31	69
GCS 8–11	45	62	38

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion																																																								
Coplin et al. (11)	29	III GCS 3-9	Decompressive craniectomy	GOS at discharge; FIM at admission and discharge from rehabilitation	Retrospective review of 29 consecutive patients with GCS $\leq$ 9 and CT scans with midline shift greater than the volume of a surgically amenable lesion to evaluate the safety and feasibility of decompressive craniectomy with duraplasty versus traditional craniotomy as the initial surgical procedure. Exclusion criteria included age < 16 yr, injury > 24 h before admission, Diffuse Injury II (TCDB classification), isolated EDH, preexisting illness limiting life expectancy to < 1 yr from ictus, age > 40 yr with extensor posturing, and bilateral unreactive pupils $\geq$ 4 mm in diameter.	<ul style="list-style-type: none"> <li>• No significant difference between craniotomy and craniectomy groups with regards to age (<math>P = 0.8</math>), gender (<math>P = 1.0</math>), admission GCS score (<math>P = 0.07</math>), time to surgery (<math>P = 1.0</math>), serum ethanol concentration (<math>P = 0.6</math>).</li> <li>• Craniectomy group had significantly lower GCS score at surgery (<math>P = 0.04</math>).</li> <li>• Craniectomy group had significantly more patients with Diffuse Injury III and IV (56% versus 9%).</li> <li>• No significant difference between groups with respect to mortality, GOS, acute hospital length of stay, initial FIM score, change in FIM score, or length of rehabilitation stay.</li> <li>• Despite significantly lower GCS at time of surgery and worse injury classification by CT scan, decompressive craniectomy with duraplasty afforded equivalent outcome to traditional craniotomy, establishing its safety and feasibility as a first-line surgical intervention as opposed to its traditional role as a "salvage" procedure.</li> <li>• Authors emphasize importance of craniectomy extending down to the floor of the middle cranial fossa at the root of the zygoma.</li> </ul>																																																								
						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>CR/MD (%)</th> <th>SD/VS/D (%)</th> </tr> </thead> <tbody> <tr> <td>GCS 12-15</td> <td>45</td> <td>87</td> <td>13</td> </tr> <tr> <td>Volume 1-5 cm<sup>3</sup></td> <td>26</td> <td>81</td> <td>19</td> </tr> <tr> <td>Volume 6-15 cm<sup>3</sup></td> <td>58</td> <td>57</td> <td>43</td> </tr> <tr> <td>Volume 16-35 cm<sup>3</sup></td> <td>67</td> <td>46</td> <td>54</td> </tr> <tr> <td>Volume &gt;36 cm<sup>3</sup></td> <td>51</td> <td>33</td> <td>67</td> </tr> <tr> <td>Neurological deterioration</td> <td>135</td> <td>47</td> <td>53</td> </tr> <tr> <td>No neurological deterioration</td> <td>49</td> <td>80</td> <td>20</td> </tr> <tr> <td>Initial GCS &lt; 4</td> <td>20</td> <td>0</td> <td>100</td> </tr> <tr> <td>Frontal</td> <td>93</td> <td>48</td> <td>52</td> </tr> <tr> <td>Parietal</td> <td>12</td> <td>50</td> <td>50</td> </tr> <tr> <td>Temporal</td> <td>63</td> <td>63</td> <td>37</td> </tr> <tr> <td>Occipital</td> <td>9</td> <td>67</td> <td>33</td> </tr> <tr> <td>Central</td> <td>25</td> <td>20</td> <td>80</td> </tr> </tbody> </table>		No. of patients	CR/MD (%)	SD/VS/D (%)	GCS 12-15	45	87	13	Volume 1-5 cm <sup>3</sup>	26	81	19	Volume 6-15 cm <sup>3</sup>	58	57	43	Volume 16-35 cm <sup>3</sup>	67	46	54	Volume >36 cm <sup>3</sup>	51	33	67	Neurological deterioration	135	47	53	No neurological deterioration	49	80	20	Initial GCS < 4	20	0	100	Frontal	93	48	52	Parietal	12	50	50	Temporal	63	63	37	Occipital	9	67	33	Central	25	20	80
	No. of patients	CR/MD (%)	SD/VS/D (%)																																																											
GCS 12-15	45	87	13																																																											
Volume 1-5 cm <sup>3</sup>	26	81	19																																																											
Volume 6-15 cm <sup>3</sup>	58	57	43																																																											
Volume 16-35 cm <sup>3</sup>	67	46	54																																																											
Volume >36 cm <sup>3</sup>	51	33	67																																																											
Neurological deterioration	135	47	53																																																											
No neurological deterioration	49	80	20																																																											
Initial GCS < 4	20	0	100																																																											
Frontal	93	48	52																																																											
Parietal	12	50	50																																																											
Temporal	63	63	37																																																											
Occipital	9	67	33																																																											
Central	25	20	80																																																											
						<table border="1"> <thead> <tr> <th></th> <th>All</th> <th>Craniotomy</th> <th>Craniectomy</th> <th>P value</th> </tr> </thead> <tbody> <tr> <td>No. of patients</td> <td>29</td> <td>17</td> <td>12</td> <td></td> </tr> <tr> <td>Discharge GOS</td> <td>4</td> <td>4</td> <td>4</td> <td>0.8</td> </tr> <tr> <td>Mortality (%)</td> <td>34.5</td> <td>41.2</td> <td>25</td> <td>0.4</td> </tr> <tr> <td>Hospital length of stay (d)</td> <td>17</td> <td>18</td> <td>18</td> <td>0.4</td> </tr> <tr> <td>Admission FIM</td> <td>63</td> <td>64</td> <td>38</td> <td>0.7</td> </tr> <tr> <td>Discharge FIM</td> <td>90</td> <td>92</td> <td>71</td> <td>0.6</td> </tr> <tr> <td>Change in FIM</td> <td>27</td> <td>27</td> <td>23</td> <td>1.0</td> </tr> </tbody> </table>		All	Craniotomy	Craniectomy	P value	No. of patients	29	17	12		Discharge GOS	4	4	4	0.8	Mortality (%)	34.5	41.2	25	0.4	Hospital length of stay (d)	17	18	18	0.4	Admission FIM	63	64	38	0.7	Discharge FIM	90	92	71	0.6	Change in FIM	27	27	23	1.0																
	All	Craniotomy	Craniectomy	P value																																																										
No. of patients	29	17	12																																																											
Discharge GOS	4	4	4	0.8																																																										
Mortality (%)	34.5	41.2	25	0.4																																																										
Hospital length of stay (d)	17	18	18	0.4																																																										
Admission FIM	63	64	38	0.7																																																										
Discharge FIM	90	92	71	0.6																																																										
Change in FIM	27	27	23	1.0																																																										
Coraddu et al. (12)	37	III GCS 3-5 to >7	Stereotactic evacuation	Mortality at discharge	Retrospective series of 37 patients with traumatic ICH treated by stereotactic evacuation through an enlarged burr hole to evaluate outcome using this technique. Surgical indications included coma, deteriorating consciousness, or focal neurological deficit.	<ul style="list-style-type: none"> <li>• No mortality with admission GCS &gt; 7.</li> <li>• Admission GCS related inversely to mortality (no statistics).</li> <li>• Temporal location had higher mortality (no statistics).</li> </ul>																																																								
						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>ICH, total</td> <td>37</td> <td>30</td> </tr> <tr> <td>GCS 3-5</td> <td>10</td> <td>80</td> </tr> <tr> <td>GCS 6-7</td> <td>12</td> <td>25</td> </tr> <tr> <td>GCS &gt; 7</td> <td>15</td> <td>0</td> </tr> <tr> <td>Frontal</td> <td>18</td> <td>5.5</td> </tr> <tr> <td>Temporal</td> <td>18</td> <td>55</td> </tr> </tbody> </table>		No. of patients	D (%)	ICH, total	37	30	GCS 3-5	10	80	GCS 6-7	12	25	GCS > 7	15	0	Frontal	18	5.5	Temporal	18	55																																			
	No. of patients	D (%)																																																												
ICH, total	37	30																																																												
GCS 3-5	10	80																																																												
GCS 6-7	12	25																																																												
GCS > 7	15	0																																																												
Frontal	18	5.5																																																												
Temporal	18	55																																																												

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion																																																																																																															
De Luca et al. (14)	22	III GCS 3-12	Decompressive craniectomy	GOS at discharge	Retrospective review of 22 patients who underwent decompressive craniectomy for cerebral edema with refractory intracranial hypertension or for "imminent herniation" during the evacuation of a space-occupying traumatic lesion. Surgical methods are described.	<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR/MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>22</td> <td>41</td> <td>18</td> <td>23</td> <td>18</td> </tr> </tbody> </table>	No. of patients	GR/MD (%)	SD (%)	VS (%)	D (%)	22	41	18	23	18																																																																																																					
No. of patients	GR/MD (%)	SD (%)	VS (%)	D (%)																																																																																																																	
22	41	18	23	18																																																																																																																	
Diaz et al. (15)	9	III Unknown	Surgery	Mortality at discharge	Retrospective series of 9 patients with DTICH to evaluate clinical characteristics and outcome.	<ul style="list-style-type: none"> <li>• 55.6% mortality.</li> <li>• Included with n = 9 for well-presented data on DTICH.</li> <li>• Definition of DTICH: 1) history of head injury with head in motion producing transient or permanent LOC, focal neurological findings, or cranial fracture; 2) interval between injury and development of DTICH of &lt;2 wk.</li> </ul> <table border="1"> <thead> <tr> <th>No. of patients</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>9</td> <td>55.6</td> </tr> </tbody> </table>	No. of patients	D (%)	9	55.6																																																																																																											
No. of patients	D (%)																																																																																																																				
9	55.6																																																																																																																				
Gaab et al. (17)	37	III Unknown	Decompressive craniectomy	GOS 1 yr	Prospective series of 37 patients who underwent decompressive craniectomy because of traumatic brain swelling to assess efficacy of treatment protocol at 1 yr after trauma. Surgical indications and exclusion criteria detailed.	<ul style="list-style-type: none"> <li>• 14.7% mortality using standardized protocol.</li> <li>• Initial GCS <math>\geq</math> 6, day 1 GCS <math>\geq</math> 8 associated with better outcome (no statistics).</li> <li>• All patients with GOS 1-2 had GCS 3 with pupillary abnormality.</li> <li>• Represents early report of Guerra et al. study.</li> </ul>																																																																																																															
Gennarelli et al. (19)	1107	III GCS 3-8	Surgery and nonsurgical	GOS 3 mo	Multicenter prospective series of 1107 patients with severe blunt traumatic brain injury (GCS $\leq$ 8) to examine differences in type of injury, severity of injury, and GOS at 3 mo. Subgroups of patients with "other focal injury" (n = 205) and "diffuse injury" (n = 487) were examined for this chapter. Of note, "other focal injury" included ICH, contusions, but also depressed cranial fracture, SDH, and EDH that were not the primary lesions, and combined EDH/SDH in which the most important lesion was unknown.	<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>205</td> <td>21</td> <td>18</td> <td>19</td> <td>3</td> <td>39</td> </tr> </tbody> </table> <table border="1"> <thead> <tr> <th>No. of patients</th> <th>Full (%)</th> <th>Disabled (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>34</td> <td>41.2</td> <td>35.3</td> <td>8.8</td> <td>14.7</td> </tr> </tbody> </table>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	205	21	18	19	3	39	No. of patients	Full (%)	Disabled (%)	VS (%)	D (%)	34	41.2	35.3	8.8	14.7																																																																																									
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																																																																																
205	21	18	19	3	39																																																																																																																
No. of patients	Full (%)	Disabled (%)	VS (%)	D (%)																																																																																																																	
34	41.2	35.3	8.8	14.7																																																																																																																	
<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Other focal lesion, total</td> <td>205</td> <td>21</td> <td>18</td> <td>19</td> <td>3</td> <td>39</td> </tr> <tr> <td>GCS 3-5</td> <td>112</td> <td>7</td> <td>14</td> <td>18</td> <td>4</td> <td>56</td> </tr> <tr> <td>GCS 6-8</td> <td>93</td> <td>38</td> <td>23</td> <td>19</td> <td>2</td> <td>18</td> </tr> <tr> <td>Operated</td> <td>71</td> <td>21</td> <td>15</td> <td>20</td> <td>4</td> <td>39</td> </tr> <tr> <td>Operated, GCS 3-5</td> <td>35</td> <td>9</td> <td>9</td> <td>20</td> <td>6</td> <td>57</td> </tr> <tr> <td>Operated, GCS 6-8</td> <td>36</td> <td>33</td> <td>22</td> <td>19</td> <td>3</td> <td>22</td> </tr> <tr> <td>Nonoperated</td> <td>134</td> <td>21</td> <td>19</td> <td>18</td> <td>3</td> <td>39</td> </tr> <tr> <td>Nonoperated, GCS 3-5</td> <td>77</td> <td>6</td> <td>17</td> <td>17</td> <td>4</td> <td>56</td> </tr> <tr> <td>Diffuse injury, total</td> <td>487</td> <td>34</td> <td>17</td> <td>11</td> <td>5</td> <td>32</td> </tr> <tr> <td>GCS 3-5</td> <td>237</td> <td>16</td> <td>10</td> <td>14</td> <td>8</td> <td>51</td> </tr> <tr> <td>GCS 6-8</td> <td>250</td> <td>52</td> <td>24</td> <td>9</td> <td>2</td> <td>13</td> </tr> <tr> <td>Coma 6-24 h</td> <td>92</td> <td>63</td> <td>15</td> <td>2</td> <td>1</td> <td>15</td> </tr> <tr> <td>Coma 6-24 h, GCS 3-5</td> <td>27</td> <td>52</td> <td>7</td> <td>7</td> <td>4</td> <td>30</td> </tr> <tr> <td>Coma 6-24 h, GCS 6-8</td> <td>65</td> <td>68</td> <td>18</td> <td>5</td> <td>0</td> <td>9</td> </tr> <tr> <td>Coma &gt;24h</td> <td>395</td> <td>28</td> <td>17</td> <td>13</td> <td>6</td> <td>36</td> </tr> </tbody> </table>							No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Other focal lesion, total	205	21	18	19	3	39	GCS 3-5	112	7	14	18	4	56	GCS 6-8	93	38	23	19	2	18	Operated	71	21	15	20	4	39	Operated, GCS 3-5	35	9	9	20	6	57	Operated, GCS 6-8	36	33	22	19	3	22	Nonoperated	134	21	19	18	3	39	Nonoperated, GCS 3-5	77	6	17	17	4	56	Diffuse injury, total	487	34	17	11	5	32	GCS 3-5	237	16	10	14	8	51	GCS 6-8	250	52	24	9	2	13	Coma 6-24 h	92	63	15	2	1	15	Coma 6-24 h, GCS 3-5	27	52	7	7	4	30	Coma 6-24 h, GCS 6-8	65	68	18	5	0	9	Coma >24h	395	28	17	13	6	36
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																																																																																
Other focal lesion, total	205	21	18	19	3	39																																																																																																															
GCS 3-5	112	7	14	18	4	56																																																																																																															
GCS 6-8	93	38	23	19	2	18																																																																																																															
Operated	71	21	15	20	4	39																																																																																																															
Operated, GCS 3-5	35	9	9	20	6	57																																																																																																															
Operated, GCS 6-8	36	33	22	19	3	22																																																																																																															
Nonoperated	134	21	19	18	3	39																																																																																																															
Nonoperated, GCS 3-5	77	6	17	17	4	56																																																																																																															
Diffuse injury, total	487	34	17	11	5	32																																																																																																															
GCS 3-5	237	16	10	14	8	51																																																																																																															
GCS 6-8	250	52	24	9	2	13																																																																																																															
Coma 6-24 h	92	63	15	2	1	15																																																																																																															
Coma 6-24 h, GCS 3-5	27	52	7	7	4	30																																																																																																															
Coma 6-24 h, GCS 6-8	65	68	18	5	0	9																																																																																																															
Coma >24h	395	28	17	13	6	36																																																																																																															

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion																																																														
Gentleman et al. (20)	23	III Coma versus coma	Surgery and not nonsurgical	GOS 6 mo	Retrospective review of 2701 head-injured patients distilled to a cohort of 23 patients with DTICH. Volume of hematoma and timing of repeat CT scan are related to management. Operative versus nonoperative management is related to GOS at 6 mo.	<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Coma &gt;24 h, GCS 3-5</td> <td>210</td> <td>11</td> <td>10</td> <td>15</td> <td>9</td> <td>54</td> </tr> <tr> <td>Coma &gt;24 h, GCS 6-8</td> <td>185</td> <td>46</td> <td>25</td> <td>10</td> <td>3</td> <td>15</td> </tr> <tr> <td>Coma &gt;24 h, not decerebrate</td> <td>219</td> <td>38</td> <td>21</td> <td>12</td> <td>5</td> <td>24</td> </tr> <tr> <td>Coma &gt;24 h, not decerebrate, GCS 3-5</td> <td>77</td> <td>21</td> <td>8</td> <td>13</td> <td>12</td> <td>47</td> </tr> <tr> <td>Coma &gt;24 h, not decerebrate, GCS 6-8</td> <td>142</td> <td>47</td> <td>29</td> <td>11</td> <td>1</td> <td>11</td> </tr> <tr> <td>Coma &gt;24 h, not decerebrate</td> <td>176</td> <td>15</td> <td>13</td> <td>14</td> <td>7</td> <td>51</td> </tr> <tr> <td>Coma &gt;24 h, not decerebrate, GCS 3-5</td> <td>136</td> <td>6</td> <td>12</td> <td>16</td> <td>9</td> <td>57</td> </tr> <tr> <td>Coma &gt;24 h, not decerebrate, GCS 6-8</td> <td>40</td> <td>48</td> <td>15</td> <td>8</td> <td>3</td> <td>28</td> </tr> </tbody> </table> <ul style="list-style-type: none"> <li>Operative versus nonoperative lesions differed in average volume (24 versus 7 cm<sup>3</sup>) and time to repeat CT based on clinical criteria (41 versus 99 h). Outcome between groups was not statistically significant.</li> <li>39% of DTICH required evacuation; criteria not described.</li> <li>All patients with DTICH had abnormal CT on admission.</li> <li>Definition of DTICH: "lesion of increased attenuation developing after admission to hospital, in a part of the brain which the admission CT scan had suggested was normal."</li> </ul>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Coma >24 h, GCS 3-5	210	11	10	15	9	54	Coma >24 h, GCS 6-8	185	46	25	10	3	15	Coma >24 h, not decerebrate	219	38	21	12	5	24	Coma >24 h, not decerebrate, GCS 3-5	77	21	8	13	12	47	Coma >24 h, not decerebrate, GCS 6-8	142	47	29	11	1	11	Coma >24 h, not decerebrate	176	15	13	14	7	51	Coma >24 h, not decerebrate, GCS 3-5	136	6	12	16	9	57	Coma >24 h, not decerebrate, GCS 6-8	40	48	15	8	3	28
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																															
Coma >24 h, GCS 3-5	210	11	10	15	9	54																																																														
Coma >24 h, GCS 6-8	185	46	25	10	3	15																																																														
Coma >24 h, not decerebrate	219	38	21	12	5	24																																																														
Coma >24 h, not decerebrate, GCS 3-5	77	21	8	13	12	47																																																														
Coma >24 h, not decerebrate, GCS 6-8	142	47	29	11	1	11																																																														
Coma >24 h, not decerebrate	176	15	13	14	7	51																																																														
Coma >24 h, not decerebrate, GCS 3-5	136	6	12	16	9	57																																																														
Coma >24 h, not decerebrate, GCS 6-8	40	48	15	8	3	28																																																														
Gower et al. (21)	10	III GCS	<8 Subtemporal decompression versus pentobarbital coma	Mortality 1 yr	Retrospective series of 115 patients with GCS < 8 with no operative mass lesion at time of admission managed on a standard treatment protocol. 10 patients underwent subtemporal decompression because of medically refractory intracranial hypertension, and outcome was compared with those patients managed by pentobarbital coma without surgery. Technique described.	<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR/MD (%)</th> <th>SD/VS/D (%)</th> </tr> </thead> <tbody> <tr> <td>Operated DTICH</td> <td>9</td> <td>56</td> <td>44<sup>b</sup></td> </tr> <tr> <td>Nonoperated DTICH</td> <td>14</td> <td>57</td> <td>43</td> </tr> <tr> <td>DTICH, total</td> <td>23</td> <td>57</td> <td>43</td> </tr> </tbody> </table> <p><sup>b</sup> P = n.s.</p> <ul style="list-style-type: none"> <li>Patients undergoing subtemporal decompression had significantly lower mortality than those treated with pentobarbital coma despite having lower (but not statistically significant) average admission GCS (4.9 versus 6.1). However, 10 patients undergoing pentobarbital coma were determined not to be operative candidates and subsequently died; biases results in favor of operative group.</li> <li>70% of operated patients had average decrease in ICP of 34%.</li> </ul>	No. of patients	GR/MD (%)	SD/VS/D (%)	Operated DTICH	9	56	44 <sup>b</sup>	Nonoperated DTICH	14	57	43	DTICH, total	23	57	43																																															
No. of patients	GR/MD (%)	SD/VS/D (%)																																																																		
Operated DTICH	9	56	44 <sup>b</sup>																																																																	
Nonoperated DTICH	14	57	43																																																																	
DTICH, total	23	57	43																																																																	
Gudeman et al. (22)	III 12	GCS 4-10	Surgery and nonsurgical	GOS 3 mo, 1 yr	Prospective study of 162 patients with severe head injury, 12 of whom developed DTICH by CT scan. These patients were analyzed for clinical associations and outcomes to better understand this lesion.	<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Pentobarbital coma</td> <td>17</td> <td></td> <td></td> <td></td> <td>82.4</td> </tr> <tr> <td>Subtemporal decompression</td> <td>10</td> <td></td> <td></td> <td></td> <td>40<sup>c</sup></td> </tr> </tbody> </table> <p><sup>c</sup> P = 0.039.</p> <ul style="list-style-type: none"> <li>92% DTICH detected &lt;48 h from injury.</li> <li>50% developed after decompressive surgery.</li> <li>DTICH had 50% mortality; higher but not statistically significant compared with all severe head injuries during the same period.</li> <li>Significantly higher incidence of secondary systemic insults at time of admission in DTICH patients versus all severe CHI patients (92% versus 44%).</li> <li>No significant temporal relationship between development of DTICH, ICP levels, neurological change.</li> <li>Authors believe that development of DTICH is an epiphenomenon of secondary insult to a damaged brain as opposed to a causative factor for neurological deterioration—thus, treatment should be aimed at prevention of secondary hypoxic insults.</li> <li>Definition of DTICH: new parenchymal high density lesion when no lesion or a negligible abnormality was present on initial CT.</li> </ul>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Pentobarbital coma	17				82.4	Subtemporal decompression	10				40 <sup>c</sup>																																												
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																															
Pentobarbital coma	17				82.4																																																															
Subtemporal decompression	10				40 <sup>c</sup>																																																															

