

Guidelines for the Treatment of Head Injury in Adults

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Abstract

The workshop of scientific medical faculties (Arbeitsgemeinschaft wissenschaftlicher medizinischer Fakultäten [AWMF]) of Germany has asked societies of specific medical disciplines to jointly publish guidelines on the treatment of diseases and injuries. On behalf of the Deutsche Gesellschaft für Neurochirurgie, its commission on guidelines initiated an interdisciplinary approach to publish guidelines on the treatment of head injury in adults. These guidelines were published in German by the AWMF in late 2015. Because these guidelines have received widespread attention in Germany and became fundamental for research in head injuries, we have translated the German version into English to make it accessible to the international scientific community.

Keywords

- ▶ head injury
- ▶ guidelines
- ▶ adults

Introduction

Head injury is the most frequent cause of death up to early adulthood. Because brain tissue has the shortest tolerance to hypoxia of all organs, urgent treatment is required to maximize survival and to minimize permanent brain damage in survivors.

These guidelines are designed to outline the current methods of diagnosis and care for patients with head injury at the site of the accident, during transport, and at the hospital. They

are aimed at anyone treating patients with head injuries. Basic medical knowledge, however, is a prerequisite.

The basis of these guidelines is the grade of evidence as published in the scientific literature. This evidence is classified into five grades.¹

Three levels of recommendation, A, B, or 0, are based on these grades of evidence of statements according to the national program of guidelines of medical service.²

The definition of these levels of recommendation is A, strong recommendation; B, recommendation; and 0, recommendation uncertain.

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Table 1 Therapeutic studies

Level of recommendation	Grade of evidence	Type of study
A	1a	Systemic review of randomized controlled studies
	1b	Minimum of one randomized controlled study
B	2a, b	Systematic review of comparing cohort studies
	3a, b	Systematic review of case-controlled studies or at least one adequate controlled study
0	4	Case series and inadequate case-controlled studies justified expert opinion
	5	Opinion without critical appraisal

The authors developing these guidelines reached a consensus attributing the respective level of recommendation. In some cases the grade of evidence and the level of recommendation did not coincide when an excessively high degree of certainty was considered evident. Accordingly, the surgical measures in intracranial space-occupying hematomas were attributed a high level of recommendation in the absence of appropriate clinical studies. For decades, respective observations of their efficacy have never been questioned but confirmed by everyday experience and good clinical practice. Most of the recommendations are classified according to the subsequent grades of evidence¹ (► **Tables 1** and **2**).

Definition

A *head injury* is caused by the effect of force to the head that may have caused a dysfunction of the brain and in addition may have led to an injury of the brain, scalp, skull bone, vessels, and dura.

An *open head injury* involves a laceration of the dura and soft tissue leading to open access of the subdural space to the outside.

Primary brain injury is a lesion caused by the initial effect of force leading to reversible or irreversible dysfunction of

brain cells. These primary lesions, however, can lead to a cascade of reactions causing delayed *secondary brain injuries*, which may be prevented by urgent and effective measures that are the object of medical and other treatment of head injuries.

Epidemiology

In Germany there are ~ 332 head injuries per 100,000 inhabitants. Of these, 91% are classified as mild, 4% are moderate, and 5% are severe. Altogether ~ 280,000 patients experienced a head injury, of whom 2,750 die. The overall cost for society has been estimated to reach €2.8 billion per year.³

Symptoms and Signs

Symptoms after head injury are headache, dizziness, sickness, diplopia, deafness, and amnesia. Signs after head injury are bruises, laceration of scalp, deformities of the skull, bleeding from the scalp, mouth, nose, and/or ear. Further signs raising suspicion of a head injury are decreased wakefulness, vomiting, seizures, posturing, disorder of orientation, speech, and/or coordination, cranial nerve function, and/or vegetative or motor function. A disorder of

Table 2 Diagnostic studies

Level of recommendation	Grade of evidence	Type of study
A	1a	Systematic review of adequate studies type Ib
	1b	Double-blind objective independent randomized study involving all subjects of target group
B	2a, b	Systematic review of diagnostic study or at least one selective randomized study with independent blinded and objective reference test of target population
	3a, b	Systematic review of diagnostic studies or at least one study without inclusion of all patients in the reference test
0	4	Case-controlled study or studies without independent blinded or objective reference test
	5	Opinion without critical appraisal

consciousness indicates a serious dysfunction of the brain. Full consciousness may be differentiated from impaired consciousness and unconsciousness.

