KEY POINTS

- The headache associated with intracranial hypotension caused by leakage of cerebrospinal fluid after a lumbar puncture is treated with an autologous epidural blood patch.
- The diagnosis of spontaneous intracranial hypotension is associated with the contrast-enhanced brain-MRI finding of diffuse nonnodular pachymeningeal enhancement in nearly 80% of patients.

Trigeminal Neuralgia

Trigeminal neuralgia is the most common and most intense cranial neuralgia. Incidence increases with advanced age. Multiple sclerosis should be considered in those with onset before age 50 years. Pain is typically unilateral and localized to the maxillary and mandibular branches of the trigeminal nerve (cranial nerve V). The pains are brief, "shock-like," or electric, lasting seconds to minutes. Paroxysms may be spontaneous or triggered by innocuous stimulation of the face. Refractory periods are common after a series of paroxysms. Ipsilateral autonomic features are rare. Neurologic examination is typically normal. Contrast-enhanced brain MRI detects nonvascular structural pathology (such as compressing and demyelinating causes) in 15% of patients. In the other 85%, brain MRA often identifies neurovascular contact between a loop of the superior cerebellar artery and the trigeminal nerve. Contact causing displacement or atrophy of the nerve has been associated with a greater likelihood of symptom development.

Management of trigeminal neuralgia begins with carbamazepine administration. Pain may resolve within a few days, although mild dizziness and drowsiness are common. Other adverse effects of the medication, including hyponatremia and agranulocytosis, are less common but necessitate intermittent monitoring. Alternative drugs are oxcarbazepine, baclofen, gabapentin, and lamotrigine. Approximately 30% of patients do not respond to trials of monotherapy or combined therapy. For those refractory to two-drug trials, surgical intervention should be considered. Nonsurgical options are effective in 50% of patients and include percutaneous radiofrequency coagulation, glycerol injection, or focused stereotactic (Gamma Knife) radiation. Posterior fossa microvascular decompression of the neurovascular contact zone is a more invasive and more effective option. It should be prioritized in those with low surgical risk.

KEY POINTS

- Contrast-enhanced brain MRI detects nonvascular structural pathology in 15% of patients with trigeminal neuralgia, and brain magnetic resonance angiography often identifies neurovascular contact between a loop of the superior cerebellar artery and the trigeminal nerve in the other 85%.

(Continued)

KEY POINTS *(continued)*

- Initial management of trigeminal neuralgia is with carbamazepine; if medical therapy fails, focused stereotactic (Gamma Knife) radiation, glycerol injection, or posterior fossa microvascular decompression may be effective.

Medication-Induced Headache

Headache is a potential effect of medication exposure or withdrawal. Oral contraceptives, phosphodiesterase inhibitors, β-adrenergic agonists, and nitrates are among the most commonly implicated drugs; they may cause headache de novo or aggravate an existing primary headache disorder. Withdrawal from caffeine or antidepressants also frequently provokes headache. Medication overuse headache (MOH), previously called "rebound" headache, is a highly prevalent condition affecting 1% of adults. This clinical syndrome may result from overtreatment with acute medication in patients with underlying migraine or tension-type headache. Use of triptans, ergot alkaloids, opioids, or combination analgesics for 10 or more days per month or simple analgesics for 15 or more days per month constitutes medication overuse. Affected patients often report daily or near-daily headache that is refractory to numerous treatment options. MOH is more common in midlife, in women, and in those with high baseline headache frequency. In those with migraine, opioids are associated with a 44% increase and butalbital compounds with a 70% increase in the risk of headache progression. These medications should be avoided in patients with recurrent primary headache disorders. Management of MOH involves discontinuation of the overused medication and the introduction of appropriate preventive medication directed at the primary headache disorder.

KEY POINT

- Medication overuse headache can result from use of triptans, ergot alkaloids, opioids, or combination analgesic agents for 10 or more days per month or simple analgesic agents for 15 or more days per month; treatment includes discontinuation of the overused medication and use of appropriate preventive medication directed at the primary headache disorder.