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion																					
Guerra et al. (23)	57	III	GCS4-6 Decompressive craniectomy	GOS 1 yr	Prospective series of 57 patients who underwent decompressive craniectomy under a standardized treatment protocol and standardized surgical technique for posttraumatic diffuse brain swelling. The patients were divided into a group undergoing primary decompression (n = 39) and a group undergoing secondary decompression because of therapy-resistant intracranial hypertension after evacuation of a surgical mass lesion (n = 18). Surgical indications and methods are detailed. Patients with GCS 3 and/or bilateral fixed/dilated pupils were excluded. Age >30 yr, >40 yr, and >50 yr variably used as exclusion criteria throughout study.	<p>● 37% GOS 5, 19% mortality overall.</p> <p>● GCS on posttrauma day 1 predictive of outcome.</p> <p>● Significantly better outcome with no loss of B waves and presence of plateau waves on ICP monitoring.</p> <p>● Inclusion of SEP, ICP/ICPP, and AEP monitoring techniques helped to define indications for the authors.</p> <p>● Authors conclude that decompressive craniectomy should be 1st among second-tier therapies for resistant intracranial hypertension.</p>																					
						<table border="0"> <tr> <td></td> <td>No. of patients</td> <td>GR/MD (%)</td> <td>SD/VS/D (%)</td> </tr> <tr> <td>Primary decompression</td> <td>38</td> <td>58</td> <td>42</td> </tr> <tr> <td>Secondary decompression</td> <td>17</td> <td>65</td> <td>35</td> </tr> </table> <p>P = n.s.</p>		No. of patients	GR/MD (%)	SD/VS/D (%)	Primary decompression	38	58	42	Secondary decompression	17	65	35									
	No. of patients	GR/MD (%)	SD/VS/D (%)																								
Primary decompression	38	58	42																								
Secondary decompression	17	65	35																								
						<table border="0"> <tr> <td></td> <td>No. of patients</td> <td>GR (%)</td> <td>MD (%)</td> <td>SD (%)</td> <td>VS (%)</td> <td>D (%)</td> </tr> <tr> <td>All patients</td> <td>55</td> <td>37</td> <td>21</td> <td>11</td> <td>9</td> <td>19</td> </tr> </table>		No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	All patients	55	37	21	11	9	19							
	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																					
All patients	55	37	21	11	9	19																					
Katayama et al. (28)	21	III	Unknown	Mortality	Retrospective case control study of 21 patients with frontal lobe contusions and medically intractable intracranial hypertension >40 mm Hg, comparing mortality between the group treated medically (n = 8) and the group treated with surgical evacuation of contused brain (n = 13). Authors state that age, sex, GCS, and ICP levels were comparable between groups, but do not provide statistics.	<p>● Mortality was significantly decreased in the surgical group (22% versus 88%).</p> <p>● Authors note that progressive ICP increase and neurological deterioration often continued past the timing of maximal enlargement of intracerebral lesions on CT.</p> <p>● Authors advocate surgical excision of contused brain in the setting of medically uncontrollable ICP elevation.</p>																					
						<table border="0"> <tr> <td></td> <td>No. of patients</td> <td>D (%)</td> </tr> <tr> <td>Nonoperative</td> <td>8</td> <td>88</td> </tr> <tr> <td>Surgery</td> <td>13</td> <td>22<sup>d</sup></td> </tr> </table> <p>d P &lt; 0.01</p>		No. of patients	D (%)	Nonoperative	8	88	Surgery	13	22 <sup>d</sup>												
	No. of patients	D (%)																									
Nonoperative	8	88																									
Surgery	13	22 <sup>d</sup>																									
Kaufman et al. (29)	8	III	ALLGCS Surgery and nonsurgical	Mortality at discharge	Retrospective series of 12 patients with DTICH or recurrent hematoma after head injury to examine the association of disseminated intravascular clotting and fibrinolysis with occurrence of these lesions. A subset of 8 patients with DTICH was evaluated for this chapter.	<p>● 7 of 8 DTICH patients had abnormal clotting parameters.</p> <p>● Included with n = 8 because of presentation of data and evaluation of clotting parameters.</p>																					
						<table border="0"> <tr> <td></td> <td>No. of patients</td> <td>D (%)</td> <td>Clotting abnormalities (%)</td> </tr> <tr> <td>DTICH, total</td> <td>8</td> <td>37.5</td> <td>87.5</td> </tr> </table>		No. of patients	D (%)	Clotting abnormalities (%)	DTICH, total	8	37.5	87.5													
	No. of patients	D (%)	Clotting abnormalities (%)																								
DTICH, total	8	37.5	87.5																								
Kotwica and Jakubowski (31)	48	III	ALLGCS Surgery and nonsurgical	GOS, not specified	Retrospective series of 136 patients aged 70-91 yr with TBI to evaluate outcome from TBI in an aged population. 48 patients with ICH/contusion reviewed.	<p>● Overall mortality from ICH/contusions was 50%; 8 of 13 in operated cases, and 16 of 35 in nonoperated cases.</p> <p>● Mortality correlated with admission GCS (no statistics).</p> <p>● Authors feel that because the mortality rate for patients &gt;70 yr with GCS &lt; 9 is so high (&gt;80%), limited attempts should be made at resuscitation, intensive care, and surgery.</p>																					
						<table border="0"> <tr> <td></td> <td>No. of patients</td> <td>CR (%)</td> <td>MD (%)</td> <td>SD (%)</td> <td>VS (%)</td> <td>D (%)</td> </tr> <tr> <td>ICH + contusion, surgery</td> <td>13</td> <td>7.7</td> <td>7.7</td> <td>15.4</td> <td>7.7</td> <td>61.5</td> </tr> <tr> <td>ICH + contusion, no surgery</td> <td>35</td> <td>11.4</td> <td>11.4</td> <td>22.9</td> <td>8.6</td> <td>45.7</td> </tr> </table>		No. of patients	CR (%)	MD (%)	SD (%)	VS (%)	D (%)	ICH + contusion, surgery	13	7.7	7.7	15.4	7.7	61.5	ICH + contusion, no surgery	35	11.4	11.4	22.9	8.6	45.7
	No. of patients	CR (%)	MD (%)	SD (%)	VS (%)	D (%)																					
ICH + contusion, surgery	13	7.7	7.7	15.4	7.7	61.5																					
ICH + contusion, no surgery	35	11.4	11.4	22.9	8.6	45.7																					

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class GCS	Treatment	Outcome	Description	Conclusion																																																																		
Kumchev et al. (32)	79	III GCS 3-6 to 12-15	Surgery and nonsurgical	Mortality	Retrospective review of 79 patients with intracerebral hematomas, comparing location of hematoma with mortality, and outcome of surgery versus nonsurgical therapy. Treatment protocol was not standardized; surgical indications were not reported.	<ul style="list-style-type: none"> <li>Those patients with temporal ICH were more likely to die.</li> <li>Surgery did not alter mortality. Surgically managed patients were more likely to show clinical improvement.</li> </ul> <table border="1"> <thead> <tr> <th>Location</th> <th>No. of patients</th> <th>D (%)</th> <th>P values</th> </tr> </thead> <tbody> <tr> <td>Frontal</td> <td>16</td> <td>6</td> <td>&lt;0.001</td> </tr> <tr> <td>Temporal</td> <td>36</td> <td>57.6</td> <td>&lt;0.01</td> </tr> <tr> <td>Parieto-occipital</td> <td>14</td> <td>18.2</td> <td>&gt;0.01</td> </tr> <tr> <td>Multiple sites</td> <td>13</td> <td>18.2</td> <td>&gt;0.01</td> </tr> </tbody> </table>	Location	No. of patients	D (%)	P values	Frontal	16	6	<0.001	Temporal	36	57.6	<0.01	Parieto-occipital	14	18.2	>0.01	Multiple sites	13	18.2	>0.01																																														
Location	No. of patients	D (%)	P values																																																																					
Frontal	16	6	<0.001																																																																					
Temporal	36	57.6	<0.01																																																																					
Parieto-occipital	14	18.2	>0.01																																																																					
Multiple sites	13	18.2	>0.01																																																																					
Kunze et al. (33)	28	III All GCS	Decompressive craniectomy	GOS 1 yr	Retrospective study of 28 patients undergoing uni/bilateral decompressive craniectomy for posttraumatic edema after "maximal" medical therapy.	<ul style="list-style-type: none"> <li>57% overall favorable outcome (GOS 4-5) at 1 yr.</li> <li>Mean ICP decreased from 41.7 to 20.6 mm Hg after decompression.</li> <li>Patients with "vast" primary lesions, hypoxia, ischemic infarction, brainstem injury, "central" herniation, and primary anisocoria excluded.</li> </ul> <table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR/MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Decompressive craniectomy</td> <td>28</td> <td>57</td> <td>18</td> <td>14</td> <td>11</td> </tr> </tbody> </table>	No. of patients	GR/MD (%)	SD (%)	VS (%)	D (%)	Decompressive craniectomy	28	57	18	14	11																																																							
No. of patients	GR/MD (%)	SD (%)	VS (%)	D (%)																																																																				
Decompressive craniectomy	28	57	18	14	11																																																																			
Lee et al. (34)	29	III GCS ≤8	Subtemporal decompression ± temporal lobectomy	GOS 1 yr	Retrospective series of 29 patients operated on for acute SDH and severe contusion/swelling of temporal lobe with uncal herniation. 16 patients underwent subtemporal decompression and debulking of contused brain whereas 13 subsequent patients underwent temporal lobectomy in addition. These two groups were compared with respect to outcome at 1 yr or longer.	<ul style="list-style-type: none"> <li>Addition of temporal lobectomy to subtemporal decompression and debulking of contused brain significantly decreased mortality from 56% to 8% and significantly improved average GOS from 2.2 to 4.0. Preoperative GCS, age, and sex were not significantly different between groups.</li> <li>Incidence of motor recovery, cognitive deficits, and seizures were not significantly different between groups.</li> </ul> <table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR/MD (%)</th> <th>SD/VS/D (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>STD, debulking</td> <td>16</td> <td>25</td> <td>75</td> <td>56</td> </tr> <tr> <td>STD, debulking, TL</td> <td>13</td> <td>77</td> <td>23</td> <td>8*</td> </tr> <tr> <td>STD, debulking, complete TL</td> <td>10</td> <td>100</td> <td>0</td> <td>0</td> </tr> <tr> <td>STD, debulking, anterior TL</td> <td>3</td> <td>0</td> <td>100</td> <td>33</td> </tr> </tbody> </table> <p>* P &lt; 0.01.</p>	No. of patients	GR/MD (%)	SD/VS/D (%)	D (%)	STD, debulking	16	25	75	56	STD, debulking, TL	13	77	23	8*	STD, debulking, complete TL	10	100	0	0	STD, debulking, anterior TL	3	0	100	33																																										
No. of patients	GR/MD (%)	SD/VS/D (%)	D (%)																																																																					
STD, debulking	16	25	75	56																																																																				
STD, debulking, TL	13	77	23	8*																																																																				
STD, debulking, complete TL	10	100	0	0																																																																				
STD, debulking, anterior TL	3	0	100	33																																																																				
Lobato et al. (35)	277	III GCS 3-7	Surgery and nonsurgical	GOS 6 mo	Prospective series of 277 severely head-injured patients classified by CT scan into 8 injury types to examine GOS at 6 mo related to lesion type. ICP was monitored in all patients. Patients brain dead on arrival were excluded. A standardized management protocol was used and is described. GCS, ICP, and interval to operation varied between CT groups.	<ul style="list-style-type: none"> <li>Patients with pure extracerebral hematoma, single brain contusion, general brain swelling, and normal CT scans had a significantly better outcome than patients with extracerebral hematoma and acute hemispheric swelling, multiple brain contusions, and patients with diffuse axonal injury.</li> <li>Acute hemispheric swelling significantly increased mortality.</li> </ul> <table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Single contusion (SC)</td> <td>25</td> <td>72</td> <td>8</td> <td>4</td> <td>12</td> </tr> <tr> <td>SC + EDH</td> <td>7</td> <td>14</td> <td>86</td> <td>0</td> <td>0</td> </tr> <tr> <td>SC + SDH</td> <td>13</td> <td>15</td> <td>46</td> <td>15</td> <td>23</td> </tr> <tr> <td>MUC</td> <td>20</td> <td>0</td> <td>15</td> <td>10</td> <td>75</td> </tr> <tr> <td>MUC + SDH</td> <td>12</td> <td>0</td> <td>17</td> <td>8</td> <td>75</td> </tr> <tr> <td>Multiple bilateral contusion</td> <td>42</td> <td>19</td> <td>26</td> <td>5</td> <td>50</td> </tr> <tr> <td>GBS</td> <td>25</td> <td>76</td> <td>8</td> <td>4</td> <td>12</td> </tr> <tr> <td>GBS + EDH</td> <td>16</td> <td>88</td> <td>6</td> <td>0</td> <td>6</td> </tr> <tr> <td>DAI</td> <td>43</td> <td>5</td> <td>9</td> <td>21</td> <td>53</td> </tr> <tr> <td>Normal CT</td> <td>28</td> <td>39</td> <td>29</td> <td>18</td> <td>11</td> </tr> </tbody> </table>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Single contusion (SC)	25	72	8	4	12	SC + EDH	7	14	86	0	0	SC + SDH	13	15	46	15	23	MUC	20	0	15	10	75	MUC + SDH	12	0	17	8	75	Multiple bilateral contusion	42	19	26	5	50	GBS	25	76	8	4	12	GBS + EDH	16	88	6	0	6	DAI	43	5	9	21	53	Normal CT	28	39	29	18	11
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																																			
Single contusion (SC)	25	72	8	4	12																																																																			
SC + EDH	7	14	86	0	0																																																																			
SC + SDH	13	15	46	15	23																																																																			
MUC	20	0	15	10	75																																																																			
MUC + SDH	12	0	17	8	75																																																																			
Multiple bilateral contusion	42	19	26	5	50																																																																			
GBS	25	76	8	4	12																																																																			
GBS + EDH	16	88	6	0	6																																																																			
DAI	43	5	9	21	53																																																																			
Normal CT	28	39	29	18	11																																																																			