Impaired consciousness includes reduced wakefulness, disorder or absence of orientation in terms of time, place, and self-awareness. Eyes can be open.

Unconsciousness (synonymous with coma): Absence of awareness of surroundings and self. The clinical signs are a nonarousable state, the patient does not open eyes spontaneously or on painful stimuli and does not follow commands. Movements upon painful stimuli or spontaneously are possible. A Glasgow Coma Scale (GCS) score < 8 represents coma. Clinical signs of a life-threatening deterioration of patients with impaired consciousness or coma are the loss of pupillary function, hemiparesis, acute circulatory disorders, and extensor response.⁴⁻⁹

Classification of the Severity of Head Injury

Head injury is a dynamic disorder in the acute phase. Because rapid deterioration or improvement is common, the original assessment of the injury must be adjusted to the clinical course of events.

An internationally most frequently used classification is the differentiation of three grades of minor, moderate, and severe head injury according to the sum score of the GCS. The point in time of when the GCS is recorded (e.g., time of admittance to the hospital, site of accident, the least favorable score within the first 48 hours) has not been defined uniformly. In addition to the methodological weaknesses of the GCS sum score, the reliability and the validity of this classification has been viewed with reservations.

The severity of the head injury is of secondary relevance for the actual treatment, which is governed by the actual neurologic findings and the course of events, particularly in the early phase of the course that requires frequent repeat investigations.¹⁰⁻¹⁷

A classification popular in Germany by Tönnis and Loew¹⁷ suggested three degrees of severity based on the duration of persisting neurologic disorders and may therefore only be applied retrospectively.

Additional Injuries of the Patient with Head Injury

Additional life-threatening injuries must be suspected in any comatose patient after a head injury. The search for multiple injuries is urgent and often difficult because the comatose patient will not be able to talk or understand or indicate the location where he or she is in pain. Particularly disorders of respiratory function and/or severe external or internal blood loss need urgent attention because they may lead to cerebral hypoxia and increase cerebral lesions.

Approximately 15% of patients with severe head injuries suffer additional spinal injuries. Unless proven otherwise, any comatose patient should therefore be suspected to have an additional spinal injury.

In addition to the head injury, there may also be injuries of cerebral vessels, such as dissection of arteries, traumatic aneurysms, and arteriovenous fistula.

First Aid at the Site of Accident: Preclinical Care

Urgent Treatment

An unfavorable outcome after head injury is related to hypoxia and arterial hypotension. Any measures to counteract hypoxia or hypotension have absolute priority. Because spontaneous respiration may become inadequate any time after a brain injury, preventive measures to ensure adequate oxygen supply of the brain are urgent.

A

Patients in a coma (GCS of 8 or lower) are to be intubated. Disorders of respiration, pneumothorax, and hemothorax are to be treated urgently.¹⁸

B

Normal oxygenation and normocapnia are desirable. Arterial oxygen saturation should not drop < 90% if possible. Obvious hemorrhage must be stopped to ensure adequate cardiovascular function. Blood pressure and pulse rate should be monitored and lost fluids substituted.

B

Normal blood pressure is desirable; a blood pressure < 90 mm Hg is to be avoided.¹⁹⁻²⁰

History

A

In addition to the neurologic findings, the medical history of the patient is essential because it may offer clues indicating an intracranial injury. It is essential to record the history of the patient. Details on the type of damage to the car or the height of the fall either from the patient or from a bystander may indicate the type and direction of force suffered at the time of injury and may be relevant to the management of the patient. Information on current medication (e.g., anticoagulation) of the patient or drug abuse is also important.

Neurologic Examination

The assessment and documentation of the following is considered essential:

A

- Full consciousness, impaired consciousness, unconsciousness
- Function of pupils^{11,12}
- Motor function of each arm and leg, differentiating no, partial, and complete paralysis. Without spontaneous movements, motor function upon painful stimuli must be tested. Special attention should be directed to hemiparesis and flexor or extensor response.