Primary Headache

Migraine

Diagnosis

The International Classification of Headache Disorders (ICHD), third edition (beta version), recognizes several subtypes of migraine, including migraine without aura, migraine with aura, and chronic migraine. Each is characterized by episodes of disabling headache lasting hours to days. Accuracy of diagnosis and management is enhanced by the use of formal ICHD criteria, although the POUND mnemonic (Pulsatile quality of headache, One-day duration, Unilateral location, Nausea or vomiting, Disabling intensity) is a helpful means of recalling

CONT.

the symptoms typically associated with migraines. Because of the extensive phenotypic variation, nearly half of migraine presentations are misdiagnosed. Neck pain (75%) and "sinus" symptoms, such as tearing or nasal drainage (50%), are both more common than features felt to be characteristic of migraine, such as vomiting or aura. In the presence of a stable clinical pattern of migraine and a normal neurologic examination, brain imaging is not indicated.

Migraine without aura is the most prevalent migraine subtype (**Table 5**). Although clinicians often emphasize unilateral location or pulsatile characteristics, moderate to severe pain is the most sensitive feature, and worsening by routine physical activity is the most specific element among the pain criteria. Photophobia, phonophobia, and nausea are each reported by approximately 75% of patients with migraine without aura.

The diagnosis of migraine with aura is made after two discrete aura episodes have occurred (**Table 6**). Aura may occur in 20% to 30% of patients with migraine. It frequently precedes pain but may occur during or without head discomfort. Aura symptoms involve positive and negative neurologic phenomena developing gradually and evolving over a period of 5 to 60 minutes. Resolution is gradual and complete. ICHD criteria recognize a number of aura subtypes. Typical aura involves any combination of homonymous visual, hemisensory, or language symptoms. Brainstem aura is defined by the presence of two of the following brainstem symptoms: dysarthria, vertigo, tinnitus, hypacusis, diplopia, ataxia, or decreased level of consciousness. Hemiplegic aura comprises any aura involving motor weakness. Both hemiplegic and brainstem auras are listed as contraindications for triptan use. Retinal aura involves monocular visual compromise. These auras need to be distinguished from ocular pathologies, such as retinal ischemia or detachment. Given an associated increased risk of stroke, estrogen-containing oral contraceptives should be avoided in women with migraine aura of all subtypes.

TABLE 5. International Headache Society Criteria for Migraine Without Aura

A. At least five attacks fulfilling criteria B-D

B. Headache attacks lasting 4-72 hours (untreated or unsuccessfully treated)

C. Headache with at least two of the following four characteristics:

1. Unilateral location
2. Pulsating quality
3. Moderate or severe pain intensity that inhibits or prohibits daily activities
4. Aggravation by walking up or down stairs or similar routine physical activity

D. During headache, occurrence of at least one of following symptoms:

1. Nausea/vomiting
2. Photophobia/phonophobia

E. Headache not better accounted for by another ICHD-3 diagnosis

ICHD-3 = International Classification of Headache Disorders, 3rd edition (beta version).

TABLE 6. International Headache Society Criteria for Migraine With Aura

A. At least two attacks fulfilling criteria B and C

B. One or more of the following fully reversible aura symptoms:

1. Visual
2. Sensory
3. Speech and/or language
4. Motor
5. Brainstem
6. Retinal

C. At least two of the following four characteristics:

1. At least one aura symptom spreads gradually over >5 minutes, and/or two or more symptoms occur in succession
2. Each individual aura symptom lasts 5-60 minutes
3. At least one aura symptom is unilateral
4. The aura is accompanied, or followed within 60 minutes, by headache

D. Headache not better accounted for by another ICHD-3 diagnosis, and transient ischemic attack has been excluded

ICHD-3 = International Classification of Headache Disorders, 3rd edition (beta version).

Chronic migraine is defined as headache for 15 or more days per month (**Table 7**). Transformation from acute to chronic migraine occurs in the general population at an annual rate of 3%. Older age, female sex, head trauma, major life changes or stressors, obesity, chronic pain, mood and anxiety disorders, and inadequate acute migraine management are risk factors. Acute migraine medication or caffeine overuse and exposure to nicotine may also raise the risk of or exacerbate chronic migraine; some patients with chronic migraine may have a secondary diagnosis of MOH. Patients with chronic migraine are more disabled and more likely to report migraine-related comorbidities (mood or anxiety disorders, sleep dysfunction, irritable bowel syndrome, fibromyalgia) than are those with episodic migraine.