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion																																																								
Lobato et al. (36)	587	III CCS 3-8	Surgery and nonsurgical	GOS 6 mo	Retrospective series of 587 patients with GCS $\leq$ 8 and at least one control CT scan (average 36.7 h after initial CT) to evaluate incidence of changes in CT classification over time and to assess the relative prognostic significance of a second CT scan versus the first.	<ul style="list-style-type: none"> <li>51.2% developed significant CT changes requiring change in diagnostic category.</li> <li>Final outcome was more accurately predicted by control CT diagnosis than by initial CT diagnosis.</li> <li>19% of diffuse injuries evolved to mass lesions.</li> </ul>																																																								
Mandera et al. (37)	31	III CCS 3-8 to 13-15	Surgery and nonsurgical	GOS, not specified	Retrospective study of 31 children with traumatic ICH to examine factors related to surgical decision making and assess outcome.	<ul style="list-style-type: none"> <li>Outcome did not differ between surgical and conservative groups despite the fact that more patients with severe injury were treated surgically.</li> </ul>																																																								
Marshall (39)	414	III CCS 3-8	Nonsurgical	GOS 11 d-2 yr	Prospective series of 746 patients from TCDB to identify patients at risk for developing intracranial hypertension in the absence of significant mass lesions on initial CT. The authors propose a new classification of head injury based on CT and relate the new classification categories to outcome. Nonsurgical diffuse injury categories are included.	<ul style="list-style-type: none"> <li>CT diagnosis was a highly significant independent predictor of mortality when age and motor score were included in the model.</li> <li>In Diffuse Injury III, highest ICP and postresuscitation pupillary reactivity were significant predictors of outcome.</li> </ul>																																																								
Marshall et al. (40)	502	III CCS 3-8	Surgery (ICH) and nonsurgical (Diffuse Injury I-IV)	GOS 11 d-2 yr	Prospective series of 746 patients selected from TCDB to assess importance of prognostic variables in determining outcome from severe head injury. Subgroups of nonoperated patients with Diffuse Injury patterns I-IV and patients with evacuated ICH are included.	<ul style="list-style-type: none"> <li>90% of deaths occurred within 2 wk.</li> <li>Intracranial diagnosis strongly correlated with outcome; e.g., higher % of Diffuse Injury III/IV patients were vegetative.</li> <li>In Diffuse Injury II group, age <math>&gt;</math>40 yr strongly correlated with poor outcome.</li> <li>GOS for evacuated ICH patients correlated negatively with age <math>&gt;</math>40 yr.</li> <li>For all patients, postresuscitation GCS, pupillary reactivity, and age correlated with outcome.</li> </ul>																																																								
<table border="1"> <thead> <tr> <th colspan="2">CT diagnosis<sup>f</sup></th> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Diffuse Injury I</td> <td>52</td> <td>27</td> <td>34.6</td> <td>19.2</td> <td>9.6</td> <td>9.6</td> <td>9.6</td> </tr> <tr> <td>Diffuse Injury II</td> <td>177</td> <td>8.5</td> <td>26</td> <td>40.7</td> <td>11.3</td> <td>13.5</td> <td>13.5</td> </tr> <tr> <td>Diffuse Injury III</td> <td>153</td> <td>3.3</td> <td>13.1</td> <td>26.8</td> <td>22.9</td> <td>34</td> <td>34</td> </tr> <tr> <td>Diffuse Injury IV</td> <td>32</td> <td>3.1</td> <td>3.1</td> <td>18.8</td> <td>18.8</td> <td>56.2</td> <td>56.2</td> </tr> <tr> <td>Diffuse Injury II, age <math>\leq</math> 40 yr</td> <td>153</td> <td>10</td> <td>28.7</td> <td>41.1</td> <td>11.1</td> <td>9.1</td> <td>9.1</td> </tr> <tr> <td>Diffuse Injury II, age <math>&gt;</math>40 yr</td> <td>24</td> <td>0</td> <td>8.3</td> <td>37.5</td> <td>12.5</td> <td>41.7</td> <td>41.7</td> </tr> </tbody> </table> <p><sup>f</sup> <math>P &lt; 0.001</math></p>							CT diagnosis <sup>f</sup>		No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Diffuse Injury I	52	27	34.6	19.2	9.6	9.6	9.6	Diffuse Injury II	177	8.5	26	40.7	11.3	13.5	13.5	Diffuse Injury III	153	3.3	13.1	26.8	22.9	34	34	Diffuse Injury IV	32	3.1	3.1	18.8	18.8	56.2	56.2	Diffuse Injury II, age $\leq$ 40 yr	153	10	28.7	41.1	11.1	9.1	9.1	Diffuse Injury II, age $>$ 40 yr	24	0	8.3	37.5	12.5	41.7	41.7
CT diagnosis <sup>f</sup>		No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																							
Diffuse Injury I	52	27	34.6	19.2	9.6	9.6	9.6																																																							
Diffuse Injury II	177	8.5	26	40.7	11.3	13.5	13.5																																																							
Diffuse Injury III	153	3.3	13.1	26.8	22.9	34	34																																																							
Diffuse Injury IV	32	3.1	3.1	18.8	18.8	56.2	56.2																																																							
Diffuse Injury II, age $\leq$ 40 yr	153	10	28.7	41.1	11.1	9.1	9.1																																																							
Diffuse Injury II, age $>$ 40 yr	24	0	8.3	37.5	12.5	41.7	41.7																																																							



TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Mean GCS	Treatment	Outcome	Description	Conclusion														
Mathiesen et al. (41)	218	III	6.9	Surgery and nonsurgical	GOS 6 mo	Retrospective review of data accumulated in prospective, randomized, double-blind HFT-2 study on nimodipine in head injury. 218 patients not following commands within 24 h of trauma with intracerebral lesions only on CT were chosen for analysis. Patients with extracerebral lesions, normal CT, gunshot wounds, pregnancy, bilaterally fixed/dilated pupils, GCS 3 for >2 h, and previous drug administration interfering with neurological assessment were excluded. Clinical management was not standardized. Patients were divided into 4 subgroups based on CT appearance within 24 h of trauma: 1) contusions; 2) fracture contusions (directly underlying depressed fx); 3) DAI; and 4) ICH (>10 cm <sup>3</sup> ). A subgroup of patients with GCS $\geq$ 6 and lesion $\geq$ 20 cm <sup>3</sup> were analyzed for the effect of surgery and timing of surgery on outcome. A subgroup of patients who "talked and died" were analyzed to determine the value of early surgery.	<ul style="list-style-type: none"> <li>Initial CT characteristics associated with neurological deterioration, defined as fall in GCS by 2, compressed/absent cisterns.</li> <li>Secondary (<math>\geq</math> 5 d) deterioration correlated with SAH on initial CT, hypoxic events.</li> <li>For the 4 groups:                             <ol style="list-style-type: none"> <li>Contusions: outcome adversely affected by presence of IVH.</li> <li>Fracture/contusions: outcome significantly worse than other groups; outcome adversely affected by presence of SAH.</li> <li>DAI: mean admission GCS significantly worse than other groups. No patients operated.</li> <li>ICH: mean admission GCS significantly better than other groups. Outcome adversely affected by presence of IVH.</li> </ol> </li> <li>Patients with lesions <math>\geq</math> 20 cm<sup>3</sup> and admission GCS <math>\geq</math> 6:                             <ol style="list-style-type: none"> <li>Patients operated without previous deterioration (n = 9) had better outcome than those not operated on or operated on after deterioration (n = 66).</li> <li>Patients with radiological signs of mass effect (compression/obliteration of cistern and/or midline shift <math>\geq</math> 5 mm) had better outcome with surgery versus no surgery, and those operated before deterioration had better outcome than those operated after deterioration.</li> <li>Surgery did not influence outcome in patients admitted with GCS <math>\leq</math> 5.</li> </ol> </li> <li>11 patients with temporal contusion, radiological signs of mass effect, admission GCS <math>\geq</math> 10: outcome significantly better in those operated before or immediately after deterioration compared with delayed or no surgery.</li> <li>Authors' points:                             <ol style="list-style-type: none"> <li>Patients with frontal or temporal contusions with midline shift or cisternal compression had statistically better outcome with surgery.</li> <li>Surgical outcome was better the earlier it was performed related to deterioration.</li> <li>Patients with temporal contusions and radiological signs of mass effect should be operated on before deterioration.</li> <li>Authors offer indications for surgery: all patients with contusions <math>\geq</math> 20 cm<sup>3</sup> with radiological signs of mass effect or with any lesion <math>\geq</math> 50 cm<sup>3</sup>.</li> </ol> </li> </ul>														
	All patients						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD (%)</th> <th>D (%)</th> <th>Mean GOS</th> </tr> </thead> <tbody> <tr> <td></td> <td>218</td> <td>56</td> <td>27.5</td> <td>3.2</td> </tr> </tbody> </table>		No. of patients	GR/MD (%)	D (%)	Mean GOS		218	56	27.5	3.2				
	No. of patients	GR/MD (%)	D (%)	Mean GOS																	
	218	56	27.5	3.2																	
	Contusions						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>122</td> <td>24</td> <td>30</td> <td>11</td> <td>5</td> <td>30</td> </tr> </tbody> </table>		No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)		122	24	30	11	5	30
	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)															
	122	24	30	11	5	30															
	Contusions/depressed cranial fracture <sup>g</sup>						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>24</td> <td>12</td> <td>17</td> <td>2.5</td> <td>4</td> <td>42</td> </tr> </tbody> </table>		No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)		24	12	17	2.5	4	42
	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)															
	24	12	17	2.5	4	42															
	DAI						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>39</td> <td>23 25 30</td> <td>5 17</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		39	23 25 30	5 17						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	39	23 25 30	5 17																		
	ICH						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>32</td> <td>31 28 16</td> <td>3 22</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		32	31 28 16	3 22						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	32	31 28 16	3 22																		
	GCS $\geq$ 6, volume $\geq$ 20 cm <sup>3</sup>						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>42</td> <td>50</td> <td>50</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		42	50	50						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	42	50	50																		
	No surgery						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>6</td> <td>100</td> <td>0</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		6	100	0						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	6	100	0																		
	Surgery before deterioration <sup>g</sup>						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>9</td> <td>67</td> <td>33</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		9	67	33						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	9	67	33																		
	Surgery on day of deterioration						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>9</td> <td>55</td> <td>44</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		9	55	44						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	9	55	44																		
	Surgery $\geq$ 1 d after deterioration						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>19</td> <td>26</td> <td>74</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		19	26	74						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	19	26	74																		
	GCS $\geq$ 6, volume $\geq$ 20 cm <sup>3</sup> , CT mass effect						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>6</td> <td>100</td> <td>0</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		6	100	0						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	6	100	0																		
	No surgery						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>8</td> <td>62.5</td> <td>37.5</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		8	62.5	37.5						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	8	62.5	37.5																		
	Surgery before deterioration <sup>g</sup>						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>1</td> <td>0</td> <td>100</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		1	0	100						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	1	0	100																		
	Surgery on day of deterioration						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>14</td> <td>36</td> <td>64</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		14	36	64						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	14	36	64																		
	Surgery $\geq$ 1 d after deterioration						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>3</td> <td>100</td> <td>0</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		3	100	0						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	3	100	0																		
	GCS $\geq$ 6, volume $\geq$ 50 cm <sup>3</sup>						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>10</td> <td>70</td> <td>30</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		10	70	30						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	10	70	30																		
	No surgery						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>1</td> <td>0</td> <td>100</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		1	0	100						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	1	0	100																		
	Surgery before deterioration <sup>g</sup>						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>8</td> <td>0</td> <td>100</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		8	0	100						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	8	0	100																		
	Surgery on day of deterioration <sup>g</sup>						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>3</td> <td>100</td> <td>0</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		3	100	0						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	3	100	0																		
	Surgery $\geq$ 1 d after deterioration						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>10</td> <td>70</td> <td>30</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		10	70	30						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	10	70	30																		
	GCS $\geq$ 10 cm <sup>3</sup> , temporal contusion, CT mass effect						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>1</td> <td>0</td> <td>100</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		1	0	100						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	1	0	100																		
	No surgery						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>8</td> <td>0</td> <td>100</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		8	0	100						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	8	0	100																		
	Surgery before or at time of deterioration <sup>g</sup>						<table border="1"> <thead> <tr> <th></th> <th>No. of patients</th> <th>GR/MD/SD (%)</th> <th>VS/D (%)</th> </tr> </thead> <tbody> <tr> <td></td> <td>3</td> <td>100</td> <td>0</td> </tr> </tbody> </table>		No. of patients	GR/MD/SD (%)	VS/D (%)		3	100	0						
	No. of patients	GR/MD/SD (%)	VS/D (%)																		
	3	100	0																		

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion
Meier et al. (42)	38	III All GCS	Surgery	GOS at discharge	Retrospective series of 936 patients who underwent surgery for TBI, 38 of whom were children with ICH/contusion as primary pathology. GOS is related to admission GCS.	<p>● Children with admission GCS 6-8 had better overall outcome than adults with admission GCS 6-8.</p> <p>Children with contusions</p> <p>No. of patients: 38 GR/MD (%): 53 SD/VS (%): 10 D (%): 37</p>
Meier et al. (43)	19	III GCS 3-12	Decompressive craniectomy	GOS at discharge	Retrospective series of 19 patients with intractable intracranial hypertension undergoing decompressive craniectomy to evaluate factors related to prognosis. Surgical indications included failure of conservative intervention for intracranial hypertension, age <50 yr without multiple trauma, age <30 yr in presence of major extracranial injuries, severe brain swelling on CT (or intraoperatively). 11 patients underwent decompressive craniectomy because generalized brain edema was evident during evacuation of a space-occupying lesion; 8 patients underwent decompressive craniectomy secondary to failure of conservative treatment.	<p>No. of patients: 19 GR/MD (%): 26 SD (%): 5 VS (%): 26 D (%): 43</p> <p>All Operated Nonoperated 63 27</p> <p>● 2 patients &gt;60 yr had GOS 2 (VS). ● Age and GCS related to GOS (no statistics). ● Mortality increased with the presence of extracranial injuries (no statistics).</p>
Miller et al. (44)	20	III GCS 3-8	Surgical and nonsurgical	GOS 3 mo	Prospective series of 225 patients with severe head injury managed by standardized protocol to identify prognostic variables and to validate results of a previous study. Subgroups with surgical and nonsurgical lesions are identified and outcome is specified for each lesion. The definition of ICH included >5 mm midline shift, and, thus, all were taken to surgery.	<p>No. of patients: 20 GR/MD (%): 26 SD/VS (%): 16 D (%): 58</p> <p>ICH Contusion, no surgery 31 Diffuse, no surgery 101 GR/MD (%): 64 SD/VS (%): 13 D (%): 23 GR/MD (%): 73 SD/VS (%): 10 D (%): 17</p> <p>● Correlation between age, abnormal motor response, bilateral loss of light reflex, impaired/absent oculocephalic reflex, and outcome in nonoperatively managed patients. ● Parenchymal lesions of increased density on CT were associated with severe disability/vegetative state compared with other lesions. ● 31% incidence of uncontrollable postoperative elevated ICP in ICH patients. Surgical and nonsurgical groups were not comparable.</p>
Munch et al. (45)	49	III GCS < 8 to ≥ 8	Decompressive craniectomy	GOS 6 mo	Retrospective series of 49 patients undergoing unilateral decompressive craniectomy for TBI under standardized treatment protocol to evaluate preoperative and postoperative CT characteristics, ICP/ CPP change, and to assess outcome. A subdivision into rapid and delayed groups was also analyzed according to outcome. Surgical indications detailed: Patients with bilateral intracranial lesions; bilaterally fixed and dilated pupils; and life-threatening concomitant medical disease were excluded. All patients undergoing rapid decompression had acute SDH. All patients undergoing delayed decompression had diffuse brain swelling.	<p>No. of patients: 49 GR/MD (%): 28 SD/VS (%): 72</p> <p>At discharge from ICU 49 At 6 mo 41 GR/MD (%): 41 SD/VS (%): 59</p> <p>● 41% good outcome at 6 mo. ● Visibility of mesencephalic cisterns and midline shift improved significantly after craniectomy; gyral pattern and visibility of ventricles did not. ● No change in mean therapeutic intensity level before versus after decompression. ● Age &lt;50 yr, GCS ≥ 8 correlated significantly with better outcome. ● GOS at 6 mo significantly better in rapid versus delayed decompression groups, but GOS at discharge from ICU not significantly different. ● No significant difference between before and after decompression ICP and CPP. ● Change in mesencephalic cistern visibility correlated with distance between lower craniectomy border and cranial base (more than area of decompression).</p> <p>Mean GOS 3.9 GCS &lt; 8 1.4<sup>b</sup> GCS ≥ 8 3.1 Rapid 3.1 Delayed 1.9<sup>f</sup> Age &lt;50 yr 3.0 Age ≥ 50 yr 1.9<sup>f</sup></p> <p><sup>h</sup> P = 0.023, <sup>i</sup> P = 0.046.</p>



TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion																																																						
Polin et al. (51)	35	III Mean GCS 5.62	Bifrontal decompressive craniectomy	GOS at discharge	Retrospective case-control series of 35 patients undergoing bifrontal decompressive craniectomy for refractory posttraumatic cerebral edema comparing preoperative and postoperative ICP, age versus outcome, and comparing the group as a whole with the group from TCDB matched for age, admission GCS, sex, and maximal ICP in terms of outcome. Only TCDB patients with diffuse injury, no significant mass lesion, no midline shift (Marshall et al. [39]) were eligible for consideration as controls.	<ul style="list-style-type: none"> <li>Admission GCS related to outcome.</li> <li>Operation &lt;48 h associated with favorable outcome (46%) compared with &gt;48 h (9%).</li> <li>Significant change in ICP before versus after decompression.</li> <li>Significant difference in postoperative ICP versus control ICP 48–72 h after injury.</li> <li>Coagulopathy did not affect outcome in surgical group.</li> <li>ICP &gt; 40 mm Hg sustained and operation &gt;48 h after injury had significantly less favorable outcomes.</li> <li>Age &lt;18 yr had higher rate of favorable outcome in surgical group.</li> <li>Surgery age &lt;18 yr had higher rate of favorable outcome than matched controls if operated &lt;48 h and ICP &lt; 40 mm Hg.</li> <li>Medical management alone carried 3.86 times relative risk of unfavorable outcome compared with decompressive craniectomy.</li> <li>Maximum benefit is achieved in patients operated on &lt;48 h after injury and with ICP elevations sustained &lt;40 mm Hg (60% favorable versus 18% in matched controls).</li> </ul>																																																						
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Surgery<sup>k</sup></td> <td>35</td> <td>11.4</td> <td>25.7</td> <td>31.4</td> <td>8.6</td> <td>22.9</td> </tr> <tr> <td>Case control</td> <td>92</td> <td>7.6</td> <td>8.8</td> <td>35.2</td> <td>18.7</td> <td>30.8</td> </tr> </tbody> </table>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Surgery <sup>k</sup>	35	11.4	25.7	31.4	8.6	22.9	Case control	92	7.6	8.8	35.2	18.7	30.8																																		
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																							
Surgery <sup>k</sup>	35	11.4	25.7	31.4	8.6	22.9																																																						
Case control	92	7.6	8.8	35.2	18.7	30.8																																																						
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR/MD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Coagulopathy</td> <td>18</td> <td>33<sup>l</sup></td> <td>28</td> <td>18</td> </tr> <tr> <td>No coagulopathy</td> <td>17</td> <td>41</td> <td></td> <td></td> </tr> <tr> <td>ICP &gt; 40 mm Hg, OR &gt;48 h</td> <td>15</td> <td>6.7<sup>m</sup></td> <td></td> <td></td> </tr> <tr> <td>ICP &lt; 40, OR &lt;48 h</td> <td>20</td> <td>60</td> <td></td> <td></td> </tr> <tr> <td>Age &lt;18 yr</td> <td>18</td> <td>44</td> <td></td> <td></td> </tr> <tr> <td>Age &gt;18 yr</td> <td>17</td> <td>29</td> <td></td> <td></td> </tr> <tr> <td>OR &lt;48 h, ICP &lt;40</td> <td>20</td> <td>60<sup>n</sup></td> <td></td> <td></td> </tr> <tr> <td>ICP &lt; 40, control</td> <td>58</td> <td>18</td> <td></td> <td></td> </tr> <tr> <td>Age &lt;18, ICP &lt;40, OR &lt;48 h</td> <td>10</td> <td>80<sup>o</sup></td> <td></td> <td></td> </tr> <tr> <td>Age &lt;18, ICP &lt;40, control</td> <td>25</td> <td>24</td> <td></td> <td></td> </tr> </tbody> </table>	No. of patients	GR/MD (%)	VS (%)	D (%)	Coagulopathy	18	33 <sup>l</sup>	28	18	No coagulopathy	17	41			ICP > 40 mm Hg, OR >48 h	15	6.7 <sup>m</sup>			ICP < 40, OR <48 h	20	60			Age <18 yr	18	44			Age >18 yr	17	29			OR <48 h, ICP <40	20	60 <sup>n</sup>			ICP < 40, control	58	18			Age <18, ICP <40, OR <48 h	10	80 <sup>o</sup>			Age <18, ICP <40, control	25	24		
No. of patients	GR/MD (%)	VS (%)	D (%)																																																									
Coagulopathy	18	33 <sup>l</sup>	28	18																																																								
No coagulopathy	17	41																																																										
ICP > 40 mm Hg, OR >48 h	15	6.7 <sup>m</sup>																																																										
ICP < 40, OR <48 h	20	60																																																										
Age <18 yr	18	44																																																										
Age >18 yr	17	29																																																										
OR <48 h, ICP <40	20	60 <sup>n</sup>																																																										
ICP < 40, control	58	18																																																										
Age <18, ICP <40, OR <48 h	10	80 <sup>o</sup>																																																										
Age <18, ICP <40, control	25	24																																																										
Servadei et al. (54)	724	III GCS 3–12	Surgery and nonsurgical	GOS 6 mo	Prospective study of 724 patients with moderate-to-severe TBI to determine the frequency of changes in CT findings and their prognostic implications.	<ul style="list-style-type: none"> <li>16% of diffuse injuries on initial CT demonstrated deterioration on subsequent CT.</li> <li>12% of diffuse injuries evolved to mass lesions.</li> <li>Evolution from diffuse injury without swelling or shift (Type II) to a mass lesion was associated with significant increase in risk of unfavorable outcome.</li> <li>Trend towards more favorable outcome in patients with nonevacuated versus evacuated mass lesions.</li> </ul>																																																						
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR/MD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Diffuse injury I, no change</td> <td>75</td> <td>87</td> <td>13</td> <td></td> </tr> <tr> <td>Diffuse injury I, evolution</td> <td>5</td> <td>80</td> <td>20</td> <td></td> </tr> <tr> <td>Diffuse injury II, no change</td> <td>185</td> <td>62</td> <td>38</td> <td></td> </tr> <tr> <td>Diffuse injury II, evolution<sup>p</sup></td> <td>34</td> <td>38</td> <td>62</td> <td></td> </tr> <tr> <td>Diffuse injury III, no change</td> <td>57</td> <td>33</td> <td>67</td> <td></td> </tr> <tr> <td>Diffuse injury III, evolution</td> <td>7</td> <td>57</td> <td>43</td> <td></td> </tr> <tr> <td>Diffuse injury IV, no change</td> <td>14</td> <td>21</td> <td>79</td> <td></td> </tr> <tr> <td>Diffuse injury IV, evolution</td> <td>4</td> <td>75</td> <td>25</td> <td></td> </tr> <tr> <td>Mass lesion</td> <td>343</td> <td>43</td> <td>57</td> <td></td> </tr> </tbody> </table> <p><sup>p</sup> P &lt; 0.01.</p>	No. of patients	GR/MD (%)	VS (%)	D (%)	Diffuse injury I, no change	75	87	13		Diffuse injury I, evolution	5	80	20		Diffuse injury II, no change	185	62	38		Diffuse injury II, evolution <sup>p</sup>	34	38	62		Diffuse injury III, no change	57	33	67		Diffuse injury III, evolution	7	57	43		Diffuse injury IV, no change	14	21	79		Diffuse injury IV, evolution	4	75	25		Mass lesion	343	43	57						
No. of patients	GR/MD (%)	VS (%)	D (%)																																																									
Diffuse injury I, no change	75	87	13																																																									
Diffuse injury I, evolution	5	80	20																																																									
Diffuse injury II, no change	185	62	38																																																									
Diffuse injury II, evolution <sup>p</sup>	34	38	62																																																									
Diffuse injury III, no change	57	33	67																																																									
Diffuse injury III, evolution	7	57	43																																																									
Diffuse injury IV, no change	14	21	79																																																									
Diffuse injury IV, evolution	4	75	25																																																									
Mass lesion	343	43	57																																																									
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Diffuse injury I</td> <td>80</td> <td>74</td> <td>12</td> <td>9</td> <td>0</td> <td>5</td> </tr> <tr> <td>Diffuse injury II</td> <td>219</td> <td>36</td> <td>22</td> <td>20</td> <td>2</td> <td>21</td> </tr> <tr> <td>Diffuse injury III</td> <td>64</td> <td>20</td> <td>16</td> <td>9</td> <td>2</td> <td>53</td> </tr> <tr> <td>Diffuse injury IV</td> <td>18</td> <td>11</td> <td>22</td> <td>11</td> <td>0</td> <td>56</td> </tr> </tbody> </table>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Diffuse injury I	80	74	12	9	0	5	Diffuse injury II	219	36	22	20	2	21	Diffuse injury III	64	20	16	9	2	53	Diffuse injury IV	18	11	22	11	0	56																				
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																							
Diffuse injury I	80	74	12	9	0	5																																																						
Diffuse injury II	219	36	22	20	2	21																																																						
Diffuse injury III	64	20	16	9	2	53																																																						
Diffuse injury IV	18	11	22	11	0	56																																																						