When the patient is not unconscious, the recording of orientation, function of cranial nerves, speech, and coordination is mandatory.

All these essential neurologic findings must be documented with the corresponding time. They govern the subsequent management.

B

Short-term repeat examinations are advisable to detect any deterioration.

B

The neurologic findings should be registered in a standardized fashion. The GCS has been accepted internationally. The limitations of this scale, misleading signs of improvement, limitations in intubated and sedated patients, and so on, must be considered.

The GCS has been widely used to describe and encode a disorder of brain function. In spite of its universal application, difficulties have been reported. The GCS is a standardized classification of three phenomena: eye opening, verbal communication, and best motor function of arms. Its use may lead to misjudgments because the signs differentiating a deterioration of prognosis in coma are limited to the best motor function. Thus the most important signs of acute life-threatening herniation in coma, loss of pupil function, hemiparesis, and the unconditional registration of flexor or extensor response, are not discerned with the GCS. The sum score of the GCS may therefore be misleading. It is clearly inferior to a detailed neurologic investigation.^{10-12,16,21}

Head Injury Caused by a Disorder of Consciousness of Nonrelated Origin

An acute onset of impaired consciousness from various causes may precipitate head injury. A disturbance of blood glucose is easy to detect and treat. Coma may be of endocrine, metabolic, cardiovascular, cerebrovascular, or other origin (e.g., intoxication or hypothermia).¹¹

Indication for In-Hospital Treatment

With any of the subsequent signs the patient should be admitted to the hospital:

A

- Coma
- Impaired consciousness
- Amnesia
- Other neurologic signs
- Seizures
- Clinical or radiologic signs of skull fracture or penetrating injury, suspected nasal or petrous bone cerebrospinal fluid (CSF) fistula

In any of the subsequent clinical signs a referral to a hospital appears advisable:

B

- Vomiting in close temporal relationship to the injury
- Evidence of a disorder of coagulation
- If in doubt

A

The choice of hospital depends on the time necessary to reach it and on the facilities it offers. Patients comatose after head injury or with deteriorating con-

sciousness, disorder of pupil function, paresis, or seizures should be transported to an institution with constantly available neurosurgical care.¹¹

Transport

Neither analgesia, sedation, nor relaxation may be recommended because there is no adequate evidence in the literature to confirm any benefit. Although some patients may require analgesia or sedation to ensure adequate respiration, it may be at the expense of the realization of clinical signs of deterioration.

The perforating object should not be removed if possible in transfixing injuries. An additional spinal injury should be suspected in any comatose patient. During transportation a stiff collar to protect the cervical spine and respective caution is recommended until appropriate diagnostic measures can be taken.¹²

Measures to Protect the Brain

A

Glucocorticoids should not be given because an increased 14-day mortality has been reported.²²

O

Mannitol or hyperosmolar saline can lower the intracranial pressure (ICP) for a short time (up to 1 hour). It can be recommended when transtentorial herniation is suspected even without verification of increased ICP. There is no evidence beyond this indication.¹⁹

O

Hyperventilation may be an option with signs of transtentorial herniation, anisocoria posturing, and deterioration of consciousness. Twenty breathing cycles per minutes in adults appear adequate.

O

Barbiturates have been recommended in noncontrollable ICP in earlier guidelines. However, there is no sufficient evidence for its effectiveness. Barbiturates are administered at the expense of monitoring of neurologic findings. They have a negative inotropic side effect and can possibly cause a detrimental drop in blood pressure.

B

Anticonvulsive agents inhibit seizures in the first week after injury. Early seizures, however, have no effect on long-term outcome.²³

From clinical studies there is no evidence of the protective effect of any further drugs that have been considered effective in experimental studies, such as 21-aminosteroids, calcium antagonists, glutamate receptor antagonists, and so on.^{11,12,19, 22-23}

Documentation

A

Details of the mechanism of the injury and the initial examination and follow-up of neurologic

findings are fundamental. These findings should be documented.

First Aid after Hospital Admission

Because a comatose patient after a head injury may have suffered multiple injuries, a multidisciplinary team should await the patient in the emergency department.