Acute Migraine Management

The acute treatment of migraine aims to eliminate pain and restore function. Goals of care involve freedom from pain,

TABLE 7. International Headache Society Criteria for Chronic Migraine

A. Headache (tension-type–like and/or migraine-like) on at least 15 days per month for greater than 3 months and fulfilling criteria B and C

B. Occurring in a patient who has had at least five attacks fulfilling criteria B-D for migraine without aura and/or criteria B and C for migraine with aura

C. On at least 8 days per month for greater than 3 months, fulfilling any of the following:

1. Criteria C and D for migraine without aura
2. Criteria B and C for migraine with aura
3. Believed by the patient to be a migraine at onset and relieved by a triptan or ergot alkaloid derivative
4. The aura is accompanied, or followed within 60 minutes, by headache

D. Headache not better accounted for by another ICHD-3 diagnosis

ICHD-3 = International Classification of Headache Disorders, 3rd edition (beta version).

CONT.

nausea, and sensory sensitivities within 1 to 2 hours and maintenance of such control through at least 24 hours. Because attack characteristics show significant inter-individual and intra-individual variability, different treatment options and strategies are required (**Table 8**). Guidelines recommend tailoring the treatments to the severity and symptomatology of the attack. Migraines awakening a patient from sleep or those associated with nausea and vomiting may require medication offered in parenteral or nasal formulations. Because consistency of response to any treatment rarely approaches 100%, most patients benefit from availability of two or more acute migraine therapies. Administration of medication at the time of mild pain has been shown to improve therapeutic outcomes when compared to treating moderate to severe headache; therefore, treatment should be started as early as possible in the disease course. Treatment should be limited to 10 days per month to avoid MOH.

Evidence-based guidelines recommend several simple and combination analgesic agents as first-line therapies for acute migraine. Acetaminophen has established efficacy only in migraine of mild to moderate intensity; data suggest the response may be enhanced by coadministration with metoclopramide. Aspirin administered alone or in combination with acetaminophen and caffeine also has established efficacy in acute migraine. The effectiveness of the NSAIDs ibuprofen, naproxen sodium, and diclofenac potassium is supported by strong evidence. Special formulations of these products have been shown to be more rapidly absorbed and effective than their standard tablet counterparts. These formulations include effervescent aspirin, solubilized ibuprofen, and diclofenac powder for oral solution.

Triptans are selective agonists at 5-hydroxytryptamine 1B and 1D receptors. They are migraine-specific agents with direct impact on trigeminovascular activation associated with migraine attacks. Triptans reverse intracranial vasodilation (1B) and provide neuronal inhibition at peripheral and central trigeminal nerve circuitry (1D). Guidelines recommend the use of triptans in patients with moderate to severe migraine who have not responded to NSAID therapy over a series of at least three migraine attacks. Current evidence suggests that all oral triptans possess nearly similar clinical efficacy. Orally dissolvable tablets are intestinally absorbed; their only advantage is use without the need to drink liquids. Nasal spray options may have more rapid onset, bypassing the gastrointestinal tract. Outcomes are similar to those of oral agents, but unpleasant taste, nasal congestion, or burning may be limiting factors. Subcutaneous sumatriptan provides the most rapid onset and achieves the highest response rates among all triptan agents and formulations. Local site reactions and more prominent "triptan" sensations characteristic of the class (flushing, chest or throat tightness, paresthesias) may be noted. All triptans are

TABLE 8. Acute Migraine Therapies

Drug	Recommended Dose
NSAIDs[a]	
Aspirin	325-900 mg
Ibuprofen	400-800 mg
Naproxen sodium	250-1000 mg
Combination of acetaminophen-aspirin-caffeine	2 tablets
Diclofenac potassium (oral solution)	50 mg
Migraine-Specific Oral Agents[a]	
Almotriptan	6.25-12.5 mg
Eletriptan	20-40 mg
Frovatriptan	2.5 mg
Naratriptan	1-2.5 mg
Rizatriptan	5-10 mg
Sumatriptan	25-100 mg
Sumatriptan-naproxen	85-500 mg
Zolmitriptan	2.5-5 mg
Nonoral Agents[a]	
Dihydroergotamine	1 mg nasally
Dihydroergotamine	1 mg subcutaneously
Prochlorperazine	10 mg intravenously
Sumatriptan	5-20 mg nasally
Sumatriptan	4-6 mg subcutaneously
Zolmitriptan	5 mg nasally

[a]Doses listed may be administered once or twice daily.