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion
Singounas (57)	16	III GCS 8-10	Unknown	Mortality	Retrospective review of 48 children with severe head injury (<14 yr) with subdural hematomas excluded to comment on relatively good prognosis. Mortality for 16 patients with brain edema only was reported.	Diffuse edema, no focal lesion No. of patients: 16 D (%): 12.5
Soloniuk et al. (58)	35	III GCS ≤ 8 or > 8	Surgery	GOS 6 mo	Retrospective review of 35 patients undergoing craniotomy for evacuation of ICH to evaluate characteristics of timing of appearance of ICH and factors related to outcome. ICH defined as homogeneous coalescent area of high attenuation measuring 2 cm or greater.	<ul style="list-style-type: none"> <li>Only 26% of ICH requiring evacuation are present on CT within 6 h of injury.</li> <li>49% overall 1-yr mortality; 71% with GCS ≤ 6.</li> <li>Mortality varies with location, with parietal, temporal, and frontal rates of 80%, 57%, and 37%, respectively.</li> </ul> ICH, operated No. of patients: 35 D (%): 49
Sprick et al. (59)	34	III GCS 3 to > 7	Surgery and nonsurgical	GOS, not specified	Retrospective series of 34 patients with DTICH of at least 2.5-cm diameter in previously normal area on initial CT to evaluate outcome and clinical characteristics.	<ul style="list-style-type: none"> <li>53% DTICH developed within 24 h after admission and 79% within 48 h</li> </ul> All No. of patients: 34 CR (%): 17.6 D (%): 26.5 Operated No. of patients: 7 CR (%): 14.3 D (%): 42.9
Statham et al. (60)	18	III GCS 5-14	Surgery (n = 1) and nonsurgical	GOS 6 mo	Retrospective review of 18 patients with primary CT diagnosis of frontal contusion, defined as regions of mixed high and low or low attenuation in the frontal lobes, to attempt to determine factors that may lead to neurological deterioration and worse outcome.	<ul style="list-style-type: none"> <li>Good outcome was achieved in 72% of patients with frontal contusion.</li> <li>Both cases of deterioration were associated with extensive bilateral injury (no statistics).</li> </ul> No. of patients: 18 GR (%): 72.2 MD (%): 11.1 SD (%): 5.6 VS (%): 0 D (%): 11.1 Unilateral contusion: 10 Limited bilateral contusions: 5 Extensive bilateral contusions: 3 Frontal contusions, total: 18
Taylor et al. (61)	27	II GCS 4-11	Decompressive bitemporal craniectomy and nonsurgical	GOS 6 mo; Health State Utility Index 6 mo	Prospective, randomized, controlled trial of 27 children with TBI and sustained intracranial hypertension to evaluate effect of early decompressive bitemporal craniectomy on outcome and on control of ICP.	<ul style="list-style-type: none"> <li>Trend towards greater decrease in ICP in decompression group (P = 0.057).</li> <li>Trend towards shorter ICU stay in decompression group (P = 0.12).</li> <li>Trend towards improved outcome in decompression group (P = 0.046 [P &lt; 0.0221 required for significance]).</li> </ul> No. of patients: 27 Favorable (%): 53.8 <sup>†</sup> Unfavorable (%): 46.1 Decompression: 13 Control: 14
Teasdale et al. (62)	37	III GCS 3-8	Nonsurgical	GOS 6 mo	Retrospective series of 37 patients diagnosed with severe diffuse head injury (without midline shift of >5 mm) to determine the relationship between compression of basal cisterns/3rd ventricle and elevated ICP.	<ul style="list-style-type: none"> <li>Highly significant relationship between status of basal cisterns/III ventricle and level of ICP.</li> <li>Presence versus absence of cisterns/III ventricle seemed to correlate with outcome within GCS groups (no statistics).</li> </ul> No. of patients: 37 GR/MD (%): 61.5 SD (%): 15.4 VS/D (%): 23.1 GCS 3-5, absent cisterns/3rd ventricle: 11 GCS 3-5, present cisterns/3rd ventricle: 5 GCS 6-8, absent cisterns/3rd ventricle: 8 GCS 6-8, present cisterns/3rd ventricle: 13

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion	
Tseng (63)	32	III All GCS	Surgery and nonsurgical	GOS 1 yr	Retrospective series of 32 patients with DTIC to evaluate clinical and radiologic factors affecting outcome.	<ul style="list-style-type: none"> <li>Hematoma volume, cisternal effacement, timing of detection, occurrence of clinical deterioration, and GCS at time of follow-up CT were related significantly to outcome.</li> <li>16% overall mortality.</li> </ul>	
					<p>GCS 3-7 at follow-up CT<sup>a</sup></p> <p>GCS 8-12 at follow-up CT</p> <p>GCS 13-15 at follow-up CT</p> <p>Cistern effacement<sup>b</sup></p> <p>No cistern effacement</p>	<p>No. of patients</p> <p>GR/MD (%)</p> <p>SD/VS/D (%)</p> <p>12 33 67</p> <p>16 100 0</p> <p>4 100 0</p> <p>5 0 100</p> <p>27 88.9 11.1</p>	
Tseng (64)	32	III GCS 5-7	Craniotomy ± manual temporal lobe reduction	GOS 3 mo	Retrospective study of 32 patients with GCS 5-7. CT evidence for uncal herniation, anisocoria, and hemiparesis treated either with standard surgical procedure (i.e., craniotomy with hematoma evacuation and contusion resection) or with standard procedure plus manual reduction of herniated temporal lobe. GOS measured at ≥3 mo postop to compare outcome between groups. Reduction procedure described as gentle elevation of temporal lobe until tentorium is visualized and CSF egress is noted.	<ul style="list-style-type: none"> <li>Addition of temporal lobe reduction to the standard surgical procedure seemed to result in better outcome (no statistics; selection bias).</li> </ul>	
					<p>No. of patients</p> <p>CR (%)</p> <p>MD (%)</p> <p>SD (%)</p> <p>VS (%)</p> <p>D (%)</p> <p>10 20 70 0 10</p> <p>22 0 27 9 55</p>		
Uzzell et al. (65)	117	III GCS 4-6	Nonsurgical	GOS 6 mo; neuro-psychological mean 4.8 mo (n = 30)	Retrospective series of 117 patients with DAI, diffuse swelling, or focal injury by CT to characterize differential outcome. Neuropsychological testing was performed in a subset of 30 patients to characterize differential neuropsychological sequelae based on lesion type. Patients with SDH, EDH, SAH, or development of a second major lesion were excluded. DAI defined as "midline hemorrhages or multiple small hemorrhages at gray-white matter interfaces"; diffuse swelling defined as "narrowing or obliteration of ventricles or basilar cisterns"; focal injuries defined as "unilateral intracerebral hemorrhages or contusions."	<ul style="list-style-type: none"> <li>Patients with DAI had significantly higher mortality and significantly less "good recovery" than other groups.</li> <li>Memory and learning score but not intelligence or visuospatial speed score significantly differed among CT groups.</li> <li>Levels of memory, learning, and visuospatial speed were higher after diffuse swelling injuries, but improvement was less over time.</li> <li>Patients with DAI had greater improvement over time of memory, learning, and visuospatial speed.</li> <li>Patients with focal injuries had improvement over time in visuospatial speed, but not recall or learning.</li> <li>Diagnostic groups did not differ significantly in age, years of education, GCS, timing of neuropsychological testing, sex, handedness, or GOS at 6 mo.</li> </ul>	
					<p>CT group<sup>a</sup></p> <p>DAI</p> <p>Diffuse swelling</p> <p>Focal injury</p>	<p>No. of patients</p> <p>CR (%)</p> <p>MD (%)</p> <p>SD (%)</p> <p>VS (%)</p> <p>D (%)</p> <p>33 18 15 15 40</p> <p>34 62 17 6 15</p> <p>50 48 15 14 22</p>	
Vollmer et al. (66)	321	III All GCS	Surgery and nonsurgical	GOS 6 mo	Prospective study from 1984-1987 of patients with GCS 3-15 to evaluate effect of age on outcome in different populations, including patients with large (>15 cm <sup>3</sup> ) ICH requiring evacuation (n = 54) and patients with <15 cm <sup>3</sup> ICH (n = 267).	<ul style="list-style-type: none"> <li>Presence of ICH and of large ICH (&gt;15 cm<sup>3</sup>) increased significantly with age.</li> <li>Increasing age was independently predictive of poor outcome in large and small ICH subgroups.</li> </ul>	
					<p>ICH &lt;15 cm<sup>3</sup><sup>a</sup></p> <p>Age 16-25 yr</p> <p>Age 26-35 yr</p> <p>Age 36-45 yr</p> <p>Age 46-55 yr</p> <p>Age &gt;56 yr</p>	<p>No. of patients</p> <p>VS/D (%)</p> <p>267</p> <p>31.8</p> <p>27.7</p> <p>42.4</p> <p>62.2</p> <p>82.2</p>	

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class GCS	Treatment	Outcome	Description	Conclusion																																																	
Whitfield et al. (67)	26	III GCS 3-13	Bifrontal decompressive craniectomy	GOS 6 mo	Retrospective review of outcome for 26 patients undergoing bifrontal decompressive craniectomy for refractory intracranial hypertension (ICP > 30 mm Hg with CPP < 70 mm Hg, or ICP > 35 mm Hg irrespective of CPP), managed using a standardized CP/CRP treatment algorithm. Continuous CP, ABP, and CPP data were analyzed for 8 patients to examine effect of bifrontal decompressive craniectomy on these variables. Surgical technique described.	<ul style="list-style-type: none"> <li>61% of severe TBI subgroup had favorable outcome (GOS 4-5).</li> <li>ICP significantly reduced from 37.5 to 18.1 mm Hg (P = 0.003).</li> <li>CPP significantly increased.</li> <li>Amplitude of ICP waveform, amplitude of slow waves, and RAP coefficient (reflecting increased compensatory reserve) were significantly reduced by surgery in a subgroup of eight patients.</li> <li>Timing of surgery was not predictive of outcome.</li> </ul> <p>No. of patients</p> <table border="1"> <tr><td>GR/MD (%)</td><td>69</td><td>SD (%)</td><td>8</td><td>VS (%)</td><td>0</td><td>D (%)</td><td>23</td></tr> <tr><td></td><td>83.3</td><td></td><td>0</td><td></td><td>0</td><td></td><td>16.7</td></tr> <tr><td></td><td>61.1</td><td></td><td>11.1</td><td></td><td>0</td><td></td><td>27.8</td></tr> <tr><td></td><td>100</td><td></td><td>0</td><td></td><td>0</td><td></td><td>0</td></tr> <tr><td></td><td>66.7</td><td></td><td>0</td><td></td><td>0</td><td></td><td>33.3</td></tr> </table> <p>Timing of surgery</p> <table border="1"> <tr><td>No. of patients</td><td>Favorable (%)</td><td>Unfavorable (%)</td></tr> <tr><td>20</td><td>60</td><td>40</td></tr> <tr><td>6</td><td>100</td><td>0</td></tr> </table>	GR/MD (%)	69	SD (%)	8	VS (%)	0	D (%)	23		83.3		0		0		16.7		61.1		11.1		0		27.8		100		0		0		0		66.7		0		0		33.3	No. of patients	Favorable (%)	Unfavorable (%)	20	60	40	6	100	0
GR/MD (%)	69	SD (%)	8	VS (%)	0	D (%)	23																																																
	83.3		0		0		16.7																																																
	61.1		11.1		0		27.8																																																
	100		0		0		0																																																
	66.7		0		0		33.3																																																
No. of patients	Favorable (%)	Unfavorable (%)																																																					
20	60	40																																																					
6	100	0																																																					
Wu et al. (68)	35	III All GCS	Surgery	GOS 6 mo	Retrospective series of 489 patients treated surgically for intracranial hematomas to assess prognostic factors related to GOS at 6 mo. A subgroup of 35 patients with ICH is included.	<ul style="list-style-type: none"> <li>Postresuscitation GCS, preoperative change in pupillary size, No. of operations, No. of hematomas, and type of lesion were significantly related to surgical outcome.</li> <li>Good outcome: single, evacuated EDH &gt; ICH &gt; SDH.</li> </ul> <p>No. of patients</p> <table border="1"> <tr><td>GR (%)</td><td>MD (%)</td><td>SD (%)</td><td>VS (%)</td><td>D (%)</td></tr> <tr><td>60</td><td>22.9</td><td>8.6</td><td>2.8</td><td>5.7</td></tr> </table> <p>Acute ICH, evacuated</p>	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	60	22.9	8.6	2.8	5.7																																							
GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																			
60	22.9	8.6	2.8	5.7																																																			
Yamaki et al. (69)	48	III Unknown	Surgery and nonsurgical	GOS; time of maximal hematoma diameter	Retrospective review of 48 patients with traumatic ICH > 3-cm diameter whose first CT occurred within 6 h of injury to evaluate the timing of hematoma evolution and to assess outcome. CT scans of 32 nonoperated patients were used to assess natural history of hematoma evolution.	<ul style="list-style-type: none"> <li>All ICH &gt; 3-cm diameter developed within 24 h of injury.</li> <li>Only 56% of ICH &gt; 3-cm diameter developed within 6 h of injury.</li> <li>ICH reached maximal size in 84% of patients within 12 h.</li> </ul> <p>No. of patients</p> <table border="1"> <tr><td>GR (%)</td><td>MD (%)</td><td>SD (%)</td><td>VS (%)</td><td>D (%)</td></tr> <tr><td>58</td><td>14</td><td>2</td><td>3</td><td>28</td></tr> </table> <p>ICH, total</p> <table border="1"> <tr><td>No. of patients</td><td>GR (%)</td></tr> <tr><td>26</td><td>35</td></tr> </table> <p>ICH, operated</p> <table border="1"> <tr><td>No. of patients</td><td>GR (%)</td></tr> <tr><td>32</td><td>13</td></tr> </table> <p>Nonoperated</p> <table border="1"> <tr><td>No. of patients</td><td>GR (%)</td></tr> <tr><td>26</td><td>46</td></tr> </table> <p>Operated</p> <table border="1"> <tr><td>No. of patients</td><td>GR (%)</td></tr> <tr><td>16</td><td>31</td></tr> </table> <p>Admitted immediately after TBI</p> <table border="1"> <tr><td>No. of patients</td><td>GR (%)</td></tr> <tr><td>10</td><td>70</td></tr> </table> <p>Admitted &gt; 6 h after TBI</p>	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	58	14	2	3	28	No. of patients	GR (%)	26	35	No. of patients	GR (%)	32	13	No. of patients	GR (%)	26	46	No. of patients	GR (%)	16	31	No. of patients	GR (%)	10	70																			
GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																			
58	14	2	3	28																																																			
No. of patients	GR (%)																																																						
26	35																																																						
No. of patients	GR (%)																																																						
32	13																																																						
No. of patients	GR (%)																																																						
26	46																																																						
No. of patients	GR (%)																																																						
16	31																																																						
No. of patients	GR (%)																																																						
10	70																																																						
Young et al. (72)	15	III Grady Coma Scale 1-5	Surgery	Modified GOS; not specified	Retrospective series after introduction of CT of 15 patients treated surgically for DTICH to examine clinical and radiologic correlates, and to relate clinical status to outcome.	<ul style="list-style-type: none"> <li>Morbidity/mortality associated with coma score on admission (no statistics).</li> <li>80% occipital/parietal/occipital impact; contrecoup frontotemporal lesions.</li> <li>Definition of DTICH: develops in area of previous contusion.</li> </ul> <p>No. of patients</p> <table border="1"> <tr><td>Intact (%)</td><td>Mild disability (%)</td><td>Significant disability D (%)</td></tr> <tr><td>6</td><td>83.3</td><td>16.7</td></tr> <tr><td>9</td><td>0</td><td>22.2</td></tr> <tr><td>15</td><td>33.3</td><td>6.7</td></tr> </table> <p>Grady 1-2</p> <table border="1"> <tr><td>No. of patients</td><td>Intact (%)</td><td>Significant disability D (%)</td></tr> <tr><td>6</td><td>83.3</td><td>16.7</td></tr> </table> <p>Grady 3-4</p> <table border="1"> <tr><td>No. of patients</td><td>Intact (%)</td><td>Significant disability D (%)</td></tr> <tr><td>9</td><td>0</td><td>22.2</td></tr> </table> <p>DTICH, total</p> <table border="1"> <tr><td>No. of patients</td><td>Intact (%)</td><td>Significant disability D (%)</td></tr> <tr><td>15</td><td>33.3</td><td>6.7</td></tr> </table>	Intact (%)	Mild disability (%)	Significant disability D (%)	6	83.3	16.7	9	0	22.2	15	33.3	6.7	No. of patients	Intact (%)	Significant disability D (%)	6	83.3	16.7	No. of patients	Intact (%)	Significant disability D (%)	9	0	22.2	No. of patients	Intact (%)	Significant disability D (%)	15	33.3	6.7																			
Intact (%)	Mild disability (%)	Significant disability D (%)																																																					
6	83.3	16.7																																																					
9	0	22.2																																																					
15	33.3	6.7																																																					
No. of patients	Intact (%)	Significant disability D (%)																																																					
6	83.3	16.7																																																					
No. of patients	Intact (%)	Significant disability D (%)																																																					
9	0	22.2																																																					
No. of patients	Intact (%)	Significant disability D (%)																																																					
15	33.3	6.7																																																					
Zumkeller et al. (73)	53	III GCS < 5 to 11-15	Surgery and nonsurgical	Poor versus good, not specified	Retrospective series of 104 patients with ICH; 53 of whom presented with traumatic ICH, comparing operative versus nonoperative treatment with outcome.	<ul style="list-style-type: none"> <li>No statistical difference in outcome was found between operated and nonoperated groups.</li> </ul> <p>No. of patients</p> <table border="1"> <tr><td>Operated</td><td>No. of patients</td><td>Good (%)</td><td>Poor (%)</td></tr> <tr><td>31</td><td>31</td><td>71</td><td>29</td></tr> <tr><td>22</td><td>22</td><td>40.9</td><td>59.1</td></tr> <tr><td>17</td><td>17</td><td>70.6</td><td>29.4</td></tr> <tr><td>8</td><td>8</td><td>75</td><td>25</td></tr> </table> <p>Single lobe, operated</p> <p>Single lobe, non-operated</p> <p>P = n.s.</p>	Operated	No. of patients	Good (%)	Poor (%)	31	31	71	29	22	22	40.9	59.1	17	17	70.6	29.4	8	8	75	25																													
Operated	No. of patients	Good (%)	Poor (%)																																																				
31	31	71	29																																																				
22	22	40.9	59.1																																																				
17	17	70.6	29.4																																																				
8	8	75	25																																																				