After ensuring vital functions, resuscitation, and recording of the clinical findings diagnostic imaging is urgent. Identification of bleeding into the major cavities of the body, skull, thorax, or abdomen has priority over the diagnosis of nonvital injuries. In comatose patients, life-threatening injuries must be suspected not only in the head because they may be multiple and anywhere. Hints to facilitate their diagnosis are obtained from the history and mechanism of the injury and the first clinical findings.

Imaging after Head Injury

Computed tomography (CT) is currently considered the gold standard in patients who have suffered a head injury. CT after acute head injury is necessary, an absolute indication, in:

A

- Coma
- Impaired consciousness
- Amnesia
- Other neurologic disorders
- Vomiting in close temporal relation to the injury
- Seizure
- Clinical or radiologic signs of skull fracture
- Suspected depressed or perforating skull fracture
- Suspected CSF fistula
- Signs of a disorder of coagulation (history, prolonged bleedings, etc.)
- When in doubt, CT is optional, for example, in:

B

- History of head injury unclear
- Severe headache
- Intoxication with alcohol or drugs

B

- History of a high-energy injury such as motor vehicle accident of > 60 km/hour, deformation of the vehicle, deformation of the passenger cabin of > 30 cm, duration of rescue from the vehicle of > 20 minutes, fall from height higher than 6 m, rolled-over injury, pedestrian or motorcycle collision of > 30 km/hour, cyclist knocked off the cycle.³⁴

Because the instant removal of an intracranial hemorrhage may be lifesaving, there is no justification to delay urgent cranial CT scanning as long as cardiovascular and breathing functions are stable. This is equally relevant for the initially noncomatose patient who was sedated and

intubated prior to transportation, in whom a developing intracranial hemorrhage can be differentiated from the effect of medication only with CT. The current fastest and most efficient imaging technique in multiple injuries is a spiral CT of the head, thorax, and abdomen. After diagnosis or exclusion of acutely life-threatening injuries, further diagnostic procedures may focus on injuries of bone and other nonimmediate life-threatening injuries.

When the indication for CT is optional, close monitoring of neurologic findings is another option. A cranial CT may not be necessary with serum S100 levels < 0.14 µg/L.³⁵ In a case where CT is not available, the patient should be referred to a hospital with neurosurgical expertise when a fracture is identified with conventional radiography. The absence of a skull fracture, however, does not rule out intracranial hemorrhage.

CT, respectively repeat CT, is also necessary in deterioration of neurologic findings or when the patient fails to recover or is in a persisting coma after 4 to 8 hours.³⁴⁻³⁷

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is usually not feasible in acute injuries in terms of availability and time.³⁸ It is, however, far superior to CT in detecting lesions of brain tissue and is particularly recommended when CT offers no explanation for neurologic deficits.³⁹

Indication for Admission to Hospital

Admission to hospital is indicated in:

A

- Lesions requiring surgical treatment
- Disorder of consciousness, coma
- Neurologic deficits
- Skull fracture
- CSF leakage, open head injury
- Lesions depicted by CT

In addition, admission to hospital may be advisable in:

B

- Additional disorders requiring treatment
- Severe headache, vertigo, intoxication with drugs or alcohol

Therapy

The purpose of treatment after head injury is to limit secondary brain lesions and support recovery of lost function. Lesions in urgent need of operation should be identified promptly.

Therapy begins with first aid at the site of accident and is continued in the hospital.

Emergency Surgery

The indication for surgical removal of a traumatic space-occupying lesion has never been confirmed in a

prospective randomized controlled study. Several retrospective analyses have confirmed the benefit of surgical decompression. In keeping with unchallenged experiences for many decades, the surgical removal of space-occupying traumatic intracranial lesions can be viewed as good clinical practice and has never been questioned.^{40–42}

A

Space-occupying intracranial lesion is an absolute indication for urgent surgical removal. This applies to traumatic intracranial hemorrhages (extradural, subdural, or intracerebral hematomas) and space-occupying depressed skull fractures. The degree of space occupancy is defined by the displacement of midline structures, particularly the third ventricle. In addition to the degree of midline shift, location of hematoma, and volume of hematoma, the neurologic findings determine the indication and urgency of surgery. The outcome may depend on minutes when neurologic signs of transtentorial herniation develop.