H CONT. contraindicated in the presence of coronary, cerebral, or peripheral vascular disease; uncontrolled hypertension; or migraine with brainstem or hemiplegic auras. Different formulations of one triptan may be used safely within the same day, but using a different triptan or an ergot alkaloid should be delayed by 24 hours. Despite labeling precautions, the concurrent use of triptans and selective serotonin reuptake inhibitors or serotonin-norepinephrine reuptake inhibitor antidepressants is safe in most cases.

Other agents also are effective in the management of acute migraine. Ergot alkaloids have been used for years but have been largely replaced by triptans, which have preferable safety and tolerability profiles; parenteral and nasal formulations of dihydroergotamine are the most available and effective options among the ergot alkaloids. Dopamine D_2 receptor antagonists (metoclopramide, prochlorperazine) are often used as adjuncts to analgesics or triptans. In addition to antiemetic properties, these agents can reduce migraine pain when delivered parenterally. Guidelines recommend avoiding the use of opioids and butalbital-containing compounds for acute migraine. In addition to the potential for dependence or addiction, use of these agents has been linked to an increased risk of transformation from episodic to chronic migraine. They should be used sparingly and only when more appropriate acute therapies are contraindicated.

Migraine with a duration lasting longer than 72 hours is known as status migrainosus. Many patients with this condition can be treated with several days of glucocorticoids. Severe cases of acute migraine or status migrainosus may require emergency department or inpatient management. The cornerstone of care in these settings is intravenous delivery of a dopamine antagonist. These are typically combined with intravenous diphenhydramine (to limit dystonic reactions), intravenous ketorolac, and hydration. Opioids may be associated with prolonged length of hospital stay and should be avoided. A more extended course of treatment involving repetitive intravenous dihydroergotamine with antiemetics over 2 to 3 days is very effective in the management of refractory status migrainosus. H

Migraine Prevention

The goals of migraine prevention are the reduction of migraine frequency, intensity, and duration. None of the medications used for migraine prevention were designed for this specific purpose. The best agents may reduce migraine frequency by half in approximately half of the patients treated. Nonpharmacologic preventive measures are not only optimal but required. Trigger identification and avoidance are often helpful. Stress management techniques, such as relaxation therapy or biofeedback, have established efficacy. Regulation of sleep patterns, intake of small frequent meals, adequate hydration, and daily aerobic exercise are all extremely helpful. Regular work and school schedules should be encouraged. Stimulants (such as caffeine and nicotine) must be eliminated or limited. Diet should be modified to avoid additives or preservatives, such as monosodium glutamate and artificial sweeteners. There is good evidence to support the use of certain supplements, such as petasites (butterbur), magnesium, riboflavin, and feverfew.

Pharmacologic prophylaxis should be considered when the headache frequency reaches 5 days per month and almost always is initiated when the frequency exceeds 10 days per month. Data suggest preventive medication can reduce attack frequency and intensity, patient disability, and medical cost. Several weeks or months are often required before maximum benefit is achieved. Once a response occurs, the medications should be continued for a period of 6 to 12 months, at which point dose reduction or drug elimination may be considered. Evidence-based guidelines for pharmacologic prevention of episodic migraine have established Level A evidence supporting the use of five medications: three β-adrenergic blockers (propranolol, timolol, metoprolol) and two antiepileptic drugs (divalproex sodium and topiramate). Level B evidence is available for atenolol, two antidepressants (amitriptyline and venlafaxine), and several NSAIDs. No evidence supports the use of calcium channel blockers or selective serotonin reuptake inhibitor antidepressants in migraine prevention. Studies involving patients with chronic migraine are more limited, but efficacy in prevention has been shown with topiramate and onabotulinum toxin A. Treatment selection is informed by previous therapeutic trials, the presence of coexisting medical conditions, and patient preference.