<sup>a</sup> GCS, Glasgow Coma Scale; GOS, Glasgow outcome score; ICH, intracerebral hemorrhage; ICP, intracranial pressure; CT, computed tomographic; GR, good recovery; MD, moderate disability; VS, vegetative state; D, death; SD, severe disability; DTICH, delayed traumatic intracerebral hematoma; SDH, subdural hematoma; n.s., not significant; FIM, functional independence measure; EDH, epidural hematoma; LOC, loss of consciousness; TCDB, Traumatic Coma Data Bank; SEP, somatosensory evoked potential; CPP, cerebral perfusion pressure; AEP, acoustic evoked potential; STD, subtemporal decompression; TL, temporal lobectomy; MUC, multiple unilateral contusion; GBS, generalized brain swelling; DA, diffuse axonal injury; TBI, traumatic brain injury; SAH, subarachnoid hemorrhage; IVH, intraventricular hemorrhage; ABP, arterial blood pressure; RAP, renal artery pressure; ICU, intensive care unit; OR, operation.

**M. Ross Bullock, M.D., Ph.D.**

Department of Neurological Surgery,  
Virginia Commonwealth University  
Medical Center,  
Richmond, Virginia

**Randall Chesnut, M.D.**

Department of Neurological Surgery,  
University of Washington  
School of Medicine,  
Harborview Medical Center,  
Seattle, Washington

**Jamshid Ghajar, M.D., Ph.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David Gordon, M.D.**

Department of Neurological Surgery,  
Montefiore Medical Center,  
Bronx, New York

**Roger Hartl, M.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David W. Newell, M.D.**

Department of Neurological Surgery,  
Swedish Medical Center,  
Seattle, Washington

**Franco Servadei, M.D.**

Department of Neurological Surgery,  
M. Bufalini Hospital,  
Cesena, Italy

**Beverly C. Walters, M.D., M.Sc.**

Department of Neurological Surgery,  
New York University  
School of Medicine,  
New York, New York

**Jack Wilberger, M.D.**

Department of Neurological Surgery,  
Allegheny General Hospital,  
Pittsburgh, Pennsylvania

**Reprints requests:**

Jamshid Ghajar, M.D., Ph.D.,  
Brain Trauma Foundation,  
523 East 72nd Street,  
New York, NY 10021.  
Email: ghajar@braintrauma.org

## SURGICAL MANAGEMENT OF POSTERIOR FOSSA MASS LESIONS

### RECOMMENDATIONS

(see *Methodology*)

#### Indications

- Patients with mass effect on computed tomographic (CT) scan *or* with neurological dysfunction *or* deterioration referable to the lesion should undergo operative intervention. Mass effect on CT scan is defined as distortion, dislocation, or obliteration of the fourth ventricle; compression or loss of visualization of the basal cisterns, or the presence of obstructive hydrocephalus.
- Patients with lesions and no significant mass effect on CT scan and without signs of neurological dysfunction may be managed by close observation and serial imaging.

#### Timing

- In patients with indications for surgical intervention, evacuation should be performed as soon as possible because these patients can deteriorate rapidly, thus, worsening their prognosis.

#### Methods

- Suboccipital craniectomy is the predominant method reported for evacuation of posterior fossa mass lesions, and is therefore recommended.

**KEY WORDS:** Cerebellum, Coma, Computed tomographic parameters, Contusion, Head injury, Occipital, Posterior fossa, Surgical technique, Timing of surgery, Traumatic brain injury

*Neurosurgery* 58:S2-47-S2-55, 2006

DOI: 10.1227/01.NEU.0000210366.36914.38

www.neurosurgery-online.com

### OVERVIEW

Posterior fossa injury is rare, occurring in less than 3% of head injuries in most published series (8, 12, 22). The vast majority of these series deal exclusively with posterior fossa epidural hematomas (EDH), representing 1.2 to 12.9% of all EDH (7, 20, 21, 30). A small number of observational studies address subdural and intraparenchymal hematomas of the posterior fossa (5, 8, 17, 22, 24), representing 0.5 to 2.5% and 1.7% of all subdural hematomas and intraparenchymal hematomas, respectively (22, 24). Additionally, there is a separate literature that focuses on parturitional hemorrhages, up to 48% of which primarily involve the posterior fossa (27). Because of the physiology and anatomy of the neonate, and the unique mechanism of these injuries, this subgroup of patients warrants independent analysis, and will not be addressed in these *Guidelines*.

Despite the rarity of these lesions, the importance of timely recognition and surgical evacu-

ation, when indicated, cannot be overstated. Many patients can undergo rapid clinical deterioration because of the limited size of the posterior fossa and the propensity for these lesions to produce brainstem compression.

### PROCESS

A MEDLINE computer search using the following key words: "posterior fossa" or "cerebellum" or "cerebellar" or "occipital" and "subdural" or "epidural" or "extradural" or "intradural" or "parenchymal" or "intraparenchymal" or "intracerebellar" or "fracture" between 1975 and 2001 was performed. A total of 1828 documents were found. The search was narrowed to include the key words: "surgery" or "operative" or "craniotomy" or "craniectomy" or "decompressive craniectomy" or "repair" and "trauma" or "traumatic" or "TBI" or "CHI." A total of 430 articles were found. A tertiary search adding the key words "contu-



sion," "hemorrhagic contusion," "surgical decompression," "craniostomy," "TICH," and "DTICH" was performed, yielding 421 articles. The secondary and tertiary searches were combined, yielding a total of 433 articles. In addition, the reference lists of selected articles were reviewed, and 24 articles were selected for critical analysis. The results of this analysis were incorporated into the review presented here. Papers primarily addressing the following topics were not included: nontraumatic lesions, patients with associated posterior fossa anomalies (e.g., Chiari malformation), posttraumatic aneurysms, chronic subdural hematomas, vertebral artery dissection, patients undergoing anticoagulation therapy, patients with associated illnesses (e.g., acquired immunodeficiency syndrome, idiopathic thrombocytopenia purpura, hemophilia, arteriovenous malformation, after craniotomy, or von Willebrand's disease), pre-CT era reports, and book chapters. In general, papers with the following characteristics were also excluded: case series with less than 10 patients evaluated by CT scan and with incomplete outcome data (mortality or Glasgow outcome score [GOS]), case reports, and operative series with operations occurring longer than 14 days from injury. Several articles with case series of less than 10 patients were examined and reviewed because of the limited number of patient series evaluating primary traumatic posterior fossa mass lesions that exist in the literature. Selected articles were evaluated for design, prognostic significance, therapeutic efficacy, and overall outcome. In addition, several articles were reviewed for the purposes of historical perspective.

### SCIENTIFIC FOUNDATION

Because of the rapid and life-threatening nature of neurological deterioration secondary to expanding mass lesions in the limited compartment of the posterior fossa, surgery is generally viewed as required therapy in symptomatic patients with progressive dysfunction. Because of the potential adverse consequences of withholding or delaying surgery for such patients, studies depend on retrospective analyses. As a result, there is no Class I or Class II evidence to support recommendations for the surgical management of these injuries. However, the predominantly observational studies that were reviewed yield an important and relatively clear picture of the prognosis for the patient with a posterior fossa mass lesion as patients are currently managed. Admission Glasgow Coma Scale (GCS) score (5, 7, 8, 12, 24, 26) and GCS score at surgery (6, 14, 19, 20, 22) correlate with outcome (GOS and mortality). D'Avella et al. (5) retrospectively reviewed the clinical and radiographic characteristics of 81 patients with traumatic intracerebellar hemorrhages. Subjecting their data to multivariate analysis, they found that only GCS and the presence of concomitant supratentorial lesions independently predicted outcome at 6 months. Outcome was favorable (GOS, 4 or 5) in 95% of patients with admission GCS score of at least 8, whereas outcome was poor (GOS, 1-3) in 81% of patients with a GCS score less than 8. Class III data suggests that a neurologically deteriorating patient should undergo emergent evacuation of the mass lesion.

Neurologically intact patients with a posterior fossa lesion and no CT evidence for mass effect (compression of cisterns, distortion of 4th ventricle, hydrocephalus) have been successfully managed nonoperatively with close observation and serial imaging (1, 5, 15, 25, 28).

Wong (28) conducted a retrospective study of 25 patients with posterior fossa EDH and compared clinical and radiological characteristics and outcomes between 17 patients undergoing early surgery and 8 patients managed nonoperatively. Patients with a posterior fossa EDH of at most 10 cm<sup>3</sup> in volume, at most 15 mm in thickness, and responsible for at most a 5-mm midline shift had excellent survival rates with either surgical or nonsurgical treatment. Patients managed nonsurgically with a posterior fossa EDH greater than 10 cm<sup>3</sup> in volume, greater than 15 mm in thickness, and responsible for greater than 5 mm of midline shift had a significantly greater mortality than those with similar CT characteristics undergoing early surgery. The disparity in mortality, however, is confounded by the strong correlation between the presence of an associated frontal lesion (which was found more commonly in the latter group) and death. This report raises the concept of conservative management for patients with posterior fossa lesions on the basis of objective CT characteristics. This concept is also supported by a two-center study performed by Bozbuga et al. (1). The authors divided patients into management groups on the basis of CT characteristics. All patients (n = 14) without evidence of mass effect on CT scan, defined by obliteration of the perimesencephalic cisterns, compression and/or displacement of the fourth ventricle, or the presence of hydrocephalus, were managed nonoperatively and had a good outcome (GOS, 5). According to the authors, these objective criteria were "earlier, more predictive, and more reliable" than the clinical findings, although no statistics regarding this statement were performed. Several other case series add additional support (9, 16, 25).

There are several prognostic factors that adversely affect outcome regardless of management. These include the presence of associated intracranial lesions (4, 5, 12, 14, 18-22, 24, 28), extension of an infratentorial lesion into the supratentorial compartment (19), the location of the lesion (e.g., intraparenchymal versus extra-axial, and midline versus hemispheric) (17, 22), the presence or absence of associated hydrocephalus (7, 8, 14), and the acuity of presentation, with subacute presentation portending a better outcome than acute presentation (4, 14, 19, 23-25). There are no controlled studies measuring the impact of these variables on surgical versus nonsurgical management of posterior fossa mass lesions.

### SUMMARY

There are no controlled, prospective clinical trials of treatment using surgical versus nonsurgical management of posterior fossa mass lesions. The available data support rapid evacuation of posterior fossa mass lesions that 1) show CT evidence of mass effect, or 2) result in progressive neurological dysfunction. Moreover, data support expectant management

with serial imaging for select cases in which there is neurological stability and no radiological evidence for mass effect.

## KEY ISSUES FOR FUTURE INVESTIGATION

There are several patient groups in which the distinction between surgical and conservative management is blurred. One such group includes patients who present with neurological deficit and a traumatic posterior fossa mass lesion without clinical evidence for neurological deterioration or radiological evidence for mass effect. Conversely, another group includes the neurologically intact patient with radiological evidence for mass effect from an offending hematoma. These groups have not been adequately addressed in the current literature, and, when reported, are managed at the discretion of the individual neurosurgeon, thus, precluding an accurate assessment of efficacy of treatment. The literature contains methodological problems outlined in this supplement that preclude the establishment of management standards, and even of treatment guidelines, for posterior fossa injury. Most series present prognostic data regarding outcome after either conservative or surgical treatment of posterior fossa mass lesions. In those few studies that attempt to compare outcomes, important prognostic factors known to be relevant to TBI outcome, such as cardiorespiratory instability, other systemic injuries, comorbidities, etc. (2), are not controlled between the surgical and nonsurgical cohorts. As a result, we have important prognostic information regarding operatively and nonoperatively managed posterior fossa injury, but no means for valid, direct comparison between the two. This comparison is essential if we are attempting to establish a standard of care. Thus, attention needs to be directed to controlled studies of patients with similar CT and clinical characteristics who are managed with operative versus nonoperative intervention.

## REFERENCES

- Bozbuga M, Izgi N, Polat G, Gurel I: Posterior fossa epidural hematomas: Observations on a series of 73 cases. *Neurosurg Rev* 22:34-40, 1999.
- Brain Trauma Foundation, American Association of Neurological Surgeons, Joint Section on Neurotrauma and Critical Care: Guidelines for the management of severe traumatic brain injury. *J Neurotrauma* 17:457-462, 2000.
- Brambilla G, Rainoldi F, Gipponi D, Paoletti P: Extradural haematoma of the posterior fossa: A report of eight cases and a review of the literature. *Acta Neurochir (Wien)* 80:24-29, 1986.
- Ciurea AV, Nuteanu L, Simionescu N, Georgescu S: Posterior fossa extradural hematomas in children: Report of nine cases. *Childs Nerv Syst* 9:224-228, 1993.
- d'Avella D, Servadei F, Scerrati M, Tomei G, Brambilla G, Angileri FF, Massaro F, Cristofori L, Tartara F, Pozzati E, Bruni P, Delfini R, Tomasello F: Traumatic intracerebellar hemorrhages: A clinicoradiological analysis of 81 cases. *Neurosurgery* 50:16-25, 2002.
- Ersahin Y, Mutluger S: Posterior fossa extradural hematomas in children. *Pediatr Neurosurg* 19:31-33, 1993.
- Holzschuh M, Schuknecht B: Traumatic epidural haematomas of the posterior fossa: 20 new cases and a review of the literature since 1961. *Br J Neurosurg* 3:171-180, 1989.
- Karasawa H, Furuya H, Naito H, Sugiyama K, Ueno J, Kin H: Acute hydrocephalus in posterior fossa injury. *J Neurosurg* 86:629-632, 1997.
- Kawakami Y, Tamiya T, Tanimoto T, Shimamura Y, Hattori S, Ueda T, Ishida T: Nonsurgical treatment of posterior fossa epidural hematoma. *Pediatr Neurol* 6:112-118, 1990.
- Koc RK, Pasaoglu A, Menku A, Oktem S, Meral M: Extradural hematoma of the posterior cranial fossa. *Neurosurg Rev* 21:52-57, 1998.
- Lui T, Lee S, Chang C, Cheng W: Epidural hematomas in the posterior cranial fossa. *J Trauma* 34:211-215, 1993.
- Mahajan R, Sharma B, Khosla V, Tewari M, Mathuriya S, Pathak A, Kak V: Posterior fossa extradural haematoma—Experience of nineteen cases. *Ann Acad Med Singapore* 22:410-413, 1993.
- Mohanty A, Kolluri V, Subbakrishna D, Satish S, Mouli B, Das B: Prognosis of extradural haematomas in children. *Pediatr Neurosurg* 23:57-63, 1995.
- Neubauer UJ: Extradural haematoma of the posterior fossa. Twelve years experiences with CT-scan. *Acta Neurochir (Wien)* 87:105-111, 1987.
- Otsuka S, Nakatsu S, Matsumoto S, Sato S, Motozaki T, Ban S, Yamamoto T: Study on cases with posterior fossa epidural hematoma—Clinical features and indications for operation. *Neurol Med Chir (Tokyo)* 30:24-28, 1990.
- Pang D, Horton J, Herron J, Wilberger JJ, Vries J: Nonsurgical management of extradural hematomas in children. *J Neurosurg* 59:958-971, 1983.
- Pozzati E, Grossi C, Padovani R: Traumatic intracerebellar hematomas. *J Neurosurg* 56:691-694, 1982.
- Pozzati E, Tognetti F, Cavallo M, Acciarri N: Extradural hematomas of the posterior cranial fossa. Observations on a series of 32 consecutive cases treated after the introduction of computed tomography scanning. *Surg Neurol* 32:300-303, 1989.
- Prusty GK, Mohanty A: Posterior fossa extradural haematoma. *J Indian Med Assoc* 93:255-258, 1995.
- Rivano C, Altomonte M, Capuzzo T, Borzone M: Traumatic posterior fossa extradural hematomas. A report of 22 new cases surgically treated and a review of the literature. *Zentralbl Neurochir* 52:77-82, 1991.
- Roda J, Gimenez D, Perez-Higueras A, Blazquez M, Perez-Alvarez M: Posterior fossa epidural hematomas: A review and synthesis. *Surg Neurol* 19:419-424, 1983.
- Sripairojkul B, Saeheng S, Ratanalert S, Pheunpathom N, Sriplung H: Traumatic hematomas of the posterior cranial fossa. *J Med Assoc Thai* 81:153-159, 1998.
- St John JN, French BN: Traumatic hematomas of the posterior fossa. A clinicopathological spectrum. *Surg Neurol* 25:457-466, 1986.
- Tsai FY, Teal JS, Itabashi HH, Huprich JE, Hieshima GB, Segall HD: Computed tomography of posterior fossa trauma. *J Comput Assist Tomogr* 4:291-305, 1980.
- Vrankovic DJ, Splavski B, Hecimovic I, Kristek B: Acute traumatic hematomas of the posterior fossa: Experience with eleven cases. *Neurol Croat* 43:21-30, 1994.
- Wang EC, Lim AY, Yeo IT: Traumatic posterior fossa extradural haematomas (PFEDH). *Singapore Med J* 39:107-111, 1998.
- Welch K, Strand R: Traumatic parturitional intracranial hemorrhage. *Dev Med Child Neurol* 28:156-164, 1986.
- Wong CW: The CT criteria for conservative treatment—but under close clinical observation—of posterior fossa epidural haematomas. *Acta Neurochir (Wien)* 126:124-127, 1994.
- Zuccarello M, Andrioli G, Fiore D, Longatti P, Pardatscher K, Zampieri P: Traumatic posterior fossa haemorrhage in children. *Acta Neurochir (Wien)* 62:79-85, 1982.
- Zuccarello M, Pardatscher K, Andrioli GC, Fiore DL, Iavicoli R, Cervellini P: Epidural hematomas of the posterior cranial fossa. *Neurosurgery* 8:434-437, 1981.