Operation with Delayed Urgency

B

Open or closed depressed fractures without midline shift, perforating injuries, and basal fractures with CSF leakage may be considered in need of neurosurgery of delayed urgency. The timing of the operation may depend on many factors and must be determined by the neurosurgeon.

B

Nonvital operations of concomitant injuries in the comatose patient should only be performed as far as they are necessary to ensure adequate intensive care. Operations with significant loss of blood or volume shift should be avoided. Principles of “damage control surgery” have been suggested. Further operations should be delayed until the state of the patient appears adequately stable.^{14,42,43}

Decompressive Craniectomy

0

Decompressive craniectomy and duraplasty is the most powerful option to lower increased ICP. Mostly the indication becomes apparent with increasing brain edema often some days after the injury, in some cases immediately after the accident due to swelling. Some studies have reported good results, but this procedure is still an object of research.^{44–46}

Nonoperative Management of Intracranial Hematomas

0

Nonoperative management of intracranial hematomas may be warranted in certain cases when the neurologic

findings remain stable. Neurosurgical evacuation must be readily available in case of deterioration or increasing volume of the hematoma.⁴⁰

Recording of Intracranial Pressure

B

ICP has been recorded with increasing frequency in the acute management of comatose patients after head injury. The recording has been implemented in several national and international guidelines. For pathophysiologic reasons, the recording of ICP appears useful because the clinical monitoring of many cerebral functions is limited in the comatose and eventually sedated patient and may be an early warning of an impending herniation of the mesencephalon from swelling or intracranial hematomas and thus may allow for preventive measures in time.

To date there is no evidence from a prospective randomized controlled study that ICP monitoring has any effect on outcome. Several recent cohort studies and clinical practice, however, indicate its practical value in neurosurgical intensive care. The implementation of guidelines, which among others recommend ICP monitoring, has led to increasingly favorable outcomes of patients after a head injury. Neurosurgeons record ICP in comatose patients after a head injury after consideration of the clinical findings and imaging data to monitor and control ICP. The practical benefit for the patient from thus invasively obtained data is controversial, however, because the specific ICP level, which needs to be treated, has not been identified with scientific data, and the effect from medication, considered to have an ICP lowering effect, remains obscure. The overall benefit from ICP recording performed for pathophysiologic considerations must be weighed against its risk of complication.^{14,19,22,47–60}

Sufficient perfusion of brain tissue requires adequate cerebral perfusion pressure (CPP), which is the difference of the mean arterial blood pressure and ICP. When the ICP is elevated, management remains uncertain from the evidence available in the literature. Should the ICP be lowered or should the CPP be elevated? The evidence available suggests:

B

- CPP should not drop below 50 mm Hg¹⁹

B

- CPP should not be pushed above 70 mm Hg¹⁹

B

- The continuous monitoring of CPP requires invasive recording of ICP. As long as there are no slit ventricles, ICP monitoring with ventricular drainage offers the option to withdraw CSF to lower ICP.

Simultaneous data on brain perfusion, oxygen saturation, and/or brain metabolism are required to determine the optimum CPP in the individual case. To define this value, regional recordings with parenchymal sensors, transcranial

Doppler, and perfusion imaging are currently being investigated.^{19,61,62}

Nonsurgical Management

Patients after head injury require substitution of lost functions (respiration, nutrition). Current scientific data suggest achieving a state of homeostasis (normoxygenation, normotension, avoiding hyperthermia) and avoiding complications. Septicemia, pneumonia, and a disorder of coagulation are predictors of an unfavorable outcome.