KEY POINTS

- Because of the extensive phenotypic variation, nearly half of migraine presentations are misdiagnosed; the POUND mnemonic (Pulsatile quality of headache, One-day duration, Unilateral location, Nausea or vomiting, Disabling intensity) is a helpful means of recalling the symptoms typically associated with migraines. **HVC**
- In the presence of a stable clinical pattern of migraine and a normal neurologic examination, brain imaging is not indicated. **HVC**
- Given an associated increased risk of stroke, estrogen-containing oral contraceptives should be avoided in women with migraine aura of all subtypes.
- Triptans are migraine-specific agents that are useful in moderate to severe migraine that has not responded to NSAID therapy; they are contraindicated in the presence of coronary, cerebral, or peripheral vascular disease; uncontrolled hypertension; or migraine with brainstem or hemiplegic auras.
- Pharmacologic prophylaxis for migraine should be considered when headache frequency reaches 5 days per month and almost always is initiated when the frequency exceeds 10 days per month; propranolol, timolol, metoprolol, divalproex sodium, and topiramate are most effective for migraine prophylaxis.

Tension-Type Headache

The most prevalent primary headache condition, tension-type headache is defined by clinical criteria as a headache disorder that, unlike migraine, is mild to moderate in intensity and is not associated with nausea, severe sensory sensitivities, or neurologic symptoms (**Table 9**). Imaging is not indicated. Subclassification is based on monthly headache frequency: infrequent episodic (<1 day), frequent episodic (1-14 days), and chronic (15 or more days). Acetaminophen, aspirin, NSAIDs, and caffeine-containing compounds are effective acute treatments for tension-type headache. Amitriptyline and stress management techniques have modest benefit in prevention of tension-type headache, and some data support the use of acupuncture. Muscle relaxants, benzodiazepines, opioids, and onabotulinum toxin A have no role in the management of tension-type headache.

KEY POINTS

HVC
- Imaging is not indicated for tension-type headache.
- Acetaminophen, aspirin, NSAIDs, and caffeine-containing compounds are effective acute treatments for tension-type headache, but muscle relaxants, benzodiazepines, opioids, and onabotulinum toxin A have no role in the management of tension-type headache.

Trigeminal Autonomic Cephalalgias

Trigeminal autonomic cephalalgias (TACs) are the most severe and stereotypic primary headache disorders. Pain is severe, localized to the periorbital or temporal areas, and associated with pronounced ipsilateral cranial autonomic features, such as nasal congestion or rhinorrhea and ptosis or miosis. The TACs, which include cluster headache, chronic paroxysmal hemicrania (CPH), and short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing (SUNCT), are differentiated by episode duration, frequency, and periodicity.

Cluster headache may last 15 to 180 minutes and recur one to eight times daily over a span of weeks to months; the shorter duration distinguishes cluster headache from migraine (**Table 10**). This headache is characterized by a cyclical nature in which periods of recurrent headache activity are interrupted by months to years of headache remission. Many of the attacks are nocturnal, and some may be provoked by alcohol ingestion. Episodes of CPH last 2 to 30 minutes and recur up to 40 times per day, whereas those of SUNCT last 1 to 600 seconds and may recur more than 100 times daily. SUNCT may be confused with trigeminal neuralgia, which is more likely mandibular or maxillary and lacks autonomic features. Unlike cluster headache, both CPH and SUNCT typically continue without periods of remission. Brain MRI should be performed initially to exclude structural lesions mimicking TACs.

Cluster headache has several acute and preventive options. Oxygen inhalation and subcutaneous sumatriptan are both effective in the treatment of attacks. A 2-week course of glucocorticoids may help reduce attack frequency at the onset

TABLE 9. International Headache Society Criteria for Tension-Type Headache

A. At least 10 attacks fulfilling criteria B-E

B. Headache attacks (untreated or unsuccessfully treated) lasting from 30 minutes to 7 days

C. Headache with at least two of the following four characteristics:
   1. Bilateral location
   2. Pressing/tightening (nonpulsating) quality
   3. Mild or moderate intensity
   4. Not aggravated by walking stairs or similar routine physical activity

D. Headache characterized by both of the following:
   1. No nausea/vomiting
   2. No more than one episode of photophobia or phonophobia

E. Not better accounted for by another ICHD-3 diagnosis

ICHD-3 = International Classification of Headache Disorders, 3rd edition (beta version).

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TABLE 10. International Headache Society Criteria for Cluster Headache

A. At least five attacks fulfilling criteria B-D

B. Severe or very severe unilateral orbital, supraorbital, and/or temporal pain lasting 15-180 minutes (when untreated)

C. Either or both of the following:
   1. At least one of the following symptoms or signs, ipsilateral to the headache:
      a) conjunctival injection and/or lacrimation
      b) nasal congestion and/or rhinorrhea
      c) eyelid edema
      d) forehead and facial sweating
      e) forehead and facial flushing
      f) sensation of fullness in the ear
      g) miosis and/or ptosis
   2. A sense of restlessness or agitation

D. Attack frequency from one every other day to eight per day when the disorder is active

E. Headache not better accounted for by another ICHD-3 diagnosis

ICHD-3 = International Classification of Headache Disorders, 3rd edition (beta version).