TABLE 1. Surgical management of posterior fossa mass lesions\*

Authors (ref. no.)	No. of patients	Inclusion Class	GCS	Treatment	Outcome	Description	Conclusion
Bozbuga et al. (1)	73	III	Unknown	Surgery and nonsurgical	GOS, not specified	Prospective study of 73 patients with PFEDH managed by CT criteria either surgically or nonsurgically based on presence of signs of mass effect on CT.	<ul style="list-style-type: none"> <li>Patients with PFEDH and no associated CT evidence for mass effect may be managed effectively without surgery.</li> <li>No patient with mass effect on CT was managed nonsurgically, thus, precluding direct comparison between groups.</li> <li>No morbidity or mortality in subacute cases (presentation 24 h–7 d).</li> </ul>
Brambilla et al. (3)	8	III	GCS 4–15	Surgery	Death versus complete recovery, not specified	Retrospective series of 8 patients surgically treated for PFEDH to evaluate clinical characteristics and outcome.	<ul style="list-style-type: none"> <li>Authors emphasize high mortality rate thought secondary to concomitant brainstem and basal ganglia injury seen at autopsy. *Note: paper included with n = 8 because of limited number of case series with ≥10 patients.</li> </ul>
Ciurea et al. (4)	9	III	GCS 13–14	Surgery	GOS, not specified	Retrospective series of 9 patients aged 2–12, surgically treated for PFEDH to evaluate clinical characteristics and outcome.	<ul style="list-style-type: none"> <li>PFEDH</li> <li>Subacute presentation predominated (88.8%) and, thus, may have biased outcome data.</li> <li>Associated intracranial injuries seemed associated with worse outcome (no statistics). Note: paper included with n = 9 because of limited number of case series with ≥10 patients.</li> </ul>
d'Avella et al. (5)	81	III	GCS 3–5 to 14–15	Surgery and nonsurgical	GOS 6 mo	Retrospective series of 81 patients with traumatic intracerebellar hemorrhage/contusion to evaluate prognostic factors and examine clinico-radiological presentation.	<ul style="list-style-type: none"> <li>44.4% poor result (GOS 1–3); 55.6% favorable result (GOS 4–5).</li> <li>GCS, presence of supratentorial lesion, status of basal cisterns and fourth ventricle, mechanism of injury, and clot size correlated with outcome.</li> <li>GCS and presence of concomitant supratentorial lesion were independent prognostic factors.</li> <li>9.4 times relative risk of poor outcome with GCS &lt; 8.</li> <li>2.3 times relative risk of poor outcome with associated supratentorial lesion.</li> <li>Traumatic intracerebellar clots observed to increase in size up to 4 d after injury—authors advocate repeat CT scans until lesion stabilizes.</li> <li>Authors recommend surgery for patients with larger clots causing posterior fossa mass effect.</li> </ul>
Holzschuh and Schuknecht (7)	20	III	GCS 3–15	Surgery	GOS 6 mo	Retrospective series of 20 patients surgically treated for PFEDH to evaluate clinical and radiologic characteristics and outcome.	<ul style="list-style-type: none"> <li>GCS correlated with GOS (no statistics)</li> <li>Mortality of acute (&lt;24 h) and subacute (24 h–7 d) cases was 50% and 20%, respectively.</li> <li>Authors emphasize importance of CT for patients with signs of occipital trauma to diagnose PFEDH before clinical deterioration. Note: CT in 18/20 cases.</li> </ul>

No. of patients	GR (%)	MD (%)	D (%)
Nonsurgical	100	0	0
Surgical	86	7	7
Acute (<24 h)	54	85	7
Subacute (24 h–7 d)	19	100	0

No. of patients	GR (%)	MD (%)	D (%)
PFEDH	8	62.5	37.5
PFEDH + associated lesion	9	89	11
PFEDH + associated lesion	4	75	25

No. of patients	GR (%)	MD (%)	D (%)
Intracerebellar hemorrhage/contusion, total	81	55.6	44.4
GCS ≥ 8	39	94	6
GCS < 8 <sup>b</sup>	42	19	81
OR <sup>c</sup>	27	48	52

No. of patients	GR (%)	MD (%)	D (%)
PFEDH, total	20	45	20
GCS 15–14	2	100	0
GCS 13–8	8	62.5	12.5
GCS 7–3	10	20	30
Acute (<24 h)	14	28.6	21.4
Subacute (24 h–7 d)	5	60	20
Chronic (>7 d)	1	100	0

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion																																																																																										
Karasawa et al. (8)	53	III GCS 3-8 to 13-15	Surgery and nonsurgical	GOS at discharge	Retrospective series of 53 patients with posterior fossa injury, comparing those with and without associated ICH to evaluate CT scans associated with the development of HCP and the differences in outcome between subgroups with and without HCP.	<ul style="list-style-type: none"> <li>• Supratentorial extension, hematoma thickness &gt;15 mm, and abnormal mesencephalic cisterns were significantly associated with HCP (<math>P &lt; 0.05</math>).</li> <li>• Bilateral spongiform degeneration and inability to visualize the fourth ventricle were significantly associated with HCP in intracerebellar hematomas/contusions.</li> <li>• No significant difference in mortality between patients with PFEDH with or without HCP.</li> <li>• HCP increased mortality in patients with ICH/contusion (100% versus 15.4%, <math>P &lt; 0.05</math>).</li> <li>• Surgery did not alter mortality in patients with associated HCP, though number of patients was too small for comparison.</li> <li>• Admission GCS was inversely related to mortality (<math>P = n.s.</math>).</li> <li>• Authors advocate external ventricular drainage in patients with ICH/contusion and HCP, not with PFEDH.</li> </ul>																																																																																										
Koc et al. (10)	11	III GCS 7-15	Surgery	GCS at discharge	Retrospective series of 14 patients (11 with CT scans) treated surgically for PFEDH to examine clinical and radiologic features and to assess outcome.	<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Posterior fossa injury, total</td> <td>53</td> <td>74</td> <td>7</td> <td>2</td> <td>2</td> <td>15</td> </tr> <tr> <td>EDH</td> <td>25</td> <td>88</td> <td>8</td> <td>0</td> <td>0</td> <td>4</td> </tr> <tr> <td>ICH/contusion</td> <td>13</td> <td>46.2</td> <td>0</td> <td>7.7</td> <td>7.7</td> <td>38.5</td> </tr> <tr> <td>SAH</td> <td>7</td> <td>100</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> </tr> <tr> <td>SDH</td> <td>5</td> <td>80</td> <td>0</td> <td>0</td> <td>0</td> <td>20</td> </tr> <tr> <td>Brainstem hemorrhage</td> <td>3</td> <td>0</td> <td>66.7</td> <td>0</td> <td>0</td> <td>33.3</td> </tr> <tr> <td>EDH + surgery + HCP</td> <td>7</td> <td>86</td> <td>14</td> <td>0</td> <td>0</td> <td>0</td> </tr> <tr> <td>EDH + surgery, no HCP</td> <td>7</td> <td>71</td> <td>14</td> <td>0</td> <td>0</td> <td>14</td> </tr> <tr> <td>ICH + surgery + HCP</td> <td>2</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> <td>100</td> </tr> <tr> <td>ICH + surgery, no HCP</td> <td>1</td> <td>100</td> <td>0</td> <td>0</td> <td>0</td> <td>0</td> </tr> <tr> <td>HCP + surgery</td> <td>9</td> <td>66.7</td> <td>11.1</td> <td>0</td> <td>0</td> <td>22.2</td> </tr> <tr> <td>HCP, no surgery</td> <td>3</td> <td>66.7</td> <td>0</td> <td>0</td> <td>0</td> <td>33.3</td> </tr> </tbody> </table> <p>Authors report that lesion size, presence of coexisting intracranial lesions, and increasing age are not associated with prognosis, however, all morbidity and mortality occurred in patients without CT scans.</p>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Posterior fossa injury, total	53	74	7	2	2	15	EDH	25	88	8	0	0	4	ICH/contusion	13	46.2	0	7.7	7.7	38.5	SAH	7	100	0	0	0	0	SDH	5	80	0	0	0	20	Brainstem hemorrhage	3	0	66.7	0	0	33.3	EDH + surgery + HCP	7	86	14	0	0	0	EDH + surgery, no HCP	7	71	14	0	0	14	ICH + surgery + HCP	2	0	0	0	0	100	ICH + surgery, no HCP	1	100	0	0	0	0	HCP + surgery	9	66.7	11.1	0	0	22.2	HCP, no surgery	3	66.7	0	0	0	33.3
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																																																											
Posterior fossa injury, total	53	74	7	2	2	15																																																																																										
EDH	25	88	8	0	0	4																																																																																										
ICH/contusion	13	46.2	0	7.7	7.7	38.5																																																																																										
SAH	7	100	0	0	0	0																																																																																										
SDH	5	80	0	0	0	20																																																																																										
Brainstem hemorrhage	3	0	66.7	0	0	33.3																																																																																										
EDH + surgery + HCP	7	86	14	0	0	0																																																																																										
EDH + surgery, no HCP	7	71	14	0	0	14																																																																																										
ICH + surgery + HCP	2	0	0	0	0	100																																																																																										
ICH + surgery, no HCP	1	100	0	0	0	0																																																																																										
HCP + surgery	9	66.7	11.1	0	0	22.2																																																																																										
HCP, no surgery	3	66.7	0	0	0	33.3																																																																																										
Lui et al. (11)	89	III All GCS	Surgery	GOS, 1 yr; mortality, 1 wk	Retrospective review of 89 patients treated surgically for PFEDH to evaluate outcome related to CT characteristics, age, GCS.	<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>PFEDH with CT</td> <td>11</td> <td>100</td> <td></td> <td></td> <td>0</td> </tr> <tr> <td>PFEDH, total</td> <td>89</td> <td>74.2</td> <td>5.6</td> <td>1.1</td> <td>1.1</td> </tr> <tr> <td>GCS (surgery) 3-5</td> <td>17</td> <td></td> <td></td> <td></td> <td>17.9</td> </tr> <tr> <td>GCS 6-8</td> <td>19</td> <td></td> <td></td> <td></td> <td>64.7</td> </tr> <tr> <td>GCS 9-12</td> <td>29</td> <td></td> <td></td> <td></td> <td>5.2</td> </tr> <tr> <td>GCS 13-15</td> <td>24</td> <td></td> <td></td> <td></td> <td>13.8</td> </tr> <tr> <td>Age &lt;16 yr</td> <td>29</td> <td></td> <td></td> <td></td> <td>0</td> </tr> <tr> <td>Age ≥16 yr</td> <td>60</td> <td></td> <td></td> <td></td> <td>10.3</td> </tr> <tr> <td></td> <td></td> <td></td> <td></td> <td></td> <td>21.7</td> </tr> </tbody> </table> <p> <ul style="list-style-type: none"> <li>• Mortality 17.9%.</li> <li>• Mortality inversely related to GCS before surgery.</li> <li>• Age &lt;16 yr associated with decreased mortality.</li> <li>• Presence of mixed lesion (i.e., extension into occipital region) and concomitant, noncontiguous supratentorial lesion associated with increased mortality.</li> </ul> </p>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	PFEDH with CT	11	100			0	PFEDH, total	89	74.2	5.6	1.1	1.1	GCS (surgery) 3-5	17				17.9	GCS 6-8	19				64.7	GCS 9-12	29				5.2	GCS 13-15	24				13.8	Age <16 yr	29				0	Age ≥16 yr	60				10.3						21.7																														
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																																																											
PFEDH with CT	11	100			0																																																																																											
PFEDH, total	89	74.2	5.6	1.1	1.1																																																																																											
GCS (surgery) 3-5	17				17.9																																																																																											
GCS 6-8	19				64.7																																																																																											
GCS 9-12	29				5.2																																																																																											
GCS 13-15	24				13.8																																																																																											
Age <16 yr	29				0																																																																																											
Age ≥16 yr	60				10.3																																																																																											
					21.7																																																																																											

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion																																																
Mahajan et al. (12)	19	III GCS < 7 to 13-15	Surgery	GOS, not specified	Retrospective series of 19 patients treated surgically for PFEDH to examine clinical characteristics and outcome.	<ul style="list-style-type: none"> <li>● Mortality 15.8%.</li> <li>● Admission GCS correlated with outcome (no statistics).</li> <li>● Both patients with concomitant supratentorial intracranial lesions died.</li> </ul>																																																
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>PFEDH, total</td> <td>19</td> <td>57.9</td> <td>26.3</td> <td>0</td> <td>15.8</td> </tr> <tr> <td>GCS (admission) 15-13</td> <td>7</td> <td>100</td> <td>0</td> <td>0</td> <td>0</td> </tr> <tr> <td>GCS 12-8</td> <td>7</td> <td>57.1</td> <td>28.6</td> <td>0</td> <td>14.3</td> </tr> <tr> <td>GCS 7-3</td> <td>5</td> <td>0</td> <td>60</td> <td>0</td> <td>40</td> </tr> </tbody> </table>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	PFEDH, total	19	57.9	26.3	0	15.8	GCS (admission) 15-13	7	100	0	0	0	GCS 12-8	7	57.1	28.6	0	14.3	GCS 7-3	5	0	60	0	40																		
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																	
PFEDH, total	19	57.9	26.3	0	15.8																																																	
GCS (admission) 15-13	7	100	0	0	0																																																	
GCS 12-8	7	57.1	28.6	0	14.3																																																	
GCS 7-3	5	0	60	0	40																																																	
Mohanty et al. (13)	24	III GCS 3-5 to 13-15	Surgery	GOS at discharge	Retrospective series of 489 patients with surgically evacuated EDH comparing prognosis between children and adults and evaluating difference in associated clinical parameters between children and adults. A subgroup of 24 patients with surgically treated PFEDH is included.	<ul style="list-style-type: none"> <li>● Incidence of PFEDH/EDH higher in children versus adults (11% versus 3%).</li> <li>● PFEDH had better outcome versus supratentorial EDH in both age groups (no statistics).</li> </ul>																																																
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>CR/MD (%)</th> <th>SD/VS/D (%)</th> </tr> </thead> <tbody> <tr> <td>PFEDH, total</td> <td>24</td> <td>87.5</td> <td>12.5</td> </tr> <tr> <td>age ≤ 15 yr</td> <td>11</td> <td>91</td> <td>9</td> </tr> <tr> <td>age ≥ 16 yr</td> <td>13</td> <td>85</td> <td>15</td> </tr> </tbody> </table>	No. of patients	CR/MD (%)	SD/VS/D (%)	PFEDH, total	24	87.5	12.5	age ≤ 15 yr	11	91	9	age ≥ 16 yr	13	85	15																																	
No. of patients	CR/MD (%)	SD/VS/D (%)																																																				
PFEDH, total	24	87.5	12.5																																																			
age ≤ 15 yr	11	91	9																																																			
age ≥ 16 yr	13	85	15																																																			
Neubauer (14)	18	III GCS 3-14	Surgery	GOS not specified	Retrospective series of 18 patients surgically treated for PFEDH to evaluate outcome relative to acuity of presentation, presence of hydrocephalus, and presence of associated lesions.	<ul style="list-style-type: none"> <li>● Mortality 22%—all presented with acute course.</li> <li>● GCS immediately before surgery, acute presentation, and presence of hydrocephalus seemed to correlate with mortality (no statistics).</li> <li>● Concomitant lesions did not alter mortality but correlated with increased disability (no statistics).</li> </ul>																																																
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>PFEDH, total</td> <td>18</td> <td>55</td> <td>11</td> <td>5.5</td> <td>22</td> </tr> <tr> <td>With associated lesion</td> <td>8</td> <td>25</td> <td>25</td> <td>12.5</td> <td>25</td> </tr> <tr> <td>No associated lesion</td> <td>10</td> <td>80</td> <td>0</td> <td>0</td> <td>20</td> </tr> <tr> <td>Acute (&lt;24 h)</td> <td>10</td> <td></td> <td></td> <td></td> <td>40</td> </tr> <tr> <td>Subacute (2-7 d)</td> <td>8</td> <td></td> <td></td> <td></td> <td>0</td> </tr> <tr> <td>With HCP</td> <td>6</td> <td></td> <td></td> <td></td> <td>33</td> </tr> <tr> <td>No HCP</td> <td>12</td> <td></td> <td></td> <td></td> <td>16</td> </tr> </tbody> </table>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	PFEDH, total	18	55	11	5.5	22	With associated lesion	8	25	25	12.5	25	No associated lesion	10	80	0	0	20	Acute (<24 h)	10				40	Subacute (2-7 d)	8				0	With HCP	6				33	No HCP	12				16
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																	
PFEDH, total	18	55	11	5.5	22																																																	
With associated lesion	8	25	25	12.5	25																																																	
No associated lesion	10	80	0	0	20																																																	
Acute (<24 h)	10				40																																																	
Subacute (2-7 d)	8				0																																																	
With HCP	6				33																																																	
No HCP	12				16																																																	
Otsuka et al. (15)	11	III All GCS	Surgery and nonsurgical	GOS at discharge	Retrospective series of 11 patients with PFEDH to evaluate clinical and radiologic features, and to compare outcome between operated and nonoperated groups.	<ul style="list-style-type: none"> <li>● Mortality 18.2%.</li> <li>● Outcome was similar between groups when surgical indications were deterioration/nonimprovement of neurological signs and symptoms of headache, nausea, and vomiting in presence of CT parameters of maximal hematoma thickness &gt;15 mm, poor visualization of posterior fossa cisterns, marked deformity/displacement of IV ventricle, and extension of PFEDH into supratentorial region (no statistics).</li> </ul>																																																
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>MD (%)</th> <th>SD (%)</th> <th>VS (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Surgery</td> <td>6</td> <td>66.7</td> <td>16.7</td> <td>0</td> <td>16.7</td> </tr> <tr> <td>Nonsurgical</td> <td>5</td> <td>80</td> <td>0</td> <td>0</td> <td>20</td> </tr> </tbody> </table>	No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	Surgery	6	66.7	16.7	0	16.7	Nonsurgical	5	80	0	0	20																														
No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)																																																	
Surgery	6	66.7	16.7	0	16.7																																																	
Nonsurgical	5	80	0	0	20																																																	
Pozzati et al. (17)	32	III GCS < 7 to 14 (14 point scale)	Surgery (n = 30) and nonsurgical (n = 2)	Modified GOS, not specified	Retrospective series of 32 patients with PFEDH, 30 of whom underwent surgery, to evaluate clinical characteristics and outcome.	<ul style="list-style-type: none"> <li>● Mortality, 15.6%.</li> <li>● Presence of associated intracranial lesion correlates with poor outcome (i.e., disability, mortality) compared with "pure" PFEDH (no statistics).</li> <li>● All patients with GOS &lt; 5 had associated lesions.</li> <li>● Nonsurgical management was successful in 2 patients with small clots.</li> </ul>																																																
						<table border="1"> <thead> <tr> <th>No. of patients</th> <th>GR (%)</th> <th>Disabled (%)</th> <th>D (%)</th> </tr> </thead> <tbody> <tr> <td>Pure PFEDH, operated</td> <td>14</td> <td>100</td> <td>0</td> <td>0</td> </tr> <tr> <td>Associated lesion, operated</td> <td>16</td> <td>50</td> <td>18.8</td> <td>31.3</td> </tr> <tr> <td>Small, nonsurgical</td> <td>2</td> <td>100</td> <td>0</td> <td>0</td> </tr> </tbody> </table>	No. of patients	GR (%)	Disabled (%)	D (%)	Pure PFEDH, operated	14	100	0	0	Associated lesion, operated	16	50	18.8	31.3	Small, nonsurgical	2	100	0	0																													
No. of patients	GR (%)	Disabled (%)	D (%)																																																			
Pure PFEDH, operated	14	100	0	0																																																		
Associated lesion, operated	16	50	18.8	31.3																																																		
Small, nonsurgical	2	100	0	0																																																		