In the absence of evidence for clinical benefit ensuring an adequate CPP, adequate brain perfusion is a fundamental pathophysiologic prerequisite of homeostasis. To ensure adequate CPP with increased ICP, a fall in blood pressure must be avoided, eventually requiring catecholamines. There are several options to lower increased ICP:

- 0**
 - Osmodiuretics (e.g., mannitol) may lower the ICP for a short time.¹⁹ Serum osmolality and kidney function must be monitored. There are no data to recommend administration of albumin.
- 0**
 - Hyperventilation may transiently lower the ICP by vasoconstriction and thus decreased intracranial blood volume. The effect may be helpful in acute transtentorial herniation. Prolonged hyperventilation will lead to decreased brain perfusion and unfavorable results and should therefore be restricted to exceptional use.
- 0**
 - Elevation of head and upper body to 30 degrees has been recommended, although it does not affect the CPP. Excessively high ICP, however, is reduced.
- 0**
 - Analgesia/Sedation has no direct effect on ICP. In agitated patients with inadequate spontaneous respiration, sedation may enhance a lowering of ICP and ensure adequate oxygenation.
- 0**
 - Hypertonic saline has been considered to have a brain-protective effect, but there is currently no adequate evidence.
- 0**
 - Barbiturates had been recommended for the treatment of uncontrollably high ICP in earlier guidelines, but there is currently no sufficient evidence for this. The negative inotropic and hypotensive effects and the impossibility to monitor the neurologic state of the patient must be considered.
- 0**
 - Hyperbaric oxygen has not been demonstrated to be effective.
- 0**
 - Hypothermia has no proven beneficial effect.

- 0**
 - Hemostatic agents have no proven beneficial effect. Tranexamic acid may be considered an option.
- 0**
 - Antibiotic prophylaxis in frontobasal fractures with CSF leakage has been controversial up to date. There is no evidence available to offer proof of its efficacy.
- A**
 - Steroids after head injury have been controversial for many years. They should not be administered because they were associated with an increased 14-day mortality after head injury.
- B**
 - Anticonvulsants lower the likelihood of seizures in the first week after injury. An early seizure, however, does not have a bearing on clinical outcome. Anticonvulsants beyond the second week after head injury are not associated with a reduction of late seizures.
- 0**
 - Prophylaxis of thrombosis with physical measures is not controversial to prevent secondary complications. Heparins and analogs bear the risk of additional hemorrhage and an increase of intracranial hematomas. Their benefit must be assessed in each individual case. Because they are not certified for the treatment of head injury, their off-label use is not possible without consent of the patient or the legal representative.
- 0**
 - Brain protection by medication: Several further agents (e.g., 21-aminosteroids, calcium antagonists, glutamate receptor antagonists, tris buffer) have been considered to have a brain-protective effect, but no scientific evidence is available for their benefit.
- A**
 - Mydriatic agents are contraindicated in patients in posttraumatic coma because the onset of the loss of pupil reactivity as an early indicator of intracranial herniation cannot be identified.^{11,12,19,22–33,63–76}

Prognosis

Early after head injury, the prognosis is usually uncertain. Essential factors related to outcome have been demonstrated to be coma and concomitant neurologic disorders, duration of coma, and age. Additional investigations of high predictive value among others are evoked potentials and MRI; lesions of the brainstem in particular indicate a poor prognosis.

Pituitary insufficiency is one of the often overlooked long-term sequelae after head injury that must be looked for. Weeks and months after a minimal head injury, a chronic subdural hematoma may develop, particularly in elderly and predisposed patients.^{39,77–80}

Long-Term Care after Head Injury

B

Reimplantation of bone flaps after craniectomy appears advisable for protection and cosmetic reasons, particularly in patients with neurologic deficits, who are likely to fall. There is no evidence on the best timing.

Patients after craniectomy may develop secondary sequelae associated with particular pathophysiologic phenomena (e.g., increase of headache, pareses, seizures attributed to the “sinking skin flap syndrome”), mostly after ventricular drainage. Replacement of bone flaps may lead to immediate neurologic recovery. Some patients without the prospect for recovery (e.g., in a persistent vegetative state) may not benefit from reimplantation.

Long-term care is determined by the neurologic deficits and the prospect of recovery. Improvement of neurologic deficits is mostly observed within the first 2 years after the injury.

A structural adaptive neuroplasticity has been viewed as the basis of recovery, which may be supported by targeted exercise. To avoid complications (e.g., muscle contractions), rehabilitation (e.g., physiotherapy) should begin in the acute phase. Details of rehabilitation can be viewed under “Leitlinie Multiprofessionelle neurologische Rehabilitation” of the Deutsche Gesellschaft für Neurologie.^{81–85}

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