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of the cycle. Verapamil is the drug of choice for longer-term prevention of cluster headache. CPH is uniquely and universally responsive to indomethacin. Reports have suggested a small benefit from lamotrigine, but SUNCT is largely refractory to medical management.

KEY POINTS

- Trigeminal autonomic cephalalgias are the most severe and stereotypic primary headache disorders and include cluster headaches, chronic paroxysmal hemicrania, and short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing.
- Cluster headache is treated with oxygen inhalation and subcutaneous sumatriptan.
- Chronic paroxysmal hemicrania is universally responsive to indomethacin; short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing is largely refractory to medical management.

Other Primary Headache Syndromes

Primary stabbing headaches ("ice-pick headaches") are episodes of stabbing head pain lasting seconds and occurring in isolation or in series. There are no associated autonomic features. The location of pain is fixed in one third of patients and extratrigeminal in most patients. Those with migraine may be more likely to describe these attacks. Indomethacin can be helpful during cycles of more frequent attacks.

Cough headache develops abruptly with cough or Valsalva maneuvers and typically lasts seconds to minutes. Mild headache may continue for 1 to 2 hours. The severity of pain correlates with cough frequency. Advancing age and male sex may be risk factors. Brain MRI is indicated because secondary pathologies, most commonly a Chiari malformation, may be found in half of those affected. Indomethacin may reduce headache frequency during cycles of increased activity.

KEY POINT

- Brain MRI is indicated in patients with cough headache to identify possible secondary pathologies (such as a Chiari malformation), which may be found in 50% of affected patients.

Head Injury

Traumatic Brain Injury

Traumatic brain injury (TBI) results from biomechanical forces applied to the structures of the head and neck. Damage may be temporary or permanent and may arise from functional or structural alterations in the central nervous system. Severity is determined by clinical findings and imaging results. When indicated, noncontrast CT is the imaging modality of choice because it is more sensitive for bony injuries, less expensive, and more widely available than MRI. Mild TBI is associated with Glasgow Coma Scale (GCS) scores of 13 to 15 with no or only a brief initial loss of consciousness; results of head CT are typically normal. Moderate TBI is associated with GCS scores of 9 to 12 and/or an initial loss of consciousness of 30 minutes to 24 hours. Severe TBI is associated with GCS scores of 3 to 8 and/or an initial loss of consciousness of more than 24 hours (**Table 11**). In moderate and severe TBI, head CT may reveal a skull fracture, cerebral contusions and edema, and intracranial hemorrhage.

Mild Traumatic Brain Injury

The terms mild TBI and concussion are often used interchangeably. Mild TBI is the most common presentation of TBI and frequently results from accidents, athletic activity, or military service activities. Guidelines for the use of head CT in mild TBI have been published by the Centers for Disease Activity and Prevention (**Table 12**). Although yet to be incorporated into management guidelines, serum measurements of brain-specific biomarkers released after mild TBI can help predict which patients may have intracranial lesions visible on CT scan. Elevated levels of ubiquitin carboxy-terminal hydrolase L1 and glial fibrillary acidic protein can be detected as early as 20 minutes after head injury. A recent study of patients within 12 hours of mild TBI (GCS scores of 9 to 15) showed that these elevated serum levels combined with certain clinical information had a sensitivity of 97.5% for predicting lesions visible on head CT scan; negative predictive value was 99.6%. Patients

TABLE 11. Glasgow Coma Scale

Eye Opening	Score
Spontaneous	4
Response to verbal command	3
Response to pain	2
No eye opening	1
Best Verbal Response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
No verbal response	1
Best Motor Response	
Obeys commands	6
Localizing response to pain	5
Withdrawal response to pain	4
Flexion to pain	3
Extension to pain	2
No motor response	1
Total	

Data from: Teasdale G, Jennett B. Assessment of coma and impaired consciousness. A practical scale. Lancet 1974 Jul 13;2(7872): 81-4. [PMID: 4136544]