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion
Pozzati et al. (18)	7	III GCS 5-11	Surgery and nonsurgical	GOS at discharge	Retrospective series of 7 patients to examine the role of CT in conservative versus surgical treatment of isolated intracerebellar hematoma.	<ul style="list-style-type: none"> <li>• Poorer outcome was associated with midline versus hemispheric clots (no statistics).</li> <li>• CT allowed successful conservative management of selected patients.</li> <li>• No good outcome was achieved with clot &gt; 3 cm. Note: paper included with n = 7 because of limited number of case series with ≥ 10 patients.</li> </ul>
Prusty and Mohanty (19)	17	III GCS 4-15	Surgery (n = 16) and nonsurgical (n = 1)	GOS at discharge and 6 mo	Retrospective series of 17 patients with PFEDH. 16 of whom underwent surgery, to examine clinical characteristics and outcome.	<ul style="list-style-type: none"> <li>• Mortality 17.6%—all patients who died presented acutely (&lt;24 h from injury) and had concomitant intracranial lesions.</li> <li>• GOS 5 at 6 mo for all patients presenting with subacute or chronic course. Note: CT performed in 14/17 patients.</li> </ul>
Rivano et al. (20)	22	III GCS 4-15	Surgery	GOS at discharge	Retrospective series of 22 patients undergoing surgery for PFEDH to evaluate clinical and radiologic features with respect to outcome.	<ul style="list-style-type: none"> <li>• Mortality 13.6%—all patients who died presented with acute clinical course.</li> <li>• GCS at surgery related to outcome (no statistics).</li> <li>• Presence of associated intracranial lesions decreased good outcome, increased morbidity, and increased mortality compared with patients with isolated PFEDH (no statistics). Note: 2/22 patients evaluated by angiography.</li> </ul>
Sripairojki et al. (22)	22	III GCS 3T-15	Surgery	GOS 6 mo	Retrospective series of 22 patients with surgically treated posterior fossa hematomas evaluating outcome by age, type of hematoma, and interval between injury and surgery.	<ul style="list-style-type: none"> <li>• Mortality 38%.</li> <li>• GCS &lt; 9 at time of surgery correlated with poor outcome.</li> <li>• Interval between injury and surgery &lt; 1 d correlated with better outcome than surgery &gt; 6 h—this may be caused by a selection of patients with subacute presentation.</li> <li>• Concomitant supratentorial and infratentorial lesions increased morbidity and mortality (mortality 20% versus 0%).</li> </ul>

TABLE 1. Continued

Authors (ref. no.)	No. of patients	Inclusion Class	Treatment	Outcome	Description	Conclusion						
						No. of patients	GR (%)	MD (%)	SD (%)	VS (%)	D (%)	
St. John and French (23)	8	III	Surgery	GOS, not specified	Retrospective series of 8 patients surgically treated for traumatic posterior fossa hematoma to evaluate clinical characteristics and outcome.	Age <40 yr	16	75	6.2	6.2	0	12.5
						Age ≥40 yr	6	16.7	16.7	0	50	
						GCS (surgery) 3-8	9	33.3	11.1	0	44.4	
						GCS 9-12	6	66.7	0	16.7	0	16.7
						GCS 13-15	7	85.7	14.3	0	0	0
						EDH	7	100	0	0	0	0
						EDH + associated lesion	9	55.6	22.2	0	0	22.2
						SDH	1	100	0	0	0	0
						SDH + ICH	1	0	0	100	0	0
						ICH	2	0	0	50	0	50
						ICH + supratentorial lesion	2	0	0	0	0	100
Tsat et al. (24)	57	All GCS	Surgery and nonsurgical	GOS variable	Retrospective series of 57 patients with primary posterior fossa injury to evaluate CT characteristics and correlate type of injury with prognosis.	Injury to OR ≤6 h	9	55.6	0	11.1	0	33.3
						Injury to OR 6 h-1 d	7	42.9	14.3	14.3	0	28.6
						Injury to OR >1 d	6	83.3	16.7	0	0	0
Vrankovic et al. (25)	11	Unknown	Surgery and nonsurgical	GOS at discharge	Retrospective series of 11 patients with ATPFH treated either surgically (n = 8) or nonoperatively (n = 3) to evaluate clinical and radiologic characteristics and outcome.	♂ Odds ratio, 0.057 (statistically significant). ● Mortality 25%. ● Subacute presentation seemed to correlate with better outcome, but number of patients too small to evaluate. Note: paper included with n = 8 because of limited number of case series with n ≥ 10 patients.						
						All patients	8	50	12.5	12.5	0	25
						Acute (<24 h)	6	33.3	16.7	16.7	0	33.3
						Subacute (24 h-7 d)	2	100	0	0	0	0
						● PFEDH patients who were comatose on admission died with or without surgery.						
						● 2/2 PFSDH patients with associated injury were comatose on admission, did not undergo surgery, and died.						
						● Evidence for brainstem injury, posterior fossa swelling/cistern obliteration correlated with increased mortality (no statistics).						
						PFEDH	14	50	0	0	0	50
						PFSDH	8	62.5	12.5	0	0	25
						Cerebellar injury	14	0	0	28.6	14.3	57.1
						Brainstem injury	21	0	0	0	23.8	76.2
● 9 of 11 patients had occipital fractures and 8.1% of patients with occipital fractures had ATPFH, emphasizing importance of CT for patients with signs of occipital trauma.												
● 3 patients with intracerebellar hematomas and no signs of mass effect on CT were successfully managed without surgery.												
● Authors advocate immediate surgery for all cases of PFEDH because of unpredictability of deterioration.												
All patients	11	54.5	36.4	0	0	9.1						





**M. Ross Bullock, M.D., Ph.D.**

Department of Neurological Surgery,  
Virginia Commonwealth University  
Medical Center,  
Richmond, Virginia

**Randall Chesnut, M.D.**

Department of Neurological Surgery,  
University of Washington  
School of Medicine,  
Harborview Medical Center,  
Seattle, Washington

**Jamshid Ghajar, M.D., Ph.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David Gordon, M.D.**

Department of Neurological Surgery,  
Montefiore Medical Center,  
Bronx, New York

**Roger Hartl, M.D.**

Department of Neurological Surgery,  
Weil Cornell Medical College of  
Cornell University,  
New York, New York

**David W. Newell, M.D.**

Department of Neurological Surgery,  
Swedish Medical Center,  
Seattle, Washington

**Franco Servadei, M.D.**

Department of Neurological Surgery,  
M. Bufalini Hospital,  
Cesena, Italy

**Beverly C. Walters, M.D., M.Sc.**

Department of Neurological Surgery,  
New York University  
School of Medicine,  
New York, New York

**Jack Wilberger, M.D.**

Department of Neurological Surgery,  
Allegheny General Hospital,  
Pittsburgh, Pennsylvania

**Reprints requests:**

Jamshid Ghajar, M.D., Ph.D.,  
Brain Trauma Foundation,  
523 East 72nd Street,  
New York, NY 10021.  
Email: ghajar@braintrauma.org

## SURGICAL MANAGEMENT OF DEPRESSED CRANIAL FRACTURES

### RECOMMENDATIONS

(see *Methodology*)

#### Indications

- Patients with open (compound) cranial fractures depressed greater than the thickness of the cranium should undergo operative intervention to prevent infection.
- Patients with open (compound) depressed cranial fractures may be treated nonoperatively if there is no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression greater than 1 cm, frontal sinus involvement, gross cosmetic deformity, wound infection, pneumocephalus, or gross wound contamination.
- Nonoperative management of closed (simple) depressed cranial fractures is a treatment option.

#### Timing

- Early operation is recommended to reduce the incidence of infection.

#### Methods

- Elevation and debridement is recommended as the surgical method of choice.
- Primary bone fragment replacement is a surgical option in the absence of wound infection at the time of surgery.
- All management strategies for open (compound) depressed fractures should include antibiotics.

**KEY WORDS:** Antibiotic prophylaxis, Burr hole, Cranial fracture, Craniotomy, Depressed cranial fracture, Depressed skull fracture, Head injury, Skull fracture, Surgical technique, Traumatic brain injury

*Neurosurgery* 58:S2-56-S2-60, 2006

DOI: 10.1227/01.NEU.0000210367.14043.0E

www.neurosurgery-online.com

### OVERVIEW

The presence of a cranial fracture has consistently been shown to be associated with a higher incidence of intracranial lesions, neurological deficit, and poorer outcome (4, 8, 12, 14). Indeed, Chan et al. (4) found cranial fracture to be the only independent significant risk factor in predicting intracranial hematomas in a cohort of 1178 adolescents. Macpherson et al. (12) found that 71% of 850 patients with a cranial fracture had an intracranial lesion (i.e., contusion or hematoma), compared with only 46% of 533 patients without a cranial fracture. Hung et al. (8) determined that patients with both loss of consciousness and cranial fracture were at significantly greater risk of developing a "surgically significant intracranial hematoma" than those with one or

neither condition. Servadei et al. (14) showed the importance of cranial fracture in predicting the presence of intracranial lesions, even in minor head injuries (Glasgow Coma Scale score 14 or 15). These studies underscore the importance of cranial fractures as indicators of clinically significant injuries, as well as the importance of computed tomographic (CT) scans in evaluation of all patients with known or clinically suspected cranial fractures.

Depressed cranial fractures may complicate up to 6% of head injuries in some series (7), and account for significant morbidity and mortality. Compound fractures account for up to 90% of these injuries (3, 6, 17), and are associated with an infection rate of 1.9 to 10.6% (9, 13, 16, 17), an average neurological morbidity of approximately 11% (6), an incidence of late epilepsy of up to 15% (10), and a

mortality rate of 1.4 to 19% (3, 5–7, 17). By convention, compound depressed cranial fractures are treated surgically, with debridement and elevation, primarily to attempt to decrease the incidence of infection. Closed (“simple”) depressed cranial fractures undergo operative repair if the extent of depression is greater than the full thickness of the adjacent calvarium, with the theoretical benefits of better cosmesis, a diminution in late-onset posttraumatic epilepsy, and a reduction in the incidence of persistent neurological deficit. There is, however, very little literature to support these management strategies, despite their widespread, and theoretically sound, practice. There is Class III literature that addresses the efficacy of surgical management of these injuries, and it argues against automatic surgical treatment of all compound fractures (7).

Most of the literature reviewed focuses predominantly on infectious complications, seizures, surgical technique (e.g., bone fragment replacement versus removal), or the predictive power of cranial fracture for the presence of other intracranial pathology. Several large studies of patients with cranial fracture shed light on the breadth of issues associated with such lesions and are discussed below, under Scientific Foundation. However, some of these studies were conducted before the CT-scan era, and thus, although important for our understanding of the injury itself, are not included for critical analysis.

## PROCESS

A MEDLINE computer search using the following key words: “skull” and “fracture” and “depressed” between 1975 and 2001 was performed. A total of 224 documents were found. The search was narrowed to include the key words: “surgery” or “operation” or “elevation”. A total of 122 articles were found, 5 of which met the criteria for critical analysis. In addition, the reference lists of all articles were reviewed, and additional articles were selected for background information. The results of this analysis were incorporated into the review presented here. Papers primarily addressing the following topics were not included: patients with associated medical illnesses, sinus fractures, cranial base fractures, isolated orbital or facial fractures, and pre-CT era reports. In general, papers with the following characteristics were also excluded: case series with less than 10 patients evaluated by CT scan and with incomplete outcome data (mortality or Glasgow outcome score), case reports, operative series with operations occurring longer than 14 days from injury. Several articles with case series of less than 10 patients were examined and reviewed because of the limited number of patient series evaluating the acute surgical management of depressed cranial fractures in the CT era. Selected articles were evaluated for design, prognostic significance, therapeutic efficacy, and overall outcome. In addition, several articles were reviewed for the purposes of historical perspective.

## SCIENTIFIC FOUNDATION

Closed, linear cranial fractures are considered nonoperative lesions unless associated with surgical intracranial masses.

Controversy surrounds appropriate management of depressed cranial fractures. Compound depressed cranial fractures are depressed fractures with an overlying scalp laceration in continuity with the fracture site and with galeal disruption, and have conventionally been treated with debridement and surgical elevation (3, 6, 9, 14). Simple depressed cranial fractures have no galeal disruption and are traditionally managed with surgical elevation only if the extent of depression equals or exceeds the thickness of adjacent, intact bone, or if there is an associated intracranial hematoma with mass effect that requires evacuation.

The rationale for aggressive treatment of depressed cranial fractures stems from their association with infection and late epilepsy. Cosmetic deformity also plays a role in surgical decision making. Such complications, and their potential sequelae, are well documented. In a series of 359 patients with compound cranial fractures, Jennett and Miller (9) documented a 10.6% incidence of infection, which was associated with a significantly higher incidence of persistent neurological deficit, late epilepsy (defined as seizures longer than 1 wk from injury), and death. Operative debridement reduced the incidence of infection to 4.6% in their series. Operative delay greater than 48 hours from injury dramatically increased the incidence to 36.5%. There was no difference in infection rate between surgical cohorts who had bone fragments replaced versus removed—results supported by a series of 225 patients with depressed cranial fracture reported by Braakman (3), and a treatment strategy reported as early as Macewan in 1888 (9). In a separate report of 1000 patients with nonmissile depressed cranial fractures, Jennett et al. (10) documented a 15% incidence of late epilepsy, which was significantly associated with posttraumatic amnesia longer than 24 hours, torn dura, the presence of focal neurological signs, and the presence of early epilepsy (i.e., within 1 wk of injury). In the closed-fracture patients in this series, there was no difference in incidence of epilepsy between the elevated and nonelevated cases. Additionally, there was a higher incidence of late epilepsy in patients with elevated compound fractures. The authors explain this finding by documenting a higher incidence of those factors independently associated with late epilepsy, such as dural tearing and long posttraumatic amnesia, in the elevated-fracture patient cohort. These series were reported before the CT era, however, they offer us a clear picture of both the range of complications associated with nonmissile depressed cranial fractures and the controversies surrounding management strategies.

The primary question facing the neurosurgeon regarding depressed cranial fracture is whether to operate. Heary et al. (7) reported a group of patients with compound depressed cranial fractures in which nonsurgical therapy was used for a subgroup of 26 patients without clinical or radiographic evidence of dural violation or significant underlying brain injury. They concluded that patients with open (compound) depressed cranial fractures may be treated nonoperatively if there is no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression greater

than 1 cm, frontal sinus involvement, gross deformity, wound infection, pneumocephalus, or gross wound contamination. No infectious complications occurred. Similarly, van den Heever and van der Merwe (16) reported an equally low incidence of infection in a group of nonoperatively treated patients that included 139 compound depressed fractures. Surgical indications in their series included clinical characteristics of the wound. CT scans were not routinely used unless a neurological deficit was present on admission.

Although these studies are retrospective and nonrandomized, and, thus, subject to inherent biases, they clearly demonstrate that at least a select group of patients with compound depressed cranial fractures will do well without surgery.

Another challenge to traditional thinking that has surfaced in the literature involves the proper surgical management of compound depressed cranial fractures with respect to the bone fragments. Conventional treatment involves operative debridement, elevation of the fracture, removal of bone fragments, and delayed cranioplasty. However, this subjects the patient to a second operation (i.e., cranioplasty), with its attendant risks and complications. Kriss et al. (11), Jennett and Miller (9), and Braakman (3) showed that infectious complications are not increased by primary bone fragment replacement. Wylen et al. (17) retrospectively reviewed a series of 32 patients who underwent elevation and repair of a compound depressed cranial fracture with primary replacement of bone fragments within 72 hours of injury. Patients treated longer than 72 hours after injury and patients who presented with existing infection were excluded from the study. There were no infectious complications. Blankenship et al. (2) also demonstrated a 0% infection rate in 31 children with compound depressed cranial fractures treated with primary bone fragment replacement, regardless of the degree of contamination of the wound at the time of surgery. Thirty patients in this series were treated within 16 hours of injury. Likewise, Adeloye and Shokunbi (1) report the success of immediate bone replacement, without infectious sequelae, in 12 patients with compound depressed fractures, 11 of whom were treated within 10 hours of injury. Four patients in their series were treated with free-fragment removal secondary to the greater severity of parenchymal injury, suggesting benefit from the decompression that bone removal would provide. Despite the retrospective, uncontrolled, nonrandomized design of these observational studies, they clearly demonstrate the feasibility of immediate bone fragment replacement without a corresponding increase in infectious sequelae, thus, obviating the need for a second surgical procedure.

### **SUMMARY**

The majority of studies are case series. No controlled, prospective clinical trials of treatment using surgical versus nonsurgical management have been published. The majority of data support debridement and elevation of grossly contaminated compound depressed cranial fractures as soon as possible after injury. However, several retrospective studies demonstrate successful nonoperative management of some patients with less-severe compound depressed cranial fractures on the basis of CT and clinical

criteria. In the absence of gross wound infection at the time of presentation, immediate replacement of bone fragments seems not to increase the incidence of infection if surgery is performed expeditiously, and this replacement eliminates the need for subsequent cranioplasty and its attendant risks and complications. No controlled data exist to support the timing of surgery or the use of one technique over another.

### **KEY ISSUES FOR FUTURE INVESTIGATION**

To improve the strength of recommendations above the option level, well-controlled trials of surgical technique are warranted, and should examine issues of bone fragment replacement versus removal, dural laceration repair, etc., and their respective relationship to outcome variables, such as incidence of infection, incidence of epilepsy, need for reoperation, surgical complications, and, most importantly, neurological and neuropsychological outcomes.

### **REFERENCES**

1. Adeloye A, Shokunbi MT: Immediate bone replacement in compound depressed skull fractures. *Cent Afr J Med* 39:70-73, 1993.
2. Blankenship JB, Chaddock WM, Boop FA: Repair of compound-depressed skull fractures in children with replacement of bone fragments. *Pediatr Neurosurg* 16:297-300, 1990.
3. Braakman R: Depressed skull fracture: Data, treatment, and follow-up in 225 consecutive cases. *J Neurol Neurosurg Psychiatry* 35:395-402, 1972.
4. Chan KH, Mann KS, Yue CP, Fan YW, Cheung M: The significance of skull fracture in acute traumatic intracranial hematomas in adolescents: A prospective study. *J Neurosurg* 72:189-194, 1990.
5. Colak A, Berker M, Ozcan OE: Occipital depression fractures in childhood. A report of 14 cases. *Childs Nerv Syst* 7:103-105, 1991.
6. Cooper PR: Skull fracture and traumatic cerebrospinal fluid fistulas, in Cooper PR (ed): *Head Injury*. Baltimore, Williams and Wilkins, 1993, pp 115-136 ed 3.
7. Heary RF, Hunt CD, Krieger AJ, Schulder M, Vaid C: Nonsurgical treatment of compound depressed skull fractures. *J Trauma* 35:441-447, 1993.
8. Hung CC, Chiu WT, Lee LS, Lin LS, Shih CJ: Risk factors predicting surgically significant intracranial hematomas in patients with head injuries. *J Formos Med Assoc* 95:294-297, 1996.
9. Jennett B, Miller J: Infection after depressed fracture of skull. Implications for management of nonmissile injuries. *J Neurosurg* 36:333-339, 1972.
10. Jennett B, Miller J, Braakman R: Epilepsy after nonmissile depressed skull fracture. *J Neurosurg* 41:208-216, 1974.
11. Kriss F, Taren J, Kahn E: Primary repair of compound skull fractures by replacement of bone fragments. *J Neurosurg* 30:698-702, 1969.
12. Macpherson BC, MacPherson P, Jennett B: CT evidence of intracranial contusion and haematoma in relation to the presence, site and type of skull fracture. *Clin Radiol* 42:321-326, 1990.
13. Mendelow AD, Campbell D, Tsementzis SA, Cowie RA, Harris P, Durie TB, Gillingham FJ: Prophylactic antimicrobial management of compound depressed skull fracture. *J R Coll Surg Edinb* 28:80-83, 1983.
14. Servadei F, Ciucci G, Pagano F, Rebutti GG, Ariano M, Piazza G, Gaist G: Skull fracture as a risk factor of intracranial complications in minor head injuries: A prospective CT study in a series of 98 adult patients. *J Neurol Neurosurg Psychiatry* 51:526-528, 1988.
15. Steinbok P, Flodmark O, Martens D, Germann ET: Management of simple depressed skull fractures in children. *J Neurosurg* 66:506-510, 1987.
16. van den Heever CM, van der Merwe DJ: Management of depressed skull fractures. Selective conservative management of nonmissile injuries. *J Neurosurg* 71:186-190, 1989.
17. Wylen EL, Willis BK, Nanda A: Infection rate with replacement of bone fragment in compound depressed skull fractures. *Surg Neurol* 51:452-457, 1999.

TABLE 1. Surgical management of depressed cranial fractures\*

Authors	No. of patients	Class	Inclusion GCS	Treatment	Outcome	Description	Conclusion
Adeloye and Shokunbi (1)	16	III	GCS 15	Bone fragment replacement versus removal	Infection rate; GOS; timing not specified	Retrospective series of 16 patients treated either with immediate bone fragment replacement (n = 12) or free fragment removal (n = 4) to compare infection rate and outcome between groups.	<ul style="list-style-type: none"> <li>● No incidence of infectious complication in either group, supporting the safety of bone fragment replacement.</li> <li>● Selection bias and small number of patients preclude comparison of outcome.</li> </ul>
Blankenship et al. (2)	31	III	Unknown	Bone fragment replacement	Infection rate; nonunion, average 26.5 mo	Retrospective review of 31 children aged 20 mo to 17 yr surgically treated with primary bone fragment replacement for compound depressed cranial fractures to examine the incidence of infectious complications. Thirty of 31 patients were treated within 16 h of injury.	<p>No. of patients: 12 (Replacement), 4 (No replacement)</p> <p>CR (%): 100 (Replacement), 100 (No replacement)</p> <p>Infection (%): 0 (Replacement), 0 (No replacement)</p> <ul style="list-style-type: none"> <li>● No instances of infectious complications and no need for subsequent cranioplasty, regardless of degree of wound contamination.</li> </ul>
Braakman (3)	225	N/A	N/A	Bone fragment replacement versus removal	Infection rate; incidence of epilepsy	Retrospective review of 225 patients with depressed cranial fracture to examine epidemiology, assess outcome with respect to incidence of epilepsy and persistent neurological deficit, and to assess the effect of primary bone fragment replacement on infection rate.	<p>No. of patients: 31</p> <p>Infection (%): 0</p> <ul style="list-style-type: none"> <li>● 4.4% incidence of early epilepsy.</li> <li>● 7.1% incidence of late epilepsy.</li> <li>● Primary replacement of bone fragments did not alter infection rate.</li> </ul>
Heary et al. (7)	54	III	Excellent, good, fair, poor	Surgery and nonsurgical	Mean GOS, 9.5 mo	Retrospective review of 54 prospectively treated patients with compound depressed cranial fractures comparing patients treated surgically versus nonsurgically based on a standardized treatment protocol involving clinical and CT criteria.	<p>No. of patients: 82 (Replacement), 27 (Partial replacement), 56 (Removal)</p> <p>Infection (%): 2.4 (Replacement), 11.1 (Partial replacement), 10.7 (Removal)</p> <ul style="list-style-type: none"> <li>● No difference in outcome between patients treated surgically versus nonsurgically with compound depressed cranial fractures that have no clinical or radiographic evidence of dural penetration, significant intracranial hematoma, depression &gt;1 cm, frontal sinus involvement, gross deformity, wound infection, or gross wound contamination.</li> <li>● No infectious complications occurred in either group with standardized treatment protocol including prophylactic antibiotics.</li> <li>● Patients with surgical lesions had significantly increased incidence of SAH and cerebral contusion (P &lt; 0.01 and P &lt; 0.05, respectively).</li> <li>● Mortality 5.6% of those analyzed, 1.9% total.</li> </ul>
Jennett and Miller (9)	359	N/A	N/A	Early versus delayed; bone fragment replacement versus removal; dura closed versus open	Infection rate, CNS signs, late epilepsy, mortality	Retrospective review of 359 patients with compound, depressed cranial fractures to examine the causes and consequences of infection and to assess the effect of surgical timing and methods on infection rate.	<p>No. of patients: 28 (Surgery), 26 (Nonsurgical)</p> <p>Excellent (%): 64.3 (Surgery), 80.8 (Nonsurgical)</p> <p>Good (%): 21.4 (Surgery), 3.8 (Nonsurgical)</p> <p>Fair (%): 10.7 (Surgery), 7.7 (Nonsurgical)</p> <p>Poor (%): 0 (Surgery), 0 (Nonsurgical)</p> <p>D (%): 3.6 (Surgery), 7.7 (Nonsurgical)</p> <ul style="list-style-type: none"> <li>● 10.6% overall infection rate.</li> <li>● Infection was significantly associated with increased mortality, prolonged CNS signs, late epilepsy.</li> <li>● Incidence of infection was significantly greater in patients with &gt;48 h delay between injury and operation.</li> <li>● No difference in infection rate between the group whose dura was closed and the group whose dura was left open.</li> <li>● Primary bone fragment replacement did not significantly affect postoperative infection rate or incidence of late epilepsy.</li> <li>● 10% incidence of early epilepsy (1 wk).</li> <li>● 15% incidence of late epilepsy (&gt;1 wk).</li> <li>● Increased risk of late epilepsy with posttraumatic amnesia &gt;24 h, torn dura, focal neurological signs, occurrence of early epilepsy.</li> <li>● Lower risk of early/late epilepsy with occipital fractures.</li> </ul>
Jennett et al. (10)	1000	N/A	N/A	Surgery versus no surgery	Incidence of early and late epilepsy	Retrospective review of 1000 patients with nonmissile depressed cranial fractures to study risk factors associated with posttraumatic epilepsy.	<ul style="list-style-type: none"> <li>● Lower risk of early/late epilepsy with occipital fractures.</li> </ul>

TABLE 1. Continued

Authors	No. of patients	Class	Inclusion GCS	Treatment	Outcome	Description	Conclusion
Mendelow et al. (13)	176	N/A	N/A	Prophylactic antibiotics versus no antibiotics	Infection rate	Retrospective series of 176 patients with compound, depressed cranial fractures to determine factors that predispose to infection and the effect of prophylactic antibiotics on infection rate.	<p>Initially clean, injury to OR &lt;36 h</p> <p>Initially clean, injury to OR &gt;48 h</p> <p>Injury to OR &gt;48 h or never</p> <p>Bone fragment replacement</p> <p>Bone fragment removal</p> <p>1 P = n.s., 2 P &lt; 0.001</p> <p>●6.3% overall infection rate.</p> <p>●Prophylactic use of ampicillin and a sulphonamide was associated with a significantly lower infection rate (1.9%) than with any other combination (15.6%) or no antibiotics at all (10.5%).</p> <p>●Dural penetration, primary versus secondary closure, sinus involvement, surgery versus no surgery, bone replacement versus removal did not significantly affect infection rate.</p>
Steinbok et al. (15)	111	III	Duration of coma	Surgery and nonsurgical	Incidence of seizures, neurological dysfunction, cosmesis; timing not specified	Retrospective series of 111 children <16 yr with simple depressed cranial fractures to examine outcome with respect to the occurrence of seizures, neurological dysfunction, and cosmesis between surgically and nonsurgically managed patients.	<p>Ampicillin + sulphonamide</p> <p>No antibiotics</p> <p>All other antibiotics</p> <p>1 P &lt; 0.05, 2 P &lt; 0.01, compared with ampicillin + sulphonamide</p> <p>●No difference in seizures, neurological dysfunction, cosmetic deformity between surgical and nonsurgical treatment. Groups differed with respect to maximum depression of fracture (P = 0.000), but not to duration of coma, age.</p> <p>●Dural laceration was significantly associated with neurological deficit.</p>
van den Heever and van der Merwe (16)	284	III	Unknown	Surgery versus nonsurgical	Septic complication; outcome of focal neurological abnormalities; mortality	Retrospective review of 284 patients whose primary injury was nonmissile depressed cranial fracture to compare outcome between surgical and nonsurgical groups. Surgical indications detailed.	<p>Surgery</p> <p>Nonsurgical</p> <p>●5.3% infection rate—2.8% conservative versus 8% surgical.</p> <p>●5% incidence of deterioration of neurological deficit—4% conservative versus 7% surgical.</p> <p>●1.4% mortality, all in surgical group—selection bias noted. Note: cannot separate patients with CT. Paper included to show comparable outcome between surgical and nonsurgical management of depressed cranial fracture.</p>
Wylen et al. (17)	32	III	GCS 6–15	Bone fragment replacement mo	Infection, average 22	Retrospective review of 32 patients surgically treated with primary bone fragment replacement for compound depressed cranial fracture within 72 h of injury to examine infection rate.	<p>Nonsurgical</p> <p>Surgery</p> <p>No. of patients</p> <p>Infection (%)</p> <p>Mortality (%)</p> <p>Deterioration (%)</p> <p>●No instances of infectious complications or new/worsened neurological deficit following primary bone fragment replacement within 72 h of injury.</p> <p>●25% incidence of persistent neurological deficit at 6 mo.</p>

<sup>a</sup> GCS, Glasgow Coma Scale; GOS, Glasgow outcome score; GR, good recovery; N/A, not applicable; CT, computed tomographic scan; SAH, subarachnoid hemorrhage; D, death; CNS, central nervous system; OR, operation; n.s., not significant.

## APPENDIX I: POST-TRAUMATIC MASS VOLUME MEASUREMENT IN TRAUMATIC BRAIN INJURY PATIENTS

1. Direct volumetric measurement with imaging software using a modern computer tomographic CT scanner is the gold standard. This has been applied only on rare occasions.
2. The "ellipsoid method" was developed to calculate the volume of arteriovenous malformations (3). It is based on the concept that the volume of an ellipsoid is approximately one-half of the volume of the parallelepiped (a six-faced polyhedron, all of whose faces are parallelograms lying in pairs of parallel planes) into which it is placed. By measuring three diameters of a given lesion in the arterial phase of an angiogram, a parallelepiped is constructed, and its volume, divided in half, is close to the actual volume of the malformation. By extending this concept from angiography to CT scanning, calculation of space-occupying lesions becomes possible (4). The "ABC" method has been described by Kothari et al. (2) for the measurement of intracerebral hemorrhages, and is also based on the concept of measuring the volume of an ellipsoid. The formula for an ellipsoid is:
 
$$V_e = 4/3 \pi (A/2) (B/2) (C/2)$$
 where A, B, and C are the three diameters.  
 For  $\pi = 3$ , the formula becomes  $V_e = ABC/2$   
 The volume of an intracerebral hemorrhage can be approximated by following the steps listed below:
  - Identify the CT slice with the largest area of hemorrhage (Slice 1)
  - A: measure the largest diameter, A.
  - B: measure the largest diameter 90° to A on the same slice, B.
  - C: count the number of 10-mm slices.
  - Compare each slice with slice 1.
  - If the hemorrhage is greater than 75% compared with slice 1, count the slice as 1.
  - If the hemorrhage is 25 to 75%, count the slice as 0.5.
  - If the hemorrhage less than 25%, do *not* count the slice.
  - Add up the total C.
3. More recently, the "Cavalieri direct estimator" method has been introduced (1). It breaks down the lesion on the CT scan into a corresponding number of points. The volume of a lesion is the product of the sum of the points that fall into the lesion, the area associated with each point, and the distance between the scan slices. A grid that is used to determine the number of points can be obtained by photocopying a template provided in the original article or by preparing a uniformly spaced point grid by computer (4).

### REFERENCES

1. Clatterbuck R, Sipos E: The efficient calculation of neurosurgically relevant volumes from computed tomographic scans using Cavalieri's Direct Estimator. *Neurosurgery* 40:339-342, 1997.
2. Kothari RU, Brott T, Broderick JP, Barsan WG, Sauerbeck LR, Zuccarello M, Khoury J: The ABCs of measuring intracerebral hemorrhage volumes. *Stroke* 27:1304-1305, 1996.
3. Pasqualin A, Barone G, Cioffi F, Rosta L, Scienza R, Da Pian R: The relevance of anatomic and hemodynamic factors to a classification of cerebral arteriovenous malformations. *Neurosurgery* 28:370-379, 1991.
4. Stocchetti N, Croci M, Spagnoli D, Gilardoni F, Resta F, Colombo A: Mass volume measurement in severe head injury: Accuracy and feasibility of two pragmatic methods. *J Neurol Neurosurg Psychiatry* 68:14-17, 2000.

### ADVERTISING

Inquiries regarding advertising in **NEUROSURGERY** should be directed to:

**Paul Tucker**  
 Lippincott Williams & Wilkins  
 351 West Camden Street  
 Baltimore, MD 21201-2436  
 TEL: 410/528-4291  
 FAX: 410/528-4457  
 EMAIL: ptucker@lww.com

**Kelly Adamitis**  
 Lippincott Williams & Wilkins  
 530 Walnut Street  
 Philadelphia, PA 19106-3621  
 TEL: 215/521-8402  
 FAX: 215/521-8411  
 EMAIL: kadamiti@lww.com

## APPENDIX II: EVALUATION OF RELEVANT COMPUTED TOMOGRAPHIC SCAN FINDINGS

Computed tomographic (CT) scanning is the imaging modality of choice for traumatic brain injury because of its widespread availability, the rapid imaging time, the low associated costs, and its safety. CT scanning measures the density of tissues using x-rays. To standardize the imaging procedure, 5-mm slices should be obtained from the foramen magnum to the sella and 10-mm slices should be obtained above the sella, parallel to the orbitomeatal line. The following early CT scan findings correlate with outcome (1):

- Status of the basal cisterns.
- Midline shift.
- Subarachnoid hemorrhage in the basal cisterns.

### Basal Cisterns at the Midbrain Level

Compressed or absent basal cisterns indicate a threefold risk of raised intracranial pressure and the status of the basal cisterns is related to outcome. The degree of mass effect is evaluated at the level of the midbrain. Cerebrospinal fluid cisterns around the midbrain are divided into three limbs, one posterior and two laterally (Fig. 1). Each limb can be assessed

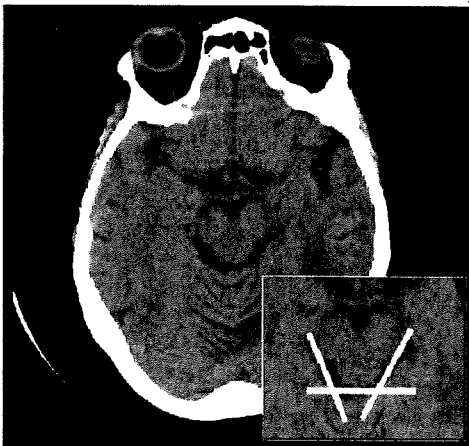


FIGURE 1. Evaluation of the basal cisterns on computed tomographic scan.

separately as to whether or not it is open or compressed. Basal cisterns can be:

- Open (all limbs open).
- Partially closed (one or two limbs obliterated).
- Completely closed (all limbs obliterated).

### Midline Shift at the Foramen of Monro

The presence of midline shift is inversely related to prognosis. However, interaction exists with the presence of intracranial lesions and other CT parameters (1). Midline shift at the level of the foramen of Monro should be determined by first measuring the width of the intracranial space to determine the midline ("A"). Next, the distance from the bone to the septum pellucidum is measured ("B") (Fig. 2). The midline shift can be determined by calculating:

$$\text{Midline shift} = (A/2) - B$$

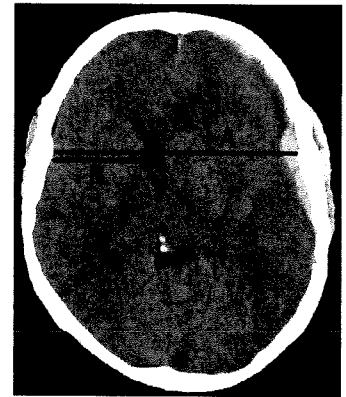


FIGURE 2. Assessment of midline shift on computed tomographic scan.

### Traumatic Subarachnoid Hemorrhage

Traumatic subarachnoid hemorrhage occurs in between 26 and 53% of all patients with severe traumatic brain injury. Mortality is increased twofold in the presence of traumatic subarachnoid hemorrhage. The presence of subarachnoid hemorrhage in the basal cisterns carries a positive predictive value of unfavorable outcome of approximately 70%.

### REFERENCE

1. Chesnut R, Ghajar J, Maas A, Marion D, Servadei F, Teasdale G, Unterberg A, von Holst, Walters B: Early indicators of prognosis in severe traumatic brain injury. *J Neurotrauma* 17:535-627, 2000.

**DISCLAIMER:** The Congress of Neurological Surgeons (CNS) is not engaged in rendering professional medical services and assumes no responsibility for patient outcomes resulting from application of these general recommendations in specific patient circumstances. Adherence to these clinical practice parameter guidelines does not necessarily assure a successful medical outcome. The information contained in these guidelines reflects published scientific evidence at the time of completion of the guidelines and cannot anticipate subsequent findings and/or additional evidence and, therefore, should not be considered inclusive of all proper procedures and tests or exclusive of other procedures and tests that are reasonably directed to obtaining the same result. Medical advice and decisions are appropriately made only by a competent and licensed physician who must make decisions in light of all the facts and circumstances in each individual and particular case and on the basis of availability of resources and expertise. Guidelines are not intended to supplant physician judgment with respect to particular patients or special clinical situations and are not a substitute for physician-patient consultation. Accordingly, the CNS considers adherence to these guidelines to be voluntary, with the ultimate determination regarding their application to be made by the physician in light of each patient's individual circumstances.

